Free ebooks ==> www.Ebook777.com

Arriving at a SURGICAL DIAGNOSIS

www.Ebook777.com

Free ebooks ==> www.Ebook777.com

Arriving at a SURGICAL DIAGNOSIS

Pramod Shrikrishna Bapat

Consultant General Surgeon
Shree Harneshwar Clinic
Talegaon (Dabhade), Pune, Maharashtra, India

Formerly

Registrar
Rajawadi Hospital, Mumbai, Maharashtra, India

Lecturer
Lokamanya Tilak Municipal Medical College
and General Hospital, Mumbai, Maharashtra, India

Professor of General Surgery
People's College of Medical Sciences, Bhopal, Madhya Pradesh, India

Consultant General Surgeon
Colonial War Memorial Hospital, Suva, Fiji Islands



JAYPEE BROTHERS MEDICAL PUBLISHERS (P) LTD

New Delhi • London • Philadelphia • Panama

www.Ebook777.com

Jaypee Brothers Medical Publishers (P) Ltd

Headquarters

Jaypee Brothers Medical Publishers (P) Ltd
4838/24, Ansari Road, Daryaganj
New Delhi 110 002, India
Phone: +91-11-43574357
Fax: +91-11-43574314
Email: jaypee@jaypeebrothers.com

Overseas Offices

J.P. Medical Ltd
83 Victoria Street, London
SW1H 0HW (UK)
Phone: +44-2031708910
Fax: +02-03-0086180
Email: info@jpmedpub.com

Jaypee-Highlights Medical Publishers Inc.
City of Knowledge, Bld. 237, Clayton
Panama City, Panama
Phone: + 507-301-0496
Fax: + 507-301-0499
Email: cservice@jphmedical.com

Jaypee Brothers Medical Publishers (P) Ltd
17/1-B Babar Road, Block-B, Shaymali
Mohammadpur, Dhaka-1207
Bangladesh
Mobile: +08801912003485
Email: jaypeedhaka@gmail.com

Jaypee Brothers Medical Publishers (P) Ltd
Shorakhute, Kathmandu
Nepal
Phone: +00977-9841528578
Email: jaypee.nepal@gmail.com

Jaypee Brothers Medical Publishers (P) Ltd
The Bourse
111 South Independence Mall East
Suite 835, Philadelphia, PA 19106, USA
Phone: + 267-519-9789
Email: joe.rusko@jaypeebrothers.com

Website: www.jaypeebrothers.com
Website: www.jaypeedigital.com

© 2013, Jaypee Brothers Medical Publishers

All rights reserved. No part of this book may be reproduced in any form or by any means without the prior permission of the publisher.

Inquiries for bulk sales may be solicited at: jaypee@jaypeebrothers.com

This book has been published in good faith that the contents provided by the author contained herein are original, and is intended for educational purposes only. While every effort is made to ensure accuracy of information, the publisher and the author specifically disclaim any damage, liability, or loss incurred, directly or indirectly, from the use or application of any of the contents of this work. If not specifically stated, all figures and tables are courtesy of the author. Where appropriate, the readers should consult with a specialist or contact the manufacturer of the drug or device.

Arriving at a Surgical Diagnosis

First Edition: **2013**

ISBN 978-93-5025-810-1

Printed at

Free ebooks ==> www.Ebook777.com

Dedicated to

Infinite patience of
my daughter **Anuprita** and wife **Vaishali**
for tolerating me, always

www.Ebook777.com

Free ebooks ==> www.Ebook777.com

Preface

'Clinical diagnosis is an art'—**I DISAGREE**. Clinical diagnosis is a science. 'Art' is a faculty, which can be mastered only by few having an inborn aptitude in a particular field of art. Clinical medicine cannot afford to be an art! Every clinician must be a master with the greatest level of competence in arriving at the clinical diagnosis; and the mastery will be achieved with a scientific approach.

What was the need for yet another book on 'clinical surgery'? Well, most of the existing books explain clinical methods, and then describe 'differential diagnoses'. There is hardly any book which explains **how to analyze clinical data systematically** and arrive at a clinical diagnosis by using **logical thinking in stepwise manner**. Operative surgery books describe procedures in stepwise manner. Why not have a clinical surgery book which explains the method of arriving at the diagnosis in the same manner?

In the present era of technological advent, clinicians seem to be requesting a battery of investigations even in straightforward cases. Such a practice tends to blunt the thinking aptitude very fast, and decay the clinical approach. Unfortunately, junior students in surgical training seem to be adopting this approach from the beginning of their apprenticeship. Hence, I thought of writing this book, which will guide them to **make a clinical diagnosis with confidence**, without having to rely on too many unnecessary investigations.

The contents, including the steps in arriving at the diagnosis in each chapter, are generally ordered in the same sequence unless special circumstances dictate otherwise. However, systems and organs naturally possess a remarkable degree of individuality with respect to anatomy, physiology and pathology; and it follows that the techniques for history-taking and examining one anatomical region / system may have to be varied when it comes to look at another. In spite of this fact, I have tried to derive a common sequence as best as possible.

I do not claim originality in all the facts presented in this book; most of these have been derived from various authentic books existing on the subject. However, some of the facts have been derived from the knowledge imparted to me by my teachers, and observations from my own clinical experience.

I am conscious of deficiencies as a single author, but I hope that this fact has helped in maintaining coherence and continuity of the methods and subject. Nevertheless, there is a possibility for inadequacies and errors to have occurred. I invite the readers to share their critical reviews and positive ideas for improvement of future editions of this book.

Pramod Shrikrishna Bapat
E-mail: bapat.asd@gmail.com

www.Ebook777.com

Free ebooks ==> www.Ebook777.com

Layout of the Book

By and large, titles given to the chapters are none other than the chief complaints with which patients present. This book focuses on history-taking in a readymade questionnaire format, and systematic step-by-step analysis of the clinical data to arrive at the diagnosis. Each chapter on individual problem is written in a standard format:

(a) Definition/s.
(b) General remarks.
(c) History-taking.
(d) Physical examination.
(e) Arriving at the diagnosis
- Principles and pitfalls
- Stepwise analysis of clinical data.

(f) Differential diagnosis.
(g) Residual problem, if any.

Clinical tests are described in a uniform format all throughout the book:

(a) Title of the test.
(b) Aim.
(c) Principle.
(d) Precaution to be observed, if any, while performing the test.
(e) Prerequisite.
(f) Procedure (clinical method).
(g) Observation and inference.
(h) Causes of false +ve and false –ve results.
(i) Controversy, if any.
(j) Therapeutic application, if any.

Justification of the diagnosis is discussed in 'to-the-point' form:

- Basic point.
- Differentiating points.
- Supporting points.

In addition, variations in clinical presentation and important relevant short notes on various conditions are also described.

www.Ebook777.com

Free ebooks ==> www.Ebook777.com

How to Use This Book

THIS IS NOT A TEXTBOOK. This is a manual. I have converted the information and knowledge from a large number of textbooks in a practically executable format in this book. To make the best use of this book, get started in the following way:

(a) Read chapters 1 and 9 at the outset.
(b) Then spend some time familiarizing with the format of the book by going through a few chapters between 15 and 49.
(c) Read chapters 2 to 8 for basic clinical methods.
(d) Read chapters 11, 12 and 13 as they are important in themselves particularly with respect to 'emergency' cases.
(e) While 'taking a clinical case':
    (i) After knowing the patient's chief complaint, open the appropriate chapter and start asking history which is given in a readymade questionnaire format. Finding the appropriate chapter will be easy as titles given to the chapters are none other than the chief complaints with which patients present.
    (ii) Examine the patient.
    (iii) Analyze the clinical data as explained in the chapter, and I am confident you will arrive at the diagnosis smoothly.
    (iv) Cross-check your diagnosis by going through the section on 'differential diagnosis'.

Arriving at the clinical diagnosis will never be a difficult problem henceforth. After reading this book, you will soon develop interest in taking even challenging cases and attempt to make clinical diagnosis.

I wish you success in your future clinical journey in the field of Surgery.

www.Ebook777.com

Free ebooks ==> www.Ebook777.com

Acknowledgments

I am greatly indebted to the following consultants who helped me in some or the other way during different stages of preparation of this book. **YR Kher**, Former Professor and Head of the Department (Retired), TN Municipal Medical College and Nair Hospital, Mumbai, Maharashtra; **Col. YG Tambay**, Senior Consultant Plastic and Reconstructive Surgeon (Retired), Indian Armed Forces; **(Late) Manohar J Joshi**, Former Honorary Professor, BJ Medical College and Sassoon Hospital, Pune, Maharashtra; **NC Joshi**, Senior Consultant Pediatrician, Mumbai, Maharashtra; **Mahendra Bendre**, Professor, DY Patil Medical College, Mumbai, Maharashtra; **AK Banerjee**, Consultant General Surgeon, Raikot, Ludhiana, Punjab; **Ashwin Apte**, Consultant Pediatric Surgeon, Bhopal, Madhya Pradesh; **Ajay Jain**, Consultant Pediatric Surgeon, Bhopal, Madhya Pradesh; **Mrudul Shahi**, Consultant Neurosurgeon, Bhopal, Madhya Pradesh; **Neelesh Risbud**, Consultant Obstetrician and Gynecologist, Pune, Maharashtra; **Ajit Oak**, Consultant Pediatrician, Dombivlee, Thane, Maharashtra; **Ashutosh Mangalgiri**, Professor of Anatomy, Chirayu Medical College and Hospital, Bhopal, Madhya Pradesh; **Shreekant and Mrs Jyotee Jategaonkar**, Consultant General Physicians, Talegaon (Dabhade), Pune, Maharashtra; **Santosh Sabnis**, Consultant Radiologist, Talegaon (Dabhade), Pune, Maharashtra; **Rajendra Bhojwani**, Consultant Radiologist, Gwalior, Madhya Pradesh; **T Laxminarayana**, Senior Consultant Radiologist, Hyderabad, Andhra Pradesh; **Rajendra Zope**, Consultant Pathologist, Talegaon (Dabhade), Pune, Maharashtra; **Pankaj Goel**, Professor and Head of the Department, Community Dentistry, People's Dental Academy, Bhopal, Madhya Pradesh.

I am also grateful to my teachers: **SV Nadkarni**, Former Head of the Department and Dean (Retired), LTM Medical College and General Hospital, Mumbai, Maharashtra; **(Late) TT Changlani**, Former Head of the Department, LTM Medical College and General Hospital, Mumbai, Maharashtra; **VG Mehendale**, Honorary Professor and Head of the Department (Retired), Rajawadi Hospital, Senior Consultant General Surgeon, and Urologist, Mumbai, Maharashtra; **MV Bhatt**, Former Honorary Professor (Retired), LTM Medical College and General Hospital, Mumbai, Maharashtra; **P Anantharam**, Former Honorary Professor (Retired), LTM Medical College and General Hospital, Mumbai, Maharashtra; **Paresh Varty**, Consultant General and GI Surgeon, Mumbai, Maharashtra; **SN Shenoy**, Senior Consultant GI, Laparoscopic and General Surgeon, Mumbai, Maharashtra.

I am thankful to **Charles Collins**, Consultant Anesthesiologist, Colonial War Memorial (CWM) Hospital, Suva, Fiji, for taking the photographs of the patient in Fig. 35-6, an unusually big and challenging case to manage from all aspects.

I am also thankful to M/s Jaypee Brothers Medical Publishers (P) Ltd, New Delhi, India, for publishing my book which has been written in unorthodox style, and also for allowing me to use photographs from their titles mentioned: 1. 'Jaypee Gold Standard Mini Atlas Series Surgical Diseases'. 1st edition, 2007. 2. 'SRB's Manual of Surgery'. 3rd edition, 2009. 3. 'Jaypee Gold Standard Mini Atlas Series Dermatology'. 1st edition, 2008. 4. 'Step by Step Imaging of Bone and Joints. 1st edition, 2008. 5. 'Textbook of Ophthalmology' 5th edition, 2008.

I would like to thank the patients with a sense of gratitude for offering their photographs for propagation of clinical teaching and learning in general; this book being a part of the process.

I would also like to thank all the technocrats related to the world of electronics and computers, without whose efforts typing, editing, typesetting and publishing books like this would not have become easy.

www.Ebook777.com

Free ebooks ==> www.Ebook777.com

Contents

SECTION 1: GENERAL SURGERY

1.	General principles of case-taking	3-6
2.	History-taking	7-14
3.	Selected common symptoms in clinical practice	15-27
4.	General examination	28-48
5.	Locoregional examination	49-51
6.	Respiratory system examination	52-58
7.	Pelvic examination	59-67
8.	Examination of the pregnant patient	68-71
9.	Diagnosis	72-77
10.	Guidelines on documentation of clinical notes	78
11.	Sudden loss of consciousness (Sudden unresponsiveness)/cardiorespiratory arrest	79-83
12.	Shock	84-96
13.	Infections / infestations	97-118
14.	Skeletal muscles in the body amenable to physical examination (Applied anatomy)	119-128
15.	Swelling—basic concepts, including differential diagnosis of acute swelling and chronic general swelling	129-195
16.	Chronic lymph node swelling	196-205
17.	Chronic bone swelling	206-218
18.	Chronic vascular swelling	219-231
19.	Chronic circumscribed swelling in the region of the head and face (Excluding swelling of the jaw and oral cavity)	232-243
20.	Chronic swelling of the jaw	244-250
21.	Chronic circumscribed swelling in the neck	251-259
22.	Goiter and thyroid cancer	260-276
23.	A reducible (irreducible) swelling in the groin	277-301
24.	A non-reducible swelling in the groin	302-309
25.	Scrotal swelling or scrotal pain	310-333
26.	Impalpable testicle or empty scrotum	334-338
27.	Chronic swelling in the back	339-340
28.	Chronic swelling in the perineum and gluteal region	341
29.	Swellings / Bulges related to vulva / Vagina / Urethra	342-343
30.	Disorders of the female breast	344-369
31.	Disorders of the male breast	370-377
32.	Disorders of the chest wall	378-380
33.	Chronic pain in abdomen (chronic abdomen)	381-429
34.	Acute pain in abdomen (acute abdomen)	430-492
35.	Lump in abdomen	493-534
36.	Distension of abdomen	535-538
37.	Jaundice	539-556
38.	Portal hypertension	557-558
39.	Difficulty in swallowing and other symptoms of esophageal disorder	559-574
40.	Vomiting of blood—Hematemesis	575-579
41.	Anorectal pain, bleeding per rectum and other manifestations of anorectal disorder	580-607
42.	Ulcer	608-627
43.	Sinus / fistula	628-643

www.Ebook777.com

44. (Recurrent attacks of) Discharge from the umbilicus 644-646
45. Pain in the limb 647-658
46. Acute arterial insufficiency and chronic peripheral arterial occlusive disease (PAOD) 659-680
47. Limb gangrene 681-690
48. Amputation stump 691-693
49. Varicose veins and chronic venous insufficiency 694-702
50. Limb edema 703-718
51. Oral cavity or orofacial pain 719-742
52. Temporomandibular joint 743-744
53. Backache 745-756

SECTION 2: UROGENITAL CASE

54. Urogenital Case 759-814
(For disorders of the scrotum, refer to chapters 25, 26, 85)

SECTION 3: NEUROSURGERY

55. Intracranial space-occupying lesion 817-820

SECTION 4: TRAUMA (ESSENTIALLY SOFT TISSUE)

56. Acute mechanical trauma—general principles 823-848
57. (Acute) Head injury 849-868
58. Facial (maxillofacial) trauma 869-878
59. Soft-tissue injuries of the neck 879-882
60. Trauma to the chest 883-891
61. Esophageal injury 892-897
62. Ingested foreign body 898-904
63. Abdominal trauma (including pelvic trauma) 905-917
64. Urogenital trauma 918-926
65. Anorectal and perineal trauma 927-928
66. Spinal trauma 929-931
67. Hand injury 932-934
68. Vascular trauma (particularly in the limbs and neck) 935-937
69. Burn injury—Thermal, chemical, electrical 938-944

SECTION 5: PEDIATRIC SURGERY

70. History-taking in a child 947-948
71. Surgical examination of the child 949-950
72. Respiratory distress in the newborn 951-955
73. Acute abdominal disorders 956-964
74. Colic (Colicky pain in abdomen) 965-966
75. Neonatal intestinal obstruction 967-971
76. Recurrent pain in abdomen 972
77. Vomiting in the neonate 973-974
78. Non-bile-stained vomiting in infancy 975-976
79. Gastrointestinal (GI) bleeding 977-978
80. Jaundice / cholelithiasis / portal hypertension 979-980
81. Discharge from the umbilicus 981-983
82. Anorectal malformation 984-985
83. Urinary tract infection (UTI) or urinary stones 986-987

Free ebooks ==> www.Ebook777.com

84.	Disorders of the lower urinary tract	988
85.	Acute scrotum	989-991
86.	Disorders of the external genitalia	992
87.	Acute pain in the limb (acute limb)	993-994
88.	Disorders of the mouth	995-996
89.	Specific regional swellings/lesions in the face	997
90.	Swelling in the neck	998
91.	Torticollis (Wryneck)	999
92.	Disorders of the breast	1000-1001
93.	A big head / hydrocephalus / assessing a VP shunt	1002-1005
94.	Spinal dysraphism—spina bifida	1006-1009
95.	Foreign bodies	1010-1012

Bibliography *1013-1014*

Index *1015-1022*

www.Ebook777.com

Free ebooks ==> www.Ebook777.com

Abbreviations

→ : Lead to / leading to
⇒ : Imply / implies / implying
↑ed : Increased
↓ed : Decreased
↓ : Under
± : May be either present or absent
× : For

A

AAA : Abdominal aortic aneurysm
ABPI : Ankle brachial pressure index
ADH : Antidiuretic hormone
AF : Atrial fibrillation
AFB : Acid fast bacilli
AIN : Acute interstitial nephritis
ALI : Acute limb ischemia
AK : Above knee
AKT : Anti-Koch's treatment
ANIML : Acute nonspecific ileocecal mesenteric lymphadenitis
ARDS : Acute respiratory distress syndrome (Older terminologies: Adult respiratory distress syndrome, shock lung)
ARF : Acute renal failure
ATN : Acute tubular necrosis
ATT : Antituberculous treatment
AV : Arteriovenous
AVF : Arteriovenous fistula
AVM : Arteriovenous malformation

B

BCC : Basal cell carcinoma
BEP : Benign enlargement of prostate
BK : Below knee
BNC : Bladder neck contracture
BNH : Bladder neck hypertrophy
BOO : Bladder outflow obstruction
BPE : Benign prostatic enlargement
BPH : Benign prostatic hyperplasia/ hypertrophy
BTF : Blood transfusion

C

C & D : Cleaning and dressing
C/F : Clinical feature
C/M : Clinical manifestation
C/o : Case of
CAPD : Continuous ambulatory peritoneal dialysis
CA : Carcinoma
CBD : Common bile duct
CDH : Congenital diaphragmatic hernia
CHF : Congestive heart failure
CHPS : Congenital hypertrophic pyloric stenosis
CML : Chronic myeloid leukemia
COAD : Chronic obstructive airway disease
CP : Cardiopulmonary
CRF : Chronic renal failure
CRPS : Complex regional pain syndrome
CT : Connective tissue
CTD : Connective tissue disorder
CVA : Cerebrovascular accident
CVD : Cerebrovascular disease
CVI : Chronic venous insufficiency
CVU : Chronic venous ulcer / ulceration
CW Doppler : Continuous wave Doppler

D

D1 : 1st part of duodenum
D2 : 2nd part of duodenum
DAI : Diffuse axonal injury
D & C : Dilatation and curettage
DI : Diabetes insipidus
DJ : Duodeno jejunal
DL scopy : Direct laryngoscopy
DM : Diabetes mellitus
DPL : Diagnostic peritoneal lavage
DSD : Detrusor-sphincter dyssynergia
DU : Duodenal ulcer

E

EB : Epstein-Barr
ECF : Extracellular fluid
ECFV : Extracellular fluid volume
EMS : Emergency medical services
ERCP : Endoscopic retrograde cholangiopancreatography
ET : Endotracheal
EUA : Examination under anesthesia

www.Ebook777.com

F

F/b : Followed by
F : Female(s)
FAP : Familial adenomatous polyposis
FAST : Focused assessment with sonography in trauma
FBC : Full blood count
FDP : Flexor digitorum profundus
FDS : Flexor digitorum superficialis
FFD : Fixed flexion deformity
FPL : Flexor pollicis longus

G

GA : General anesthesia
GCS : Glasgow coma scale
GE : Gastroenteritis
GI : Gastrointestinal
GIT : Gastrointestinal tract
GN : Glomerulonephritis
GU : Gastric ulcer

H

H/o : History of
HCC : Hepatocellular carcinoma
HRT : Hormone replacement therapy
HS purpura : Henoch-Shonlein purpura
HT : Hypertension

I

I & D : Incision and drainage
IBD : Inflammatory bowel disease
IBS : Irritable bowel syndrome
IC : Ileo-cecal
ICH : Intracerebral hematoma
ICU : Intensive care unit
IDL : Indirect laryngoscopy
IHD : Ischemic heart disease
IJV : Internal jugular vein
IL : Inferolateral
IM : Inferomedial
IMA : Inferior mesenteric artery
IP : Interphalangeal
IQ : Intelligence quotient
IUD : Intrauterine contraceptive device
IVP : Intravenous pyelography
IVU : Intravenous urography

K

K-B analysis : Kleihauer-Betke analysis
K/c/o : Known case of

L

LA : Local analgesia
LLQ : Left lower quadrant
LOC : Level of consciousness
LP : Lumbar puncture
LUQ : Left upper quadrant
LSV : Long saphenous vein
LUT : Lower urinary tract
LVF : Left ventricular failure

M

M : Male(s)
MP : Metacarpophalangeal
MRC : Medical Research Council
MI : Myocardial infarction
MNG : Multinodular goiter
MVA : Motor vehicle accident
MVT : Mesenteric vein thrombosis
M-W tear : Mallory-Weiss tear

N

NAD : No abnormality detected
NEC : Necrotizing enterocolitis
NG : Nasogastric
NTG : Nitroglycerine

O

OA : Osteoarthrosis
OPD : Outpatient department
OPG : Orthopantomogram

P

PCN : Percutaneous nephrostomy
PCNL : Percutaneous nephrolithotomy
PD : Peritoneal dialysis
PE : Pulmonary embolism
PFT : Pulmonary function test
PHT : Portal hypertension
PID : Pelvic inflammatory disease
PIH : Pregnancy-induced hypertension
PR : Per rectum

Free ebooks ==> www.Ebook777.com

PSA : Prostate-specific antigen
PTA : Post-traumatic amnesia
PTC : Percutaneous transhepatic cholangiography
PTD : Percutaneous transhepatic drainage
PTS : Post-thrombotic syndrome
PU : Pelviureteric
PUJ : Pelviureteric junction
PUO : Pyrexia of unknown origin

Q

QOL : Quality of life

R

RA : Rheumatoid arthritis
RCC : Renal cell carcinoma
RGP : Retrograde pyelography
RGU : Retrograde ureterography
RHD : Rheumatic heart disease
RLQ : Right lower quadrant
RTA : Road traffic accident, renal tubular acidosis
RTI : Respiratory tract infection
RUQ : Right upper quadrant

S

S/o : Suggestive of
SA : Spinal anesthesia
SAB : Subarachnoid bleed
SAH : Subarachnoid hemorrahge
SBE : Subacute bacterial endocarditis
SCC : Squamous cell carcinoma
SCM : Sternocleidomastoid (muscle)
SDH : Subdural hematoma
SF : Saphenofemoral
SFJ : Saphenopopliteal junction
SI : Sacroiliac
SIADH : Syndrome of inappropriate ADH secretion
SL : Superolateral
SM : Superomedial
SMA : Superior mesenteric artery
SOL : Spaceoccupying lesion
SP : Saphenopopliteal
SPJ : Saphenopopliteal junction
SPC : Suprapubic catheter/catheterization
SSV : Short saphenous vein
STN : Solitary thyroid nodule
SVC : Superior vena cava
Syn. : Synonym

T

TAO : Thromboarteritis obliterans
TB : Tuberculosis
TBSA : Total body surface area
TCC : Transitional cell carcinoma
TE : Thromboembolism
TEF : Tracheoesophageal fistula
TM : Temporomandibular
TO : Tubo-ovarian
TOS : Thoracic outlet syndrome
TURP : Transurethral resection of prostate

U

UOQ : Upper outer quadrant
URTI : Upper tespiratory tract infection
US : Ultrasound
UTI : Urinary tract infection
UUT : Upper urinary tract

V

VF : Ventricular fibrillation
VOD : Veno-occlusive disease
VP shunt : Ventriculo-peritoneal shunt
VSD : Ventricular septal defect
VT : Venous thrombosis / ventricular tachycardia
VTE : Venous thromboembolism
VU : Vesicoureteric
VVF : Vesicovaginal fistula

www.Ebook777.com

SECTION 1

General Surgery

1. General principles of case-taking
2. History-taking
3. Selected common symptoms in clinical practice
4. General examination
5. Locoregional examination
6. Respiratory system examination
7. Pelvic examination ['PS/E', 'PV/E', 'DRE' and anoscopy (Proctoscopy)]
8. Examination of the pregnant patient
9. Diagnosis
10. Guidelines on documentation of clinical notes
11. Sudden loss of consciousness (Sudden unresponsiveness) / cardiorespiratory arrest
12. Shock
13. Infections / infestations
14. Skeletal muscles in the body amenable to physical examination (Applied anatomy)
15. Swelling—basic concepts
16. Chronic lymph node swelling
17. Chronic bone swelling
18. Chronic vascular swelling
19. Chronic circumscribed swelling in the region of the head and face
20. Chronic swelling of the jaw
    Chronic oral swelling: Refer to chapter 51
21. Chronic circumscribed swelling in the neck
22. Goiter and thyroid cancer
    Swelling in the region of the breast: Refer to chapters 30 and 31
23. A reducible (/irreducible) swelling in the groin
24. A non-reducible swelling in the groin
25. Scrotal swelling / scrotal pain
26. Impalpable testicle / intermittently palpable testicle / empty scrotum
27. Chronic swelling in the back
    Swelling in the chest wall and upper back: Refer to chapter 32
28. Chronic swelling in the perineum, and gluteal region
29. Swellings / bulges related to vulva / vagina / urethra
30. Disorders of the female breast
31. Disorders of the male breast
32. Disorders of the chest wall
33. Chronic pain in abdomen
34. Acute pain in abdomen
35. Lump in abdomen
36. Distension of abdomen
37. Jaundice
38. Portal hypertension
39. Difficulty in swallowing i.e. dysphagia, and other symptoms of (chronic) esophageal disorder
40. Vomiting of blood—hematemesis
41. Anorectal pain, bleeding per rectum, and other manifestations of anorectal disorder
42. Ulcer
43. Sinus / fistula
44. (Recurrent attacks of) Discharge from the umbilicus
45. Limb pain
46. Limb ischemia—acute and chronic peripheral arterial occlusive disease
47. Limb gangrene
48. Amputation stump
49. Tortuous veins in the limbs—varicose veins
    Chronic venous insufficiency
50. Limb edema
51. Oral cavity / orofacial pain
52. Temporomandibular joint
53. Backache

Free ebooks ==> www.Ebook777.com

www.Ebook777.com

1 GENERAL PRINCIPLES OF CASE-TAKING

A clinician must gain mastery over **4 abilities.**

1. Good history-taking. It involves free and efficient communication with the patient.
2. Detect abnormal physical signs.
3. Recognize life-threatening / limb-threatening / organ-threatening disorder.
4. Confirm normality.

Symptom means a subjective feeling of manifestation of a disease e.g. pain, nausea, itching, palpitations etc.

Sign is an objective manifestation of a disease which can be detected and verified by the examiner, and sometime by the patient himself/herself e.g. tenderness, jaundice etc.

History is the subjective data. Physical findings form the objective data. Objective data also includes investigation reports.

The comprehensive subjective and objective data make up the core elements of the patient's database.

GENERAL SCHEME of a 'clinical case'

History

↓

Ask yourself: '**What am I dealing with**?'

↓

Physical **examination**: - General
- Locoregional
- Systemic

↓

Clinical diagnosis

↓

Management

– Investigation, if indicated
– Treatment. Anticipate possible postoperative events and plan the management accordingly.

↓

Progress, Termination and Follow-up.

History and physical examination constitute 'clinical case-taking'. **2 aims of case-taking** may be considered:

1. To arrive at the 'basic diagnosis' which localizes the disease and also determines the pathological nature of the disease.

2. To make a 'complete diagnosis' which includes all the relevant clinical information to support the basic diagnosis and plan a safe management. Remember, it is the patient who is treated, not the disease alone!

In the practical examination, a student is expected to present the clinical case in the (traditional) format as required by the university/board. In traditional teaching consequently, a beginner student is taught a large number of headings in history-taking and physical examination from the outset, which often gives a wrong impression that all headings are equally important. The student's mind gets tired and confused by various minute details and finally fails to sort out the most important relevant data that is useful for making the basic diagnosis which is of paramount importance.

Free ebooks ==> www.Ebook777.com

The headings of clinical case-taking which are most useful for arriving at the basic diagnosis:

1. Age and gender
2. Chief complaint/s
3. H/o organ / tissue dysfunction } Help to localize the site of the disease.
4. H/o present illness : Helps to determine the pathological nature of the disease.
5. Partial general examination:
   - Posture, gait and general activity?
   - Any sign of distress?
   - Eyes and facial expression?
   - Apparent state of health?
   - Tongue?
   - Hands, feet?
   - Vital parameters?
6. Loco-regional examination
7. Any additional points that emerge from the previous ones.

HISTORY

- History means the story of the patient's illness. The word 'story' implies a detailed account
- In a significant proportion of patients, diagnosis can be made on the basis of history alone; for example h/o a reducible swelling in the groin most probably suggests hernia; painless bright red bleeding during defecation in an adult patient suggests piles; sharp cutting pain in the anus during defecation with only minimal bleeding suggests fissure
- History-taking can be mastered by practice. In taking history and determining more important and less important facts, the knowledge and experience of a clinician are put to test most rigorously
  Poor technique of history-taking leads to confusion of the patient and obtaining wrong history.

Nonspecific symptoms may not give clue to a specific anatomical location of the disease. These symptoms relate to diversified organ/systems **and/or pathological nature** of disease. It is rather difficult to arrive at a clinical diagnosis only with the help of nonspecific symptoms and in the absence of signs (abnormal physical findings). In such situation, more reliance may have to be placed on investigation to arrive at a diagnosis. Examples of nonspecific symptoms: Anorexia, nausea, vomiting, fever, etc.

Specific symptoms relate to disease in specific organ/system e.g. dysphagia (the signature symptom of esophageal disorder).

Purposes served by history-taking:

1. It helps to make a diagnosis.
2. It forms the basis of clinical examination.
3. A reassuring relationship develops between the doctor and the patient.
4. It is worthwhile obtaining as much information as possible **before** any intended surgery, as most surprises that occur 'on table' are unhappy ones.

Principles and pitfalls in history-taking

1. Individuals vary in their capacity to observe and describe their complaints.
2. History is not mere mechanical recording or dictation-taking of the data, but each statement must be scrutinized for exaggeration or underestimation.
   a. Symptoms may be exaggerated by prisoners, hysterical persons, patients wanting leave, exemption from active service, medicolegal purpose in injury cases, etc.
   b. Reasons for underestimation: Fitness for a job, reduction in a insurance-premium, etc.

www.Ebook777.com

3. Failure by a patient, to mention a symptom or a point in history is no proof that it has not occurred.
4. Leading questions should not be asked initially; they are permitted only during revaluation.
5. Technique should be kept flexible. Case-taking yields a variety of information; it should be organized into *formal written format* only after the interview and examination are completed.

'WHAT AM I DEALING WITH'?

While beginning to listen to the patient's history, begin to formulate an answer to the question 'what am I dealing with?' The goal while attempting to answer this question is to yield possible anatomical location and pathological nature of disease. This is in preparation for clues which may be waiting later in the history and physical examination. Visual clues should also be sought during early phase of the interview.
If the question 'what am I dealing with' is answered, then innumerable irrelevant alternatives are eliminated even before physical examination is conducted; in fact it allows prediction of the physical signs which should be sought on examination. These signs may be positive or negative. Pertinent negative signs are important because they help to eliminate alternative diagnoses.

PHYSICAL EXAMINATION

Good lighting and a quiet environment are essential. Tangential lighting is much better than perpendicular lighting.
Show concern for privacy. Draw curtains / close nearby doors before the examination begins.
Drape the patient with a sheet in an appropriate manner. The goal of draping is to visualize one area of the body at a time. This preserves the patient's modesty. Examples: 1. During breast examination, after comparison of various attributes of both breasts is over, only the breast being examined should be exposed, the other half of the chest being covered by a drape. 2. For the abdominal examination, only the abdomen should be exposed. The sheet/gown should be adjusted to cover the chest, and a sheet placed to cover the external genitals and lower limbs completely (including feet). Examination of the private parts should be left at the end.
Briefly describe the plans before starting the segments of examination that might be awkward. As the examination proceeds, keep the patient informed, especially when embarrassment or discomfort is anticipated. After completing the examination, general impression should be told to the patient. The examination bed should be lowered if it was raised. Restraints should be reapplied if they were removed, and the bedrails should be raised if needed. Before leaving, waste materials should be disposed in appropriate manner, and hands washed.

SCOPE OF THE EXAMINATION

"*HOW COMPLETE THE EXAMINATION SHOULD BE* ?" There is no simple answer to this common question.

Clinical case-taking may be classified for the practical purpose, into a **comprehensive** case-taking, and a more flexible **focused or problem-oriented** case-taking.
All new patients seen for the first time ideally merit a comprehensive case-taking.

From a practical viva examination for student's point of view, clinical cases are often classified into 'a long case' and 'a short case'. A long case can be equated to a comprehensive case-taking, and a short case can be equated to a focused (problem-oriented) case-taking for making the basic diagnosis.

Components of a 'short case': Refer to the headings for making the basic diagnosis on page 4.

MINIMUM EQUIPMENT NECESSARY FOR A CLINICAL CASE-TAKING

- A paper and a pen
- A ruler and flexible tape measure preferably marked in centimeters
- A flashlight or penlight
- A watch with a second hand
- A lens to see fine details if required (for example in a case of thorn prick)

Free ebooks ==> www.Ebook777.com

- A sphygmomanometer
- A stethoscope
- Tongue depressors
- A reflex hammer
- Gloves
- Lubricant

} For vaginal, rectal, and possibly oral examinations

- Vaginal specula
- Anoscopes (Proctoscope)
- Fistula probe
- A weighing scale.

www.Ebook777.com

2 HISTORY-TAKING

For the purpose of convenience, history may be obtained under 4 basic headings: Basic information, narrative history, interrogative history, and background information.

Basic Information

1. *Date and time.*
2. *Source of reference*, if appropriate.
3. *Source of history and reliability*
   - Source of the history: Usually the patient, but can be a family member, friend, bystander, or medical record
   - Reliability varies according to the patient's age, memory, mood and IQ.
4. *Identifying data*
   - Name
   - Age
   - Gender
   - Residence
   - Occupation
   - Diet
   - Marital status.

Narrative History

= Chief complaint/s + whatever patient narrates in the flow.

Interrogative History

1. *H/o the present illness*
   - Elaboration of each chief complaint
   - Additional relevant symptoms
   - Review of systems: Common symptoms related to each major body system
   - *Gynecologic, obstetric, and contraceptive history*
   - Treatment taken (medications/any other), and response to it
   - *Any long-term medication* on which the patient has been.
2. *Past history*
   - Is this the first episode or a recurrent episode? Details of previous episode/s, if any
   - Childhood illnesses
   - Adult illnesses with dates, for at least 4 categories: Medical, surgical, obstetric / gynecologic, and details of hospital stay if any.
3. *Addictions:* Smoking, alcohol, other.
4. *Hypersensitivity.*

Free ebooks ==> www.Ebook777.com

Completing the Background Information

1. *Health maintenance practices.*
2. *Family history.*
3. *Personal and social history.*

During actual interview, the history will not, and cannot be expected to spring forth in this order. In other words, the order shown above should not dictate the sequence of the interview. Usually, the interview will be more fluid and will follow the patient's leads and cues. With practice, one quickly learns to identify where to fit in the different aspects of the patient's story.

DETAILED ANALYSIS OF HISTORY

DATE AND TIME

– It is a good practice to document the date and time not only of the first interview, but also the subsequent visits
– It has medicolegal significance as well.

SOURCE AND RELIABILITY

The judgment of reliability is usually made at the end of the interview. It reflects the quality of the information provided by the patient. An example of a comment: "The patient is vague when describing symptoms, and unable to specify details".

NAME

Name gives a personal identity.

AGE

Some disorders / diseases occur at specific age-groups. This fact can help to make or support a diagnosis.

– Congenital disorders: Cleft lip / palate, meningocele, teratoma, polycystic disease, congenital hernia, congenital hydrocele, etc. However, congenital disorders need not always manifest at birth. 'Congenital' basically means that pathologic anatomy is present since birth; the time of clinical manifestation in life may vary according to the degree of severity of the abnormality and the organ involved e.g. polycystic kidney manifests later in life; not since birth
– Diseases which occur in infants: Wilm's tumor, acute osteomyelitis, etc.
– Sarcoma is common in young age
– Typical disorders of old age: OA, BEP, cataract, etc.

Definitions of age-groups

- Up to 1 year = infancy
- 1–14 years = childhood
- 14–16 years = puberty
- 16–22 years = adolescence
- 22–40 years = adulthood (22–30 = young adult; 30–40 = adult)
- 40–55 years = middle age
- 55–75 years = old age
- Above 75 years = aged

GENDER

– Visceroptosis, hysteria, thyroid diseases, gallstone disease, postcricoid CA, etc. are more common in women
– Hemophilia occurs only in men.

www.Ebook777.com

RESIDENCE

This information may play a supportive role in arriving at the diagnosis. Some disorders are endemic in certain areas.

OCCUPATION

Diagnosis cannot be based on the occupation alone, but it may form supporting evidence, as certain occupations are known to have higher risk for developing certain disorders. Examples: Varicose veins, hepatitis, AIDS, TB, IHD, hernia, etc.

MARITAL STATUS, with number of children produced and their age

- This information has more of a therapeutic implication
- Some of the diseases of genital tract may require resection of the germ-producing organ viz. the testis / ovary
- Some of the operative procedures may damage the genital tract leading to infertility e.g. operation upon the inguinoscrotal region
- *The patient should be well-informed about these facts and* appropriate special consent should be obtained.

DIET

Non-vegetarian diet predisposes to cancer of the colon, pancreatitis, piles, etc.

CHIEF COMPLAINT / S

- Chief complaints should be noted down in *chronological order* if there are multiple complaints.
- *Exact duration* and *site* should be verified.
- *Additional clarification* may have to be obtained in some situations; for example in a c/o swelling—"Is it pulsatile/is it reducible"?

Two symptoms should never be mentioned / written in one sentence.
Example:
- Avoid 'Pain in abdomen and fever—since 4 days'
- Prefer
  - Pain in abdomen—since 4 days
  - Fever—since 4 days'.

A long list of symptoms should not be written under the heading of chief complaints.
A surgical patient usually has only 1 or 2 chief complaints; occasionally 3 or 4, and rarely more than 4.
Basically, a chief complaint is the symptom which bothers the patient most.
Chief complaint often helps to localize the site of the disease, which in-turn guides through further steps (interrogative) of history-taking. On many occasions, it may even suggest the most probable diagnosis straightaway e.g. inguinal hernia, piles, fissure, etc.

HISTORY OF THE PRESENT ILLNESS

A. **EACH CHIEF COMPLAINT SHOULD BE ELABORATED** with respect to the following points:
- Mode of onset?
- Progress / regress / fluctuation?
- Quantity-severity-intensity?
- Quality (consistency, color, odor, nature, etc; whichever appropriate)?
- Precipitating / aggravating factors?
- Relieving factors?
- Any peculiar attribute/s specific to the symptom?

Free ebooks ==> www.Ebook777.com

'Mode of onset' may be sudden, insidious, or over-and-above a pre-existing condition

i. *Sudden* (= dramatic, or sometimes explosive).
   Criteria:
   – The symptom gets established in few minutes / hours
   – Intensity of symptoms, signs and loss of function is marked.
   Examples: Angioedema, anaphylaxis; hematoma, fracture / dislocation; acute appendicitis, perforative peritonitis, acute intestinal obstruction, ureteric colic; acute DVT, embolic obstruction of peripheral arteries, acute rupture of an aneurysm, acute rupture of an ectopic pregnancy, acute inflammatory swellings like cellulitis, boil, abscess, etc.
ii. *Insidious* = pathology has been present for some length of time before symptom is noticed by the patient himself / herself, or by a friend / relative.
   Criteria: Apparent duration in days / weeks / months / years. Intensity of symptoms and sign is mild.
   Examples: Sebaceous cyst, lipoma, fibroma, etc.
iii. *Over-and-above a pre-existing condition*: Examples: Sinus / ulcer following a cold abscess; malignant transformation of a benign swelling, etc.

Progress means increase in the severity of the main symptom / sign, or development of a new symptom / sign.

i. *Dramatic* progress = progress within minutes / hours.
   Examples: Anaphylaxis; acute pancreatitis; spasms, perforation / rupture / kinking; acute vascular catastrophe like embolic occlusion, etc.
ii. *Rapid* progress = progress over days or weeks.
   Acute inflammatory conditions progress over days, malignant diseases progress over weeks.
iii. *Slow* progress = progress over months or years.
   Examples: chronic inflammatory conditions, cysts, chronic vascular diseases, endocrinal diseases, deficiency diseases, degenerative diseases.
iv. *Stationary*: Examples: Benign tumors like lipoma; sometimes strictures e.g. peptic stricture of esophagus.
v. *Slow progress -f/b- stationary -f/b- rapid progress* suggests malignant transformation of a benign condition.

B. **ADDITIONAL RELEVANT SYMPTOMS**

Examples

a. H/o **fever**?
   Causes of fever: Dehydration, infection, occult intracoelomic (e.g. intra-abdominal) abscess, autoimmune disorder, hematologic malignancy like lymphoma, etc.
b. H/o **loss of appetite**? How severe it is?
   – Nonspecific anorexia suggests TB, malignancy in general
   – Nonspecific anorexia especially if severe suggests malignancy.
c. '(Significant) **Loss of weight**?
   – If objective weight readings are not available, then ask:
   – How many percent of weight do you feel you have lost?
   – Have the previously tight-fitting clothes become loose?

C. **REVIEW OF SYSTEMS**

The *review of systems* questions may uncover problems that the patient has overlooked.
The list can include a large number of questions. Only those questions are listed below which are of practical importance and which should not be omitted / forgotten even in a busy practice.

– SOB, diminished exercise tolerance?: These symptoms are common to both respiratory and cardiac disorders.
– Chronic cough, hemoptysis?
– Chest pain, palpitations, syncope, HT, IHD, CVD, or any other cardiac disease?
– Fatigue?
– H/o 'easy bruising / bleeding, excess time required for natural hemostasis'?
– Headache, giddiness, visual disturbance (blurring, diplopia), blackouts, convulsions?

www.Ebook777.com

– DM, thyroid disorder?
– Appetite, usual weight, and any recent change in weight?
– Frequency of micturition, color of urine?
– Skin disorder, joint pains?
– H/o any immune-compromising illness / status?

D. **GYNECOLOGICAL HISTORY**

– How old were you when your periods began? i.e. the age at **menarche**?
  When talking to an adolescent girl, the opening question may be: "How did you first learn about monthly periods?"

Details of **menstrual periods**

– LMP and possibly the one before that?
– Frequency?
– Duration?
– Color of the blood—dark red or bright red?
– Do you pass 'clots'?
– How many pads do you need to use daily? Do the pads spot lightly or get soaked?

Abnormalities of menstruation

– Amenorrhea?
– Primary or secondary?
– H/o heavy bleeding followed by a period of amenorrhea?
– Oligomenorrhea?
– Dysmenorrhea?
– Premenstrual syndrome?
– Polymenorrhea?
– Menorrhagia?
– Metrorrhagia?
– Postcoital bleeding?

Menopause (where appropriate)

– Age at menopause?
– Menopausal symptoms?: Hot flashes, flushing, sweating, disturbances of sleep?
– Postmenopausal bleeding?

Vulvovaginal symptoms

– Vaginal discharge?: Duration, amount, consistency, color, odor?
– Local itching?
– Any sore or swelling in the vulval area?
– H/o sexual contacts
– The number of sexual partners in the prior month?

} Where appropriate.

Past H/o venereal disease

Herpes, gonorrhea, syphilis, PID?

DEFINITION OF TERMS, INTERPRETATION AND DISCUSSION

- **Age at menarche** is variable, depending upon genetic endowment, socioeconomic status, and nutrition
- **The dates of previous periods** alert about possibility of pregnancy or menstrual irregularities
- Significance of LMP with reference to a planned pelvic operation: Planned pelvic operations like an interval appendicectomy should be performed after bleeding phase of menstrual cycle is over. Reason: Pelvic congestion during bleeding phase may lead to more oozing / bleeding
- **Frequency of normal menstrual periods**: 24–32 days. It is measured by the intervals between the first days of successive periods
- **Duration of normal menstrual periods**: 3–7 days. H/o color of blood, passage of 'clots' and details of pads helps to determine whether the periods are 'heavy'

Free ebooks ==> www.Ebook777.com

- **Color of normal menstrual discharge is dark red. Bright** red color suggests excessive flow
- Passage of **clots** suggests excessive flow. Clots are not true blood clots, but they are fibrin clots
- **Amenorrhea** = the absence of periods
  - Primary amenorrhea = failure to initiate periods
  - Secondary amenorrhea = cessation of periods after they have been established. Physiological causes: Pregnancy, lactation and menopause. Other causes of secondary amenorrhea: Low body weight from any cause including malnutrition and anorexia nervosa, stress, chronic illness
  - Amenorrhea followed by heavy bleeding suggests threatened abortion or dysfunctional uterine bleeding related to lack of ovulation
- **Oligomenorrhea** = infrequent periods, which may also be irregular. This pattern is common for as long as 2 years after menarche, and it also occurs before menopause
- **Dysmenorrhea** = pain with menstruation. The pain is felt as a bearing down, aching, or cramping sensation in the lower abdomen and pelvis
- **Premenstrual syndrome** = a complex of symptoms occurring 4–10 days before a period
  - Tension, nervousness, irritability, depression, mood swings, headache, abdominal bloating, edema, tenderness of the breasts, and weight gain
- **Polymenorrhea** = abnormally frequent periods
- **Menorrhagia** = increased amount or duration of flow
- **Metrorrhagia** = intermenstrual bleeding i.e. bleeding occurring between periods
- Causes of polymenorrhea / menorrhagia / metrorrhagia: Systemic or dysfunctional
  Causes of **postcoital bleeding**:
  - Cervical disease e.g. polyps, cancer, etc.
  - In an older woman it may be due to atrophic vaginitis
- **Menopause** = the absence of menses for 12 consecutive months. Usually occurs between 45 and 52
- **Postmenopausal bleeding** raises the question of endometrial cancer, although it also has other causes.

OBSTETRIC HISTORY

- Have you ever been / How often have you been pregnant?
- Any difficulties with the (earlier) pregnancies?
  (Pastobstetrical problems tend to recur)
  - Complications of pregnancy?
  - Problems with labor or delivery?
- Have you ever had a miscarriage? How often?
- Was the abortion spontaneous or induced?
- How many living children do you have?

If amenorrhea suggests current pregnancy, then inquire about the common early symptoms

- Nausea and vomiting?
- Do you get fatigued?
- Have you lost weight? How much?
- Increased frequency of urination?
- Tingling / tenderness / increased size of the breasts?

INTERPRETATION AND DISCUSSION

- *Common concerns during pregnancy* having physiological basis

– Amenorrhea	Throughout
– Nausea and vomiting	1st trimester
– Fatigue	1st and 3rd trimester
– Loss of weight	1st trimester
– Increased frequency of urination	1st and 3rd trimester
– Tingling / tenderness / increased size of the breasts	1st trimester
– Pain in the groin / lower abdomen	2nd trimester (14–20 weeks)
– Edema	3rd trimester
– Heartburn	Throughout
– Constipation	Throughout

www.Ebook777.com

– Backache — Throughout
– Leukorrhea (asymptomatic milky white vaginal discharge) — Throughout.

E. **MEDICATIONS / ANY OTHER TREATMENT** taken, and **response** to them.
– Name, dose and route of allopathic / ayurvedic medicines consumed?
– Alternative medicines / herbal medicines / home remedies?
– Medicines borrowed from family members / friends?
– Vitamins, minerals?
– OC pills?

F. **ANY LONG-TERM MEDICATION** on which the patient has been for sometime e.g. antihypertensive, antidiabetic, antitubercular, antiretroviral, etc. Details?

PAST HISTORY

1. Is this **the first episode** of the illness **OR** Is this a **recurrent episode**?
   Relevant details of the previous episode/s?
2. Childhood illnesses?
3. Illnesses through adulthood?
   – Medical e.g. pneumonia, TB, depression / any other psychiatric illness, peptic ulcer, jaundice, etc.
   – Surgical e.g. abscess, cyst, hernia, appendicitis, colitis, gallbladder disease, urinary stones / any other urinary illness; any surgery, injury, accident?
   – Gynecological / obstetric?
   – Details of hospital stay, if any?

SMOKING / CONSUMPTION OF TOBACCO in any other form?

- If smoking cigarettes, ask 'how many packs a day?'
- Cigarettes are often reported in pack-years. For example, a person who has smoked 1.5 packs a day for 10 years has a 15-pack-year history
- Tobacco is literally a poison which leads to a vast number of diseases. There is a direct relationship between the prevalence of disease and the number of cigarettes smoked e.g. in smoker's arteritis, cancer of the larynx, cancer of the lung, cancer of the urinary bladder, etc.

ALCOHOL?

- One unit is equivalent to half a pint of beer / 1 glass of wine / 1 measure (pub) of spirits / 1 glass of sherry
- Clinically significant problems are unlikely, unless patient has been drinking > 50 units / week.

OTHER ADDICTIONS? Details?

HYPERSENSITIVITY? Details?

HEALTH MAINTENANCE PRACTICES?

– Immunization: For tetanus, etc.
– Screening tests: Such as pap smears, mammogram, stools for occult blood, etc.

FAMILY HISTORY

– Age, health and cause of death (if applicable) of siblings, parents and grandparents?
– Common hereditary diseases that are known to run in families—HT, IHD, DM, polycystic kidney, CA-breast, etc?
– Communicable diseases may occur in family members e.g. TB, scabies, etc.
– Non-communicable diseases like food poisoning, peptic ulcer, etc. may occur in family members.

Free ebooks ==> www.Ebook777.com

PERSONAL AND SOCIAL HISTORY

- Educational level?
- Family of origin?
- Personal interests and lifestyle?
- Sleep?
- Bowel habit?
  Criterion of constipation: Decrease in the frequency of stools regardless of consistency and volume, to < 2 stools per week.
- Bladder habit?
  Normal frequency is a function of the normal rate of formation of urine (0.5 to 1 mL / kg body weight / hour) and the normal functional (physiological) capacity of the bladder (300 mL in adult). For example, the normal frequency in a person weighing 70 kg will be ~ 3–6 times / 24 hours.

www.Ebook777.com

3 SELECTED COMMON SYMPTOMS IN CLINICAL PRACTICE

PAIN

Pain is the commonest symptom.

- Original site (pinpointing with a finger), shifting, radiation, extension?
- Duration, mode of onset, progress / regress / fluctuation?
- Intensity: Mild / moderate / severe?
- Nature: Throbbing, twisting, colicky, constricting, burning, shooting, vague?
- Precipitating / aggravating / relieving factors?
- Periodicity?
- Associated manifestations? viz. vomiting, pallor, excessive sweating and cold extremities ⇒ excruciating pain s/o acute serious pathology e.g. dissecting aneurysm, etc.

FEVER

- Duration?
- Any particular pattern of onset, rise and fall?
- Night sweats?
  - TB, cancer. Feelings of heat and sweating also accompany menopause
- Accompanied by rigors (shivering throughout the body and chattering of teeth)?
  - Rigors suggest more extreme swings in temperature, and suggest more serious infection. Chills or chilliness is subjective, and will accompany any fever even of minor prognostic significance. Rigors carry more significance than chills
- Ask h/s/o various patterns of infectious diseases
- Did you travel of late?
- Did you have contact with sick person/s at home or office?
- Have you been taking any medicine/s?
  - Medicines may cause fever. In contrast, recent ingestion of aspirin, acetaminophen, corticosteroids and NSAIDs may mask fever.

Notes

- Feeling cold, and shivering accompany a rising temperature
- Feeling hot and sweating accompany a falling temperature
- Malaise, headache, and pains in the muscles and joints (body aches) often accompany fever.

SHORTNESS OF BREATH (*Syns*. BREATHLESSNESS, DYSPNEA, DIFFICULTY IN BREATHING)

- Duration?
- Mode of onset?
- Progress?
- Is this the 1st episode or recurrent episode?

Free ebooks ==> www.Ebook777.com

- Details of the previous episodes?
- Which treatment have you been taking?

Further history to be asked in acute SOB

- Previous h/o any thrombotic episode/s?
- Have you been suffering from any major medical illness e.g. IBD etc?
- Have you been suffering from any cancer?
- Have you been on chemotherapy?
- Have you been on estrogen?

} – Factors predisposing to DVT.

- Have you been suffering from any chronic lung disease?
- Fatigue?
- Orthopnea?
- Paroxysmal nocturnal dyspnea?

} – Suggest CHF.

- Pain in the chest?
  - – Is it retrosternal oppressing? ⇒ Acute PE.
  - – Does it increase on breathing / coughing? ⇒ Pleuritic pain.
- Feeling of tightness in the chest? ⇒ Asthma.
- Cough?
- Expectoration?
- Hemoptysis?
- Wheezing?

} – Associated symptoms s/o respiratory disorder.

- Palpitation?
- Syncope?
- Known to have HT/IHD/CVD?

} – Associated symptoms s/o cardiac disorder.

Further history to be asked in chronic SOB

- Do you smoke?
- Are you exposed to air pollution?
- Recurrent attacks of RTI?
- Are you suffering from a heart disease?
- Precipitating / aggravating factors?
- Does it occur even at rest?

Clinical setting, and possible cause of acute SOB

– Postpartum or postoperative period
– The presence of factor/s predisposing to DVT (clinically obvious DVT in the limbs cannot be expected, as the thrombus would be floating at this juncture)
– The presence of a chronic lung disease
– CHF

↓

Think of acute PE

Additional specific clinical features which may be present

– A feeling of impending doom
– Restosternal oppresing pain.- Suggests massive occlusion
– Pleluritic chest pain } Suggests pulmonary infarction
– Hemoptysis }
– Patient looks anxious

– SOB occurring at night time
– Episodic
– Exposure to allergen
– Precipitated by emotional stress/exercise
– The presence of respiratory infection
– Tightness in the chest
– Wheezing

↓

Think of asthma

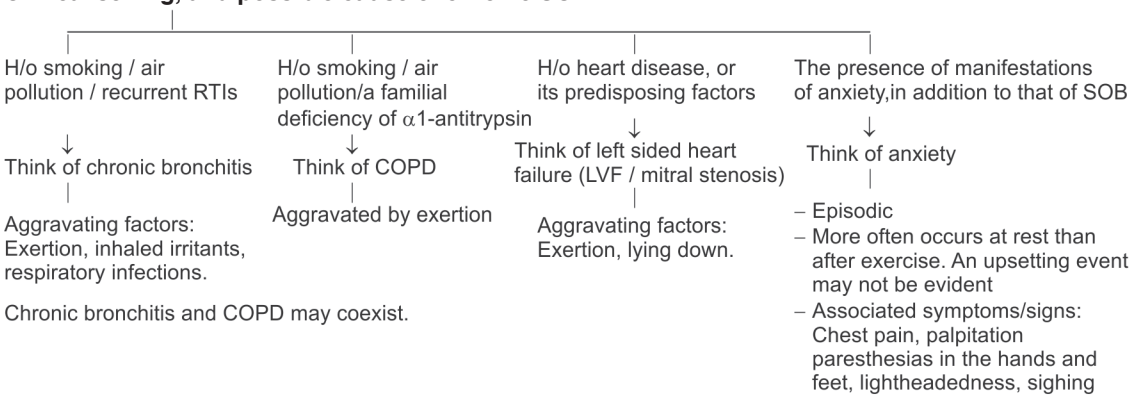
Previously healthy person

↓

Think of – Pneumonia
– Spontaneous pneumothorax

www.Ebook777.com

Clinical setting, and possible cause of chronic SOB



CAUSES and DD of **COUGH** (± HEMOPTYSIS)

- Duration?

History to be asked in acute cough

- Character? - barking / brassy, etc?
  - Barking cough ⇒ laryngeal infection, particularly epiglottal
  - Brassy cough ⇒ tracheobronchial infection.
- Is it associated with
  - Hoarseness of voice?
  - Running nose, scratchy throat, pain in the throat?
  - Burning discomfort behind the chest bone?
  - Pain in the chest?
  - Intensity?
  - Is it oppressive?
  - SOB?
  - Wheezing?
- Were you exposed to any irritant?
- Scratchy throat?
- Watering of the eyes, nose?

} For cough caused by exposure to irritating particles / chemicals / gases.

History to be asked in chronic cough

- Is it constant or intermittent (i.e. episodic)? – Episodic cough suggests bronchitis, asthma
- Is it associated with SOB or / and wheezing? – May suggest chronic bronchitis/asthma
- Is any allergen known to precipitate an episode of cough? – May suggest allergic bronchitis/asthma
- Have you been suffering from common cold / sinusitis?
- Do you sense a postnasal drip?
- Do you have to make repeated attempts to clear the throat?

} For postnasal drip

- Loss of appetite?
- Loss of weight?
- Low-grade fever in the evening?
- Night sweats?

} For pulmonary TB

Free ebooks ==> www.Ebook777.com

- What is the amount of expectoration?
- Is it foul-smelling?
- Have you been suffering from recurrent LRTI?
- Is it precipitated/aggravated by changing position?

– Copious, foul-smelling expectoration, and recurrent attacks of LRTI may be suggestive of bronchiectasis
– Foul-smelling expectoration precipitated / aggravated by changing position suggests lung Abscess

- Have you been smoking for a 'long' time? – May suggest chronic bronchitis / CA lung

- Does it occur specifically at night/in the early morning?
- Hoarseness of voice early in the morning?
- Repeated attempts to clear the throat?
- Heartburn and regurgitation?

For GE reflux.

- Does it occur at night / is it precipitated by exertion?
- Is it associated with SOB / orthopnea / paroxysmal noctural dyspnea?

For LVF/mitral stenosis.

***Causes** of cough*	*Associated features*	*Remarks*
Acute inflammation		
Laryngitis	Hoarseness of voice	Often associated with viral nasopharyngitis
Tracheobronchitis	Burning retrosternal discomfort	Often viral illness
Pneumonias—mycoplasma / viral	– Constitutional symptoms – SOB	An acute febrile illness
Pneumonias—bacterial	– Constitutional symptoms – SOB – Chest pain	– An acute febrile illness, often preceded by an acute URTI – Typically occurs in older alcoholic men – In *Pneumococcal pneumonia*, the sputum is mucoid or purulent; it may be blood-streaked, pinkish, or rusty – In *Klebsiella pneumonia* the sputum is similar to that as in *Pneumococcal pneumonia*; or it may be sticky, red, and jelly-like
Chronic inflammation		
Postnasal drip	– Chronic rhinitis with/without sinusitis – Repeated attempts to clear the throat – Postnasal drip may be sensed by patient or seen in the pharynx	-
Chronic bronchitis	– SOB – Wheezing	– Long-standing cigarette smoking – Recurrent superimposed infections
Pulmonary TB	– Initially no other symptom – Later, loss of appetite, loss of weight, and low-grade fever in the evening ± night sweats	-

Contd...

www.Ebook777.com

Contd...

***Causes** of cough*	*Associated features*	*Remarks*
Bronchiectasis	– Recurrent bronchopulmonary infections common – Sinusitis may coexist.	Sputum—purulent, often copious and foul-smelling, may be blood-streaked or even bloody
Lung abscess	– Often poor dental hygiene – A prior episode of impaired consciousness	A febrile illness Sputum—purulent and foul-smelling, may be blood-streaked or even bloody
Asthma	– Episodic SOB and wheezing, but cough may occur alone – Often a h/o allergy	-
CA lung	– Usually a long h/o cigarette smoking – Associated manifestations are numerous	-
Gastroesophageal reflux	– Wheezing especially at night (often mistaken for asthma) – Early morning hoarseness – Repeated attempts to clear the throat – Often a h/o heartburn and regurgitation ±	-
Cardiovascular disorders		
LVF or mitral stenosis	SOB, orthopnea, paroxysmal nocturnal dyspnea	Cough—often dry; especially comes on exertion, or at night Sputum—may become pink frothy suggestive of pulmonary edema, or frank hemoptysis
PE	– Feeling of impending doom – Retrosternal oppressive pain ⇒ massive occlusion – Pleuritic pain – Patient looks anxious *Predisposing clinical setting* – Postpartum / postoperative period – Clinical settings predisposing to develop DVT. C/Fs of DVT usually absent as the thrombus is floating – Chronic lung disease – CHF	Sputum that is dark, bright red, or mixed with blood ⇒ pulmonary infarction
Irritating particles / chemicals / gases	- Exposure to irritant - Eyes, nose, and throat may be affected	There may be a latent period between exposure and symptoms

Free ebooks ==> www.Ebook777.com

CAUSES and DD of **PAIN IN THE CHEST**

Intrathoracic disorders

Referred from extra-thoracic structures
- Neck: Arthritis, etc.
- Abdomen: Subdiapragmatic abscess, acute cholecystitis, biliary colic

Causes	*Pathological process*	*Site*	*Duration*	*Intensity*	*Nature*	*Aggravating factors*	*Relieving factors*	*Associated symptoms*
Angina pectoris	Temporary myocardial ischemia	Retrosternal, or across the anterior chest; may radiate to shoulders / arm / neck / lower jaw / upper abdomen	A few minutes. May be prolonged up to 20 minutes	Mild to moderate. Sometimes perceived as discomfort rather than pain	Variable: pressing / squeezing / tightness / heaviness / burning	Exertion Meals Emotional stress However may commence even at rest	Rest NTG	SOB Nausea Sweating
MI	Prolonged myocardial ischemia → irreversible muscle damage (necrosis)	Same as angina	20 minutes to several hours	Often but not always severe. In diabetics, attack may be even painless	Same as in angina	-	-	Nausea Vomiting Sweating Weakness
Pericarditis	Unclear	– Precordial, may radiate to the tip of the shoulder and to the neck – Retrosternal	Persistent Persistent	Often severe Severe	Sharp knife-like Crushing	Breathing, coughing, sometimes swallowing. Changing position	Sitting forward may relieve it	Of the underlying illness
Dissecting aortic aneurysm	A splitting within the layers of the aortic wall, allowing passage of blood to dissect a channel	Anterior chest, radiating to the neck, back or abdomen	M/o onset: Abrupt Dramatic progress Persists for hours or more	Very severe	Ripping, tearing	HT	-	Syncope. Hemiplegia or paraplegia

Contd...

www.Ebook777.com

Contd...

Causes	*Pathological process*	*Site*	*Duration*	*Intensity*	*Nature*	*Aggravating factors*	*Relieving factors*	*Associated symptoms*
Reflux esophagitis	Inflammation of the esophageal mucosa by reflux of gastric acid	Retrosternal High epigastrium/ xiphoid region ± between the scapulae or either lumbar region	Variable	Mild to severe	Burning May be squeezing	Large meal Stooping Lying down flat	Assuming sitting position	Excessive belching, ± specific symptoms of esophageal disorder Tenderness high in the epigastrium ±
Diffuse esophageal spasm	Motor dysfunction of the esophageal muscle	Retrosternal; may radiate to the back / neck / jaw	Variable	Mild to severe	Usually squeezing	Swallowing of cold liquid Emotional stress	Sometimes NTG	Dysphagia
Tracheobronchitis	Inflammation of trachea and large bronchi	Upper sternal or parasternal	Variable	Mild to severe	Burning	Coughing	-	Cough
Pleural pain	Pleuritis due to pleuracy / pneumonia / pulmonary infarction / tumor	Chest wall overlying the pathological process	Persistent	Often severe	Sharp knife-like	Breathing Coughing Movements of the trunk	Lying on the involved side	Of the underlying illness
Chest wall pain	Variable; often unclear	Often below the left breast or along the costal cartilages; also elsewhere	Fleeting Hours or days	Variable	Stabbing / aching / vague	Movements of chest, trunk, arm	-	Often local tenderness
Anxiety	Unclear	Precordial / below the left breast / across the anterior chest	Fleeting Hours or days	Variable	Stabbing / aching / vague	Emotional stress	-	SOB Palpitation Weakness Anxiety

Free ebooks ==> www.Ebook777.com

FATIGUE

It refers to a sense of weariness or loss of energy that patients describe in various ways.

Causes
- Hard work, sustained stress, grief.
- Chronic disease of the lungs, heart failure
- DM, hypothyroidism, adrenal insufficiency, panhypopituitarism
- Moderate to severe anemia, nutritional deficiency, electrolyte imbalance, chronic disease of liver, chronic disease of kidney
- Medications
- Infections—TB, hepatitis, infectious mononucleosis, etc.
- Cancer
- Depression, anxiety.

Because fatigue is a normal response to hard work, sustained stress, or grief, try to elicit the life circumstances in which it occurs. When such situations are ruled out, investigation is needed.
Infants and children cannot describe fatigue verbally. Changes in behavior such as withdrawal from normal activities, irritability, loss of interest in their surroundings, and excessive sleeping may indicate fatigue.

WEAKNESS

- **Weakness is different from fatigue.** It denotes demonstrable loss of muscle power
- Weakness, especially if localized in neuroanatomic pattern, suggests possible neuropathy or myopathy.

BLEEDING FROM GUMS

- Do you have any lesion in the gum?
- Do you have a tendency to bleed or bruise elsewhere?

Most common cause: Gingivitis.

EPISTAXIS

= bleeding from the nose
- Duration, mode of onset, progress, regress, fluctuation, amount, color, any known precipitating factor?
- H/o nose picking / trauma / foreign body?
- H/s/o infective-inflammatory pathology in the nose / nasopharynx / paranasal sinuses?
- Is this the first episode OR is it a recurrent episode? Details of the previous episodes, if any?

Causes
- Local:
  - Trauma (nose picking, injury, foreign body)
  - Inflammation, drying, crusting of the nasal mucosa
  - Tumor
- Regional: Nasopharyngitis, paranasal sinusitis, # anterior cranial fossa
- Bleeding disorder / patient on anticoagulant therapy.

HEADACHE

- **Duration?**
  - Few hours to days = 'Short' duration
  - > a week, may be months or even years = 'Long' duration
    - Constant or episodic?
- **Mode of onset**?
  - Sudden mode of onset may suggest meningitis / SAH / acute glaucoma / cluster headaches / migraine, etc.

www.Ebook777.com

- **Intensity**?
  - Very severe in meningitis, SAH
  - Severe in cluster headaches
  - Mild ache or feeling of tightness and pressure may suggest tension headache
  - Rest of all headaches may be of variable intensity, and consequently it may not be possible to differentiate them on the basis of intensity alone
- H/o **head injury**?
  - When?
  - A few hours before the onset of headache, OR weeks to months earlier?
- Have you noticed any particular **precipitating / aggravating factors / relieving factors**?
- **Fever**?
  - May suggest meningitis, SAH, paranasal sinusitis, toxic vascular headache, giant cell arteritis, etc.
- **Vomiting**?
  - May suggest meningitis, SAH, acute glaucoma, raised ICP, migraine, etc.
- **Specific associated symptoms to be asked in severe/very severe headaches with sudden mode of onset**
  - Fever, neck pain/stiffness, deterioration of LOC?
    - For meningitis/SAH
  - Diminished vision?
    - For acute glaucoma, giant cell arteritis
  - Unilateral stuffy running nose, reddening and tearing of the eye?
    - For cluster headaches
- **Specific associated symptoms to be asked for head injury as a probable cause of headache**

  A pitfall: An injury (particularly when old) is often forgotten.
  - In c/o headache commencing within a few hours of injury: Poor concentration, giddiness/vertigo, irritability, restlessness, fatigue?
  - In a suspected c/o post-traumatic chronic SDH: Alteration of LOC, changes in personality, hemiparesis?
- **Specific associated symptoms to be asked in rest of the cases**
  - Nasal discharge, nasal congestion, local tenderness?
    - For paranasal sinusitis
  - Preceding visual disturbances e.g. flashes of light, blind spots?
  - Neurological symptoms e.g. weakness, sensory disturbances, etc?

    } For migraine
  - Clear-cut h/o an error of refraction OR h/o eye fatigue/'sandy' sensations in the eyes / redness of conjunctiva?
  - Anxiety, depression, tension?
    - For tension headache.
  - Anorexia, fatigue, malaise, muscular aches and stiffness, tenderness of the scalp, visual loss / blindness
    - For giant cell arteritis.
  - Forgetfulness, slow comprehension, decreased intellectual power, inability of sustained mental activity, undue irritability, social lapses?
  - Emotional instability?
  - Epilepsy?

    } For intracranial SOL—brain tumor

Causes of headache

Short duration	Long duration
– (Acute) Paranasal sinusitis	– Errors of refraction
– Migraine	– Tension headache
– Meningitis	– Toxic vascular headaches
– Acute SAH	– Giant cell arteritis
– Acute glaucoma	– Post-traumatic chronic SDH
– Cluster headaches	– Post-concussion syndrome
	– Brain tumor (Intracranial SOL)

***DD** of headache of surgical relevance*

Cause	*Site*	*Duration*	*Mode of onset*	*Course*	*Precipitating / aggravating factors*	*Relieving factors*	*Associated specific symptoms*	*Remarks*
(Acute) Paranasal sinusitis	Overlying the frontal sinus / maxillary sinus	Several hours at a time; recurring over days or longer	Variable.	– Recurring daily – Starts in the morning in frontal sinusitis – Starts in the afternoon in maxillary sinusitis	Coughing/sneezing/ jarring the head	Nasal decongestion	Nasal discharge, congestion, local tenderness	Intensity—variable Nature—aching / throbbing
(Acute) SAH	Generalized	Few days	Sudden	Persistent	-	-	– Nausea – Vomiting – Pain in the neck – Loss of consciousness ±	Intensity—very severe
Acute glaucoma	In and around one eye	Variable	Sudden	Variable	Sometimes by drops that dilate the pupils	-	– Diminished vision ± Nausea, vomiting	Intensity—often severe
Post-traumatic chronic SDH	Variable	Variable	Insidious Weeks/ months after injury	Progressively severe, but may be obscured by clouded consciousness	-	-	– Alterations in the LOC – Changes in personality – Hemiparesis	**The injury is often forgotten**
Post-concussion syndrome	May be, but not necessarily localized to the injured area	Weeks/ months /even years	Within a few hours of injury	Tends to diminish over time	Mental and physical exertion, straining, stooping, emotional excitement, alcohol	Rest	Poor concentration, giddiness or vertigo, irritability, restlessness, fatigue	Intensity—variable
Brain tumor (Intracranial SOL)	Varies with the location of the tumor	Variable Often brief	Variable	Often intermittent, but on the whole, progressive	Coughing / sneezing / sudden movements of the head	-	Symptoms s/o intracranial SOL	-

www.Ebook777.com
Free ebooks ==> www.Ebook777.com

VOMITING

- Duration?
- Frequency?
- Contents?
- Color?
- Odor?
- Is it 'effortless'?
- Is it preceded by nausea?
- Is it related to food?

Arriving at the diagnosis of vomiting

– Could it be physiological? – For example, due to pregnancy / obnoxious smell / taste / tragic scene / emotion / motion sickness?

– Is vomiting one of the symptoms of a symptom-complex? – Arriving at the diagnosis may be relatively easier

OR

Is it the only chief complaint without any other associated symptom? – Arriving at the diagnosis may be difficult. – Unusual causes may have to be considered

– Vomiting is a distressing symptom; it is usually though not necessarily is associated with an acute / subacute illness

– Arriving at the diagnosis in case of vomiting of long duration (which may be episodic or constant) when associated with other symptoms, will not pose much difficulty.

Pathological causes of vomiting

System affected	*Pathology/pathogenic agent*
– NS	– Migraine, meningitis, encephalitis, raised ICP, bulbar paralysis – Causes of reflex vomiting: Severe deep pain, internal ear disease, acute glaucoma
– Endocrine	Thyroid crisis, hyperparathyroidism, Addison's disease, Simmond's disease
– Cardiac	Acute MI (especially inferior wall), heart failure
– Digestive	Pharyngeal irritation, food poisoning, drug-induced gastritis, blood-induced gastritis (blood in the stomach from any cause), intestinal obstruction, visceral inflammation, peptic ulcer disease, 'indigestion', etc.
– Metabolic	Acidosis
– Urinary	Uremia, renal lithiasis
– Immune	– Bacterial / viral / parasitic infections of the GIT – Systemic (acute) infections especially in children
– Mind	Psychogenic

Definitions of terms

- *Nausea* = the imminent desire to vomit
- *Vomiting* = forceful expulsion of gastric contents through the mouth
- *Retching* = labored rhythmic respiratory activity that precedes vomiting
- *Regurgitation* = expulsion of gastric/esophageal contents in the absence of nausea and abdominal diaphragmatic muscular contraction.

VERTIGO

- Do you feel that the room is spinning around you?
- Do you feel pulled to the ground or off to one side?

} ⇒ 'True' vertigo.

Free ebooks ==> www.Ebook777.com

- Duration, mode of onset, course?
- Hearing loss?
- Tinnitus?
- Have you been consuming a drug (e.g. aminoglycoside)?
- H/s/o alcohol intoxication?
- Nausea, vomiting?
- Any symptoms s/o pressure on CN V, VI and VII?
- Any symptoms s/o brainstem disorder?
- Any symptoms s/o cerebellar disorder?

Causes of vertigo: Benign positional vertigo, vestibular neuronitis (acute labyrinthitis), Ménière's disease, drug toxicity, tumor pressing on the CN VIII, disorder of the brainstem/cerebellum.

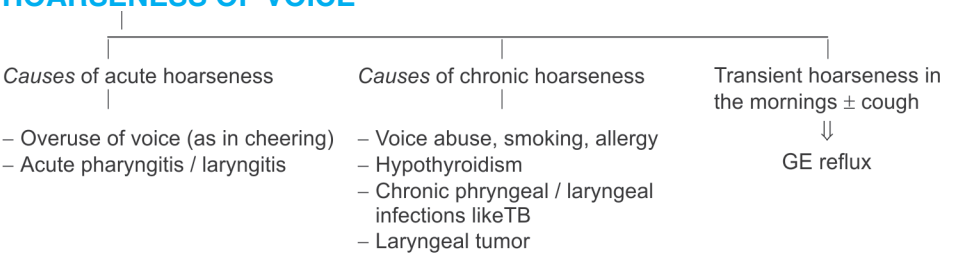
DIPLOPIA (DOUBLE VISION)

- Do you get it with both eyes or with only one eye, the other eye having been closed?
- Horizontal/vertical?

Causes of diplopia (in adults)

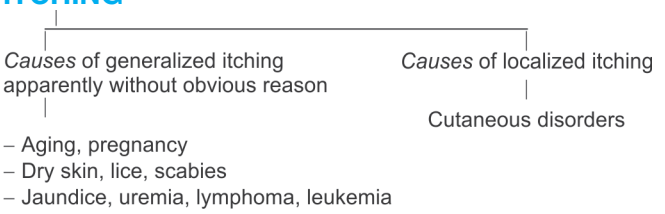
– Diplopia in one eye, with the other eye closed ⇒ problem in the cornea or lens
– Lesion in the brainstem or cerebellum
– Weakness/paralysis of one or more extraoccular muscles:
  - Horizontal diplopia ⇒ CN III/VI palsy
  - Vertical diplopia ⇒ CN III/IV palsy.

HOARSENESS OF VOICE



Hoarseness lasting > 2 weeks should be thoroughly examined and investigated if necessary.

ITCHING



EAR SYMPTOMS

Pain

Ask the following points in addition to the usual details about pain.

- Fever?
- Concurrent URTI
  – Sore throat/cough, etc?

www.Ebook777.com

Causes of pain in the ear
– Earache without concurrent URTI ⇒ otitis externa
– Earache associatd with concurrent URTI ⇒ otitis media
– Fever ⇒ infection.

Discharge

- Duration, mode of onset, progress / regress / fluctuation, quantity, quality (consistency, color, odor, nature, etc.)?
- H/o trauma which could be suggestive of fracture base of the skull?
- H/o injury with a sharp object / attempt at removal of wax or a foreign body with a sharp object?
- Earache?

Causes of discharge from the ear
– Unusually soft wax
– Debris from inflammation or rash in the ear canal
– Acute / chronic otitis media
– Fracture of base of the skull through the middle cranial fossa.

Tinnitus

= a perceived sound that has no external stimulus
– Possible characters of the sound: Musical ringing / rushing / roaring / popping sounds originating in the TM joint / vascular noises from the neck
– It can be unilateral or bilateral
– Often remains unexplained. Increases in frequency with age
– May accompany loss of hearing
– When associated with hearing loss and vertigo, it suggests Ménière's disease.

SORE THROAT

Sore throat associated with fever, pharyngeal exudate, and anterior cervical lymphadenitis, especially in the absence of cough ⇒ streptococcal pharyngitis.

SORE TONGUE

Causes: Aphthous ulcer, atrophic glossitis.

Free ebooks ==> www.Ebook777.com

4 GENERAL EXAMINATION

- Is the patient awake, alert and responsive? In other words—is the patient conscious, cooperative, and well-oriented in time, place and person?
- Posture, gait and general activity?
- Signs of distress?
- Eyes and facial expression?
- Apparent state of health: Healthy / sick / frail and feeble / terminally ill?
- Skin?
- Hands: Dorsum, nails, and palm?
- Feet and legs: Edema, and venous filling?
- Vital parameters, JVP?
- Height, weight, BMI, body proportion?
- Skull, hair, scalp?
- Mouth: Lips, tongue, cheek, sulci, gums and teeth, throat, palate, and floor?
- Nose, ear, neck?
- Lymphadenopathy?
- Odor?

ANALYSIS IN DETAILS

IS THE PATIENT **CONSCIOUS** (i.e. **AWAKE** AND **ALERT**)?

If NOT, and if the circumstance is an acute one, then—proceed as in chapter 11.

IS THE PATIENT **COOPERATIVE?**

Causes of un-cooperation from the patient

- Extreme of ages
- Psychological / psychiatric problems
- Criminals (under arrest).

IS THE PATIENT **WELL-ORIENTED IN TIME, PLACE** AND **PERSON?**

Disorientation in time and/or place and/or person suggests that the patient may be in a confusional state or delirium.

Causes of **acute confusional state**

- Primary neurological disorders contribute to only 10% of those patients presenting with an acute confusional state
- In the elderly, postoperative confusion is particularly common.

www.Ebook777.com

Delirium

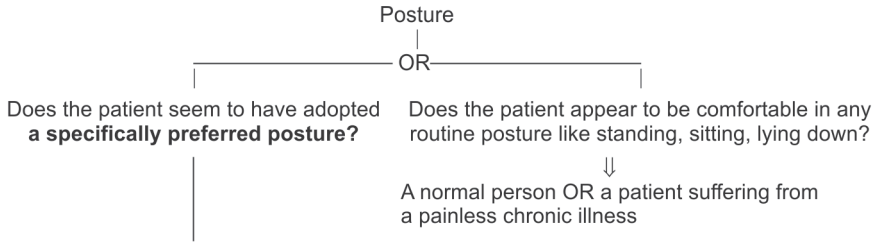
Perceptual disorders (illusions and hallucinations) may accompany the confused state—delirium. It is often associated with withdrawal and lack of awareness or with restlessness and hyperactivity.

The confusion assessment method is used to confirm delirium:

– Feature 1: Acute onset and fluctuating course.
– Feature 2: Inattention.
– Feature 3: Disorganized thinking.
– Feature 4: Altered LOC.

The presence of features 1 and 2 and either 3 or 4 are diagnostic.

POSTURE, GAIT AND GENERAL ACTIVITY?



1. A patient suffering form acute peritonitis prefers to lie supine quietly.
2. Restless, tossing/rolling about in bed in agony, unable to find a comfortable position suggest a colic e.g. ureteric colic.
3. Preferring to sit all the time with the affected limb either hanging over side of the bed, or drawn close to the body and constantly being messaged in a vein attempt to get a relief from pain suggests 'rest pain'.
4. Patients with cardiorespiratory embarrassment, pancreatic disorder are more comfortable in sitting-up position.
5. FFD at hip joint may suggest iliacus hemotoma or iliopsoas abscess.
6. A patients with pleuritis feels more comfortable in lying-down position on the affected side (as movement on the affected side is restricted).
7. In hemiplegia,the affected lower limb is extended and externally rotated.

Can the patient walk normally?

The patient may be made to walk provided s/he is not sick.

Examples of abnormal gait

– Surgical: 'Appendix shuffle' in acute appendicitis
– Medical: Spastic hemiparesis, scissors, steppage, sensory ataxia, cerebellar ataxia, Parkinsonian gait, etc.

Does the patient appear **restless?**

Common causes of restlessness: Thyrotoxicosis, anxiety.

Does the patient appear **conscious but quiet and slow to respond?**

Possible causes: Hypothyroidism, depression, presence of any significant illness.

SIGN OF DISTRESS?

– **Anxious face with a feeling of immediate impending doom**

  The latter part viz. 'the feeling of immediate impending doom' is very subtle, and although it is the patient's subjective feeling, the clinician should be able to read it on the patient's face!

  It implies an acute catastrophic condition which may lead to sudden death e.g. massive pulmonary embolism, acute MI, acute dissecting aortic aneurysm, etc.
– Anxious face with fidgety movements, inexpressive or flat affect, poor eye contact, psychomotor slowing ⇒ **anxiety/depression**
– Labored breathing, wheezing, cough ⇒ **respiratory** distress

Free ebooks ==> www.Ebook777.com

– Clutching the chest, pallor, profuse perspiration ⇒ **cardiac** distress
– Wincing, sweating, protectiveness of painful area ⇒ distress from **pain**.

EYES AND FACIAL EXPRESSION?

How is the **eye contact**—natural or decreased?
Decreased eye contact may be cultural, or may suggest anxiety, fear or sadness.

Bitot's spots, keratomalacia, suggest undernutrition.
Yellow discoloration of sclera suggests jaundice.
'**Red eye**'? *Causes of red eye*: Conjunctivitis, Corneal injury/infection, acute iritis, acute glaucoma, subconjunctival bleed.

Eyelids
Abnormalities of eyelids: Ptosis, retraction, periorbital edema, herniation of fat, ectropion, entropion, epicanthus.

- *Ptosis* = drooping of the upper lid.
  Causes: 1. Myesthenia gravis, damage to CN III, damage to the sympathetic nerve supply (Horner's syndrome). 2. Senile ptosis (due to weakened muscle, relaxed tissue, and the weight of herniated fat). 3. Congenital.
- *Retracted lid* → a wide-eyed stare.
  Causes: 1. Hyperthyroidism. 2. May be seen in normal people.
- *Periorbital edema*. Causes: Allergy, cellulitis, myxedema, fluid-retaining states such as nephrotic syndrome. Because the skin of the eyelids is loosely attached to underlying tissues, edema tends to accumulate easily in the eyelids.
- *Herniated fat* → puffy lids. This condition affects the lower lid, and medial 3rd of the upper lid. These bulges appear more often in the elderly, but may affect younger people.
  Mechanism: Fat pushes weakened fascia in the lids forwards, producing bulges.
- *Ectropion* = outward turning of the lower-lid margin. When punctum of the lower lid turns outwards, it leads to epiphora.
  Common in the elderly.
- *Entropion* = inward turning of the lid margin. → irritation of the conjunctiva and lower cornea.
  Common in the elderly.
- *Epicanthus* = a vertical fold of skin that lies over the medial canthus. - may falsely suggest a convergent squint.
  Causes: 1. Normal among many Asian people. 2. Down's syndrome and a few other congenital conditions.

Face is a mirror of one's pathological and emotional status.

Feature	*Descriptor of the facial appearance/ disorder*	*Mechanism / remark*
Sunken eyes, drooping eyelids, bony prominences stand out as sharp ridges through the lax superficial tissues	Hippocratic facies	Seen in the terminal phase of illness prior to death
Dull look Malar flush Periorbital and supraclavicular puffiness	Myxedema	Severe hypothyroidism
Staring looks (due to lid-retraction) and expression of fear (due to exophthalmos)	Hyperthyroid facies	Hyperthyroidism due to Grave's disease only
Open mouth and narrow nares with stupid appearance	Adenoid facies	–

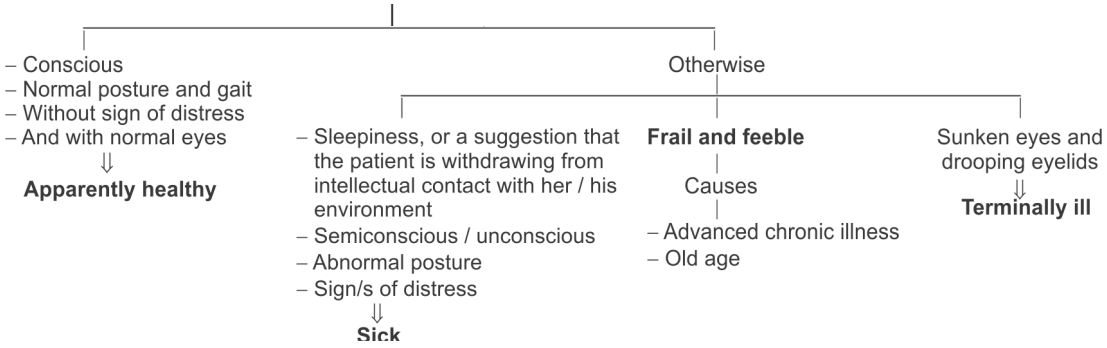
Contd...

www.Ebook777.com

Contd...

Feature	*Descriptor of the facial appearance/ disorder*	*Mechanism / remark*
Head—elongated with marked bony prominences of the forehead, nose and lower jaw Soft tissues of the nose, lips and ears also enlarged Facial features appear generally coarse	Acromegaly	Increased growth hormone → enlargement of both bone and soft tissue
Face—edematous and often pale ('pale puffy face') Edema usually appears first around the eyes, and in the morning. Severe edema → slit-like eyes	Nephrotic syndrome	–
Drooping of eyelids and corners of the mouth due to weakness of the facial muscles	Myesthenic facies	–
Plethoric rounded—'Moon-like face' Hirsutism and acne ±	Cushing's syndrome	–
Decreased facial mobility leading to blunt expression. Mask-like or expressionless face. Neck and upper trunk tend to flex forward	Parkinson's disease	–
Flat or sad expression	Depression	–

APPARENT STATE OF HEALTH?



TONGUE?

- Cyanosis ⇒ hypoxia
- Pallor ⇒ anemia
- Dry ⇒ dehydration
- Dry and brown ⇒ acute intestinal obstruction, uremia and later stages of severe illness
- Smooth and bald (devoid of papillae) = Atrophic glossitis
  Causes:
  - Deficiency of vitamin B-complex, or iron. Specific diagnosis is often difficult
  - Anticancer drugs
- 'Beefy red' ⇒ Vitamin B_{12} deficiency
- Fissured tongue
  - Fissures may appear with increasing age

Free ebooks ==> www.Ebook777.com

- Fissures of varying depth run laterally from the median groove
- Surface between the grooves is covered with normal papillae
- No pathological significance except for a sequela that food debris may get lodged and cause irritation
- *Complication*: Infection with candida albicans
- *DD*: Syphilitic tongue: Tends to be bald and with longitudinal fissures

– Hairy tongue
  - The 'hair' consist of elongated papillae on the dorsum
  - Yellowish to brown or black.
  - *Causes*: May follow antibiotic therapy. Otherwise may occur spontaneously
  - No pathological significance

– Geographic tongue
  The normal rough and coated areas together with scattered smooth red areas that are denuded of papillae → a map-like pattern that changes over time. No pathological significance.

NECK

Identify the key structures in the neck at the outset viz. SCM, thyroid cartilage, and cricoid cartilage.

– Examine the trachea for its position—central or deviated?
  - Inspect—for obvious deviation
  - Palpate: Place a finger along one side of the trachea and note the space between it and the SCM. Compare with the other side. Normally, the spaces should be symmetric. Asymmetry ⇒ tracheal deviation.

Causes of tracheal deviation: A mass in the neck, a large pneumothorax, a mediastinal mass, atelectasis.

– Deepening of infraclavicular fossa is a sign of wasting
– Cervical lymphadenopathy
– Bilateral cystic enlargement of parotids.
– May be a presenting feature of HIV infection.

SKIN

INSPECT the skin in **natural light**. Artificial light distorts color and masks jaundice.

Abnormal findings on inspection

- Discolored areas due to inflammation or ischemia
  - Reddish / bluish / brownish pale / purple / greenish black, or black
- Basic type of skin lesion
  - The subtype and exact nature with basic details viz. number, shape, size color?
  - Arrangement: Linear / clustered / annular / arciform / dermatomal?
  - Anatomical location / distribution?
- Circumscribed colored flat lesion
  - Anatomical location / distribution?
  - Shape, size, color?
  - Pulsatality?
  - Effect of pressure?
- Circumscribed colored elevated / nodular lesion
- Swelling

→ Refer to chapter 15

Palpation of the skin: Temperature, texture, moisture, thickness, and mobility and turgor.

CLINICAL METHOD / INTERPRETATION / DIFFERENTIAL DIAGNOSIS

Discoloration suggesting inflammation/ischemia

– Reddish discoloration (erythema) ⇒ inflammation or impending necrosis, **although some deep pressure sores develop without antecedent redness**
– Bluish discoloration-cyanosis ⇒ streptococcal myositis/myonecrosis
– Brownish discoloration ⇒ inflammation (non-gas-forming clostridial cellulitis)

www.Ebook777.com

– Pallor, purple discoloration, and blackening suggest successive degrees of ischemia
  - Purple discoloration ⇒ impending necrosis
  - Black discoloration ⇒ established necrosis.

Circumscribed colored lesions

Flat or only slightly raised

– Vascular
  - AVM (port wine stain, spider angioma, angiokeratoma.)
  - Cherry angioma, spider vein.
– Purpuric: - Petechiae / purpura
  - Ecchymosis
– Mole
– Actinic keratosis
– Seborrheic keratoses
– Basal celled carcinoma
– Malignant melanoma
– Dermatofibroma
– Bowen's disease.

Elevated / nodular (forming a swelling)

– AVM (venous malformation, capillary lymphatic malformation)
– Hemangioma (strawberry nevus, pyogenic granuloma)
– Keloid
– Keratoacanthoma
– Basal celled carcinoma
– Malignant melanoma
– Dermatofibrosarcoma protuberans
– TB (lupus vulgaris)
– Xanthoma

Differentiating features of vascular and purpuric lesions	*Vascular*			*Purpuric*	
	Spider angioma *Syn. spider nevus*	*Spider vein*	*Cherry angioma*	*Petechia / purpura*	*Ecchymosis*
Distribution	Face, neck, upper limbs, and upper trunk; almost never below the waist	Most often on the lower limbs	Trunk, limbs	Variable	Variable
Shape	Central body surrounded by erythema, and radiating limbs	Variable. May resemble a spider, or may be linear, irregular	Round. May be surrounded by a halo	Round or irregular	Round, oval or irregular. May have a central hematoma
Size	Very small to 2 cm	Variable. Up to few centimeters	1–3 mm	– Petechia: < 1 mm – Purpura: 1–5 mm	> 5 mm
Color	Fiery red	Bluish	Bright red; may become brownish with age	Deep red or reddish purple; fades away over time	Purple or purplish blue; fades to green, yellow, and brown with time
Pulsatality	+. Often demonstrable in the body of the spider, when pressure with a glass slide is applied	Absent	Absent	Absent	Absent

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

Differentiating features of vascular and purpuric lesions	*Vascular*			*Purpuric*	
	Spider angioma *Syn. spider nevus*	*Spider vein*	*Cherry angioma*	*Petechia / purpura*	*Ecchymosis*
Effect of pressure	Pressure on the body (i.e. center) causes blanching	Pressure on the center does not cause blanching, but diffuse pressure blanches the vein	Partial blanching ±	None	None
Significance	< 5 may be normal > 5 is regarded as pathological *Common causes*: Pregnancy, vitamin B deficiency, cirrhosis, thyrotoxicosis	~ 70% occur as isolated cases ~ 30% cases have associated varicosity of veins	– None – Increase in size and number with aging	Blood outside the vessels *Causes*: – Hematological: thrombocytopenia, leukemia, aplastic anemia – Infective: meningococcal meningitis, SBE, etc. – Nutritional: For example, scurvy	Blood outside the vessels *Causes*: – Trauma such as bruising – Hematological: Bleeding disorders

Differential diagnosis of other colored lesions of the skin is described in chapter 15.

Basic types of primary skin lesions

A. Flat non-palpable lesions

1. *Macule* = circumscribed lesion of a different color (allowing for differentiation from the surrounding skin) < 1 cm in diameter.
2. *Patch* = similar to macule but > 1 cm in diameter.

B. Elevated palpable solid masses

1. *Papule*—circumscribed, up to 1 cm in diameter. Example—an elevated nevus.
2. *Plaque*—circumscribed, > 1 cm in diameter; often formed by coalescence of papules.
3. *Nodule*—marble-like lesion > 0.5 cm in diameter, often deeper and firmer than a papule.
4. *Wheal*—a somewhat irregular transient localized skin edema. Examples—mosquito bite, hive.

C. Elevated palpable cystic masses

1. *Vesicle*—up to 1 cm in diameter, filled with serous fluid. Example—herpes simplex.
2. *Bulla* = a vesicle > 1 cm in diameter. Example—2nd degree burn.
3. *Pustule* = a cavity filled with pus. Examples—acne, impetigo.

Basic types of secondary skin lesions

1. *Erosion* = loss of superficial epidermis; surface is moist but does not bleed. Examples—a lesion resulting after rupture of a vesicle as in chickenpox.
2. *Ulcer* = a breach in the continuity due to molecular death of the tissue, cell by cell. An ulcer may bleed and scar.
3. *Fissure* = a linear ulcer. Examples—anal fissure, cracked nipple.
4. *Crust* = the residue of serum, blood or pus. Example—impetigo.
5. *Scale* = a thin flake of exfoliated epidermis. Examples—dry skin, dandruff, psoriasis.

www.Ebook777.com

Basic types of miscellaneous skin lesions

1. *Lichenification* = thickening and roughening of the skin with increased visibility of the normal skin furrows. Example—atopic dermatitis.
2. *Atrophy* = thinning of the skin with loss of the normal skin furrows; the skin looks shinier and more translucent than normal. Example—arterial insufficiency.
3. *Excoriation* = an abrasion or scratch mark.
   - It may be linear or rounded as in a scratched insect bite
   - Excoriation due to pruritus may suggest advanced CRF.
4. *Scar* = a collection of fibrous tissue replacing the normal dermal constituents.
5. *Burrow of scabies* = a minute slightly raised tunnel in the epidermis. Looks like a short (5–15 mm), linear or curved grey line and may end in a tiny vesicle. Seen better with a magnifying glass.
6. *Comedo* = the common blackhead that marks the plugged opening of a sebaceous gland, frequently seen with acne.
7. *Telangiectasias* = dilated small vessels (venules / arterioles (including spider angiomas) / capillaries) that look either red or bluish. May appear themselves or as parts of other lesions.
8. *Moles*.

Temperature

Temperature is tested with back of the fingers.

Texture

– Smooth texture is normal
– Roughness occurs in hypothyroidism, lichenification, etc.

Moisture

– Dry skin ⇒ dehydration, under-nutrition, hypothyroidism, advanced CRF.
– Moist skin ⇒ fall of high temperature in c/o fever, vasovagal shock, MI, thyrotoxic crisis.

Thickness

– Thickened in hypothyroidism, lichenification, etc.
– Thin in old age, under-nutrition.

Mobility and turgor

Lift a fold of skin and note the ease with which it lifts up (mobility), and on leaving it, note the speed with which it returns into place (turgor).

– Mobility is decreased in edema, scleroderma
– Turgor is decreased in dehydration.

DORSUM OF THE HANDS

– **Venous filling** on the back of the hand gives an idea about the degree of hydration.
  - Normally, the veins are visibly distended
  - Flat or guttered appearance suggests dehydration.

  Empty a segment of vein, and keep it isolated and empty by digital pressure (between 2 fingers). Release the anatomically distal finger and observe how fast the segment fills, or whether it remains empty for an appreciable time. This clinical test gives a fair idea about the degree of dehydration.
– **Metacarpals** – Do they look prominent? Prominent metacarpals suggest wasting.

NAILS

Inspect: Color, shape, trophic changes, splinter hemorrhages, fungal infestation, psoriasis.

Palpate for ridges, thickness, and looseness.

Free ebooks ==> www.Ebook777.com

INTERPRETATION / DIFFERENTIAL DIAGNOSIS

Blue / purple ⇒ cyanosis.

Clubbing = bulbus enlargement of soft tissues of terminal phalanx of fingers and toes. The proximal nail fold feels spongy or floating.

Causes of clubbing

- Diseases of heart, lung and alimentary tract
- Hereditary, idiopathic

Unilateral clubbing
- Aneurysm of subclavian / brachiocephalic artery
- Pancoast tumor

Unidigital clubbing
- Trauma

Koilonychia = hollowing. ⇒ anemia.

Trophic changes due to chronic peripheral arterial occlusive disease:
- May be claw-shaped
- Transversely placed ridges
- Roughened and thickened.

Splinter hemorrhages under the nails.
- May suggest systemic vasculitis, infective endocarditis.

Acute paronychia = acute cellulitis or a small abscess at the side or base of a nail.

Chronic paronychia = chronic inflammation of proximal and lateral nail folds. ⇒ fungal infestation.
- Nail folds get erythematous, swollen and often tender
- The cuticle may not be visible
- Multiple nails are often affected.

People who frequently immerse their hands in water are especially susceptible.

Onycholysis = painless separation of the nail plate from the nail bed.
- The process starts distally, enlarging the free edge of the nail to a varying degree
- Several or all nails are usually affected
- There are several causes of onycholysis.

Psoriasis
- Small pits in the nails—early sign but not specific
- 'Oil spot' lesion i.e. circumscribed yellowish tan discoloration
- Marked thickening, onycholysis.

PALM

- **Pallor**?
- **Erythema**? *Causes*: Pregnancy, thyrotoxicosis, cirrhosis, CA pancreas, high output cardiac states, rheumatoid arthritis
- Obliteration of **palmar hollow**? ⇒ wasting i.e. loss of weight.

EDEMA

- Puffiness of eyelids, particularly in the morning
- Tight ring on the finger
- Dependant edema in the lowest parts of the body viz. feet and lower legs when in upright posture (sitting / standing), or sacrum when bedridden.

www.Ebook777.com

Clinical methods for edema

1. Press firmly but gently with the thumb for at least 5 seconds—(a) over the dorsum of each foot, (b) just behind each medial malleolus (on the bone), and (c) over the shins.
   - A pit-depression caused by the thumb ⇒ edema
   - Pitting does not occur if the individual is normal, or else if edema is of non-pitting type.
2. For objective assessment, measure—(a) the circumference at the forefoot, (b) the smallest possible circumference above the ankle, (c) the largest circumference at the calf, and (d) mid-thigh circumference at a measured distance above the patella with the knee extended.
   Compare one side with the other. A difference between the 2 limbs of > 1 cm just above the ankle, or > 2 cm at the calf ⇒ edema.
   Fallacy: Muscular atrophy may cause asymmetry of the limb, and may be mistaken for edema.
3. Consistency of edematous tissue?
   - Soft ⇒ cardiac / hepatic / renal disease.
   - Firm ⇒ inflammation. Tenderness ⇒ acute inflammation, and occasionally chronic inflammation.
   - Firm-to-hard ⇒ very long-standing edema with varying degrees of fibrosis of subcutaneous tissue e.g. edema due to CVI, filariasis, etc.
4. How far up the limb does it extend?

For causes of edema and detailed clinical evaluation: Refer to chapter 50.

VEINS ON THE DORSUM OF THE FEET in A PATIENT LYING SUPINE in horizontal position

- These veins in this position are roughly at the same level as the atrium
- Normally, they are visibly distended
- Flat / guttered veins suggest dehydration (non-cardiogenic shock with a lowered CVP).

VITAL PARAMETERS AND JVP

- Temperature, pulse, respiration and BP constitute the vital parameters
- They remain constant in a normal healthy individual.

Vital parameters are so called, because:

- The vital organs govern values of these parameters. Even the slightest deviation from normal function of these organs is reflected in their values
- Significant variations may indicate life-threatening problems like insufficient consumption of O_2, blood depletion, bacterial invasion, etc.
- Changes in condition of the patient—either improvement or deterioration, can be detected by observing these values.

TEMPERATURE

Temperature is *defined* as heat maintained by the body, by achieving a balance between heat production and heat loss.
The balance is maintained with the help of heat regulating center viz. the hypothalamus.

(Pathological) **Causes of fever**

- Very high external temperature may upset the heat-regulating mechanism → hyperpyrexia e.g. sunstroke, heat stroke
- Blood disorders such as acute hemolytic anemia
- Hyperthyroidism, increased levels of progesterone → stimulation of the process of oxidation
- Drug reactions and immune disorders (such as collagen vascular disease)

Free ebooks ==> www.Ebook777.com

- Infection → fever. It is a protective mechanism. The degree of fever usually depends on the severity of infection
- Inflammation
- Trauma (surgery, crush injury)
- Cancer.

Conditions in which heat production is decreased
Shock, long-term debilitating illnesses, lowered vitality of tissues (may be old age), sleep and fasting.

A rough judgment of body temperature may be obtained by palpating with back of the fingers.
The temperature is best recorded with a mercury thermometer.

Normal body temperature = 97–99° F.
Grades of fever
- 97–95° F = Subnormal
- < 95° = Hypothermia
- 99–100° F = Mild fever
- 100–103° F = Moderate fever
- 103–105° F = High-grade fever
- > 105° F = Hyperthermia.

PULSE

Pulse is *defined* as an alternate expansion and recoil of an artery as the wave of blood is forced through it during contraction of the heart.

Sites of palpation of pulses: The basis is that a pulse is palpable where an artery crosses a bone close to the surface of skin.

The artery (Pulse)	*Bone against which the pulse is palpated*	*Site and method of palpation, and special clinical significance, if any*
Radial	Anterior surface of distal end of radius	Anterior surface of distal end of radius, lateral to the tendon of flexor carpi radialis. Forearm slightly pronated. Both sides palpated simultaneously
Ulnar	Anterolateral surface of distal end (i.e. head) of ulna	Anterior surface of distal end of ulna, **lateral** to the tendon of flexor carpi ulnaris. Forearm should be supinated
Brachial	Medial surface of humerus	-
Axillary	Medial surface of head of humerus	Arm abducted to 90°. Palpated just beyond the anterior axillary fold
Subclavian (Its 3rd part)	The 1st rib	In supraclavicular fossa in midclavicular line
Carotid	Transverse process of the 4th cervical vertebra	At the level of Adam's apple, in the groove between the larynx and medial aspect of anterior border of the SCM muscle. The head should be slightly extended. The absence of both carotid pulses is one of the indicators of cardiac arrest
Superficial temporal	Root of zygomatic process of temporal bone	Just anterosuperior to the tragus. This pulse is of much use to anesthesiologists at head end of the operation table
Femoral	Head of the femur; actually, anterior surface of the acetabulum	With the thigh slightly flexed, slightly abducted, and externally rotated, at the midpoint between pubic **symphysis** and ASIS

Contd...

www.Ebook777.com

Contd...

The artery (*Pulse*)	*Bone against which the pulse is palpated*	*Site and method of palpation, and special clinical significance, if any*
Popliteal	*In supine position*, against the popliteal surface of tibia.	*In supine position*: Slightly (~ 10–20°) abduct the thigh and slightly (~ 10–20°) flex the leg. Use the left hand for palpating the left popliteal artery, and the right hand for the right popliteal artery. Place the thumb on the tibial tuberosity and place the fingers on the inferior aspect of the popliteal fossa. Move the fingers from side to side in an attempt to feel for the pulse. If necessary, use both hands: Wrap both the hands around the knee such that both the thumbs are placed on the tibial tuberosity and fingers interdigitate deeply in the inferior aspect of popliteal fossa. Move the fingers from side to side in an attempt to feel for the pulse. I have been palpating popliteal artery pulse in this way for many years. The advantage of this method is that the tendons and fascia related to the popliteal fossa are adequately relaxed as the entire lower limb lies on the bed in relaxed position. I have not found any disadvantage of this method. Normally, this is the most difficult pulse to feel. An easily felt popliteal pulse may indicate aneurismal condition of the artery. *Crossed leg test*: The patient sits on a stool with knee of the affected limb crossed over the normal knee. Observe for oscillatory movements of the affected limb (look at the foot) synchronous with the pulsations of the popliteal artery (heart beat). The absence of oscillatory movements suggests blocked artery
Anterior tibial	Tibia, midway between the malleoli	-
Dorsalis pedis	Navicular / cuneiform / proximal end of the 1st interosseous space (~ base of the 1st MT)	Lateral to the tendon of extensor hallucis longus The absence of this pulse does not always suggest occlusive arterial disease, as in about 14% of people it may be absent due to the absence or small size of the artery
Posterior tibial	Posterior surface of medial malleolus	Midway between the tip of medial malleolus and tendocalcaneus (practically, slightly anterior to this point)

Thumb should not be used to palpate a pulse because it has its own pulse, which may be interpreted as the patient's pulse.

In infants, apical pulse is palpated to count pulse rate.

Note rate, rhythm, volume and tension of the pulse.

Rate = number of beats/min.

Age	Normal rate
Newborn	70–190
Infant	80–160
2 years	80–130
4 years	80–120
6–12 years	70–110
Adult	70–80
Old	60–70

Rate > normal is called tachycardia. Rate < normal is called bradycardia.

Free ebooks ==> www.Ebook777.com

Factors, which alter the rate

A. *Of rather physiological significance*—gender, physique, posture, food, exercise, emotions, etc.

B. *Of clinical significance*

1. Pain → tachycardia.
2. Fever, usually → tachycardia.
3. Diseases
   - Significant loss of blood, injury to viscera and most of the infections → tachycardia
   - Typhoid → a relative bradycardia
   - Cardiac disease may be associated with a normal pulse rate or tachycardia or bradycardia.
4. Drugs
   - Adrenaline, atropine, caffeine, etc. → tachycardia
   - Sedatives → bradycardia.

Rhythm = regularity of beats.

An ECG is indicated if rate / rhythm is abnormal, to identify the origin of the beats (sinus node / AV node / atrium / ventricle) and the pattern of conduction.

Cardiac arrhythmias with a regular rhythm

Pulse rate	*ECG pattern*	*Usual resting pulse rate*
Normal – 60–100.	Normal sinus rhythm 2° AV block Atrial flutter with a regular ventricular response	60–100 60–100 75–100
> 100 = tachycardia	Sinus tachycardia Supraventricular (atrial / nodal) tachycardia Atrial flutter with a regular ventricular response Ventricular tachycardia	100–180 150–250 100–175 110–250
< 60 = bradycardia	Sinus bradycardia 2° AV block Complete heart block	< 60 30–60 < 40

Cardiac arrhythmias with irregular rhythm

- Rhythmically or sporadically irregular
  - – Early beats
  - – Sinus arrhythmia
- Totally irregular
  - – Atrial fibrillation
  - – Atrial flutter with varying block

Volume = fullness of the artery.

Types of volume of surgical importance

1. Bounding: Full (= large) volume + rapid rate: ⇒ increased stroke volume.
   Causes: Anxiety, anemia, hyperdynamic stage of septic shock, hepatic failure, heart block.
2. Weak (= thready): ⇒ decreased stroke volume.
   Causes: Hypovolemic shock, later stage of septic shock.

RESPIRATION

Count the rate without alerting the patient. Breathing patterns may change if patient becomes aware that someone is watching.

Pattern of breathing to be examined: Rate, depth and regularity.

Count for 1 full minute by visual inspection of the respiratory excursions of the chest wall.

www.Ebook777.com

Normal respiration = 14–20 breaths/minute in a quiet regular pattern. An occasional sigh is normal. Normally, respiratory rate has a definite ratio with pulse rate of 1:4.

Abnormal breathing	*Nomenclature*	*Causes*
Rapid (> 24 breaths / minute), and shallow.	Tachypnea	– Shock – Elevated diaphragm – Restrictive lung disease – Pleuritic chest pain
Rapid (> 24 breaths/minute), and deep	Hyperpnea Hyperventilation	– Exercise / excitement / anxiety – Metabolic acidosis – In a comatose patient: Hypoxia / hypoglycemia / infarction of the midbrain or pons
Deep. Rate may be normal, fast or slow	Kussmaul	Metabolic acidosis
Slow i.e. < 12 breaths/minute	Bradypnea	– Diabetic coma – Drug-induced respiratory depression – Raised ICP
Periods of deep breathing alternating with periods of apnea (no breathing)	Chyne-stokes	– Normal in children and aging people during sleep – Drug-induced respiratory depression / heart failure / uremia / brain damage (typically on both sides of the cerebral hemispheres, diancephalon, or upper midbrain)
Unpredictable irregularity Breaths may be shallow or deep, and stop for short periods	Ataxic breathing	– Respiratory depression – Brain damage typically at the medullary level
Breathing punctuated by frequent sighs	Sighing respiration	– Possibility of hyperventilation syndrome—a common cause of dyspnea and dizziness – Occasional sighs are normal
Prolonged expiration	Obstructive breathing	Obstructive lung disease—asthma, chronic bronchitis, COPD

BP

Normal in adults:
- Systolic 100–130 mm Hg
- Diastolic 60–90 mm Hg.

Normal in children:
- Formula:
  - Mean systolic: 90 + age in years
  - Mean diastolic: 55 + age in years.

JVP

= the height of the venous column of blood in the internal jugular veins as measured from the sternal angle.

Pressure in the jugular veins reflects right atrial pressure, giving an important clinical indication of blood volume status, cardiac functions and right heart dynamics.

Assessing the JVP is an essential, though challenging clinical skill.

The JVP is usually estimated on the right side since the right internal jugular vein has a more direct anatomical channel into the right atrium.

It is essential to learn to identify the *pulsations* of the internal jugular vein that are transmitted to the surface of the neck. If they are not identified, then those of the external jugular vein can be used, but they are less

Free ebooks ==> www.Ebook777.com

reliable. A fallacy which must be kept in mind: Distension of the external jugular vein can be due to local kinking or obstruction. Occasionally, even bilateral distension has a local cause.

To estimate the level of the JVP, it is essential to learn to find the *highest point of oscillation in the internal jugular vein* or, if necessary, the point above which the external jugular vein appears collapsed.

Carotid arterial pulsations should not be mistaken for internal jugular venous pulsations.

Internal jugular pulsations	*Carotid pulsations*
– Soft, undulating	A more vigorous thrust
– Level usually descends with inspiration	Level not affected by inspiration
– Hardly palpable	Palpable
– Pulsations can be eliminated by applying light pressure on the vein just above the sternal end of the clavicle	Pulsations not eliminated by such pressure

The jugular veins and pulsations are difficult to see in children younger than 12 years of age, so they are not useful for clinical evaluation.

Clinical method

– The usual starting point is to elevate the head-end of the bed to 30°
– Find the highest oscillating point or meniscus of the jugular venous pulsations
– In hypovolemic patient, as a low JVP is anticipated, the head-end of the bed is lowered, sometimes even to 0°, to see the point of oscillation best
– Similarly, in volume-overloaded patient, head-end of the bed may be raised, as a high JVP is anticipated
– Place a ruler on the sternal angle and line it up with something in the background in the room that is surely vertical
– Then place a long rectangular object at exact right angle to the ruler. This constitutes the horizontal line. Move it up or down keeping it horizontal all the way such that its lower edge rests at the top of the jugular pulsations
– Read the vertical distance on the ruler.

Normal JVP: 3 to 4 cm
Causes of ↓ JVP: Dehydration, non-cardiogenic shock
Causes of ↑ JVP: ↑ blood volume, cardiac tamponade, SVC obstruction, tricuspid stenosis, hyperkinetic circulatory states and pulmonary diseases.

HEIGHT

Height should be proportional to age and gender.
In '*gigantism*', height is > 6.5'.

Causes of gigantism:
– Primary—racial, familial and constitutional
– Endocrinal, genetic and metabolic diseases.

In '*dwarfism*', height is < 4.5'.
Causes of dwarfism:
– Primary—familial and constitutional
– Endocrinal, genetic and nutritional diseases.

WEIGHT

- Weight should be proportional to height.
- In adults, weight should be roughly equal to height in cm minus 100. This approximation may be used when height-and-weight table is not readily available

www.Ebook777.com

- **Changes in weight result from changes in body tissues (fat or muscle mass) or body fluid**
- **Rapid changes in weight over a few days suggests changes in body fluids, not tissues.**

Weight gain

– Weight gain caused by changes in body tissues occurs when caloric intake exceeds caloric expenditure over a period of time and typically appears as increased body fat
  - *Generalized deposition* of fat ⇒ simple obesity
  - *Truncal obesity* with relatively thin limbs ⇒ Cushing's syndrome
– When retention of fluid is relatively mild, it may not be visible, but several kilograms of fluid usually appear as edema.

Causes of weight loss occurring over weeks / months with relatively high food intake

Hyperthyroidism, DM, malabsorption, and binge eating with clandestine vomiting.

Causes of weight loss occurring over weeks/months vis-à-vis decreased food intake

1. *GIT causes*: Major aphthous ulcer; chronic painful dental and periodontal disease; esophageal obstructive disease; gastric ulcer; worm infestation; fecal fistula; chronic vomiting and diarrhea.
2. *Hepatobiliary and pancreatic diseases*: Hepatitis, amoebic liver abscess, cirrhosis, pancreatitis.
3. TB, malignancy, AIDS and other *chronic debilitating diseases*.

Psychosocial and physiological causes of weight loss usually vis-à-vis decreased food intake

Poverty, old age, social isolation, physical disability, emotional or mental impairment, lack of teeth, ill-fitting dentures, alcohol and drug-abuse.

Symptoms associated with weight loss often suggest the cause of weight loss.

BMI (Body Mass Index)

- It gives more accurate measures of body fat than weight alone
- BMI criteria are not rigid cutpoints but guidelines for increasing risks for health and well-being
- Persons over 65 have a disproportionate risk of undernutrition when compared to younger adults.

Methods of obtaining BMI

1. Calculation from a formula: $\text{BMI} = \frac{\text{Weight (kg)}}{\text{Height (m}^2\text{)}}$
2. BMI may be obtained directly from physiological nomogram.

BMI	*Inference*
< 18.4	Underweight for height
18.5–24.9	Ideal weight for height
25–29.9	Over ideal weight for height
30–39.9	Obese
> 40	Very obese

BODY PROPORTION

In adults

Height should be equal to length of the arm span, and upper segment should be equal to lower segment.
(Upper segment = vertex ↔ pubic symphysis. Lower segment = pubic symphysis ↔ heals).

Normal infantile body proportion:

– Height is > length of the arm span
– Upper segment is > lower segment.

Infantile type of body proportions persists in cretinism and achondroplasia.

Reverse of infantile type of body proportions are seen in Marfan's syndrome, Klinefelter's syndrome.

Free ebooks ==> www.Ebook777.com

SKULL

Causes of enlarged skull: Hydrocephalus, Paget's disease, etc.

HAIR on the scalp, face, axilla, and pubis

Look for: Distribution, pattern of excess/loss, color, texture, flakes of dandruff, tiny white ovoid granules adherent to hair (nits / eggs of lice).

– Hirsutism = excessive growth of hair in females
  Causes of hirsuitism:
  1. Familial
  2. Polycystic ovary syndrome, ovarian tumor
  3. Endocrinl.
– Loss of hair of lateral 1/3 of eyebrow ⇒ Hansen's disease
– Depigmented, dry and brittle hair ⇒ undernutrition
– Fine hair—in hyperthyroidism
– Coarse hair—in hypothyroidism
– Absence / loss of axillary and pubic hair ⇒ hypogonadism, hypopituitarism.

SCALP

Part the hair in several places and look for color, scaliness, swellings (vascular, sebaceous cyst, etc.) and other lesions.
Redness and scaling is seen in seborrheic dermatitis, psoriasis.

LIPS

– *Cyanosis*?
– *Pallor* ⇒ anemia
– *Black* discoloration ⇒ smoking
– *Lingual and circumoral pigmentation* ⇒ benign intestinal polyps (P-J syndrome)
– *Hereditary hemorrhagic telangiectasia*
  - Multiple small red spots
  - These patients often have nosebleeds and GI bleeding
– *Cheilosis and angular stomatitis*. Cheilosis = fissuring and dry scaling of vermilion surface of the lips.
  Causes:
  1. Vitamin B-complex deficiency.
  2. Overclosure of the mouth e.g. in edentulous patients, ill-fitting dentures.
  Maceration of the infolded skin may → secondary infection e.g. candidiasis
– *Actinic cheilitis*:
  - Results from excessive exposure to sunlight
  - The lip becomes scaly, somewhat thickened, and slightly everted
– *Herpes simplex (cold sore)*
  - Herpes simplex virus → recurrent and painful vesicular eruptions of the lips and surrounding skin
  - A small cluster of vesicles develop first. They break and yellow brown crusts form
  - Healing ensues within 10–14 days
– *Angioedema*
  - Develops dramatically. Disappears over subsequent hours or days
  - Does not itch even if usually allergic in nature
  - Diffuse, non-pitting, tense swelling of the dermis and subcutaneous tissue

www.Ebook777.com

– *Swelling* of the lips is one of the manifestations of orofacial granulomatosis
– *Chancre* of primary syphilis.

CHEEKS

A wavy white line on the buccal mucosa where the upper and lower teeth meet—may be caused / intensified by chewing.

GUMS AND TEETH

Gingival hyperplasia → heaped-up masses that may even cover the teeth.
– Redness due to inflammation ±
– *Causes of gingival hyperplasia*: Puberty, pregnancy, leukemia, dilantin therapy.

Marginal gingivitis
– Gingival margins—reddened and swollen
– Interdental papillae—reddened, swollen and blunted
– *Plaque* = white, soft film of salivary salts, protein and bacteria that covers the teeth and leads to gingivitis—may not be readily visible
– Common among teenagers and young adults.

Acute necrotizing ulcerative gingivitis
– Mode of onset—sudden
– Constitutional symptoms
– Easy bleeding from the painful red gums
– Foul breath
– Ulcers in the interdental papillae
– Then the destructive (necrotizing) process spreads along the gum margins, where a grayish pseudomembrane develops.

Chronic gingivitis and periodontitis
Chronic untreated gingivitis may progress to periodontitis (inflammation of the deeper tissues that normally hold the teeth in place).
– The gum margins recede
– The teeth eventually loosen
– Calcified plaque contributes to inflammation.

Lead line
– A bluish-black line on the gums ⇒ chronic lead poisoning
– The line is ~ 1 mm from the gum margin, follows its contours, and is absent where there are no teeth.

Loose teeth
Therapeutic implication: It is essential to extract loose tooth / teeth before considering a case fit for general anesthesia.

THROAT

– *Large normal tonsils*
  - May be large enough to meet in the midline obscuring the pharynx
  - Color—normal. White lines may be due to light reflections and should not be mistaken for exudate
  - The individual (usually a child) is asymptomatic.

  Normal tonsils may enlarge without being infected, especially in children.

Free ebooks ==> www.Ebook777.com

– *Exudative tonsillitis*
  - Constitutional symptoms +
  - Cervical lymphadenitis

  Anterior cervical lymphadenitis ⇒ probability of group A streptococcal infection
  Posterior cervical lymphadenitis ⇒ probability of infectious mononucleosis.
– *Pharyngitis*
  - H/o sore throat, or at least a scratchy one
  - Possible causes include several viruses or bacteria
– *Diphtheria*
  - Throat is dull red
  - A gray exudate (pseudomembrane) forms on the uvula, pharynx, and tongue
  - Airway may become obstructed
– *Torus palatinus*: Although alarming at first glance, it is harmless.

NOSE AND PARANASAL SINUSES

Examine the external nose.
Test for nasal obstruction by pressing on each ala nasi in turn and asking the patient to breathe in.
Inspect the interior of the nose.
– Tenderness of the nasal tip / alae ⇒ local infection e.g. furuncle
– Edema and reddening of the nasal mucosa—in viral rhinitis
– Nasal mucosa—pale / bluish / red in allergic rhinitis
– Pale, semitranslucent mass usually coming out from the middle meatus ⇒ a polyp
– Nasal discharge, pain, fever, and local tenderness ⇒ acute sinusitis involving frontal / maxillary sinus.

EAR

– Look at the lobules—for cyanosis.

Differentiating clinical feature	*Acute otitis externa*	*Acute otitis media*
– Movements of the auricle and tragus (the 'tug test')	Painful	Painless
– Tenderness behind the ear	Absent	±
– External auditory canal	Swollen, narrowed, moist, pale, may be reddened, and tender	Normal

– Skin of the external auditory canal is itchy, red and thickened in *chronic otitis externa*.

Eardrum:
– Red, bulging in acute purulent otitis media
– Amber colored in serous effusion.

LYMPHADENOPATHY

Regional or generalized?
Refer to chapter 16.

ODOR OF THE BODY / BREATH

Alcohol, acetone (diabetes), pulmonary infections, uremia, liver failure can give odor to the body/breath.
Never assume that alcohol on a patient's breath is responsible for changes in abnormal mental status and/or neurological findings. Alcoholics may have other serious but potentially correctable problems like hypoglycemia, postictal state, or subdural hematoma.

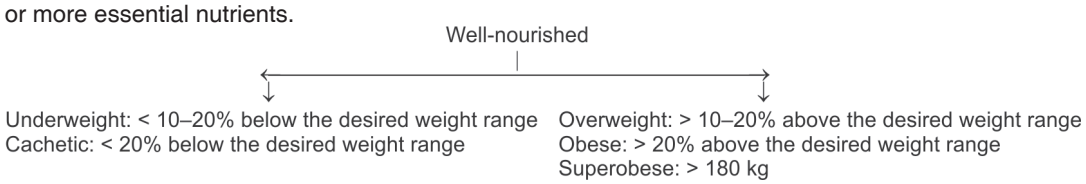
www.Ebook777.com

SIGNS OF HYPOVOLEMIA AND HYPERVOLEMIA
(Underhydration v/s overhydration)

Clinical feature	*Hypovolemia*	*Hypervolemia*
– Eyes	Sunken	Periorbital edema
– Tongue	Dry and coated	Moist
– Saliva	Thick and scanty	Excessive and frothy
– Skin	Dry, cold and clammy	Moist, warm and has a pitting edema
– Subcutaneous veins on the hands / feet	Collapsed	Full
– Temperature	Raised	No significant change
– Pulse	Rapid, weak and thready	Rapid
– Respiration	Rapid and shallow	Rapid. SOB. Rales. Frothy sputum
– BP	Low. Orthostatic hypotension	Raised
– JVP	Low	High

A NOTE ON NUTRITION

Malnutrition is defined as a pathological state resulting from a relative or absolute deficiency or excess of one or more essential nutrients.



Symptoms of malnutrition may be subtle and nonspecific such as weakness, easy fatigability, cold intolerance, flaky dermatitis, and ankle edema.

Clinical signs of wasting i.e. loss of weight
- Hollowing of cheeks
- Deepening of infraclavicular fossa
- Normal conical shape of the lower limbs is replaced by cylindrical shape
- Obliteration of the palmar hollow
- Prominence of bony margins e.g. metacarpals, spine of the scapula.

Signs of undernutrition
- Hair—depigmented and brittle
- Eyes—Bitot's spots, keratomalacia
- Mouth—cheilosis, angular stomatitis, glossitis and pallor of the tongue
- Nails—koilonychia
- Skin—shiny, flaky, thin and dry (xerosis)
- (Hypoprotienemic) edema of lower limbs.

Causes of cachexia
- In children: Severe GE, malabsorption syndromes
- In adults: Anorexia nervosa, thyrotoxicosis, DM
- In old ages: Severe cardiorespiratory disease
- Common to all ages: Prolonged severe starvation (due to any cause like extreme poverty, draught, etc.), advanced TB, advanced malignancy, AIDS.

Free ebooks ==> www.Ebook777.com



Fig 4-1 (Malignant) Cachexia

Causes of obesity

- Excessive eating—energy intake in excess of expenditure
- On steroid, oral contraceptive
- Endocrinal—hypothyroidism, Cushing's syndrome, etc.
- Genetic—polycystic ovary syndrome, hypogonadism.

Reducing weight by even 5% or 10% can improve BP, lipid levels and glucose tolerance, and reduce the risk of developing HT or DM.

www.Ebook777.com

5 LOCOREGIONAL EXAMINATION

I have deliberately used the word 'Locoregional' instead of 'Local', because local examination is never complete without examination of the surrounding area and the regional draining lymphnodes.

Locoregional examination is performed in orderly manner under the following headings: Inspection, palpation, percussion, auscultation, and special clinical methods and tests.

General methods of palpation and percussion are discussed in this chapter as they form the basis of examination in a wide variety of surgical cases. Inspection, auscultation and special clinical tests are case-specific and are not discussed here.

PALPATION

Palpating agent: Palmar surface of the fingers together. The fingers should be slightly cupped. The hand should be rolled, and moved from one area to another **in continuity.**

Fingertips may be used while 1. testing for pitting, 2. detailed palpation of any abnormality, particularly swelling, 3. palpating the abdomen which is distended due to ascites.

Methods of Palpation

1. *Light* palpation
   - Let the fingers gently mould over the area to be palpated
   - Information to be sought: Tenderness, and surface characteristics.
2. *Deep* palpation

   Thorough deep palpation may not be possible if tenderness is present. In abdominal palpation, deep palpation is carried out using 2 hands, one on top of the other. The upper hand is used to give pressure and the lower hand i.e. the one in contact with the abdomen, actually feels for abnormalities.
3. *Bimanual* palpation is indicated when 2 surfaces are accessible for palpation, as in c/o cheeks, floor of the mouth and pelvis (with simultaneous PV and/or DRE).
4. *Bidigital* palpation is indicated for small superficial swellings as in c/o lip, cheek, ear, nipple, etc. This is carried out with thumb and index finger of the same hand by gently squeezing movement.

Temperature

Temperature is checked with dorsal surface of the fingers. *Reason*: Palmar skin is warmer than and consequently not as efficient as the cooler dorsal surface to detect minor differences in temperature.

This is always the first parameter that is checked on palpation. *Reason*: As palpation proceeds, temperature of the part being palpated may increase causing wrong inference to be drawn if temperature is checked towards the end of palpation; and that will not be valid in certain cases like PAOD. Hence, checking for temperature should be the first step of palpation, **by default.**

Tenderness

This is the second step in palpation **by default.** Once the exact area of tenderness is delineated right at the beginning, unnecessary discomfort in due course of palpation can be avoided.

Tenderness is one of the cardinal signs of tissue injury, whether due to trauma / inflammation / thromobosis (leading to irreversible ischemia). Reversible ischemia may cause pain but no tenderness.

The examination is started at a site away from the site of pain and gradually carried to the expected site of tenderness, while keeping a constant watch on the patient's face. Face is the best indicator of the site and degree of tenderness.

Free ebooks ==> www.Ebook777.com

Wincing of the face and flinching of the body confirm the presence of tenderness.

Methods of eliciting tenderness in various tissues

- Skin—gently move the hand over the skin. Hyperalgesia suggests tenderness
- Subcutaneous tissue—by gently pinching a fold of skin + subcutaneous tissue between index finger and thumb
- Superficial muscle - a. Gently squeeze the muscle between fingers and palm
  b. Carry out a passive movement that stretches the muscle
- Deeper (non-accessible) muscle—carry out a passive movement that stretches the muscle.
- Bone—apply deep, firm pressure on the bone
- Joint—apply direct pressure / move
- Referred tenderness: The principle is the same as that of referred pain.
  Examples: Hyperalgesia in Sherren's triangle in appendicitis, Boa's sign in acute cholecystitis, etc.

PERCUSSION

Definition: Percussion is the procedure by which artificial vibrations are set up in a tissue by means of sharp taps delivered by finger/s, which can both be felt and heard.

Methods: Direct and mediate.

Direct method: The part under examination is directly struck by the striking finger/s.
Example: Over middle of the clavicle for examination of apex of the lung.

Mediate method

- Middle finger (called pleximeter) is placed in close and **firm** contact with the part, with **palm and other fingers being lifted off the area**
- The plexor finger delivers sharp taps on the middle phalanx of the pleximeter, and **removed immediately,** lest it should dampen the vibrations produced. The forearm should be virtually stationary. Movement should take place at the wrist joint
- Vibrations can both be felt and heard; but concentration should be focused more on 'feeling' rather than 'hearing'.

Various percussion notes (sounds)	*Examples of normal location*
1. Resonant	Normal lungs
2. Hyperresonant	None
3. Tympanic	– Puffed-out cheeks – Gastric air
4. Dull	Liver
5. Flat	Thigh

COMMON MISTAKES in physical examination which should be avoided:

1. Incomplete examination through hurry, oversight and overconfidence.
2. Improper method in eliciting physical signs.
3. Lack of knowledge of pathology (- diseases).

PREREQUISITES for locoregional examination:

1. *Proper place*
2. *Privacy*
3. *Proper attendant*: For example, a lady attendant while examining a lady patient.
4. *Proper and adequate light:*
   - In artificial light, jaundice can be missed
   - In fluorescent light nails appear cyanosed.
     The best light is natural day-light.

www.Ebook777.com

5. *Adequate exposure* is essential to avoid missing of any finding. Shy and female patients may not allow adequate exposure; this may prove to be dangerous in 'acute abdomen'. - Missing the diagnosis of obstructed / strangulated femoral hernia can be life-threatening. As it is, diagnosis of a femoral hernia in a bit fatty woman is often difficult, and femoral hernia is more common in females *cf* males.
   - Exposure × PA/E: Nipples ↔ mid-thighs
   - Exposure × breast examination: The whole body above the umbilicus, including both arms
   - Exposure × a joint examination: The joint + the proximal and distal muscles
   - Exposure × varicose veins of lower limbs: The whole limbs + the lower abdomen.
6. *Appropriate positions of the patient and part*

   Different positions are suggested for examination of different parts of the body. The position selected should bring the abnormality to the fore to its fullest extent, should achieve relaxation of the superficial tissue to facilitate palpation of the deeper tissue, and should be comfortable to both the patient and the clinician.
   - Lying-down position, by and large is the most usual position in which patients are generally examined
   - Left lateral (Sims') position is usually suitable for anorectal examination
   - Sitting: For ENT, neck, breast, shoulder and spine examination
   - Standing (with legs apart): For a groin swelling.
7. *Comparison*: Both the parts must be in symmetrical position. Tilting / obliquity of one side will make comparison useless.

REPEATED EXAMINATIONS after an interval of hours / days (depending upon the severity of the disease) help to get an idea about *progress of the disease*. - An important diagnostic aid—to differentiate one disease from another.

Free ebooks ==> www.Ebook777.com

6 RESPIRATORY SYSTEM EXAMINATION

Chest symptoms

- SOB, diminished exercise tolerance, pain in the chest: These symptoms are common to both respiratory and cardiac disorders
- Chronic cough, expectoration, hemoptysis, wheezing: These are the specific symptoms of respiratory disorder
- Palpitations, syncope, HT, IHD, CVD: These are the specific symptoms of cardiac disorder.

APPLIED ANATOMY

Describe abnormalities of the chest in 2 dimensions: Along the vertical ax is and around the circumference of the chest.

Counting the ribs and IC spaces is essential to be able to describe an abnormality in vertical location.

1. While counting the intercostal spaces, do not count them along the lateral border of the sternum; the ribs here are too close together.
   Start counting from the 2nd IC space roughly in the midclavicular line downwards, one space at a time obliquely towards the anterior axillary line as finally the 9th IC space is reached. Reason for going down in oblique direction: The IC spaces tend to be bit wider laterally and hence can be palpated more easily and confidently.
   In women, to find the IC spaces, either displace the breast laterally, or palpate a little more medially. Avoid pressing too hard on tender breast tissue.
   On palpation, costal cartilages and ribs feel identical.
2. Posteriorly, 12th rib can be another starting point.
   Press in and up against the lower border of the 12th rib, then 'walk-up' one step at a time obliquely up and around the front of the chest.
3. The inferior angle of the scapula can be another useful bony landmark. It usually lies at the level of the 7th rib/IC space.
4. When the neck is flexed, the most prominent spinous process belongs to the C7. If 2 processes are equally prominent, they belong to C7 and T1.

To locate findings around the circumference of the chest, **a series of vertical lines** are used.
The midsternal and vertebral lines are precise; the others are estimated.

1. Midsternal—the anterior midline.
2. Midclavicular—drops vertically from the midpoint of the clavicle.
3. Anterior axillary—drops vertically from the anterior axillary fold.
4. Midaxillary—drops vertically from the apex of the axilla.
5. Posterior axillary—drops vertically from the posterior axillary fold.
6. Scapular—drops from the inferior angle of the scapula.
7. Vertebral—passes along the tips of the spinous processes of thoracic vertebrae.

Normal breathing is quiet and easy.
When a healthy person lies supine, the breathing movements of the chest are relatively slight. In contrast, the abdominal movements are usually easy-to-see.
In the sitting position, movements of the chest become more prominent.

www.Ebook777.com

CLINICAL EXAMINATION

POSITION

– For examining the posterior thorax and lungs: Sitting. The arms should be folded across the chest with hands resting, if possible, on the opposite shoulders. This position moves the scapulae partly out of the way and increase the access to the lung fields.

  If the patient cannot (be made to) sit up, then roll her/him to one side and then to the other. Percuss the upper lung and auscultate both lungs in each position. Because ventilation is relatively greater in the dependant lung, the chances of hearing wheezes or crackles are greater on that side.

– For examination of the anterior thorax and lungs: Supine.

  However, the anterior thorax and lungs can also be examined satisfactorily with the patient in sitting position.

  A patient having difficulty in breathing should be examined in sitting position or with the head-end of the bed elevated to a comfortable position.

INITIAL SURVEY

- Look for **cyanosis**. Cyanosis suggests hyoxia.
- **Stridor**?

  = a high-pitched wheeze audible without the aid of a stethoscope.

  – It is an ominous sign of airway obstruction in the larynx or trachea.

INSPECTION

- **Breathing**

  Rate, rhythm, depth and effort

  ↓

  During inspiration

  ↓

  1. Inspect the neck

  – Is there contraction of SCM or other muscles of accessory respiration? } ⇒ Severe difficulty in breathing.

  – Supraclavicular retraction?

  2. Inspect the IC spaces for retraction

  – Retraction is most apparent in the lower IC spaces } ⇒ Upper airway obstruction / severe asthma / COPD

  – Supraclavicular retraction is often associated.

- **From all sides** (from a midline position behind to a midline position in front, on both sides), note the **shape of the chest and the way it moves**

  – Asymmetry, deformity?

  - (Traumatic) Flail chest → paradoxical movements of the injured segment of the thorax
  - Barrel chest: AP diameter ↑ed as compared to that in a normal adult. Normal during infancy. May be seen in normal aging. A pathological cause of barrel chest: COPD
  - Thoracic kyphoscoliosis occurs due to abnormal spinal curvatures and vertebral rotation. Distortion of the underlying lungs may make interpretation of lung findings very difficult
  - For disorders/deformities of the chest-wall, refer to chapter 32. Distortion of the underlying lungs resulting from deformity of the chest-wall may make interpretation of lung findings very difficult

  – Impairment of respiratory movements—unilateral / bilateral? } ⇒ Disease of the underlying lung or pleura.

  – Unilateral lag in movement?

PALPATION

- **Tenderness**

  Identify tender areas: Carefully palpate any area where pain has been reported or where bruises or lesions are evident.

  Intercostal tenderness may be suggestive of inflamed pleura.

Free ebooks ==> www.Ebook777.com

AP compression to distinguish fracture of ribs from soft-tissue injury: Squeeze the chest with one hand on the sternum and the other on the thoracic spine

↓

Does it lead to tenderness at a location distant from the sites at which compression is being applied? - If yes, ⇒ fracture of rib/s rather than soft tissue injury.

- **Assess any observed abnormality** such as a swelling, sinus, ulcer, etc.
- **Chest expansion**

Clinical method:

– Place the thumbs at about the level of the 10th ribs, with fingers loosely grasping and parallel to the lateral rib cage
– After positioning the hands in this way, slide them medially just enough to raise a loose fold of skin on each side between the thumb and the spine
– Ask the patient to inhale deeply
– Watch the distance between your thumbs as they move apart during inspiration, and feel for the range and symmetry of the rib cage as it expands and contracts.

Causes of unilateral decrease or delay in chest expansion:

– Chronic fibrotic disease of the underlying pleura or lung
– Pleural pain with associated splinting
– Pleural effusion
– Lobar pneumonia
– Unilateral bronchial obstruction.

PERCUSSION

Percussion helps to establish whether the underlying tissues are air-filled, fluid-filled, or solid.
Vibrations set by a plexor finger penetrate only about 5–7 cm into the chest, and therefore percussion will not help to assess lesions situated deeper than 7 cm.
Percuss one side of the chest and then the other at each level (i.e. level by level).
In c/o a woman, while aiming at the right middle lobe, displace the breast or ask the patient herself to do so.
Reason: The right middle lobe pneumonia typically occurs behind the right breast. The same may be missed if the breast is not displaced.

Percussion note	*Pathologic examples*	*Examples of normal location*
Resonant	Simple chronic bronchitis	Normal lung
Hyperresonant	*Generalized (bilateral) hyperresonance* : – Emphysema – Asthma *Unilateral hyperresonance* – Pneumothorax – An air-filled bulla in the lung	None normally
Tympanic	Large pneumothorax	– Puffed-out cheek – Gastric air bubble
Dull	– Lobar pneumonia – Pleural effusion – Empyema – Hemothorax – Fibrous tissue – Tumor	Liver
Flat	Large pleural effusion	Thigh

The hyperresonance of COPD may totally replace cardiac dullness.

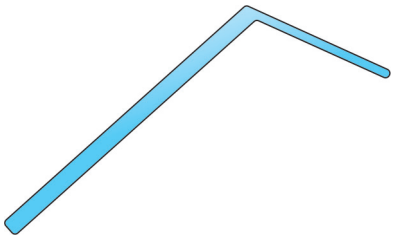
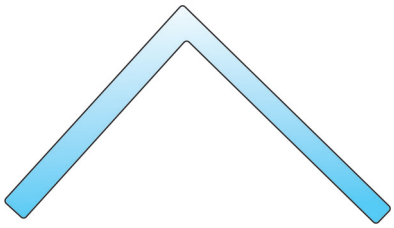
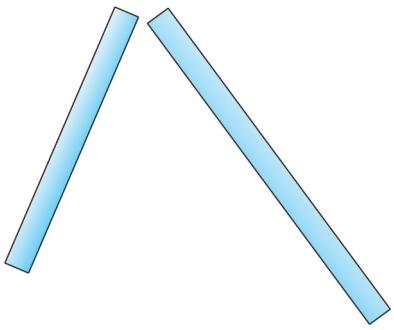
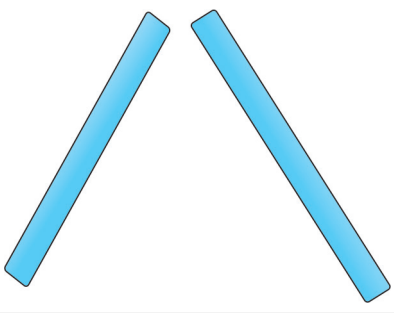
www.Ebook777.com

AUSCULTATION

This involves listening to breath sounds (lung sounds), adventitious (*syn.* added) sounds, and vocal fremitus.

Sounds which may create confusion:

– Crackling sounds from clothes
– Crackling sounds due to hair on the chest. Pressing the stethoscope harder or making the hair wet may eliminate these sounds
– Sounds due to muscle contraction—muffled, low-pitched, rumbling or roaring
  Change in the patient's position may eliminate these sounds.

Breath sounds	*Characteristic inspiratory and expiratory duration*	*Locations where heard normally*
Vesicular		Over most of both lungs
Bronchovesicular		Often in the 1st and 2nd IC spaces anteriorly, and between the scapulae
Bronchial		Over the manubrium, if heard at all
Tracheal		Over the trachea in the neck

Free ebooks ==> www.Ebook777.com

Hearing of bronchovesicular or bronchial breath sounds in locations distant from those where they are normally heard suggests replacement of air in the lungs by fluid or a solid pathology; e.g. lobar pneumonia / pulmonary edema / pulmonary bleeding, etc.

- **Transmitted voice sounds—vocal fremitus**
  If abnormally located bronchovesicular or bronchial sounds are heard, then continue on to assess transmitted voice sounds.
  Ask the patient to repeatedly say 'ninety-nine, ninety-nine, ninety-nine …..' and auscultate in symmetric areas over the chest wall.
  - Normally, the sounds transmitted through the chest wall are muffled and indistinct
  - ↑ed transmission of voice sounds suggests that the air-filled lung has become airless.
- **Breath sounds decreased** OR **absent** in a particular area?
  Causes:
  - Decreased air-flow due to obstructive lung disease or muscular weakness.
  - Poor transmission of sounds due to pleural effusion / pneumothorax / atelectasis / emphysema.
- **Adventitious sounds**

= added sounds that are superimposed on the usual breath sounds.

Discontinuous
- Crackles or rales—fine/coarse
- Pleural rub
- Mediastinal crunch

Continuous
However, they do not necessarily persist throughout the respiratory cycle
musical unlike crackles
↓
– Wheezes
– Rhonchi

Adventitious sound	*Causes*
Crackles ⇒ – abnormality of lungs—pneumonia / fibrosis / early CHF OR – abnormalities of the airways—chronic bronchitis / asthma / bronchiectasis	
Late inspiratory crackles	– Lung disease e.g. pneumonia/fibrosis – Early CHF
Early inspiratory crackles *Expiratory crackles are sometimes associated*	– Chronic bronchitis – Asthma
Pleural rub	Pleuritis Pleural rubbing sounds are produced as inflamed and roughened surfaces grate against each other during respiratory movements
Mediastinal crunch	Mediastinal emphysema (pneumomediastinum).
Wheezes ⇒ narrowed airways as in asthma, COPD, bronchitis	
Generalized wheezes	Asthma, COPD, chronic bronchitis, CHF (cardiac asthma)
Persistent localized wheeze—may be inspiratory/expiratory/both	Partial obstruction of a bronchus, as by a tumor or foreign body
(Generalized) Rhonchi	Secretions in the larger airways

Clearing of crackles / wheezes / rhonchi after coughing retrospectively suggests that secretions were the cause, as in bronchitis/atelectasis.

'Silent chest': In severe obstructive pulmonary disease the patient may no longer be able to force enough air through the narrowed bronchi to produce wheezing. The resulting silent chest should raise immediate concern and not be mistaken for improvement.

www.Ebook777.com

FORCED EXPIRATORY TIME

Purpose: to assess the expiratory phase of breathing; this is typically slowed in obstructive pulmonary disease.

Clinical method

– Ask the patient to take a deep breath in and then breathe out as quickly and completely as possible with mouth open.
– Auscultate over the trachea with the diaphragm of a stethoscope, and time the audible expiration.
– Try to get 3 consistent readings, allowing a short rest between efforts if necessary.

Inference: A forced expiratory time ≥ 6 seconds suggests obstructive pulmonary disease.

DIFFERENTIAL DIAGNOSIS OF SELECTED CHEST DISORDERS

KEY POINTS: Position of the trachea, and percussion note.

(A) Trachea

OR

Is it in the midline? | Shifted? ⇒ Atelectais / pleural effusion / pneumothorax

(B) Percussion note?

Resonant	Dull	Dull to flat	Hyperresonant
⇓	⇓	⇓	⇓
– Normal	– Consolidation	Pleural effusion	– Pneumothorax
– Chronic bronchitis	– Atelectasis		– COPD
– Left-sided heart failure (early)			– Asthma

Arriving at a preliminary gross impression by analyzing the above key points collectively

1. Shift of trachea, and a dull note both on the same side ⇒ Atelectasis on that side.
2. Shift of trachea, and abnormal percussion notes on the sides opposite to each other ⇒ the site of disease on the side opposite to that of the shift of the trachea; i.e. on the same side as that of the abnormal percussion note.

   ↓

   – Dull-to-flat ⇒ Pleural effusion.
   – Hyperresonant ⇒ Pneumothorax.

Normal findings

- Tracheobronchial tree and alveoli—clear. Pleurae—thin and close together. Mobility of the chest-wall—unimpaired
- Trachea—midline
- Percussion note—resonant
- Vocal fremitus—normal
- Breath sounds—vesicular. Bronchovesicular over large bronchi, and bronchial over trachea ±
- Adventitious sounds—none. A few transient inspiratory crackles at the bases ±.

Chronic bronchitis

- Pathology: Productive cough. Airway obstruction may develop
- Trachea—midline
- Percussion note—resonant
- Vocal fremitus—normal
- Breath sounds—vesicular
- Adventitious sounds—none or scattered coarse crackles in early inspiration and perhaps expiration, OR wheezes, OR rhonchi.

Left-sided heart failure (early)

Pathology: Increased pressure in the pulmonary veins → congestion and interstitial edema (around the alveoli). Bronchial mucosa may become edematous.

- Trachea—midline

Free ebooks ==> www.Ebook777.com

- Percussion note—resonant
- Vocal fremitus—normal
- Breath sounds—vesicular
- Adventitious sounds
  - late inspiratory crackles in the dependant position of the lungs.
  - possibly wheezes.

Consolidation

- Pathology: Alveoli fill with fluid or blood cells, as in—pneumonia, pulmonary edema, or pulmonary bleeding
- Trachea—midline
- Percussion note—dull overlying the airless area
- Vocal fremitus—increased over the involved area
- Breath sounds—bronchial over the involved area
- Adventitious sounds—late inspiratory crackles over the involved area.

Atelectasis (Lobar obstruction)

- Pathology: Obstruction of airflow in a main-stem bronchus (by a plug of mucus / a foreign body, etc.) → collapse of the affected lung tissue into an airless state
- Trachea—may be shifted towards the affected side
- Percussion note—dull over the airless area
- Vocal fremitus—usually absent. *Exception*: May be increased in right upper lobe atelectasis
- Breath sounds—usually absent. *Exception*: In right upper lobe atelectasis, tracheal sounds may be transmitted
- Adventitious sounds—none.

Pleural effusion

- Pathology: Accumulation of fluid in the pleural space, separating air-filled lung from the chest-wall
- Trachea—shifted towards the opposite side in *large effusion*
- Percussion note—dull-to-flat over the fluid
- Vocal fremitus—decreased or absent. May be increased towards the top of a large effusion
- Breath sounds—decreased or absent. Bronchial sounds may be heard near the top of a large effusion
- Adventitious sounds—none, except a possible pleural rub.

Pneumothorax

- Pathology: Leakage of air into the pleural space, usually unilaterally, separating air-filled lung from the chest-wall
- Trachea—shifted towards the opposite side if *much air*
- Percussion note—Hyperresonant or tympanitic over the pleural air
- Vocal fremitus—decreased or absent over the pleural air
- Breath sounds—decreased or absent over the pleural air
- Adventitious sounds—none, except a possible pleural rub.

COPD

- Pathology: A slowly progressive disorder. The distal air spaces enlarge and lungs become hyperinflated. Often associated with chronic bronchitis
- Trachea—midline
- Percussion note—diffusely Hyperresonant
- Vocal fremitus—decreased
- Breath sounds—decreased or absent
- Adventitious sounds—none, or the crackles, wheezes and rhonchi of associated chronic bronchitis.

Asthma

- Pathology: Widespread narrowing of the tracheobronchial tree → diminished airflow to a fluctuating degree. During attacks, airflow decreases further and lungs hyperinflate
- Trachea—midline
- Percussion note—resonant to diffusely Hyperresonant
- Vocal fremitus—decreased
- Breath sounds—often obscured by wheezes
- Adventitious sounds—wheezes. Possibly, crackles.

www.Ebook777.com

7 PELVIC EXAMINATION

GENERAL REMARKS

A patient who has never had a pelvic examination is often unsure of what to expect. Before the patient undresses, explain the relevant anatomy, and explain the steps of examination. Show her/him the speculum, proctoscope and other equipment to allay the fear about the instruments.

Male clinician must perform a pelvic examination of a female patient **in the presence of a female nurse/ other female health professional. Female clinician should also be assisted if the patient is physically or emotionally disturbed.**

Always wear gloves, both during the examination and when handling equipment and specimens. **Plan ahead**, so that any needed equipment or culture media are readily available at hand.

Make sure about the provision for adequate tissues, soap and towels **necessary after completing the examination.**

VAGINAL EXAMINATION IN A FEMALE PATIENT

Tips for a successful examination

On part of the patient

1. Intercourse/douching/use of vaginal suppositories should be avoided for at least 24 hours before examination.
2. Urinary bladder should be emptied before examination.

On part of the clinician

1. Explain the steps of examination in advance.
2. Depress the drape between the knees to provide an eye contact with the patient.
3. Avoid unexpected/sudden movements.
4. Warm the speculum.
5. Monitor comfort of the patient (by watching the patient's face).
6. Use gentle technique.

Position: – Standard lithotomy position with thighs abducted
– Drape appropriately. Depress the drape between the knees.

Steps of examination

– Inspection and palpation of the vulva
– Per speculum examination (PS/E)
– Per vaginum examination (PV/E) with bimanual palpation of bladder, uterus and adnexa
– Inspection of the anal and perianal area.
– Palpation of the perianal area
– Digital rectal examination (DRE)
– Proctoscopic examination
– **Allow the patient adequate time to wash and dress.**

Free ebooks ==> www.Ebook777.com

EXTERNAL EXAMINATION OF THE VULVA

A. Assess the sexual maturity (of an adolescent patient)
Causes of delayed puberty: Familial, chronic illness in general, abnormalities of hypothalamus/anterior pituitary/ovaries.

B. External genitalia

1. Mons pubis, labia majora, and perineum.
Look for:
- Excoriations or itchy, small, red maculopapules suggestive of pediculosis pubis.
- Nits and lice at the bases of the pubic hairs.

2. Separate the labia, and inspect the clitoris, urethral meatus, labia minora, and introitus (i.e. vaginal opening).
- Enlarged clitoris may suggest masculinizing disorder
- Look for prolapse of the urethral mucosa, caruncle
- Ask the patient to cough. Look for stress urinary incontinence
- Look for discharge, atrophic changes, erosions, ulcers, and warts; any of them could be a cause of dysuria and pelvic discomfort
- Look for swelling like Bartholin's cyst, Bartholin's gland infection/abscess, sebaceous cyst/s, malignant ulcer/ulcerated swelling.

Clinical method of palpation of swelling of Bartholin's glands, and palpation of the urethra
- Insert index finger into the vagina near the posterior end of the introitus.
- Place the thumb on the posterior part of the labium majus. Palpate bidigitally between the index finger and the thumb on each side in turn
- Look for tenderness, swelling, and extrusion of pus from opening of ducts of the glands
- Palpate the urethra with index finger in the vagina.

Feel for:
- Induration—could be due to chronic inflammation or cancer
- Diverticulum—may cause a purulent discharge from the urethra.

INTERNAL EXAMINATION OF THE VULVA

A. With the labia separated by the thumb and index finger, ask the patient to **bear down** (Valsalva maneuver). Look for a cystocele (prolapse of the bladder), cystourethrocele and rectocele (prolapse of the rectum).

B. Per speculum examination (**PS/E**): Look for cervical abnormality.

C. Per vaginum examination (**PV/E**) and bimanual examination.
Bimanual palpation is invaluable in: 1. assessment of regional extent of pathology such as a tumor, and 2. assessment of mobility of normal and abnormal structures (which cannot be obtained by any radiological technique, however sophisticated).
Lubricate the index and middle fingers of gloved hand, and from standing position, insert the fingers into the vagina, exerting the pressure primarily posteriorly. The thumb should be abducted, and the little and ring fingers flexed into the palm. Pressing inwards on the perineum with flexed fingers causes little if any discomfort and allows to position the palpating fingers correctly.
Points to palpate and note
- Tenderness, nodularity in the vaginal wall, including in the region of the urethra and bladder anteriorly. Stool in the rectum may simulate a rectovaginal mass. A differentiating point in favor of stool: Stool can be indented by digital pressure
- Cervix: Position, shape, size, mobility and tenderness (cervical excitation), surface and consistency. **Normally cervix can be moved somewhat without pain**.
  Feel the fornices around the cervix

www.Ebook777.com

- Uterus: Place the other hand on the suprapubic area midway between the umbilicus and the symphysis pubis. While elevating the cervix and uterus with the pelvic hand, press the abdominal hand in and down, trying to grasp the uterus between the 2 hands.
  Note its shape, size, mobility, consistency, tenderness and masses arising out of it.
  Now slide the pelvic fingers into the anterior fornix, and palpate the body of the uterus bimanually. In this position, the pelvic fingers feel the anterior surface, and the abdominal fingers feel a part of the posterior surface.
  Inability to feel uterus by either of these maneuvers suggests retroverted or retroflexed uterus. Slide the fingers into the posterior fornix and feel for the uterus butting against the fingertips.
  Both retroversion and retroflexion are normal variants
- Each ovary: Place the abdominal hand on the lower quadrant and the pelvic fingers into the lateral fornix of the corresponding side. Press the abdominal hand in and down, trying to push the adnexal structures towards the pelvic hand. Try to identify the ovary or any adjacent adnexal masses.
  By moving the hands slightly, slide the adnexal structures between the fingers if possible and note the details: Shape, size, mobility, tenderness and consistency.
  Normal ovaries are somewhat tender. They are usually palpable in slender, relaxed women but are difficult or impossible to feel in obese or poorly relaxed.
  3–5 years after menopause, the ovaries get atrophied and are no longer palpable. Palpable ovaries in this situation must be considered to be abnormal—cyst or tumor.
  Adnexal masses most commonly result from disorders of the uterine tubes or ovaries.
- Assess the strength of pelvic muscles: Withdraw the fingers slightly just clear of the cervix, and spread them to touch the sides of the vaginal wall. Ask the patient to squeeze her pelvic muscles around the fingers as hard and long as she can. A squeeze that compresses the fingers snugly, moves them upward and inward, and lasts ≥ 3 seconds suggests full strength of the pelvic muscles
- If urethritis/paraurethritis is suspected: With the index finger in the vagina (flexor surface facing upwards i.e. towards the urethra), milk the urethra gently from inside outward. Note any discharge from or about the urethral meatus; if present, culture it.

Remove the finger and proceed with rectal examination.

INTERNAL EXAMINATION WHEN THE INTROITUS IS SMALL

This situation is met with due to virginity, and atrophy and tightness due to old age.

(Alternative) Techniques of examination

- Use only index finger
- A small speculum may make inspection possible
- If introitus is even smaller, a fairly good bimanual examination can be performed by placing one finger in the rectum rather than in the vagina.

A practical point worth remembering regarding lubrication of gloved fingers: Let the lubricant drop on to the fingers without allowing contact between the tube and the gloves. The tube should be discarded if contaminated inadvertently.

SPECIFIC GYNECOLOGICAL NORMAL AND ABNORMAL FINDINGS, INTERPRETATION, AND DISCUSSION

Swellings of the vulva, urethral meatus and vagina: Refer to chapter 29.

Imperforate hymen

Imperforate hymen occasionally delays menarche.

Shapes of the cervical os

- Normal: Round, oval or slit-like.
- Trauma of one / more vaginal deliveries may tear the cervix, producing lacerations: Unilateral transverse / bilateral transverse / stellate.

Retention cysts: They appear as one / more translucent nodules and have no pathological significance.

Free ebooks ==> www.Ebook777.com

Abnormalities of the cervix

Cervical polyp:

- Usually arises from the endocervical canal, becoming visible when it protrudes through the os
- Bright red, soft and rather fragile
- When only the tip is seen, it cannot be differentiated clinically from a polyp originating in the endometrium
- Benign, but may bleed.

CA cervix:

- In its earlier stage, it cannot be distinguished from a normal cervix
- In a late stage, an irregular cauliflower-like growth may develop.

Mucopurulent cervicitis → purulent yellow discharge from cervical os; usually due to infection from *Chlamydia trachomatis*, *Neisseria gonorrhoeae*, or *herpes*. These infections are sexually transmitted, and may occur without symptoms and signs.

Physiological vaginal discharge is clear or white, and may contain white clumps of epithelial cells. Not malodorous.

Distinguishing vaginal from cervical discharge

Use a large cotton swab to wipe-off the cervix. If no cervical discharge is present at the os, suspect vaginal origin (due to vaginitis).

Abnormalities of vagina of surgical importance: Vaginitis, cancer, fistula (VVF, RVF).

Vaginitis types → *Symptoms and signs* ↓	*Trichomonas vaginitis*	*Candida vaginitis*	*Bacterial vaginitis*	*Atrophic vaginitis*
Symptoms (other than discharge, pruritus, soreness, and dysuria, and may be dyspareunia)	Pruritus usually as severe as with candidiasis	-	Unpleasant fishy or musty genital odor	-
Vulva	May be reddened	Vulva and even the surrounding skin are often inflamed and sometimes swollen to a variable extent	Usually normal	Atrophic
Discharge	– Yellowish green or gray, possibly frothy – Often profuse and pooled in the vaginal fornix – May be malodorous	– White and curdy – Typically thick, but may be thin – Not as profuse as in Trichomonas infection – Not malodorous	– Gray or white, thin, homogeneous; coats the vaginal walls – Usually not profuse; may be minimal – Malodorous	– Variable in color, consistency and amount – May be blood-tinged – Rarely profuse
Notes	– Caused by *Trichomonas vaginitis*, a protozoa – Often but not always acquired sexually	– Caused by *Candida albicans*, a yeast (a normal vaginal inhabitant) – There are many predisposing factors	– Exact pathogenic agent: Unknown. Probably caused by anaerobic bacteria – May be transmitted sexually	Caused due to decreased estrogen production after menopause

Uterus

- Uterine enlargement suggests pregnancy, or tumor (benign or malignant)
- Nodules on the surface of the uterus suggest myomas (fibroids)
- *Myomas (Fibroids)*: Very common benign uterine tumors. Single or multiple. Vary greatly in size, occasionally reaching massive proportions. Feel like firm irregular nodules in continuity with the uterine surface. A nodule projecting posteriorly can be mistaken for a retroflexed uterus. Occasionally, a myoma projecting laterally can be confused with an ovarian mass. Submucous myomas may be suspected because of an enlarged uterus

www.Ebook777.com

– *Prolapse of the uterus* is often associated with a cystocele and rectocele
– The cervix well within the vagina = 1st degree prolapse
– The cervix levels with the introitus = 2nd degree prolapse
– The cervix and vagina prolapsing outside the introitus = 3rd degree prolapse (procidentia).

Adnexal masses result from disorders of the uterine tubes or ovaries, and broad ligament.
Examples: Rupture of tubal (ectopic) pregnancy, salpingitis, salpingo-oophoritis, T-O abscess, ovarian cyst, broad ligament cyst, lipoma, fibroma, dermoid, malignant tumor, Krukenberg tumor.
Small (≤ 6 cm in diameter) mobile cystic masses in a young woman are usually benign and often disappear after the next menstrual period.
There are 3 conditions which are often hard to differentiate from adnexal mass
1. Pedunculated myoma of the uterus.
2. Inflammatory disease of the intestine e.g. appendicitis, diverticulitis, etc.
3. Cancer of the colon.

Causes of impaired strength of pelvic muscles: Vaginal deliveries, old age, neurological deficit. Pelvic muscle weakness may be associated with urinary stress incontinence.

RECTAL EXAMINATION

Position *of the patient*
Pelvic examination can be performed in various positions. Each position has some advantages and some disadvantages.

Position	*Particular advantage*	*Particular disadvantage*	*Particular indication / remarks*
1. Left lateral	Convenient for the patient	Bimanual examination not possible comfortably	Most routinely used in practice
2. Dorsal	Good for bimanual examination	Not so satisfactory for perianal examination	Old patients. Too ill/frail patients
3. Right lateral	– Pelvirectal junction tends to fall down and consequently may become amenable to the examining finger	–	– Indicated when pelvirectal junction pathology is suspected
	– Practically more convenient than left lateral position, for palpation of prostate		
4. Knee-elbow	Best for feeling prostate and seminal vesicles	Most awkward for the patient	Least commonly used in practice

Left lateral position: Patient lies on the left side with flexion at the 'upper' (i.e right) knee and hip. The 'lower' limb is kept straight though in a relaxed state. The hip should be near the edge of the examination bed/table.
Dorsal position: Patient lies supine with the lower limbs flexed (sole resting on the examination table).
Right lateral position: Similar to left lateral position with particulars applicable to the opposite sides.
Give the patient **enough time to get in the proper position and relax as much as possible**.
A few reassuring words before starting the examination are helpful. Place (the other) hand on the patient's shoulder. This provides subtle reassurance much needed before touching the anus.

Inspection
Separate the buttocks and examine the anal and perianal area.
– **Position of the anus**. Is it ectopic?
– **Puckering of the anus** suggests a good (normal) sphincter tone
– Any **mass/swelling related to the anus**?
  - Sentinel pile, abscess, haematoma, prolapsed internal pile

Free ebooks ==> www.Ebook777.com

- Prolapse of rectum
- Warts, condylomata

– **Fissure**?
  - Usually posteriorly in the midline
  - Anteriorly in the midline in females
  - If at any site other than anterior / posterior, then suspect anal cancer

– **Perianal area**:
  - Opening/s of anal fistula / rarely a sinus? Exact location and exact distance from the anal verge?
  - Dermatitis?
  - Ulcer?

Palpation of the perianal area after gently separating the buttocks

– Saddle anesthesia?
– Tenderness?
– Induration?
– Softening?
– Any swelling?

Digital Rectal Examination

Thoroughly lubricate the gloved index finger.

Place the index finger on the anus in such a way to exert gentle pressure with the pulp of the finger (*NOT with the tip of the finger*) till the finger slips in the anal canal. Initially, insert only the distal phalanx to give the anus time to relax and to accommodate the finger easily.

Note the following points:

1. **Tenderness, and Tone** of the anal sphincter?
   – Tenderness and spasm suggest a painful anorectal condition. It may not allow completing the rectal examination
   – Funnel shaped patulous anus is suggestive of being habituated to anal coitus
   – Flaccid or spastic anal sphincter suggests similar changes in the urinary sphincter and may be a clue to the diagnosis of neurogenic disease
   – Local causes of reduced tone—prolapsed piles and rectum.

   Wait a few seconds; the anal sphincter normally relaxes to the degree that the finger can be advanced to the knuckle without causing pain.

   Ask the patient to squeeze the examining finger within the rectum and judge the squeeze pressure.
2. **Lumen**

   Note the normal broadening of rectal cavity, and the contents.
3. **Posterior** wall
   – Note the concavity of the sacrum and coccyx.

   Abnormalities in this wall are usually outside the wall of the rectum.

   Presacral swellings—sacrococcygeal teratoma, postanal dermoid, anterior meningocele, chordoma, chromaffin tumor of the sympathetic chain
   – Is there any internal opening of a fistula?
   – In a suspected case of coccydynia, grasp the coccyx between index finger in the rectum and thumb outside, and move it in order to detect any abnormal mobility and tenderness.
4. **Lateral** walls
   – Tenderness?

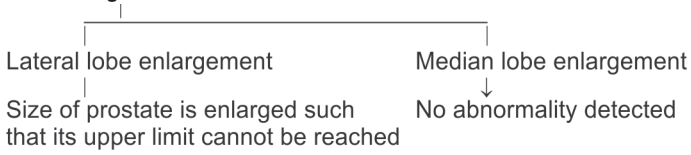
   Abnormality in the following structures / the following abnormalities may be detected"
   - Uterine tubes and ovaries
   - Pelvic appendicitis (usually on the right side)
   - Ischiorectal fossae for abscess

www.Ebook777.com

- Rarely a stone in the lower end of the ureter
- Inflammation and growth from the bony wall of the pelvis
- Rarely, central dislocation of the hip joint or fracture of the pelvic girdle
- Internal iliac arteries and internal iliac group of lymph nodes

Tenderness high up (usually on the right side) suggests appendicitis/salpingitis/salpingo-oophoritis.
Bilateral tenderness suggests salpingitis/salpingo-oophoritis.
Rotate the finger completely to palpate the anterior wall.

5. **Anterior** wall – In c/o a **female** patient
   a. **Cervix**: **Normal** cervix feels like a rounded firm mobile swelling felt anteriorly—often mistaken for a pathological swelling!
   b. **A retroverted/retroflexed uterus**: Usually mobile. Occasionally may be fixed (immobile) due to endometriosis or PID.
   c. **Pouch of Douglas**:
      - Cancer of cervix/vagina
      - Adnexal tumor.
   d. Abnormality in the **rectovesical pouch**—usually in the form of a swelling.
   e. Base of the bladder may be felt only if the bladder is greatly distended or grossly infiltrated with cancer.
   f. An indurated elevation with a central hole suggests internal opening of a fistula.
5. **Anterior** wall
   – In c/o a **male** patient
   a. **Prostate**
      *Features of a **normal** prostate*
      – Felt outside the wall of the rectum. Rectal mucosa is free
      – About the size of a chestnut
      – Consistency: Similar to that of the contracted thenar eminence (with the thumb opposed to the little finger)
      – Central groove (median sulcus)
      – Upper border *just* palpable.
      ***Abnormal** features*
      – Tenderness suggests acute prostatitis, abscess
      – Findings in BPE



      Deepening of the lateral grooves. Median sulcus is maintained.
      Surface—smooth. Consistency—firm.
      – Soft consistency ⇒ abscesss.
      – Hard consistency ⇒ cancer / calculous prostatitis.
      – Grating feel ⇒ prostatic stones.

   In children, prostate is not fully developed; hence base of the bladder is palpable.
   On bimanual palpation of bladder, a large stone in the bladder may be palpable.
   b. **Seminal vesicles**
      Normally not palpable.
      Palpably enlarged seminal vesicles suggest abnormality. Examples: Acute seminal vesiculitis, tuberculous seminal vesiculitis (irregular/granular and hard), etc.

Free ebooks ==> www.Ebook777.com

c. Abnormality in the **rectovesical pouch**—usually in the form of a swelling.
d. Base of the bladder may be felt only if—the bladder is greatly distended OR grossly infiltrated with cancer.
e. An indurated elevation with a central hole suggests internal opening of a fistula.

6. **Inferiorly**
– In c/o a **male** patient
Cowper's glands (bulbourethral glands): Try to feel for them between the index finger in the rectum and the thumb outside on each side of the median raphe.

7. **Other abnormalities** which may be felt
– Complicated internal piles (thrombosed / fibrosed, etc.)
– Polyp
– Stricture
– Tumor (including rare single and multiple tumors). Estimate mobility of rectal tumor in relation to the pelvis.
Classification based on mobility:
• The tumor moves with mucosa = Mobile
• Does not move with mucosa but moves relative to the pelvis = Tethered
• Does not move relative to the pelvis = Fixed.
– Internal opening of a fistula.

8. '**Bifinger** palpation'
a. Index finger in the rectum and thumb outside.
– Try to feel for a small mass by comparing different quadrants with each other
– Grip the coccyx and move it. Note any tenderness and excessive mobility.
While palpating with the index finger alone in the rectum, a small mass in the anorectal wall or just outside the wall may be completely missed if the mucus membrane over it is normal, and particularly if the patient is wriggling about due to discomfort / pain.
b. Index finger in the rectum and thumb in the vagina. Normally both should almost meet.
The presence of a mass (abscess, tumor etc.) will prevent them from touching each other.

9. '**Bimanual** palpation': Index finger of one hand in the rectum and the other hand in suprapubic region.
a. This will define relation of the abdominal lump with the lump palpable in the rectum.
b. Information regarding fixity of the lump coming out of the pelvis.
c. Assess mobility of normal and abnormal structures (which cannot be obtained by any radiological technique, however sophisticated).
d. A large bladder stone may be palpable.

10. **During withdrawal of the finger**
a. Flex the finger slightly and feel the rectal wall.
b. A soft swelling in the rectal wall is more likely to be felt on the withdrawal journey of the finger than on its upward course.

11. **After withdrawing** the finger,

a. Inspect the tip of the gloved finger for blood, pus and mucus.
b. **Test the stool** smearing the glove, **for occult blood** when indicated.

PROCTOSCOPIC EXAMINATION

The nomenclature for describing the appropriate instruments is confusing.
Proctoscopy means inspection of the rectum + anal canal. Hence, a proctoscope is expected to be useful for this purpose. In practice however, the so-called proctoscope is a short instrument (usually measuring not more

www.Ebook777.com

than 12 cm long) which is only really of value in examining the anal canal and lower rectum, but not the entire rectum. Such a short instrument should be ideally called ‘anoscope’.

The term *sigmoidoscope* is equally misleading as only the very distal part of sigmoid colon can be examined with it. In most cases anyway, it is impractical to pass a rigid sigmoidoscope much beyond 15 cm.

Position of the patient: Commonly left lateral.

Procedure

- Lubricate the obturator and while holding it firmly within the sheath of the proctoscope, pass it gently within the anal canal
- Push the scope towards the umbilicus for the 1st 4 cm. Beyond 4 cm, change the direction and push towards sacral promontory
- Withdraw the obturator, and clean the lumen with a swab if necessary
- With a good illumination, inspect the lumen of the rectum and anal canal while gradually withdrawing the sheath of the proctoscope
- **If the sheath is required to be pushed in again for re-inspection, the obturator must be introduced in the sheath before beginning to push the proctoscope in.**

After completing the pelvic examination, allow the patient adequate time to wash and dress.

Free ebooks ==> www.Ebook777.com

8 EXAMINATION OF THE PREGNANT PATIENT

GENERAL REMARKS

- It is important to recognize the changes in anatomy and physiology that are related to pregnancy, apart from an abnormality
- The thyroid and the breasts undergo moderate enlargement
- The abdomen's most notable change is distension. Early distension from fluid retention and relaxation of abdominal muscles may be noted before the uterus becomes an abdominal organ (12–14 weeks of gestation).

GENERAL EXAMINATION

- **Is the patient fully conscious, cooperative and well-oriented in time, place and person**?
- **Posture**
  Is there any specific posture in which she appears to be more comfortable?
- **General appearance**: Looks healthy or ill?
- **Facial appearance**?
  - 'The mask of pregnancy' and a bit of facial edema are normal
  - Facial edema after 6 months suggests PIH**.**
- **Vital parameters, JVP**?
  - In mid-pregnancy, BP is normally lower than in the non-pregnant state
  - High BP prior to 6 months suggests chronic (pre-existing) HT
  - High BP after 6 months needs evaluation to diagnose and treat PIH.
- **Height, weight, body proportion**?
  1st trimester weight-loss related to nausea and vomiting should not exceed 2–3 kg. Weight-loss of > 3 kg may be due to hyperemesis.
  Recommended total weight-gain-ranges for pregnant women

Weight-for-height category	*Recommended total gain*
Low i.e. BMI < 19.8	12–18 kg
Normal i.e. BMI 19.8–26	12–16 kg
High i.e. BMI 26–29	7.0–12 kg

  - Figures of weight-gain mentioned above are for single pregnancies
  - The range for women carrying twins is 16–20 kg
  - Young adolescent (< 2 years after menarche) should strive for gains at the upper end of the range
  - Short women (< 155 cm) should strive for gains at the lower end of the range.
- **Hair**?
  Localized patches of alopecia should not be attributed to pregnancy.
- **Eyes**?
  Look particularly for anemia.
- **Mouth**?
  Gingival enlargement and bleeding is common during pregnancy.

www.Ebook777.com

- **Throat, nose, ear**?
  Nasal congestion and nosebleeds are common during pregnancy.
- **Neck**?
  Thyroid
  – A physiological goitre is expected
  – A marked or irregular enlargement of thyroid is NOT due to pregnancy.
- **Nails**, **palm**, **hands**, **skin**, and **lower limbs**?
  – Varicose veins may begin or worsen during pregnancy.
  – Look for edema on hands, feet, ankles and leg.
  Physiological edema is common in advanced pregnancy, during hot weather, and in women who stand for long hours.
  Pathologic edema may be associated with PIH.

  Generalized lymphadenopathy?

EXAMINATION OF THE ABDOMEN

Positioning
- Semi-sitting position affords the greatest comfort
- As prolonged periods of lying on the back (i.e. supine position) may lead to hypotension (making her feel dizzy and faint), abdominal palpation should be carried out efficiently
- All other examination procedures should be done in sitting or left lateral lying position.

INSPECTION

– Movements of the anterior abdominal wall associated with respiratory excursions?
– Contour of the abdomen?
– Any asymmetry?
– Visible lump, if any? Note the features of the lump as usual
– Skin:
  - Linea nigra = a brownish black pigmented line following the midline of the abdomen
  - Purplish striae (furrows) are bands of thin wrinkled skin, initially red, becoming white with passage of time
– Dilated veins?
– Umbilicus?: Normally, it is displaced upwards by the gravid uterus
– Visible peristalsis?
– Pulsations?
– Fullness (bulging) of intercostals spaces?

PALPATION

A. First round of palpation: **Gentle 'superficial' palpation** with **flat of the fingers**.
  1. Any hyperesthesia in well-defined areas?
  2. Abdomen soft, OR contracted? If contracted: Does it appear to be due to voluntary contraction or guarding?
  3. Tenderness present or absent? If present—in which area?
  4. Any lump?
  5. Any (gross) organomegaly?

B. Next round of palpation
  – **'Deep' palpation** to obtain details.
  1. Localization of tenderness.

Free ebooks ==> www.Ebook777.com

2. Lump caused by the gravid uterus

Palpable height of the uterine fundus	*The most probable month of pregnancy*
– Just suprapubic	3 months
– Midway between the umbilicus and pubic symphysis	4 months
– At the level of umbilicus	5 months
– Almost at the level of xiphoid	9 months

Uterine contractility

The uterus contracts irregularly after 3 months and often in response to palpation. The abdomen then feels tense or firm and it is difficult to feel fetal parts.

If the hand is left resting on the fundal portion of the uterus, the fingers will sense the relaxation of the uterine muscle.

Prior to 37 weeks, regular uterine contractions with or without pain or bleeding are abnormal; they suggest preterm labor.

Diastasis recti

= separation of the rectus muscles, may be noticeable in the later trimesters of pregnancy.

If diastasis is severe, as it may be in multiparous women, only a layer of skin, fascia, and peritoneum covers most of the anterior uterine wall. The fetus is felt easily through this muscular gap.

Fetal movements

These can usually be felt after 6 months (and experienced by the mother at 5 months onwards).

If movements cannot be felt after 6 months, consider error in calculating gestation, or fetal death/ morbidity, or false pregnancy.

3. Details of a pathological lump if any, as usual.

4. Details of organomegaly, if any.

5. Pulsations of the aorta and iliac arteries.

6. Para-aortic enlarged lymph nodes.

7. 'Succusion splash': Avoid this test in a pregnant woman, however, if it is must, do it extraordinarily gently.

8. Determine the direction of blood-flow in dilated veins, if any.

9. Measurement of fundal height with a tape measure if the woman is > 5 months pregnant.

Hold the tape in the midline and measure the distance from the top of the pubic symphysis to the top of the uterine fundus. After 5 months, measurement in centimeters roughly equals the weeks of gestation.

- If fundal height is > 2 cm higher than expected, consider: Multiple gestation, a big baby, extra amniotic fluid, or uterine myomata
- If fundal height is less than expected by > 2 cm, consider: Missed abortion, transverse lie, growth retardation, or false pregnancy.

AUSCULTATION

A. Auscultate the mother's abdomen in the usual way.

B. Auscultate the fetal heart: Location, rate and rhythm.

C. With a doptone, fetal heart sounds are audible after 12 weeks.

D. With a fetoscope, they are audible after 18 weeks.

Location

- Between 12 and 18 weeks of gestation: In the midline of the lower abdomen
- After 28 weeks, they are heard best over the fetal back/chest. The location depends on the lie of the fetus.

Normal rate

- During early pregnancy: In 160s / minute
- Slows to 120s to 140s near term.

www.Ebook777.com

Interpretation of unusual / abnormal findings

- After 6 months, auscultation of > 1 FHSs with varying rates in different locations suggests > 1 fetus
- Lack of audible FHSs may indicate pregnancy of fewer weeks than expected, fetal death, or false pregnancy
- Fetal heart rate that drops noticeably near term with fetal movements could indicate poor placental circulation.

EXAMINATION OF THE CHEST

- Although women late in pregnancy sometimes report difficulty in breathing, there are usually no abnormal physical signs
- On auscultation of the heart, soft blowing murmurs are common during pregnancy
- Breasts undergo changes during pregnancy.

PELVIC EXAMINATION

Examination of the Groins

- External iliac and femoral artery pulsations
- Inguinal lymph nodes
- Hernial sites.

Inspection of the External Genitalia

- Parous relaxation of the introitus and noticeable enlargement of labia and clitoris are normal
- Look for scars of episiotomy or perineal laceration
- Some women have labial varicosities that become painful and tortuous
- Palpate Bartholin's and Skene's glands. Normally, there should be no tenderness and discharge
- Look for cystocele and rectocele. They may be pronounced due to muscle relaxation of pregnancy
- Inspect the anus for piles. They often engorge later in pregnancy. They may be painful, and bleed.

Per Speculum Examination

1. *Vagina*
   - Vagina takes on a bluish / violet color. A pink vagina suggests a non-pregnant state
   - Walls appear thicker and deeply rugated (because of increased thickness of the mucosa, loosening of the connective tissue, and hypertrophy of smooth muscle cells)
   - Secretions become thick, white and more profuse; considered normal during pregnancy
   - Vaginal pH becomes more acidic.
2. *Cervix*
   - Cervix looks cyanosed. Cyanosis appears very early after conception and continues throughout pregnancy
   - Red velvety mucosa around the os. A pink cervix suggests a non-pregnant state
   - The canal is filled with a tenacious mucus plug that protects the developing fetus from infection
   - Pronounced softening develops in the cervix during pregnancy.

Per Vaginum Examination

Tissues are soft. It may be difficult to distinguish the cervix at first because of its softer texture.

- Cervical os: A nulliparous cervix should be closed, while a multiparous cervix may admit a fingertip through the external os. The internal os should be closed in both situations
- The surface of a multiparous cervix may feel irregular due to the healed lacerations from a previous birth
- Estimate the length of cervix by palpating the lateral surface of the cervix from the cervical tip to the lateral fornix. Prior to 34–36 weeks, the cervix should retain its normal length of ~ 1.5–2 cm
  A shortened effaced cervix prior to 32 weeks suggests preterm labor
- Early in pregnancy, *the corpus luteum*, the ovarian follicle that has discharged its ovum, may be sufficiently prominent to be felt on the affected ovary as a small nodule; it disappears by midpregnancy.

Free ebooks ==> www.Ebook777.com

9 DIAGNOSIS

Definition: Diagnosis is a label to particular changes in anatomy and physiology of the body, with the resultant group of symptoms and signs.
Anatomical + Pathological diagnosis may be defined as '***basic*** *diagnosis*'.
'A ***complete*** *diagnosis*' includes the presence of associated disease, if any.
A diagnosis may be labeled as '***partial***', when all the aspects of diagnosis cannot be determined.
Diagnosis is not something which should be made for its own sake but as a means to a cure; and a favorble moment for successful treatment (operative or otherwise) should not be lost merely due to the lacking of a complete diagnosis.

'Disorder' means derangement or abnormality of function.
'Disease' is a more specific term applied for an illness characterized by a recognizable pattern of symptoms and signs.

The causative factor → a series of changes in the structure and function of tissue / organ / system / whole body. These changes become manifested as symptoms, signs and abnormal findings in investigation-results. Inference from all symptoms, signs and investigations collectively give a total picture of various changes that have occurred in the body as a result of a disease.

Try to relate every symptom and sign to anatomy, physiology and pathology. That means, from every symptom and sign, try to determine (1) The **exact site** of the disease i.e. which region-system-organ-tissue seems to have been affected by the disease; (2) **Functional disability**, which in turn may help to determine **the site of the disease again**, and also the **degree of homeostatic imbalance**; and (3) **Pathology**.
Every symptom and sign may not be able to determine all the three; but try to relate to as many of them as possible.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. Thinking process should begin at the outset of the patient encounter, not at the end. **Try to enhance the relevance and value of the data that is collected**.
2. **Organizing the clinical data poses several challenges**. One of the greatest difficulties faced particularly by the beginner student is how to cluster the clinical data.
   One needs to decide whether to cluster the patient's symptoms and signs into one problem or into several problems. The amount of data may appear unmanageable. The following guidelines may be helpful.
   a. Young people are more likely to have a single disease, while older people tend to have multiple diseases.
   b. If specific symptoms and signs occur in a single system, one disease may explain them.
   c. When multiple body systems seem to have been affected, there are 2 possible explanations—either multiple diseases, or a single disease affecting multiple systems. To select the correct option between the two, the knowledge of disease *pattern* is necessary.
3. Diagnosis is often achieved by *pattern recognition*.
4. **Occasionally patients with a chronic disease present in an acute manner**. There are two possible reasons for this. Firstly, symptoms are perceived truly late by the patients. Secondly, some patients hide

www.Ebook777.com

their symptoms perceived early during the course of the disease for reasons best known to them. Arriving at the diagnosis becomes difficult in such patients. In such a situation, ignore acute presentation. Consider it as a chronic case and review the whole situation. This alternative process may improve the chance of making the correct diagnosis.

5. In some situations, making a complete diagnosis may be difficult, and one may have to be satisfied with '*a **partial diagnosis***'; for example, in some acute cases, determination of pathology alone may be possible, the exact localization of organ / tissue being difficult or even impossible. Even then, an urgent surgical treatment may have to be executed due to (acute life-threatening) pathological nature of the disease. Such an operative procedure is primarily of exploratory nature which is logically continued as therapeutic once the diseased organ / tissue is localized 'on table'. If an urgent surgical procedure is delayed then the acute pathological process may advance rapidly resulting in permanent deformity / disability or death. Examples: Acute osteomyelitis (which sometimes may be difficult to differentiate from acute septic arthritis or acute pyomyositis), acute peritonitis, acute hemoperitoneum, (acute) cerebral compression following head injury, etc.
6. Think about an alternative diagnosis to the obvious one, perhaps especially where the patient's symptoms are not absolutely typical, where the key investigations have not identified the expected condition, or where the patient has been back to the clinic several times with apparently progressive symptoms but a diagnosis that remains elusive. No surgeon can get it right all the time.
7. Confer with colleagues and review the pertinent medical literature.
8. The final diagnosis may turn out to be totally different, that was never thought of clinically, even after making a clinical diagnosis by methodical evaluation. Surprises are known in clinical practice!

STEPWISE ANALYSIS OF DATA

STEP 1: IS THE CONDITION **ACUTE, CHRONIC OR ACUTE-ON-CHRONIC**?

- Acute = duration of symptoms up to 1 week.
  Mode of onset—usually sudden. Progress—usually rapid (hours to a couple of days). Patient may look distressed or sick
- Subacute = duration of symptoms up to 3 weeks.
  Mode of onset—usually sudden, but can be insidious
- Chronic = duration of symptoms > 3 weeks.
  Mode of onset—usually but not necessarily, insidious. Progress—over weeks or more. Disturbance of function is minimal (during the early stage of the disease)
- Acute-on-chronic = basically a known chronic case, presenting with acute manifestations due to rapid progress / modification in the existing pathology.

STEP 2: **ANATOMICAL diagnosis**?

That is, which anatomical region, body cavity, system, organ, tissue seems to have been affected?

Clinical points which may help to localize the site of the disease:

– The site of the chief complaint e.g. pain, swelling, ulcer, etc.
– Symptoms pointing to dysfunction of an organ / tissue.
– Localized tenderness.

- **Regions of the body**
  1. Head: – Cranium
     – Face.
  2. Neck.
  3. Trunk: – Thorax
     – Abdomen and pelvis
     – Back.
  4. Upper extremity: Shoulder to hand, including axilla.

Free ebooks ==> www.Ebook777.com

5. Lower extremity: Buttock to foot.

Parts of each region to be considered in further detail: Wall, lining, potential space if any, and viscera.

- **Body cavities**
  1. Ventral
     - Thoracic, and abdominopelvic.
     - Thoracic cavity contains pleural and mediastinal cavities.
  2. Dorsal
     - Cranial cavity (containing brain), and vertebral canal (containing spinal cord).
- **Systems**
  - Respiratory
  - Cardiovascular including blood (hematological, microcirculation, and lymphatic)
  - Nervous, endocrine: These systems help to regulate homeostasis. In general, actions of the nervous system are more rapid as compared to those of endocrine system
  - Digestive (GI), GU
  - Integumentary, bones, rheumatic (joints, muscles and CT)
  - Immune.
- **Organ/tissue** affected?

  In c/o an organ, a specific area/part and the layer/s (as applicable) of the organ affected?

STEP 3: PATHOLOGICAL diagnosis?

This comprises of pathological nature of the disease, stage of the disease and etiology.

A. **Pathological nature** of the disease?
- *Congenital defect*
- *Abnormal position*: Malrotation / herniation / ptosis / descent (prolapse)
- *Abnormal size:*
  - Reduction in size (due to atrophy resulting from various causes)
  - Enlargement: Can be due to some of the following causes
- *Distension, dilatation*
- *Congenital swelling* e.g. hamartoma, teratoma
- *Congestion*
- *Collection* (of fluid, blood, pus, etc.—free or encysted).
- *Inflammation* (acute / chronic) / *end result of inflammation* (suppuration / ulceration / gangrene / fibrosis)
- *Infiltration*
- *Hyperplasia*
- *Tumor*
- *Abnormal shape*: Examples—diverticulum, narrowing
- *Ischemia, gangrene, necrosis*
- *Sclerosis*
- *Degeneration*. For example, calcarial, etc.
- *Obstruction*
- *Entrapment* such as, of a nerve, artery, tendon, etc.
- *Injury*:
  - External
  - Internal e.g. dissection/rupture of aneurysm, esophageal tear
- The presence of *a foreign body*

- By and large, only alteration of physiology leads to the development of symptoms and few signs (especially those symptoms which also happen to be signs). And when pathological process leads to structural changes in the normal anatomy (that is to say when pathologic anatomy results), physical signs will appear

www.Ebook777.com

- To determine the pathological nature of the disease, select one or two symptoms/signs which are fundamental, primary, prominent, definite and easily verifiable. Consider various possibilities (pathological processes listed above) which can give rise to this symptom / sign. In actual practice, think of the common disease first. Think about rare disease only if all common possibilities are ruled out
- 'Pathognomonic symptom / sign' means a symptom / sign which is characteristic enough to clinch the diagnosis. Pathognomonic symptoms / signs are very few; an example: A swelling in the groin which reduces with gurgling clinches the diagnosis of hernia.

B. **Pathological stage including sequelae and complications**?

At what stage in the natural history of untreated disease has the patient presented to seek medical advise? It is essential to define this, as modality of treatment often varies according to the stage of the disease. Appendicitis may be quoted as a classical example in this regard. An acute appendicitis within 1st 48 hours is often treated by urgent appendicectomy whereas an appendicular lump is usually conserved. It is not uncommon to get patients presenting straight with a complication. If complication is (acute) life-threatening, it needs priority in the management. In such a situation, whether the primary disease which led to complication should be treated simultaneously with the treatment of complication or should be treated later, will vary from case to case.

There are **2 methods to proceed further**:

1. ***Positive or direct method***: This is applicable in typical cases i.e. those cases in which clinical picture matches with the textbook description of a disease. The case/diagnosis may be classified according to the number of diagnostic criteria present; e.g. if there are 10 clinical features as diagnostic criteria, then the case/diagnosis may be classified as under:
   - Classical case—if 7 or more features are present
   - Definite diagnosis—if 5 or 6 features are present
   - Probable diagnosis—if 3 or 4 features are present
   - Possible diagnosis—if 2 features are present.
2. ***Method of exclusion***: This is useful when the case is not typical. Out of the various possibilities thought of, start ruling out the conditions one by one with the help of clinical data. The last condition which cannot be ruled out may be accepted as the diagnosis even if clinical features do not match with the typical textbook description.

C. **Etiology**?

Etiology comprises of the following factors. In some cases all of them are evident, in others it may not be so.

1. **Deficiency / absence / excess of basic physical (physiological) needs**
   - O_2
   - Clean water
   - Fresh food
   - Heat
   - Pressure
   - Protection—clothing and shelter.
2. **Pathogenic agent**
   - Addiction
   - Drug (Medicine)
   - Poison: Animal / plant
   - Carcinogen
   - Injury: Mechanical / thermal / radiational / chemical
   - Infection: Bacterial / viral
   - Infestation
   - Foreign body
   - Antigen: External / self
   - Unidentified

Free ebooks ==> www.Ebook777.com

- 'Functional'
- Psychogenic.

3. **Predisposing factor**
   - Age
   - Gender
   - Race
   - Religion
   - Residence: Environment, weather
   - Diet (and malnutrition)
   - Occupation
   - Heredity
   - Pregnancy
   - Mode of contraception
   - Hypersensitivity
   - Activity / inactivity
   - Lifestyle
   - Posture.
4. **Precipitating event**.

STEP 4: IS THERE HOMEOSTATIC IMBALANCE?

The physiological derangement needs to be determined in some cases. Fluid, electrolyte and acid-base imbalance is reflected in clinical features; for example altered level of consciousness, dehydration, tachycardia, hypotension, altered urine output, etc.

STEP 5: ASSOCIATED DISORDER if any?

Closely related or otherwise?

↙ ↘

- Congenital anomalies
- Syndrome

- Congenital anomaly
- Permanent disability
- Chronic systemic disease e.g. chronic bronchitis / asthma / HT / IHD / CVD / convulsion / DM / thyroid disease / liver, spleen, kidney disease / RA / immune deficiency, etc.: Controlled / compensated / active?
- Any short duration illness e.g. cold, flu, jaundice, typhoid fever etc?

|

- Stress associated with anesthesia and surgery may worsen the associated disorder
- An uncontrolled/decompensated illness may complicate anesthesia and surgery.

Note

- If it is not possible to determine one particular step in the process of arriving at the diagnosis, go on to determine the next step. Come back to the missing step later, after drawing some conclusion about the possible diagnosis
- The steps and their order discussed above are written for giving the basic guidance on how to analyze clinical data to arrive at the diagnosis. Due to variability in specific pathology affecting different organs/ systems, this same order of steps may not be applicable in all chapters that follow in the book.

MAKE A HYPOTHESIS OF DIAGNOSIS, AND JUSTIFY IT WITH THE HELP OF THE FOLLOWING POINTS

- **Basic point**
- **Confirmatory = differentiating points**
- **Supporting points.**

www.Ebook777.com

It is the differentiating points which pinpoint the diagnosis.

This process of making a hypothesis and justifying it draws on all the knowledge and experience that have been gained through reading and (unrelenting) clinical practice.

Sometimes symptoms defy analysis, and differentiating physical findings may be absent; in such a situation, it may never be possible to move beyond simple descriptive categories e.g. 'fatigue', 'pain in abdomen under observation and investigation', 'nonspecific pain in abdomen', etc.

To give a differential diagnosis means to list the possible diagnoses in the descending order of probability. Differential diagnosis is considered when confirmatory (= differentiating) points in favor of one particular diagnosis are not available.

Always include 'the worst case scenario' in the list of differential diagnoses.

APPLIED PHYSIOLOGY

Homeostasis

Homeo = the same. Stasis = standing still.

Homeostasis means a condition in which the body's internal environment remains within certain physiological limits.

Internal environment essentially means the interstitial fluid (= intercellular fluid = tissue fluid).

Components of the internal environment: Water and certain substances dissolved into water viz. gases, nutrients and ions (Na^+ and Cl^-).

An organism is said to be in homeostasis, when it is internal environment has optimum volume essential for health of the cells, contains optimum concentration of solutes, and has an optimum temperature. Good health implies balanced homeostasis.

When one or more components of the body loose their ability to contribute to homeostasis, body processes do not function efficiently.

Degrees of homeostatic imbalance may be classified as mild, moderate and severe.

- *'Mild'* degree of imbalance may remain clinically undetectable as it is corrected quickly
- *'Moderate' imbalance* → disorder or disease
- *'Severe'* imbalance may → death.

Free ebooks ==> www.Ebook777.com

10 GUIDELINES ON DOCUMENTATION OF CLINICAL NOTES

A clear, well-organized clinical record is one of the most important adjuncts to patient care. The skill in case-documentation should evolve in parallel with the growing skills in arriving at the diagnosis.

Documentation, in fact involves proper recording of facts in successive steps of patient management.

- Clinical notes: History, physical examination findings, and diagnosis
- Investigation reports
- Progress notes
- Consent
- Operation notes
- Discharge summary
- Death notes, if applicable.
- Any other specific notes in the given case.

As this book essentially deals with 'arriving at the diagnosis', guidelines on only clinical notes will be discussed in this chapter.

The key issues

1. The order of the contents.
2. The amount of detail needed—this often poses a vexing problem. Students are required to be detailed as it helps to build up descriptive skills, vocabulary, and speed—admittedly a painful and tedious process! Subsequently as a professional however, pressures of work vis-à-vis time will ultimately force some compromises.

GUIDELINES

- Records are scientific and legal documents, so they should be clear and understandable. Use of short forms and brief phrases instead of whole words/sentences is common, but **abbreviation and symbols should be used only if they are readily understood.**
- **Handwriting** must be **legible**; otherwise all that has been recorded is worthless.
- Make the **headings clear.**
- **Omit repetitive introductory phrases** such as 'the patient says …..', 'the patient reports …..'.
- **Omit unnecessary words**, such as those in parentheses in the examples below. This saves valuable time and space.
  Examples: 'Thorax is symmetrical (bilaterally, or on comparison)', 'lungs are resonant (to percussion)', 'liver is tender (to palpation)', 'cervix is pink (in color)', etc.
- To ensure accurate evaluations and future comparisons, **make measurements in centimeters**, not in nuts, fruits, vegetables, balls or bats.
- **Diagrams** add greatly to the clarity of the record. I attach printouts of preoperative, intraoperative, and postoperative photographs, to the patient's record. It is worth making use of the technological advent (digital photography) in day-to-day practice.
- Omit the negative findings which do not seem to carry enough weight (for being useful in differential diagnosis).
- Try to describe structures in a concise, positive way. Examples: 'Temperature overlying the swelling is normal', 'surrounding area is normal', 'cervix is pink and smooth', etc.
- **Do not use words, penmanship or punctuation that is inflammatory or demeaning**.

www.Ebook777.com

11 SUDDEN LOSS OF CONSCIOUSNESS (SUDDEN UNRESPONSIVENESS) CARDIORESPIRATORY ARREST

DEFINITIONS

Unconsciousness (Complete loss of consciousness)
= deterioration of the level of consciousness to such an extent that the patient remains unarousable even after application of painful stimuli repeatedly. It implies a potentially life-threatening event.

Coma
= a prolonged and perhaps profound state of unconsciousness from which the patient cannot be aroused.

Cardiorespiratory arrest
= the cessation of respiration and circulation.
Clinically, cardiorespiratory arrest is recognized by **unconsciousness**, **cessation of heart beat (practically,** the absence of carotid artery pulsations on both sides), and **cessation of spontaneous respiration. However, respiratory movements need not be completely absent**; there may be ineffectual gasping movements, but the patient usually becomes cyanosed.

GENERAL REMARKS

The maximum length of time for which the brain can survive at normal temperature without a supply of oxygenated blood is 3–4 minutes; after this interval, the cerebral changes are irreversible and the patient is dead. This fact emphasizes the necessity for very rapid and effective management of cardiorespiratory arrest. After 1 minute of setting in an arrest, the pupils dilate and later they become unresponsive to light. If this stage persists for more than 1–2 minutes, the patient is unlikely to recover.

In cardiac arrest, the heart may fail to produce a cardiac output of blood either because it is truly arrested—*asystole*, or because its action is grossly irregular and uncoordinated—*ventricular fibrillation*.

Causes of sudden loss of consciousness

No carotid pulse
No spontaneous respiration
⇓
Primarily cardiac arrest
↓
- Ischemia—induced arrhythmia / heart block
- Cardiac tamponade
- PE
- Fat embolism
- Vasovagal faint
- Anaphylactic shock
- Neurogenic shock
- Cardiac rupture
- Intramyocardial (tumor) rupture

Carotid pulse initially present
⇓
Primarily respiratory arrest
↓
- Upper airway obstruction from any cause
- Tension pneumothorax
- Insufficient O_2 in inspired air
- Untreated respiratory failure
  - Drug overdose including narcotic depression
  - Drowning
  - Deep anesthesia
  - Failure to remove CO_2 from anesthetic close circuit
  - Loss of respiratory drive
  - Severe pulmonary disease
  - Phosgene inhalation

Both carotid pulse and spontaneous respiration initially present
⇓
Systemic (other than cardiac or respiratory) causes—may lead to loss of consciousness either suddenly or relatively insidiously. They are broadly classified into toxic / metabolic, and structural

All pulses normal, and spontaneous respiration present
⇓
A psychiatric cause mimicking loss of consciousness

Free ebooks ==> www.Ebook777.com

The mode of onset and duration of deterioration of level of consciousness will vary according to the cause and pathology.

Some of the causes may be responsible for **transient loss of consciousness**

Clinical situation / precipitating cause	*Most probable diagnosis*
• Sudden unexpected pain/unpleasant sight/smell/sound	Vasovagal syncope
• Syncope on exertion	Cardiac disorder e.g. IHD, valvular disease, etc.
• Arm exercise	Subclavian steal syndrome
• On abruptly standing from a sitting / lying down position	Orthostatic hypotension
• Prolonged standing in attention	Vasovagal syncope
• Rotational movement of the head	Carotid sinus syncope
• Changing position e.g. sitting to lying / bending / turning over in bed	Atrial myxoma / thrombus
• During / immediately after swallowing / coughing / micturition / defecation	'Situational syncope'
• Neuronal suppression	Hypoglycemia, basilar migraine
• Neuronal excitation	Epilepsy
• Hypotensive medication	Drug-induced syncope

ASSESSMENT OF A PATIENT WITH SUDDEN LOSS OF CONSCIOUSNESS

An **'arrest call'** does not necessarily mean that the patient has had a cardiac arrest, although this is the most common cause.

Is the patient awake and alert? If NOT, then

↓

Quickly assess the LOC (level of consciousness). Simultaneously,

– Start feeling for pedal and carotid pulses on both sides. Palpable pedal pulse/s rule out hypotension

And

– Assess for spontaneous respiration

• Speak to the patient by name and in a loud voice

If the patient opens eyes, responds to questions, and then falls asleep → **'Lethargic'**(= **'Drowsy'**)

No response

• Shake the patient gently, as if awakening a person from sleep

The patient opens eyes and responds albeit slowly, and may be in a confused state → **'Obtunded'**

No response

• Apply a painful stimulus such as rubbing the sternum, pressing on the eponychium with the edge of finger-nail, or rolling a pencil across a nail bed (no more stronger stimuli needed!)

If the patient opens eyes and lapses into an unresponsive state after the stimulus ceases; verbal responses slow or even absent; and motor response either normal-avoidant or stereotypic → **'Stuporose'**

No response

If remains unarousable even after

• Application of painful stimuli repeatedly → **'Unconscious'**

↓

A potentially life-threatening event affecting the 2 hemispheres, the brainstem, or both

The usual sequence of history-taking, physical examination and laboratory evaluation does not apply to this situation

↓

www.Ebook777.com

1. **Position the patient flat** preferably on a **hard surface** (for effective cardiac massage, if indicated), and with **elevation of the lower limbs** (for increasing the venous return). If deemed necessary, transfer the patient rapidly to the floor to ensure a hard surface which will provide adequate counter-pressure for external cardiac massage (if indicated).
2. Assess the **ABC** (Airway, Breathing, Circulation), and respond accordingly.

   Clinical manifestations of hypoxia:
   - Confusion, restlessness, agitation
   - Pallor, sweating
   - Tachycardia.

   Pitfalls
   - Cyanosis is uncommon and difficult to detect clinically, particularly if hypovolemia is present
   - Anemic patients do not become cyanosed. There must be at least 5 g% of reduced hemoglobin in the circulation before cyanosis can be observed.

   Noisy breathing/snoring/stridor ⇒ incomplete upper airway obstruction.

   (Stridor is absent if the airway is completely patent or completely obstructed.)

   The absence of spontaneous breathing / the presence of at the most ineffectual gasping movements ⇒ respiratory/cardiorespiratory arrest → commence resuscitation at once.

   Clinical criteria of compromised breathing
   - Patient making great respiratory efforts, but still hungry for air. The accessory muscles of respiration are put into action
   - Distension or retraction of the IC spaces
   - Cyanosis in the presence of an adequate airway
   - Tachypnea OR bradypnea
   - Paradoxical (see-saw) movement of the chest and abdomen, indicating failure of normal diaphragmatic function.

   Palpable pedal pulse/s rules out hypotension.

   Impalpable carotid pulses on both sides ⇒ cardiac/cardiorespiratory arrest → commence resuscitation at once.
3. Carry out a quick neurological and focused general examination.
4. Interview the relatives / friends / witnesses.

A QUICK NEUROLOGICAL AND FOCUSED GENERAL EXAMINATION

to find out the possible cause of deteriorated LOC, a cardiorespiratory arrest having been ruled out

1. Observe the **rate, rhythm and pattern of respiration**.

   Because neural structures that govern breathing in the cortex and brainstem overlap those that govern consciousness, abnormalities of respiration often occur in deteriorated LOC.

Respiration	*Possible cause of deteriorated LOC*
– Regular rhythm Rate may be normal, or may show hyperventilation	Toxic-metabolic
– Irregular Chyne-Stokes type	Toxic-metabolic, or structural
– Irregular ataxic type	Structural

Free ebooks ==> www.Ebook777.com

2. Observe the **posture.**
If there is no spontaneous movement, apply a painful stimulus and observe the resulting pattern of movement.

Pushes the stimulus away, or withdraws
||
Normal-avoidant

Abnormal postural responses of the trunk and extremities
||
Stereotypic

No respone i.e. flaccid paralysis
|
No response on one side ⇒ a coticospinal tract lesion
Example—hemiplegia early in its course, spasticity will develop later.The paralyzed arm and leg are slack. They fall loosely and without tone when raised and dropped on the bed. The leg may lie externally rotated. One side of the lower part of the face may be paralyzed

Abnormal flexor response = Decorticate rigidity
The upper arms are held tight to the sides. Elbows, wrists and fingers are flexed. The legs are extended and internally rotated. The feet are plantarflexed. When unilateral, this is the posture of chronic spastic hemiplegia
⇓
Destructive lesion of the corticospinal tracts within or very near the cerebral hemispheres

Abnormal extensor response = Decerebrate rigidity
The head is extended. The jaws are clenched. The arms are adducted and are stiffly extended at the elbows. Forearms pronated. Fingers flexed. The legs are stiffly extended at the knees. Feet are plantarflexed. This posture may occur spontaneously or only as response to external stimuli such as light, or pain
⇓
– Lesion in the dincephalon, midbrain or pons.
OR
– Severe metabolic cause like hypoxia or hypoglycemia.

3. Muscle tone
– Grasp each forearm, raise it to a vertical position, and observe the position of the hand.
Normal : Hand is only slightly flexed at the wrist.
Abnormal : The limp hand drops to form a right angle with the wrist ⇒ flaccid paralysis as in hemiplegia of sudden cerebral accident
– Then lower the arm to ~ a foot off the bed and drop it. Observe how (rapidly) it falls.
Normally it drops somewhat slowly.
A flaccid arm drops rapidly
– Flex both lower limbs such that the heels rest on the bed and then release them.
The *normal* limb returns slowly to its original extended position.
In acute hemiplegia, the flaccid limb falls rapidly into extension, with external rotation at the hip.

4. Pupils
– Observe the **size, equality** and **reactivity to light**; they help in assessing the cause of unconsciousness and in determining the region of the brain that is impaired
– Reactivity to light is one of the most important signs distinguishing structural from metabolic causes of unconsciousness
– **Before drawing inference, make sure to rule out the possibility of having instilled miotic drops for glaucoma or mydriatic drops for fundoscopic examination.**

Begin assesment of pupils by looking for equality

Bilaterally equal in size

Reactive to light
⇓
Toxic-metabolic disorder is likely to be the cause of unconsciousness
|
– Pinpoint (< 1 mm) ⇒ effect of narcotic drug like morphine, heroin, etc.
– Small (1–2.5 mm) ⇒ metabolic encephalopathy (a diffuse failure of cerebral function that has many causes, including drugs)
– Large ⇒ cocaine, amphetamine, LSD, other sympathetic nervous system agonists

Fixed i.e. unreactive to light
⇓
Structural damage is likely to be tha cause of unconsciousness
|
– Pinpoint ⇒ a bleed in pons
– Small ⇒ damage to the sympathetic pathways in the hypothalamus
– Midposition/slightly dilated (4–6 mm) ⇒ stuctural damage in the midbrain
– Large:
- Severe anoxia and its sympathomimetic effects e.g. as seen after cardiac arrest
- Atropine-like agents, phenothiazine, tricyclic antidepressants

Unequal
and usually fixed
i.e. unreactive to light
⇓
Structural damage
e.g. unilateral large and fixed pupil ⇒ heniation of the temporal lobe causing compression of the CN III and midbrain

www.Ebook777.com

5. Examine the **skull** and **scalp** for injuries.
6. Examine the **ear, nose, mouth and throat**.
   - Blood/CSF in the ear/nose ⇒ # base of the skull
   - Otitis media ⇒ a possibility of brain abscess
   - Tongue injury ⇒ a seizure.
7. Test for **meningeal signs** for meningitis, subarachnoid bleeding.
8. **Skin**
   - Color, moisture, bleeding disorders, needle marks, and other lesions?
   - Cyanosis, jaundice, cherry red color of CO poisoning?
9. **Unusual odor** of alcohol, liver failure, uremia?
10. **Systemic examination.**

Causes of Unconsciousness

Although there are many causes of unconsciousness, most can be classified as either **toxic-metabolic**, or **structural**.

	Toxic / metabolic	*Structural*
Pathology	Arousal centers poisoned or critical substrates depleted	Lesion destroys or compresses brainstem arousal areas, either directly or secondary to more distant expanding mass lesion
Causes	Anoxia, hypothermia, hyperthermia, ischemia Hypoglycemia, hyperglycemia Hypothyroidism Meningitis, encephalitis Liver failure, uremia Alcohol, drugs	Intracranial bleed Cerebral infarct / embolus, tumor, abscess Brainstem infarct / tumor / bleed Cerebellar infarct / tumor / bleed / abscess

For immediate life-saving measures (Response to ABCDE), refer to chapter 56.

Free ebooks ==> www.Ebook777.com

12 SHOCK

DEFINITIONS

Shock

- Shock is defined as severe impairment of tissue perfusion leading to cellular anoxia and cell-death which is incompatible with life if not recognized and treated rapidly. In other words, it is a state of *inadequate* tissue perfusion
- It is essential to understand the real meaning of 'inadequate'. Blood flow may be grossly elevated in hypercatabolic states such as trauma, sepsis, and yet inadequate to meet the metabolic demands of the tissues
- In all forms of shock, cell damage is due to an ischemic hypoxic injury.

Septicemia

= the acute toxic state produced by microorganisms, their toxins or secondary mediators, in response to an invasive infection. It need not necessarily indicate the presence of bacteriemia.

Sepsis

= the systemic response to infection, manifested by 2 or more of the following as a result of infection.
– Heart rate > 90/min
– Respiratory rate > 20/min
– Temperature > 100.4° F or < 96.8° F
– WBC count > 12000/mm³ or < 4000/mm³.

Septic shock

= sepsis (as defined above) + e/o inadequate organ perfusion (organ dysfunction) – one/more of the following.
– Acute alteration of mental status
– Hypotension: Systolic BP < 90 mm Hg, or a drop of > 30 mm Hg
– Oliguria
– Hypoxemia: PO_2 < 9.5 kPa (overt pulmonary disease not being the cause of hypoxemia)
– Hyperlactatemia.

Systemic inflammatory response syndrome (SIRS)

= systemic inflammatory response to a variety of severe clinical insults. The presence of bacteriemia, or proof of a significant infected source is not required as part of the diagnosis.
2 or more of the criteria of sepsis (as defined above) in the setting of a known cause of endothelial inflammation such as infection / pancreatitis / vasculitis / ischemia / multiple trauma and tissue injury including that due to major surgery / hemorrhagic shock/immunemediated organ injury may qualify for SIRS.

www.Ebook777.com

Multiple organ dysfunction syndrome **(MODS)** *Syn.* Multiple organ failure **(MOF)**

= the presence of altered organ function in an acutely ill patient such that homeostasis cannot be maintained without intervention.

GENERAL REMARKS

1. The term 'shock' should not be considered synonymous with 'hypotension'. Cardiac output and O_2 delivery may be critically low, even though the BP remains normal. It is important to identify this state and intervene.
2. In the absence of early and complete resuscitation, shock can be a self-perpetuating process leading to cellular injury, organ dysfunction and death.
3. All patients with shock can be considered to have an *inadequate* cardiac output; not always low, but always *inadequate* to meet metabolic demands of the tissues. There are then only 2 causes for the inadequacy.
   a. Primary pump failure → cardiogenic/obstructive shock.
   b. Peripheral circulatory failure due to

Actual loss of circulatory volume i.e. true hypovolemia	OR	A loss of peripheral vascular resistance i.e. vasodilatation, leading to an apparent hypovolemia as seen in vasovagal attack, anaphylaxis, sympathetic paralysis, neurogenic shock, adrenal insufficiency and septic shock

   Detailed discussion on pathophysiology of various types of shock is beyond the scope of this book.
4. In low output shock (true hypovolemia), particularly of acute onset, clinical manifestations are clear-cut and easily recognized e.g. mental confusion, sweating, pallor, cold extremities, tachycardia, hypotension, oliguria. But in sepsis, some cases of trauma, and shock of slow onset, early clinical manifestations may not be clear-cut and easily recognized. Patients may be in a state of shock despite normal vital parameters.
   In sepsis, patients can be significantly shocked with cardiac output being 4–5 times normal.
5. Compensatory mechanisms (e.g. vasoconstriction) are a normal physiological response to low output shock and are essential for survival, helping to maintain blood flow and perfusion to vital organs. In the short-term these mechanisms/effects are protective, but ultimately → breakdown of cellular integrity. In other words, paradoxically, it is these very compensatory mechanisms themselves ultimately lead to organ damage.
6. Cellular dysfunction occurs in all cells, but susceptibility of different cells to hypoxia is variable.
   There is no doubt however, that the most sensitive organ is the brain. Brain cells cannot use anaerobic metabolism, and are therefore exquisitely sensitive to a lack of blood supply that brings them O_2. If circulation is completely arrested for 3 minutes, irreversible changes occur in the brain.
7. The early, catecholamine-induced vasoconstrictor response is not uniform throughout the body. There is redistribution of blood flow in favor of 'certain vital' organs notably the brain and the heart. Therefore, the main brunt of initial microvascular changes affects the skin, muscle, GIT. Kidney response varies according to rapidity of the onset of hypovolemia. The magnitude of reduction of blood flow in some of the 'non-vital' organs is not reflected in any of the routine clinical hemodynamic measurements and this is not appreciated.
8. *Irreversible shock*: Practically, this term should be avoided. Pertaining to hypovolemia, the usual reason why hypovolemia fails to respond to apparently adequate treatment is that the treatment has not been adequate! Nevertheless, theoretically, at some stage in its evolution, shock becomes irreversible due to the severity of vascular impairment and tissue damage. In medical practice, there are no indicators which declare the point at which shock state has achieved irreversibility. Recordings of BP and CVP indicate the severity of shock (vascular disturbance).
9. Resuscitation should not be delayed because of lack of a diagnosis; however, ultimate treatment success will depend largely on the detection and management of the cause of shock.
10. Typical clinical picture of shock is by no means always seen in all its details.

Free ebooks ==> www.Ebook777.com

Clinical indicators of shock in the descending order of sensitivity

1. Cerebral dysfunction.
2. The signs of circulatory disturbance.

All patients with shock, no matter how produced or how variable the details of the clinical picture may be, share the common primary factor of reduced tissue perfusion, and that the most sensitive manifestation of this factor is cerebral dysfunction. The recognition of cerebral dysfunction remains of paramount importance. However, a reduction in cerebral tissue perfusion is not the only possible cause of a disturbance of consciousness.
There are many causes of cerebral cortical dysfunction other than due to shock: Refer to chapter 11.

ASSESMENT of a case of shock is done by combination of history, physical examination, estimation of apparent/observed losses, investigations, and monitoring of certain parameters.
The most reliable clinical indices are the LOC, skin temperature, venous and capillary refill, and urine output.

CLINICAL FEATURES

1. Features of **cerebral dysfunction**
- The mildest features: Sleepiness, and a suggestion that the patient is withdrawing from intellectual contact with her/his environment; apparently inappropriate anxiety
- Later, restlessness and agitation
- Finally, delirium and a clouding of consciousness proceeding to coma.

2. Features of **circulatory dysfunction**

They vary in different types of shock, and also according to the stage in the evolution of shock state.

The points in general examination, which help to detect the circulatory dysfunction

- *Skin* color, temperature and turgor
- *The state of the veins on the dorsum of the feet in a patient lying supine in horizontal position*

  These veins in this position lie roughly at the same level as the atrium. Normally, they are visibly distended

  Flat/guttered veins could be due to dehydration/shock (non-cardiogenic shock with a lowered CVP)
- *Venous refilling time as examined in any convenient superficial vein* such as one the back of the hand.

  An appreciably increased refilling time suggests non-cardiogenic shock with a lowered CVP
- *Capillary circulation* } Monitored / 5–10 minutes in the initial stages
- *Pulse*
- *BP*
- *Respiration*
- *JVP.*

3. Features of the **underlying condition which produced the shocked state.**

4. **Urine output** reflects renal perfusion.

5. **Estimate the losses of fluid** in case of **hypovolemic shock**

Accurate quantification of losses is often difficult. However, the following *guidelines* can be useful.
- In burns: 4 ml / Kg / % TBSA burnt
- In c/o salt depletion (severe diarrhea, intestinal obstruction, high intestinal fistula): Lassitude and postural hypotension ⇒ a deficit of up to 4 L
- In c/o water depletion: Thirst ⇒ a deficit of ≥ 1.5 L
- Severe water deprivation may be associated with deficit as high as 10 liters.

Estimate the blood loss in case of **hemorrhagic shock**

A normal blood volume in adults = 80 ml/kg body weight.

Cold extremities below the knees ⇒ loss of ~ 30% of blood volume.

www.Ebook777.com

External blood loss

– Patient's history can be of some help
  - Approximate how many teaspoonfuls/tablespoonfuls/glassfuls of blood might have been lost?
  - How much area of the floor or cloth (such as bed sheet, towel etc.) was soaked with blood?

  1 f^2 area corresponds to 100 ml of blood. 1 m^2 area corresponds to 1 L of blood
– A handful of blood clot (equivalent to the size of a fist) represents about 500 ml.

Internal blood loss

– For each mass of soft tissue swelling equivalent to the size of a fist represents about 500 ml of blood loss
– Thorax or abdomen can hold up to 2–3 L of blood
– Long bone fractures also lead to significant blood loss.

Note

Provided a patient loses < 1/3rd of blood volume, can usually maintain blood pressure above 100 mm Hg.

A loss of > half of blood volume for more than a few minutes will lead to death.

BASIC (NON-INVASIVE) MONITORING AIDS TO THE CLINICAL ASSESSMENT OF SHOCK

– BP
– A Foley catheter is mandatory for assessing urine output
– CVP. Following the initial resuscitation, CVP estimation can help in deciding the requirement for further measures. It can also aid the identification of patients who may appear to be clinically normovolemic but remain volume-depleted (subcliinical hypovolemia)

  Normal values:
  - At the midaxillary level as the 0-refrence point: 5–8 cm of water; should never exceed 10
  - At the manubriosternal angle as the 0-reference point: 1–4 cm of water.
– ECG—will detect dysrhythmias and severe myocardial ischemia
– Pulse oxymetry—gives valuable information on the adequacy of arterial oxygenation and peripheral tissue perfusion

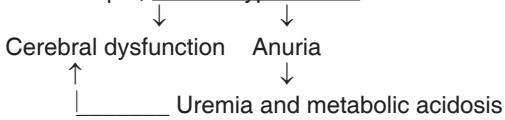
  Fallacies:
  - Readings are unreliable in hypovolemic or shocked patients, or if abnormal hemoglobins (including carboxyhemoglobin) are present.
  - Pulse oxymetry does not replace arterial blood gas analysis, as hypercapnea can occur with normal SaO_2.

In isolation, single measurements are not useful. They become useful only in light of the clinical impression. Repeated measurements over time, trends and patterns, together with the changes associated with therapeutic interventions, increase their usefulness.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. For making the diagnosis and determining the degree of shock, *do not rely on any one sign alone; look at as many as possible*.
2. Many of the conditions which can impair the level of consciousness can themselves be produced by shock. For example, <u>severe hypotension</u>



Free ebooks ==> www.Ebook777.com

Thus, even if an alternative cause of cerebral dysfunction is found, look for the signs of circulatory disturbance.

If any evidence of circulatory dysfunction is present, it is safer to assume that shock is the primary etiological factor, and to treat it vigorously.

In the absence of the most sensitive indicator of shock viz. cerebral dysfunction, one is driven to rely on circulatory signs to diagnose shock. Such a situation occurs when the patient is already unconscious for a perfectly well-understood and valid reason. A classic example: A patient who is brought in unconscious after a road traffic accident with an obvious head injury, and has a doubtfully lowered BP and quickened pulse rate.

3. Normal vital parameters do not rule out shock; i.e. the normal heart rate and BP do not necessarily reflect normal perfusion of the heart; and that is due to variable degree of vasoconstriction.

   10% reduction in blood volume occurring over a period of 1 hour may not produce alterations in vital parameters.
4. A patient with a lucid mental state, dry skin with normal temperature, normal venous and capillary refill, and a good urine output is unlikely to be significantly hypovolemic.
5. The clinical detection of blood loss and the resulting hemodynamic effects is crude. Recordings of vital parameters have limitations. Isolated readings are especially misleading; trends are of much greater value.
   – A rising pulse rate combined with a falling BP strongly suggests uncontrolled, often occult, blood loss.

   The response of an individual to blood loss can be remarkably variable. (a) With 25% loss of circulating volume, there may be no change in vital parameters, particularly in young patients who are capable of mounting intense vasoconstriction. (b) A patient may not be able to respond to hypovolemia by increasing the heart rate because of age, pre-existing cardiac disease or medications such as β-blockers. (c) If a patient's usual BP is higher, say 150/100, then a recorded BP of 100/70 may be hypotension for that patient. Unfortunately, this knowledge is rarely available in the early stages of resuscitation, and an index of suspicion, is therefore imperative.

   Conclusion: In the following situations blood loss may be misappreciated.
   - Hypothermia
   - The elderly, the pregnant, the athletes
   - Patients on concurrent drug therapy e.g. β-blockers, antihypertensives, antianginals
   - Patients with pacemaker.
6. Shock in a trauma patient initially must be assumed to be due to hypovolemia and is therefore treated with volume expansion.
7. The clinical signs of circulatory disturbance in trauma victims may be variable; the reason for this is not fully understood.
8. Diagnosis of acute pancreatitis must be considered in a patient who suddenly develops shock or anuria regardless of the presence or absence of abdominal pain.

   Acute pancreatitis should be suspected in the patient who:
   - Develops marked pain in abdomen, fever or unexplained shock following abdominal surgery
   - Presents with diabetic coma and shock
   - Has clinical features suggesting MI with abdominal distension.

STEPWISE ANALYSIS OF DATA

Step 1

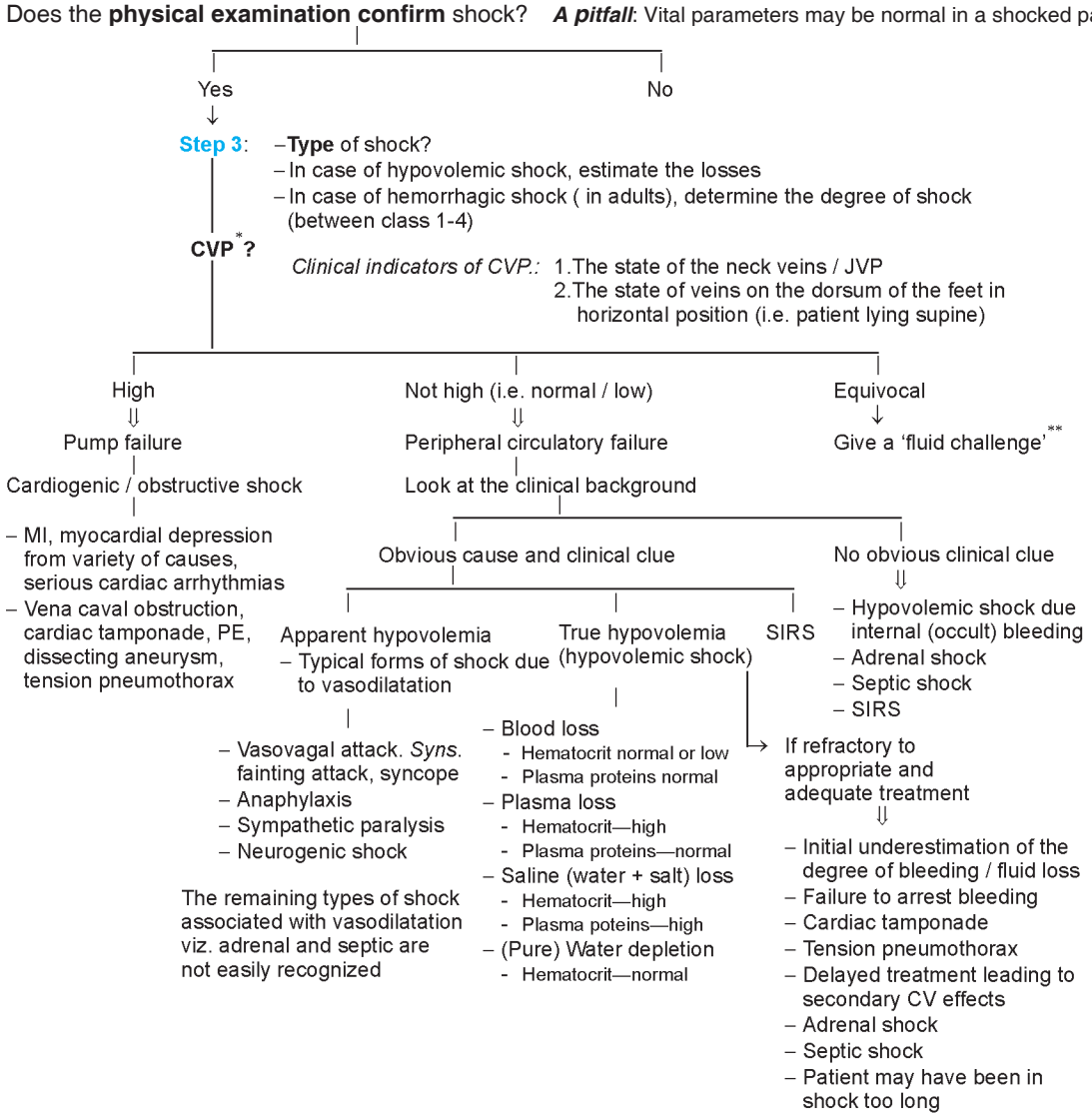
Is **shock suspected** on the basis of **cerebral dysfunction**?

Pitfalls: **1**. Exclude other causes of cerebral dysfunction.
2. If the patient is already unconscious for a perfectly well-understood and valid reason (e.g. severe head injury), then one is forced to rely on the signs of circulatory dysfunction.
3. A patient can be deceptively alert and euphoric despite being in shock.

www.Ebook777.com

Step 2

Does the **physical examination confirm** shock? ***A pitfall***: Vital parameters may be normal in a shocked patient.



* *CVP*

Pitfalls in interpreting actual measurement of CVP

1. Difficulty in establishing the base line of the right atrium.
2. One assumes the right atrial pressure accurately mirrors the left atrial pressure. It is really the left atrial pressure which relates to the systemic cardiac output which in turn may have been reduced by failure of the left ventricle or by reduced venous return. In a variety of cardiological conditions the 2 atrial pressures may be very different and to this extent the measurement of CVP may be misleading. In general however, at the extremes of measurement, the CVP gives an accurate indication of volume status.
   - –ve values suggest significant hypovolemia
   - High values suggest cardiogenic shock / fluid overload.

Between these extremes, isolated measurements become unreliable as an indicator of volume status. With minor-to-moderate hypovolemia, classic signs of shock may be absent including the CVP being within the normal range. These patients can be identified by observing the response of their CVP to a fluid challenge.

** '*Fluid challenge*'

Rapidly infuse a small amount (100–200 ml) of colloid over 2–3 minutes.

Measure BP and CVP again and note any change in values. Compare the values with those noted before giving the fluid challenge.

Examples of inferences:

– A decrease in CVP will unmask hypovolemia

– A rapid and sustained ↑in CVP suggests cardiogenic shock.

Free ebooks ==> www.Ebook777.com

Step 4

Estimate the fluid losses in case of hypovolemic shock.
Grade the hypovolemic / hemorrhagic shock.
Criteria of severe degree of hypovolemic shock
– Severe thirst
– Mental status—apathetic or comatose; little distress except thirst
– Capillary circulation—very sluggish
– Pulse—weak to imperceptible
– BP—decreased by 40% to non-recordable
– Oliguria.

Gradation of **hemorrhagic shock in adults**, **based on initial presentation** (× a 70 kg person)

Parameter	*Class 1*	*Class 2*	*Class 3*	*Class 4*
Blood loss (ml)	≤ 750	750–1500	1500–2000	≥ 2000
Blood loss (% blood volume)	≤ 15	15–30	30–40	≥ 40
Capillary refill	Normal	Delayed	Delayed	Delayed
Pulse rate	< 100	> 100	> 120	> 140
BP	Normal	Normal	↓	↓
Pulse pressure	Normal/↑	↓	↓	↓

Step 5

Organ damage and their extent?
Paradoxically, the circulatory compensatory mechanisms which maintain an adequate blood supply to the brain and keep the patient alive may themselves result in damage to important organs. The chief sensitive organs are heart, lungs, liver and kidneys. The change produced in these organs may, if sufficiently severe, become irreversible and themselves threaten life.

Heart
– Dysrhythmia
– Finally, the heart stops functioning in asystole or ventricular fibrillation.

Lungs
– Tachypnea—rapid (> 20 breaths/min) and shallow
– Cyanosis
– ↓ air entry
– Dullness and rales at the bases
– Plain X-ray findings—s/o pulmonary edema.

Acute respiratory distress syndrome (ARDS) = a life-threatening disorder resulting from a variety of causes including shock.
– Mode of onset—sudden, with the typical picture being established within 12–72 hours of the triggering event
– Initially, the physical signs in the chest are minimal despite obvious tachypnea and hypoxemia
– X-ray chest
  - Initially, may be normal or show an interstitial infiltrate
  - With progress of the condition, diffuse bilateral infiltrates (a 'white-out') appear.

Liver
– Acute liver failure. In less severe cases of shock, it may be mild: no more than a transient jaundice × perhaps 24–48 hours
– In severe cases, the patient goes rapidly downhill
– Cholangiolitis may → cholestatic jaundice

www.Ebook777.com

– ↑Serum transaminase levels ⇒ a major hepatocellular ischemic-hypoxic injury—it is more commonly observed in severe cardiogenic shock and hepatic venous congestion
– The markers of liver damage—serum transaminase level and PT. Irrespective of the etiology of shock, the elevated levels of these markers (i.e. ↑ serum transaminase levels and prolonged PT) ⇒ severe liver damage ⇒ a very significant insult ⇒ a poor prognosis
– If shock can be cured, the capacity of the liver to regenerate is great.

Kidneys

The excretory function is always disturbed in shock → ARF.

GIT

– The stomach and duodenum are particularly vulnerable. Acute ulcers ('stress ulcers') may occur which may perforate or bleed
– Acute ischemic insult to the intestines → failure of gut-barrier → transmigration of microorganisms and toxins into portal circulation.

Brain

– During the compensated phase of shock, relatively mild cerebral ischemia is associated with changes in the state of consciousness—apparently inappropriate anxiety
  Progressive hypoperfusion and hypoxia → restlessness, confusion, stupor and coma.
– BP falling to below 60 mm Hg → serious ischemic damage to the brain which can amount to actual infarction in the 'boundary zones' of the cerebral cortex and cerebellum.

Step 6

Any overlap and interplay?

Examples

– Severe noncardiogenic shock may result in cardiac damage and so an element of cardiogenic shock will be superimposed
– Vasodilatation in anaphylaxis is accompanied by saline or plasma loss from the circulation through the histamine-affected capillaries
– Hypovolemic shock may mask the characteristic features of septic shock
– Septic shock may overlap a pre-existing hypovolemic shock basically caused by burns
– General anesthesia may greatly enhance the effect of any pre-existing or immediate hypovolemia. It is most important that hypovolemic shock should be completely reversed before a patient is anesthetized for an urgent operation, and that blood loss during anesthesia should be rapidly replaced.

DIFFERENTIAL DIAGNOSIS

Cardiac tamponade

The acute form of cardiac tamponade is a surgical emergency.

Causes of acute form:

– Traumatic bleeding usually from a parasternal stab
– Rupture of a dissecting aortic aneurysm into the pericardium.

Clinical features

– Neck veins distended
  ↑JVP. ↑CVP.
– Irregular thready pulse.
  Pulsus paradoxus i.e. peripheral pulse becomes weaker during inspiration
– Profound hypotension
– ↑in the area of precordial dullness
– Heart sounds—muffled.

X-ray : Widening of the heart shadow, especially in the region of the cardiophrenic angles.

Free ebooks ==> www.Ebook777.com

2-D echocardiography can be useful both in detecting the hemopericardium and in determination of overall pump function.

The diagnosis is clinched by inserting a needle into the pericardial cavity via the 4th left IC space, 3–4 cm from the midline, and aspirating blood.

Pulmonary embolism

- Sudden SOB
- A feeling of impending doom
- Retrosternal oppressive pain suggests massive occlusion
- Pleuritic pain, cough, hemoptysis suggest pulmonary infarction
- Patient looks anxious
- The diagnosis may be aided by a predisposing clinical setting such as: Postpartum period, postoperative period, clinical settings predisposing to develop DVT (clinical evidence of DVT usually absent as the thrombus is floating), chronic lung disease, CHF
- In the absence of such clinical features, the diagnosis should be considered in any patient with acute cardiogenic shock in whom the cardiological evidence suggests a disproportionate failure of the right side of the heart compared with the left.

Such evidence includes:

- A tapping or impalpable apex beat
- A right ventricular thrust just to the right of the sternum
- An increase in the area of cardiac dullness to the right of the sternum.

X-ray : Prominent pulmonary arterial knuckle and reduced vascular markings in one or both lungs.

Vasovagal attack *Syns.* fainting attack, syncope.

- Rapid loss of consciousness within a few seconds of the onset of the disturbance → fall to the ground
- Consciousness is recovered within a few seconds / minutes
- Face looks pale
- Bradycardia, often to the tune of ~ 40/min.

Causes: Severe sharp pain of sudden onset that was often not expected, a sudden unexpected exposure to unpleasant sight/smell/sound, a sudden emotional disturbance, prolonged standing in attention.

Anaphylactic shock

- Occurs within seconds to minutes of exposure to an allergen (known/suspected/otherwise)
- Itching. Widespread urticaria
- Nausea, vomiting, diarrhea
- Rhinitis and edematous swelling of mucous membranes
- Hoarseness of voice, stridor, wheezing
- Tachycardia, hypotension.

The circulatory disturbance may be so intense that death may occur within few minutes, but immediate treatment with injection adrenaline or injection hydrocortisone is usually effective.

Sympathetic paralysis

Sympathetic paralysis, by interfering with normal vasoconstrictor tone in the peripheral circulation, renders the patient liable to recurrent attacks of fainting whenever any strain is placed on the peripheral circulation, for example by adopting the erect posture suddenly.

The clinical features are identical with those of a vasovagal attack.

Causes: Surgical sympathectomy, ganglion-blocking and adrenolytic drugs, general anesthesia.

Neurogenic shock

This is caused by major brain or spinal injury and may be associated with neurogenic pulmonary edema.

- Skin—dry and warm
- Bradycardia.

www.Ebook777.com

Hypovolemic shock

In cases such as major trauma or burns, severe (gastro) enteritis, frank acute intestinal obstruction the presence of volume loss is obvious.

In other situations like concealed blood loss, dehydration, diabetic ketoacidosis, etc; both assessment and diagnosis may be difficult.

Clinical features

- Skin—pale, cold and clammy
- The state of veins on the dorsum of the feet in a patient lying supine in horizontal position—flat or empty
- Venous refilling time—appreciably increased
- Capillary circulation—poor
- Pulse—rapid and thready
- BP - ↓. Recordings of BP indicate the severity of the vascular disturbance
- Respiration—rapid and shallow
- JVP - ↓.

Hypovolemic shock due to **blood loss**

Acute (severe) blood loss has the most dramatic and dangerous effects on the circulation.

It is one of the 'most urgent emergencies' in surgery.

Causes:

- Trauma
- Disease of the blood vessels
- Rupture of a diseased vascular organ or tissue.

When the blood loss is external the diagnosis is obvious; when internal, the difficulty in diagnosis can be great. If there are circumstances which lead to suspect blood loss, the important confirmatory evidence is obtained from the absence of physical signs of extracellular fluid depletion, and from examination of the blood and urine.

The clinical detection of blood loss and the resulting hemodynamic effects is crude. There are situations, where blood loss may be misappreciated.

In acute loss of blood, since the loss from the circulation is whole blood and there has been no time for a compensatory shift of fluid to occur from the ECF into the vascular compartment, there is no change in the composition of the residual blood. A Hb and hematocrit percentage (PCV) values are most likely to be normal. However, if the bleeding has been fairly slow and the clinical course correspondingly longer, some compensation may have occurred whereupon there might be a tendency for low values to be obtained.

Although renal function may be impaired, in really acute cases the impairment will not have lasted long enough to produce a rise in blood urea concentration. For the same reason, the initial sample of urine could be normal. In the later stages of severe shock, the blood urea concentration will rise, urine output will be low (oliguria), and urine Na and Cl concentrations will be low.

Type (cause) of ARF → investigation parameters ↓	Prerenal (hypovolemic shock)	Renal damage
Urine volume	Low	None
Plasma BUN: Serum creatinine ratio	> 20	< 20
The rate of rise of BUN	< 50 mg%/24 hours	> 50 mg%/24 hours
Specific gravity of urine	> 1.020	'Fixed'
Osmolality of urine	> 500	< 400
Urine Na	< 20 mEq/l	> 40 mEq/l
Fractions excretion of Na %	< 1	> 1–2

Effects of blood loss vary with—(a) the duration and severity of blood loss, (b) host factors like age, pre-existing anemia, IHD, etc. and (c) the speed and adequacy of resuscitation .

Free ebooks ==> www.Ebook777.com

The response of an individual to blood loss can be remarkably variable. With 25% loss of circulating volume, there may be no change in vital parameters, particularly in young patients who are capable of mounting intense vasoconstriction.

Pitfalls

- Hemodynamic condition of a shocked patient is never static. It keeps changing from moment to moment. It may get better or worse or initially better and then catastrophically worse
- Shock usually develops slowly over hours, although it can develop rapidly
- Symptoms may be out of proportion to the volume of blood lost. A small loss may occasionally cause severe shock and vice versa
- A single sign may not be reliable, so look at many
- BP recordings may be unreliable. For example, the BP of a child/young adult may not fall at all initially, until it finally falls catastrophically, when the patient has lost a third or more of blood volume
- **A rising pulse rate is an early and more reliable sign of continuing blood loss. But even pulse may not rise until late, particularly if the patient is old.**

In ***trauma*** victims, there is additional insult of tissue injury which alters neuroendocrine and metabolic response. Patients with upper GI bleeding may exhibit quite different clinical signs from patients suffering from trauma, even when the degree of bleeding is similar. In case of mild-moderate blood loss, at the time of presentation, patient may have tachycardia / bradycardia; normal BP / hypertension / hypotension. The reasons for the variability in clinical signs after trauma are currently only partly understood.

Hypovolemic shock due to **plasma loss**

Plasma loss can usually be diagnosed or strongly suspected from the clinical setting.

Causes:

- Burns. The volume and the rate of plasma loss (consequently, the severity of shock) are roughly proportional to the extent of burnt area rather than to the depth of burns
- Inflammation of large serous surfaces e.g. peritonitis
- Strangulation of a tissue / organ.

Blood picture:

- Marked rises in Hb and PCV
- Since renal excretion may have been impaired for a longer period than in acute blood loss, the blood urea is likely to be (moderately) raised.

The urinary changes are identical with those in acute blood loss.

Hypovolemic shock due to **ECF** (saline i.e. water+salt) **depletion**

ECF depletion occurs due to the loss of water and Na ions from the body.

The routes of such losses

- Most commonly the alimentary tract: Severe vomiting, diarrhea, intestinal obstruction, high output intestinal fistula, mechanical bowel preparation, etc.
- Sweat in hot climate
- Evaporation during an operation—may be easily underestimated. > 500 ml may be lost by evaporation during a 1 hour laparotomy or a similar procedure where large surface area is exposed to the atmosphere.
- Artificial ventilation leading to excessive water loss from respiration.
- Rarely, excessive loss of Na may occur in urine—hyperaldosteronism—an adrenal disorder.

Losses from the alimentary tract can be equated, in terms of their effects on the ECF as a whole, with the loss of a similar volume of 'normal' (isotonic) saline.

Loss of isotonic saline → a reduction in the size of the extracellular space and a proportionate reduction in plasma volume. Since the ECF volume in an adult is 15 liters (i.e. ~ 5 × the plasma volume), 5 liters of saline needs to be lost from the ECF before the plasma volume shrinks by as much as 1 liter. It follows that shock is slower to develop, and less severe except in the later stages.

The physical signs result from a typical feature of saline loss viz. a reduction in the pressure of the interstitial fluid (i.e. extravascular component of the ECF).

www.Ebook777.com

Clinical features
- Eyeballs: A reduction in the tension
- Skin: Decreased turgor, and feels dry
- In severe cases: Hippocratic facies.

Blood picture:
- Hematocrit ↑, however, hemoconcentration tends to be less severe than in plasma loss
- Blood urea concentration tends to be higher.

Urine picture:
- Oliguria
- High specific gravity
- Na^+ and Cl^- are virtually absent.

Primary deficit of water generally results from reduced intake rather than increased loss.
Causes:
- Inability to acquire adequate volume of water due to exhaustion or due to disturbance of consciousness (intrinsic brain pathology, patient under the effect of sedative, etc.)
- Drinking having been forbidden e.g. following an upper GI surgery
- Diabetes insipidus → water loss.

Feature	*Water depletion*	*Salt depletion*
Cause	Deficient intake	Excessive loss (especially of intestinal secretions)
Dehydration	Cellular	Extracellular
Thirst	+++	Absent
Vomiting	Absent	May be +++
Urine output	Scanty	Normal (may be even increased until late)
Lassitude	+	+++
BP	Normal until late	↓ +++
Hemoconcentration	Late and slight	Early and severe
Blood urea	↑ +	↑ +++
Serum Na^+	Slight ↑	↓ ++
Serum Cl^-	Slight ↑	↓ +++
Urine Na^+	↓ +++	↓ +++
Urine Cl^-	↓	Absent
Plasma volume	Normal until late	↓ +++
Cause of death	Uncertain	Peripheral circulatory failure

An important note of practical importance, on hypovolemic shock
The usual reason why hypovolemia fails to respond to apparently adequate treatment is that the treatment has not been adequate. Failing to grasp this point, a valuable time may be wasted in treating the remaining forms of shock (adrenal / septic); especially dangerous consequence of this is that such treatment may produce temporary improvement in the state of shock, and result in a quite unwarranted complacency, which may be followed by unexpected catastrophic deterioration.

Adrenal shock (Adrenocorticosteroid insufficiency) may be suspected on clinical grounds.
- The patient may known to have received corticosteroid treatment during the previous year or two, in which case her/his adrenal cortical response to any stress may be inadequate
- Alternatively, a picture of saline depletion without any obvious route of saline loss may suggest that Na^+ has been lost via the kidneys. Such patients may have nausea, oral mucosal pigmentation.

Free ebooks ==> www.Ebook777.com

Septic shock

– The diagnosis should always be considered in shocked patients with evidence of overt or potential infection (peritonitis, RTI, UTI, instrumentation of the urinary tract, operations upon or foreign bodies in the biliary passages, burns)
– Septic shock may complicate relatively trivial surgical procedures especially in the GIT and UT in the presence of infection
– Commonly occurs due to infections with Gram –ve bacteria but can be due to Gram +ve bacteria or fungi
– Fever with rigors.

The clinical features of sepsis are usefully considered in 2 groups—early (= hyperdynamic = warm stage) and late (= hypodynamic = cold stage). Depending on the timing of diagnosis, aggressiveness of resuscitation, and use of vasoconstrictor agents, a given patient may, at different stages exhibit differing features.

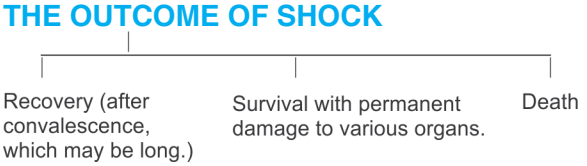
Clinical features of sepsis	*Early*	*Late*
	– Apparently inappropriate anxiety, f/b restlessness, slight confusion	Restlessness, confusion, stupor, coma i.e. progressively deteriorating LOC
	– **Skin**—suffused and dry	Skin—pale, cold and clammy
	– Tachypnea	Tachypnea
	– Tachycardia. Pulse may be bounding.	Tachycardia. Pulse—thready
	– Systolic **BP**—normal or slightly ↓	Systolic BP < 90 mm Hg
	– **CVP** maintained	CVP ↓
	– Oliguria	Oliguria

Making a clinical diagnosis in the early phase of septic shock is difficult in the absence of invasive monitoring and without measurement of blood gases or/and lactate (arterial blood)**. Such a patient may look remarkably well owing to the pink well-perfused extremities, and a normal BP and CVP. A high index of suspicion is required to make a diagnosis.**

In the postoperative patient, it is all too easy for inexperienced personnel to treat restlessness with sedation, rather than appropriate resuscitative measures.

The combination of septicemia (with its profound and widespread vasodilatation in the skin, muscles and viscera,) with hypovolemia is quite common, and particularly resistant to treatment.

THE OUTCOME OF SHOCK



Factors favoring recovery

1. Early treatment of the initiating cause and the hypovolemia
2. Young age (youth)
3. Good general health.

Factors favoring progression of shock

1. Delay in treatment
2. Failure to remove the initiating cause
3. Old age
4. Poor general health
5. Pre-existing systemic disease
6. Onset of complications especially infection and organ damage.

www.Ebook777.com

13 INFECTIONS / INFESTATIONS

Classification of infections/infestation from practical standpoint. This distinction is important for various reasons, e.g.

- Varying spectrum of infectious disease within the 2 settings
- Frequent presence of immune deficiency in hospital patients
- Significantly higher prevalence of infections by antibiotic-resistant organisms in hospital acquired infections

Community-acquired

Primarily medical

- Meningitis
- RT infections
- Viral hepatitis
- AIDS
- TB
- Typhoid, etc.

Primarily surgical

Urgent surgical treatment often though not always indicated

- Abscesses
- Acute appendicitis
- Acute cholecystitis
- Acute peritonitis
- Cellulitis / fasciitis(non-necrotic / necrotic), etc.

Hospital-acquired

(Common) postopertive infections

At the site of invasion

- Wound infection
- Dermatitis at the site of sticking tape, ostomies (ileostomy, colostomy)
- Injection / vascular line / catheter / tube-related infection e.g. injection abscess, (septic) thrombophlebitis, UTI, sinusitis, meningitis following spinal anesthesia, etc.

Systemic infections to which postoperative patients may be more prone due to various reasons

- Pneumonia
- UTI
- Enteritis
- Tetanus

- Infection of implanted prosthetic material: Infection at the site of insertion of vascular grafts / mesh / valves / pacemaker / filters, etc.
- Infection related to specific surgical procedures

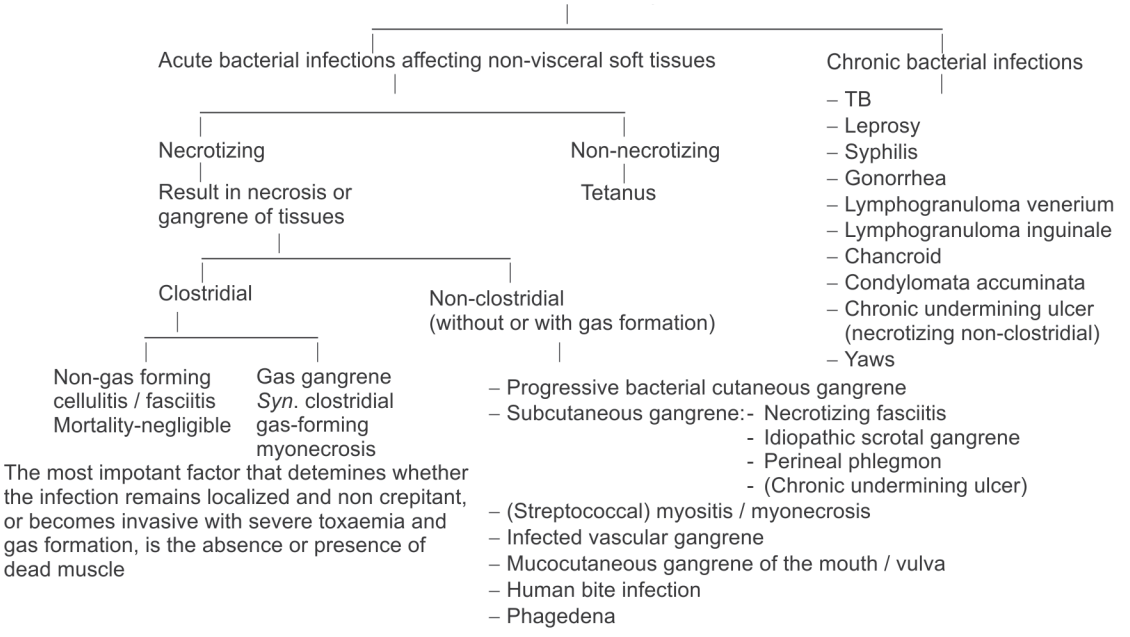
Examples:
- Head and neck : Sinustis, meningitis, ventriculitis, etc.
- Chest: Mediastinitis, empyema, etc. following cardiac surgery
- Abdomen: Anastomotic leaks, intra-abdominal abscess following GI surgery

Infections in critically ill patients

- In addition to common postoperative infections,
  - Catheter-related sepsis
  - Fungal infections

Free ebooks ==> www.Ebook777.com

SPECIFIC BACTERIAL INFECTIONS OF SURGICAL IMPORTANCE



Pathology of infective non-clostridial gangrene in brief
Organisms:
– The most common—anaerobic streptococci
– Other—*Escherichia coli*, bacteroides (*F. fusiformis*), etc.
In some well-documented cases**, a causative organism cannot be identified**
In some cases, **particularly in diabetic patients, the morbid process arises spontaneously**

ARRIVING AT THE DIAGNOSIS OF ACUTE SPECIFIC BACTERIAL INFECTIONS AFFECTING NON-VISCERAL SOFT TISSUES

Factors to be considered:
– Site
– Precipitating factor
– Predisposing factors
– Mode of onset, and progress
– Degree of toxemia.

Initially analyze each of the above factors in isolation. Then consider the inference of all collectively to arrive at a definitive clinical impression.

Site

– Parts of the body with bulky muscles such as thigh, buttock } Think of clostridial infection, provided other features agree
– Part of the body which can easily come in contact with feces

– Surgical drain site } Progressive bacterial cutaneous gangrene
Mass closure of a laparotomy wound with deep tension sutures

– Extremities / lower trunk / external genitals, perineum—necrotizing fasciitis
– The foot—Infected vascular gangrene.

www.Ebook777.com

Precipitating factor

- Wound, particularly large with devitalization of tissue, or deep puncture wound—clostridial infection.
- Trauma / operation / viral infection } Non-clostridial infection
- Spontaneous i.e. apparently without any precipitating event
- I and D of abscess, particularly in patients with DM and severe atherosclerosis—progressive bacterial cutaneous gangrene.

Predisposing factors

- Debility } Non-clostridial infection
- DM
- Protein energy malnutrition in children—mucocutaneous gangrene of the mouth / vulva.

Mode of onset, and progress

- Acute: Clostridial gas gangrene, necrotizing fasciitis
- Relatively gradual progress: Infected vascular gangrene, mucocutaneous gangrene of the mouth/vulva.

Degree of toxemia

Minimal in non-gas-forming clostridial cellulitis / fasciitis.

DIFFERENTIAL DIAGNOSIS

Non-gas-forming clostridial cellulitis/fasciitis

The disease is mild and apart from fever, there is minimal toxemia.

- Pain—not severe
- Wound—edematous and erythematous
- Brownish discoloration
- Tenderness—not severe
- Crepitus—absent.

Clostridial gas gangrene

Precipitating / predisposing factors

- Large wounds with necrotic tissue e.g. battle casualties, severe comminuted compound fractures sustained in road traffic or railway injuries, agricultural tractor injuries, etc.
- Foreign body implanted in the depth of a punctured wound e.g. shrapnel, etc.
- Following AK amputations / criminal abortions/intestinal, biliary operations
- Gas gangrene has been reported after intragluteal IM injection—the region where the skin is often contaminated by clostridial spores from the patient's feces.

Incubation period: 1 day–4 weeks. Usually < 3 days. The duration of incubation period carries inverse relationship with the severity of the illness and mortality.

Mode of onset—sudden. *Progress*—rapid.

Symptoms and signs

- Severe pain in the region of the wound
- Rapid development of toxemia with drowsiness, fever
- Tachycardia
- The affected part—edematous, tense, and extremely tender
- Discoloration occurs through a series of changes from intense white → erythematous → frank greenish-black



Fig 13-1 Clostridial gas gangrene

Free ebooks ==> www.Ebook777.com

- Discharge—serous / serosanguineous
  - Amount—variable
  - Initially odorless, subsequently becomes sweet or foul-smelling
- Ecchymosis, bullae
- Crepitus+

Progressive bacterial cutaneous gangrene

Precipitating events

- Minor trauma
- A postoperative consequence of a surgical drain, or mass closure of a laparotomy wound with deep tension sutures
- I and D of abscess, particularly in patients with DM and severe atherosclerosis.

Incubation period : 1–2 weeks.

Physical findings

- Skin surrounding the wound—edematous, red, and very tender
- As the infection extends outwards, the central area turns to eschar that ulcerates
- A thin foul-smelling discharge simulating dishwater.

At this stage, the patient becomes systemically toxic.

At operation, the infected necrosis is found to be limited to the skin and subcutaneous fat, but the fascia and underlying muscle are spared except for in advanced cases.

Microorganisms

- In classic cases—microaerophilic streptococci and aerobic staphylococci.
- Other—Bacteroides and a variety of Gram –ve aerobic bacteria can be involved.

Note

Ulcerative form of **cutaneous amoebiasis** leading to extensive phagedenic ulceration **may be mistaken** for progressive bacterial cutaneous gangrene.







Figs 13-2A to C Progressive bacterial cutaneous gangrene: **(A)** At the time of presentation i.e. on D1 consultation, **(B)** 4 days later, **(C)** Postoperative week 3

www.Ebook777.com

Necrotizing fasciitis (*syn*. hemolytic streptococcal gangrene)

Precipitating events are often not particular; could be surgery, or trauma—obvious or minor/unnoticed.

Mode of onset—sudden. *Progress*—rapid.

Constitutional symptoms are always present with fever and tachycardia. Patient may present with shock.

Symptoms and signs

- The affected part is initially very painful but later becomes numb owing to the involvement of sensory nerve fibers
- Inflammatory edema sometimes bullous
- Reddish discoloration
- Necrosis and eventual sloughing of the skin.

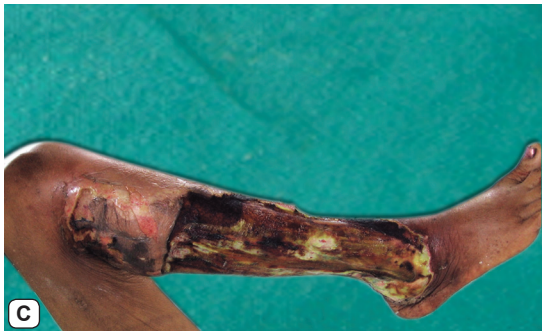
Basically, necrosis does not involve the muscle layer, and skin involvement is secondary to thrombosis of the perforating vessels coursing through the infected necrotic area.

Microorganisms

- Hemolytic streptococci and less commonly, hemolytic staphylococci
- Other—coliforms, bacteroides, diphtheroids, *Pseudomonas*.







Figs 13-3A to C Necrotizing fasciitis: **(A)** and **(B)** At the time of presentation i.e. on D1 consultation, **(C)** 5 days later

Idiopathic gangrene of the scrotum (*Syn*. Fournier's gangrene)

= Non-clostridial subcutaneous gangrene affecting the scrotum.

Perineal phlegmon

= Non-clostridial subcutaneous gangrene affecting the perineum, often with that of the scrotum and penis.

Chronic undermining ulcer: Refer to chapter 42.

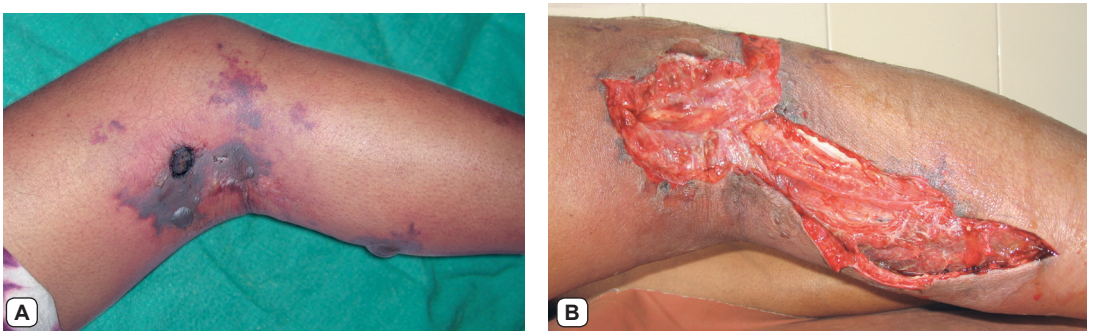
Streptococcal myositis / myonecrosis

- Cyanotic discoloration of the skin
- Blebs containing foul-smelling fluid (teeming with Gram +ve cocci)



Fig 13-4 Idiopathic gangrene of the scrotum

Free ebooks ==> www.Ebook777.com



Figs 13-5A and B Streptococcal myositis-right lower limb: **(A)** Clinical appearance, **(B)** Surgical debridement, including fasciotomy (and fasciectomy)



Fig 13-6 Infected vascular gangrene

– Islands of frankly gangrenous skin.

Myonecrosis develops only occasionally.

Prognosis is surprisingly good if infection is managed before the onset of myonecrosis.

Infected vascular gangrene

– Initial progress—relatively gradual
– The severity of toxemia is variable
– The affected part (usually the foot)—swollen, black and foul-smelling
– Crepitus+.

X-rays show extensive gas formation in the tissues.

Microorganisms: Mixed infection with fecal organisms, most commonly *Bacteroides fragilis* and *Peptostreptococcus*.

Clinical features	*Necrotizing fasciitis / myositis*	***v/s***	*Infected vascular gangrene*
– The part affected	May affect the foot. The most commonly affected areas however, are different than the foot		Usually the foot
– Mode of onset	Sudden		Insidious
– Progress	Rapid		Initial progress is relatively gradual
– Color of slough	May be black		Always black (being vascular gangrene)
– Gas formation	±		Extensive gas formation in the tissues

www.Ebook777.com



Fig 13-7 Mucocutaneous gangrene

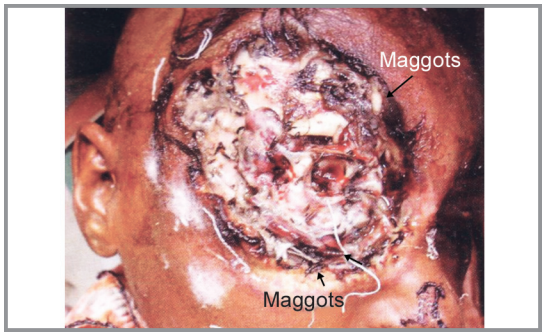


Fig 13-8 Phagedena

Clostridial myositis	***v/s***	*Non-clostridial (streptococcal) myositis*
No skin reaction		Pronounced skin reaction – Cyanotic discoloration – Blebs containing foul-smelling fluid teeming with Gram +ve cocci

Mucocutaneous gangrene of the mouth/vulva

Predisposing factor: Protein energy malnutrition in children.

– Usually preceded by an infectious illness such as measles
– Slow but relentless necrosis of the perioral or vulval tissues
– In case of mucocutaneous gangrene of the mouth, death may result from inhalation pneumonia.

Microorganisms: Mixed infection; the protagonists are either anaerobic streptococci or members of the bacteroides species.

Human bite infection

– Can be virulent and cause marked tissue destruction.

Microorganisms: *Bacteroides melaninogenicus*, *Fusobacterium species*; anaerobic cocci and spirochetes.

Phagedena = Extensive infective destruction of tissue.

It mimics carcinoma.

Points which differentiate it from carcinoma

1. It is primarily of infective origin.
2. There is no proliferation of tissue.

Tetanus

It is an exogenous infection in the vast majority.

Rare instances of endogenous infection are documented after septic abortion and operations on the GI tract.

Portals of entry

– The most common—the umbilical stump in neonates, following the application of dung—an unhygienic practice in many developing countries all over the world
– Wound, which is always penetrating in nature, commonly on the lower limbs
– Other portals/sources—skin lesions especially leg ulcers, piercing of ear lobes, tattooing, burns, parenteral injections including vaccination, nasal foreign bodies, ear infections
– In some cases, the portal of entry remains unknown.

Predictors of higher mortality

– Age: Mortality is higher at the extremes of ages
– Incubation period: It has an inverse relationship with mortality; i.e. shorter the incubation period higher the mortality
– Severity of disease: It has direct relationship with mortality.

Free ebooks ==> www.Ebook777.com

Neonatal tetanus

– Failure to suckle on the 3rd day
– Spasm of the facial muscles and masseter (lockjaw)
– Generalized clonic spasm with flexion of the arms, clenched fists, extension of the lower limbs and plantar flexion of the toes.

Tetanus in children and adults

– The majority of patients have not been previously immunized
– Incubation period—4 to 10 days
– The full-blown picture is usually reached by the 3rd day from the time the 1st symptom is experienced.

Early symptoms

– Stiffness, twitching and cramps limited to the same spinal segment as the area of infection (-local tetanus)
– Muscle-pains, headache, irritability, restlessness, constipation, sweating.



Fig 13-9 Sardonic smile

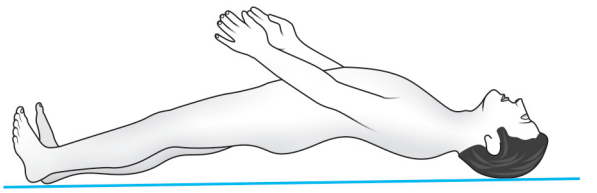


Fig 13-10 Opisthotonus

Subsequent manifestations

– Spasm of masseter (→ lockjaw), facial muscles (→ 'rhesus sardonicus'), and muscles of deglutition (→ dysphagia)
– Generalized clonic convulsions that may be triggered by mild external stimuli (sound, bright light, movement of personnel, etc.)
– Opisthotonus—the classical posture assumed by a patient suffering from tetanus, the extensor muscles being more powerful than flexors of the trunk.

Classification of tetanus

– Mild = No dysphagia. No respiratory distress
– Moderate = Presence of dysphagia and respiratory distress
– Severe = Gross spasticity and major spasms.

The feature that differentiates tetanus from strychnine poisoning:
In tetanus, the muscles do not relax between convulsive attacks.

NOTES ON SELECTED CHRONIC BACTERIAL INFECTIONS

TB

TB is a disease of malnutrition and overcrowding.
Other predisposing factors—poor general health, chronic disease, silicosis, and DM.
90% of all tuberculous infections involve the lungs, but the infection may affect practically any organ or tissue in the body.
TB is basically a generalized disease. Various lesions are localized manifestations of the basically generalized disease. Implications of this pathological fact: 1. Clinical implication: A patient suffering from TB of any organ / tissue will *usually though not necessarily always*, *experience* general non-specific symptoms viz. loss of appetite, loss of weight and low-grade fever in the evening with night sweats, in addition to symptoms specific to the organ / tissue affected. 2. Therapeutic implication: ATT must be prescribed to every case of 'surgical TB' even if the diseased organ / tissue is surgically removed.
Tuberculous lesions assume one of the *two forms*

1. Proliferative → typical tubercle follicles in solid organs / tissues.
2. Exudative—typically encountered in infection of the serous cavities (e.g. tubercuolus pleurisy/peritonitis/pericarditis) and epithelial surfaces (e.g. sterile pyuria in renal TB).

www.Ebook777.com

Objectives of surgery for TB in general

1. To obtain the diagnosis (biopsy).
2. To remove organ / tissue destroyed by the disease.
3. To treat a complication e.g. obstruction, perforation.

The spectrum of 'surgical TB'

A. Tuberculous **lymphadenitis**

- Acute—in children under 5 years of age. May be mistaken for nonspecific acute septic lymphadenitis
- Caseating—the most common type
- Hyperplastic—easily mistaken for lymphoma
- Atrophic—occurs in elderly.



Fig 13-11 A typical case of caseating lymphadenitis

B. **Abdominal** TB

- GIT, lymph nodes, solid organs (liver, spleen, pancreas, etc.), peritoneum, rarely uterine tubes and ovaries
- More common in women than in men.

1. Gastric TB
   - Generally affects younger patients
   - Usually of hyperplastic type.

   Modes of presentation
   - A long h/o chronic pain in abdomen simulating peptic ulcer
   - Gastric outlet obstruction. The case may be mistaken for CA stomach even on operation table
   - Rarely, systemic toxemia → pain, vomiting, diarrhea, fever, etc. → may lead to shock.
2. Duodenal TB
   - Generally affects younger patients
   - Ulcerative or hyperplastic
   - Generally → duodenal obstruction.

   Duodenum and stomach proximal to obstruction are considerably dilated. Such cases may have to be differentiated from annular pancreas and idiopathic chronic duodenal ileus.
3. Small intestinal TB (*syn*. ulcerative TB of the small intestine)
   - The earliest stage = mucosal ulceration → vague colicky pain in abdomen, chronic diarrhea/ recurrent attacks of diarrhea not responding to routine primary-level treatment, and fetid odor to stools
   - Subsequently, formation of stricture → subacute incomplete, or acute complete intestinal obstruction
   - Completely obstructing dense stricture may be complicated by perforation occurring just proximal to the stricture.
4. Ileocecal TB (*syn*. hyperplastic TB of the small intestine)

   → subacute/chronic intestinal obstruction ± a lump in the abdomen (in RLQ).

   Mechanisms of intestinal obstruction

   a. Stricture.

   b. Kinking of an intestinal loop due to
      - Adhesion to a caseating mesenteric lymph node

      OR • Aadhesion caused by plastic peritonitis.
5. Large intestinal TB
   - Ulcerative or hyperplastic

     → diarrhea or manifestations of (chronic) large intestinal obstruction
   - Rarely the entire colon may be affected, giving rise to thickening and fixity of the entire colon.
6. Rectal TB

   Modes of presentation
   - Ulceration / annular stricture / growth. These cases will be mistaken for CA rectum
   - External anal fistulae or internal fistulae.

Free ebooks ==> www.Ebook777.com

7. Tuberculous peritonitis
   - Tuberculous non-loculated ascites → abdominal distension
   - Tuberculous loculated ascites → lump in abdomen which may be mistaken for pancreatic pseudocyst/mesenteric cyst/ovarian cyst/giant hydronephrosis
   - Caseous peritonitis—results from rupture of caseous tuberculous mesenteric lymph nodes into the peritoneal cavity.
     Adhesions may → intestinal obstruction.
   - Plastic peritonitis ⇒ healed stage of tuberculous peritonitis. Adhesions may lead to plastering of wide areas of intestine and mesentery together. Multiple adhesions surprisingly produce very little intestinal adhesions. The disease is quiescent.
8. Tuberculous mesenteric lymphadenitis may → palpable lump/s in abdomen.
   Complications by virtue of:
   - Mass effect may → duodenal obstruction / jaundice
   - Adhesions—kink → small intestinal obstruction
   - Rupture → caseous peritonitis.
9. Tuberculosis of the liver
   → – Nonspecific changes of focal fatty infiltration and necrosis
   - Tuberculoma. - May be mistakenly diagnosed as amoebic abscess
   - Tuberculomas. - Rarely → palpable hepatomegaly. In such cases, the liver is studded with whitish nodules, varying in size from 1–3 cm.
10. Tuberculosis of the pancreas
    - May be mistaken for chronic relapsing pancreatitis. Correct diagnosis is made only on histopathology.

C. Genitourinary TB

A significant proportion of cases do not show evidence of TB elsewhere in the body.

1. Renal TB
   - Invariably hematogenous
   - A patient may suffer from one/more of the following forms of the disease: Pyelitis, hydronephrosis, pyonephrosis, perinephric abscess, a chronic discharging sinus in the loin, tuberculous autonephrectomy.
2. TB of the ureter
   - Ureter is frequently affected at its lower end
   - Hydroureter, hydronephrosis, tuberculous autonephrectomy.
3. Tuberculosis of the urinary bladder
   - Infection almost invariably descends from the kidney
   - Cystitis, chronically contracted bladder, VU reflux.
4. Tuberculous epididymitis
   - More often due to hematogenous infection than descending infection
   - Cold abscess, chronic discharging sinus.
5. Tuberculous orchitis is uncommon and secondary hydrocele is rare.

D. Tuberculous **constrictive pericarditis**

The constrictive effect of pericarditis manifests after the acute stage of pericarditis is over and healing commences.

E. TB of the **CNS**

1. Tuberculous meningitis
   - Diffuse involvement of leptomeninges
   - Results from hematogenous spread of infection in a previously non-immunized person and forms a part of the process of military TB
   - *Complications*:
     - Basal adhesions and secondary hydrocephalus
     - Cerebral and spinal arachnoiditis which mimic SOL over the brain and spinal cord.
2. Tuberculoma
   - Represents a localized form of TB giving rise to SOL in various parts of the brain, and rarely in the spinal cord.

www.Ebook777.com

F. Skeletal TB i.e. TB of bones and joints

- Skeletal TB is produced by hematogenous infection
- In case of long bones, infection usually starts in the metaphyseal region
- Typical osteolytic lesion is called 'caries'
- New bone formation is very poor as compared to pyogenic osteomyelitis. Also, the bony sequestra are never massive as seen in pyogenic osteomyelitis; small multiple feathery sequestra are characteristic
- In rare instances, tuberculous lesions are seen in diaphysis. Multiple pseudocystic type lesions develop in several bones of the body; more than one focus may also be present in the same bone. The condition is seen usually in malnourished children
- Clinical features vary considerably according to different sites.

Joint TB passes through 3 clinical stages

Stage 1: Synovitis and effusion → swelling and restriction of movements. The joint assumes a position of maximum comfort, which gives minimum tension on joint capsule.

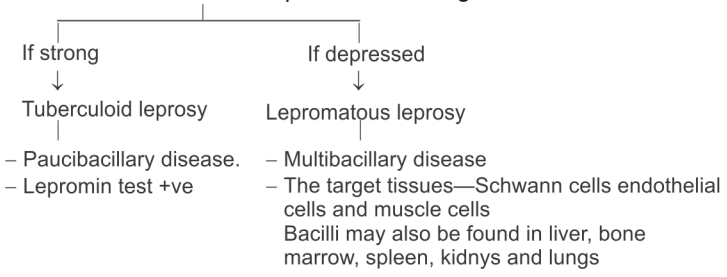
Stage 2: Destruction of articular cartilage → muscle spasm and 'night cries'. Later on, the bony ends become irregular, sinuses develop, and the joint becomes totally disorganized.

Stage 3:
- Contractures
- Dislocation
- Fibrous ankylosis → loss of mobility.

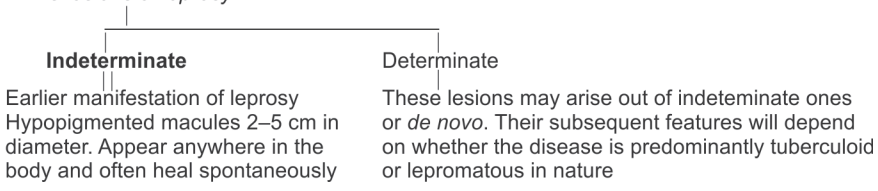
TB of the spine: Refer to chapter 53.

Leprosy

- Not a very contagious disease
- Frequently contracted in childhood/adolescence revealing itself some years later. This silent period is commonly 2–5 years and often longer
- Infection may be acquired by inhalation or through abrasions in the skin
- *The basic classification* depends on the degree of cell-mediated immunity (in response to the infection)



– The *lesions of leprosy*



Tuberculoid leprosy

A few or solitary skin lesions—2 to 5 cm in diameter, often with a raised edge.

- Hairless
- Hypopigmented in dark-skinned, and coppery in white-skinned individuals
- Dry, and anesthetic.

Thickening of local or distant cutaneous nerves.

- The most common nerves involved: Ulnar at the elbow, median at the wrist, common peroneal.

Free ebooks ==> www.Ebook777.com

Lepromatous leprosy

Widespread symmetrical macular rash

– Slightly hypopigmented or erythematous
– Sites—face, extensor surface of the limbs and the upper trunk.

Iritis ±.

Congestion and discharge from the nose, the mucosa of which is thickened and yellow.

Features with further progress of the disease

– Skin especially of the face becomes thickened and nodular
– Eyebrows—thinned out or lost
– Symmetrical enlargement of peripheral nerves, with widespread anesthesia and muscular weakness
– Painless neuropathic ulceration
– Absorption of the extremities of the digits
– Wrist drop, foot drop, claw hand.

YAWS

It is a crippling contagious disease, caused by *Treponema pertenue* and represents non-venereal form of syphilis.

It is a disease of primitive people who are scantily clothed and give little regard to personal hygiene.

Clinically, the disease shows 3 stages as seen in venereal syphilis.

Primary stage: Ulcerative lesions with regional lymphadenitis.

Secondary stage: – Papillomatous lesions erupt all over the body after a brief prodromal period of joint pains, body aches and fever. Papillomas ulcerate.

Tertiary stage: – Hyperkeratosis of soles and palms—not infectious
– Leukomelanoderma.

– Gummatous lesions → perforation of the palate, nasal septum. Gummatous destruction may → extensive necrosis of the lip, cheek and maxilla, simulating mucocutaneous gangrene of the mouth.
– Osteoperiostitis of tibia → 'sabre tibia'.

Lesions of yaws may be mistaken for several other disorders including those caused by leprosy, tropical ulcer etc.

FUNGAL INFECTIONS OF SURGICAL IMPORTANCE

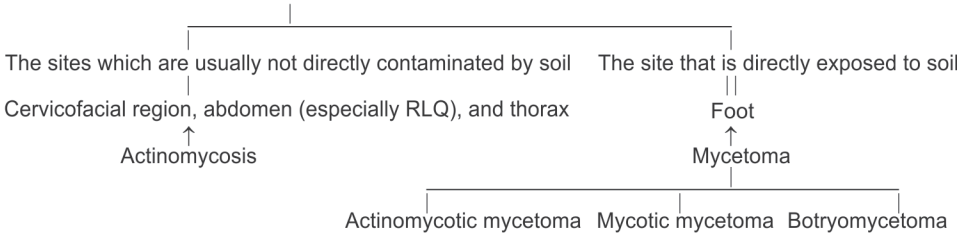
Cutaneous i.e. superficial	Subcutaneous	Deep seated	Oral / alimentary / pulmonary / muco-cutaneous candidiasis	Systemic mycosis
They have a world-wide distribution	Largely restricted to tropical and subtropical regions	May prove fatal particularly in debilitated and immunosuppressed patients		Arise from superficial and deep-seated fungal infections in immunosuppressed patients. Commonly encountered in ICU patients
– Superficial candidiasis – Ring worm – Pityriasis versicolor	– Actinomycosis – Mycetoma – Sporotrichosis	– Histoplasmosis – Cryptococcosis – Coccidiomycosis – Blastomycosis		

A Note on Actinomycosis/Mycetoma

There is a common clinical picture produced by a variety of microorganisms e.g. *Actinomyces israelii, Actinomyces bovis, Madurella mycitomi, Magnaporthe grisea, Nocardia, streptomyces,* etc; viz. indurated swelling, pigmentation, and multiple sinuses.

www.Ebook777.com

2 groups of sites in the body are affected



Definition of terms

- 'Actino' = ray
- Myc (o) / Mycete = fungus
- Actinomyces = ray-like fungus
- Actinomycosis = a disease produced by a ray-like fungus
- Mycetoma = a tumor-like mass of tangled fungal mycelia.

Features	*Actinomycosis*	*Actinomycotic mycetoma*	*Mycotic mycetoma*	*Botryomycetoma*
Microorganisms	*Actinomyces israelii. Actinomyces bovis* → a 'lumpy jaw' in animals Anaerobic	*Nocardia* *Streptomyces* Aerobic	*Madurella mycitomi, Magnaporthe grisea,* etc. Aerobic	BACTERIA. Not fungi Usually mixed flora of *Staphylococci*, *Streptococci*, *Escherichia coli*, *Pseudomonas*
Habitat	Oral cavity—tonsillar crypts, dental cavities	Soil	Soil	Ubiquitous
Granules	– Moderate size – Yellow	– Small-moderate size – Yellow-red-yellowish white	– Large – Dark / black	Nil
Filaments	Fine	Fine	Broad, segmented	Not applicable
Chlamydospores	No	No	Yes	No
Sites of involvement	– Cervicofacial region – Abdomen (especially RLQ) – Thorax	Foot However, Nocardia can affect lungs, brain, kidneys, etc.—'nocardiasis'	Foot / hand	Foot / hand
Mode of entry of organisms/ development of infection	The precise conditions that result in the development of this endogenous infection are not known; the disease often follows trauma such as extraction of a carious tooth	Direct inoculation by injury, thorn prick, etc.		

Free ebooks ==> www.Ebook777.com



Figs 13-12A to D Mycetoma of the right foot: Turned out to be a botryomycetoma on microbiological examination: **(A)**, **(B)** and **(C)** Clinical appearance, **(D)** Postexcision and skin grafting (Postoperative day 15)

PARASITIC INFESTATIONS OF SURGICAL IMPORTANCE

Amoebiasis

It is an endemic disease; however, it is widely prevalent in the developing countries of tropics.

Colonic amoebiasis

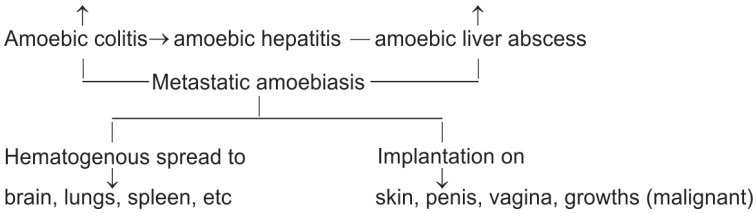
– Few patients ever present with true dysentery. Irregular bowel habits and loose motions with or without mucus are more frequently seen
– Simulates practically every chronic abdominal condition like appendicitis, cholecystitis, peptic ulcer, etc.
– The most common complaint in the chronic stage is vague abdominal pain in the epigastrium or in the lower quadrant/s
– Hardly any physical +ve sign is present in uncomplicated cases. Tenderness along the colon is common
– Narrowing of the colon is seen in cases of amoeboma but true fibrous stricture developing from ulcers is almost unknown.

The spectrum of 'surgical amoebiasis'

- Amoeboma
- Amoebic acute typhlitis
- Gangrenous amoebic colitis
- Amoebic acute perforative peritonitis (– Generalized / Localized)
- Internal fistulae
- Intussusception
- Severe melena from ulcers
- ? Amoebic appendicitis
- Pleuropulmonary amoebiasis
  – Effusion, empyema
  – Lung abscess
  – Bronchohepatic/bronchobiliary fistula
- Pericarditis
- Amoebic peritonitis due to rupture of abscess
- Fistulae

www.Ebook777.com

1.



2. Amoebic lung abscess rarely may develop in the absence of a liver abscess. Possibly, an alternative route for such infestation is direct inhalation of contaminated dust.

Intussusception

Caused by amoebic granuloma.

– Usually seen in adults
– Cecocolic / colocolic / ileocolic.

Hepatic amoebiasis = Amoebic hepatitis/amoebic liver abscess. Amoebic hepatitis, probably represents a stage prior to abscess formation.

Amoebic liver abscess

Refer to chapter 34.

Modes of clinical presentation different than the usual one

1. Typhoid type: Fever is more pronounced. Pain is comparatively less.
   Hepatic amoebiasis is perhaps a more important cause of PUO than a subdiaphragmatic abscess.
2. Pleuritic type.
3. Anginal type: An abscess in the left lobe may present with symptoms suggesting angina pectoris.
4. Dysenteric type.
5. Dyspeptic type.

The local symptoms and signs considerably depend upon the location and size of the abscess within the liver

1. An abscess in the right lobe near the lateral surface → intercostal bulging and tenderness in the lower part of the right side of the chest wall.
2. An abscess ↓ the superior surface → (referred) pain in the right shoulder.
3. An abscess in the bare area of the liver → a lump in the right loin. Rupture of such an abscess simulates a perinephric abscess.
4. An abscess in the posteroinferior quadrant of the right lobe, below the bare area → a lump simulating a renal lump.
5. An abscess near the anteroinferior surface of the right lobe → an acute inflammatory abdomen involving the RUQ, usually with a tender hepatomegaly.
6. An abscess in the left lobe of the liver → a tender epigastric lump. Rarely, it may transmit aortic pulsations and simulate an aortic aneurysm.
7. Abscesses in the caudate/quadrate lobes → symptoms due to pressure on the biliary passage and IVC.

Atypical features

1. Jaundice.
2. In alcoholic patients, abscesses tend to be multiple, large, and mortality is considerably higher.
3. Sometimes the patient may present for the first time straightway with complication viz. rupture into the chest or peritoneal cavity.

Pleuropulmonary amoebiasis

– Sometimes, initial symptoms s/o liver abscess are absent, and patient presents with chest symptoms.
– A pleural effusion may be 'sympathetic', and is due to an abscess situated in the vicinity of diaphragm. Such an effusion may be serous, serosanguinous or seropurulent.

Free ebooks ==> www.Ebook777.com

– Empyema ⇒ an abscess that has burst into pleural cavity. Amoebic empyemas tend to be loculated.
– An amoebic lung abscess tends to open into the bronchus and considerable quantity of characteristic pus is expectorated. Although this may bring partial relief, secondary infection supervenes and the lung abscess becomes chronic.

Cutaneous amoebiasis

1. Ulcerative form
   - Rapidly extending serpiginous phagedenic ulceration with a serrated margin and an undermined edge
   - Pus oozing out from ulcers may contain parasites in large numbers
   - Extensive ulceration may be mistaken for progressive bacterial cutaneous gangrene.
2. Pseudomalignant form
   - Slowly progressive papillary lesions with ulceration:
     – Firm consistency
     – Irregular heaped up border of hard consistency.

   Clinically, it may be mistaken as a malignant lesion.

   Notes

   Cutaneous amoebiasis develops when infective material directly bathes the skin.

   Predisposing situations

   – A pericolic abscess drained outside/burst spontaneously
   – Draining an appendicular abscess
   – Cecal/colonic fecal fistula
   – Chronic amoebic dysentery may lead to perianal cutaneous amoebiasis.

Cancer and amoebiasis

- Amoebiasis may itself superimpose upon cancer
- Severe amoebic infection masks the malignant characters of the tumor
- It may also add to pain, necrosis and extensive ulceration of cancerous growths of the rectum, anus, vulva and uterine cervix
- The presence of amoebae in the discharge may lead to a mistaken diagnosis of amoebic ulcer unless histopathological examination is carried out
- Antiamoebic treatment brings relief by controlling the superadded infestation, and consequently may delay the diagnosis.

Ascariasis (Round-worm infestation)

The prevalence of ascariasis is directly related to local habits and method of disposal of feces in the community, and represents a direct index of the community's progress towards a desirable level of sanitation.
Round worm infestation should be suspected in all malnourished children who complain of vague abdominal symptoms.
'Light' infestations are usually asymptomatic.
Preschool children are commonly affected.
90% of worms are located in the jejunum. Colonic environment is not conducive to their survival and hence they produce very few complications related to colon, including appendicitis.
Symptoms: Pain in abdomen, loss of appetite, dyspepsia, altered bowel habit.
Effects: Malnutrition, anemia, deficiency of vitamins A, riboflavin and C.
Surgical complications result from 4 characteristics of round worms

1. When present in significant numbers, they tend to wrap themselves around one another into tight bundles → formation of a ball, which can lead to frank mechanical obstruction.
2. An individual worm has a tendency to negotiate small orifices, once its head is engaged in them. Such engagement acts as a powerful stimulus, causing the worm to make its advance into the channel leading from that orifice.
3. Toxins released by worms can → an intense spasmodic contraction of the intestine.
4. Ova and larvae can stimulate a chronic granulomatous reaction.

www.Ebook777.com

Surgical complications that can be caused by ascariasis:
- Intestinal obstruction
- Intestinal perforation → peritonitis
- Hepatobiliary ascariasis
- Ascaris pancreatitis
- Ectopic ascariasis
- Ascaris granuloma.

Intestinal obstruction is the most common complication.

This can occur in 4 ways
1. Intense spasmodic contraction induced by toxins. This is the most common mechanism.
2. A frank mechanical obstruction by a ball of round worms.
3. Volvulus—intestinal loop loaded with a ball of worms may undergo torsion.
4. Very often peritoneal fluid appears cloudy and matting of the loops loaded with worms is seen (an intraoperative finding); associated adhesions and kinking can → obstruction.

The majority of cases present with incomplete obstruction, run a short mild course, and respond favorably to conservative treatment.

Intestinal perforation

This can occur in 3 ways
1. As a complication of obstruction, due to delay in diagnosis and treatment. Resulting peritonitis in such cases is fulminating, due to bacterial contamination associated with obstruction.
2. It is assumed and accepted, that worms can't perforate through a normal intestinal wall. They can make their way through weak spots such as inflamed Payer's patch, enteritis, ulcer, suture line, etc.
3. Toxic products from worms → inflammatory reaction and infective thrombosis of vessels → gangrene → perforation → peritonitis.

Hepatobiliary ascariasis

This complication represents one of the sinister consequences of their tendency to negotiate small orifices like ampulla of Vater.

Various pathological consequences: Ulcers in the biliary ductal mucosa f/b healing, leading to formation of stricture → obstructive jaundice, cholangitis; multiple hepatic abscesses.

Pancreatitis results from obstruction of pancreatic duct.

Ectopic ascariasis

Round worms may wander out of GIT to surprisingly unimaginable locations—renal pelvis, pleural cavity, middle ear, lacrimal duct, femoral artery, etc!

Ascaris granuloma
- Formation of granuloma is excited by ova of round worms.
- May form in the liver, pancreas, peritoneum (closely resembling tuberculous peritonitis).

Schistosomiasis (Bilharziasis)

Clinical features during the stage of invasion and maturation
- This stage lasts × 3 months
- This stage occurs in all 3 infections
- Generalized illness
  - Fever, malaise, exhaustion
  - Diarrhea and abdominal discomfort
  - Urticaria. Eosinophilia is invariably present

This is known as Katayama syndrome. Occasionally it may be severe and fatal.

Subsequent clinical manifestations differ in different species.

Schistosoma haematobium → urinary tract disease, hepatic disease, and consequently various clinical manifestations depending on the pathology.

Free ebooks ==> www.Ebook777.com

- Pathological forms and lesions are varied
- Frank hematuria—intermittent. Attacks become less severe with time
- Kidney and ureteral involvement is usually bilateral
- The most common lesion encountered in vesical schistosomiasis is the 'sandy patch'
- However, the urinary bladder may appear normal macroscopically even in fairly severe infections, and mucosal biopsies and press preparations are necessary if *Schistosoma haematobium* is suspected
- Squamous metaplasia may supervene. Association with vesical cancer has been well-established
- Urinary stones may disappear following successful medical treatment
- Portal HT: Liver function remains fairly good. Spleen is invariably massively enlarged.

Schistosoma mansoni* and *Schistosoma japonicum

→
- In severe cases, ulceration and massive bleeding (melena); this could be fatal
- Small intestinal granuloma → intestinal obstruction, and rarely mesenteric infarction
- Perforative peritonitis
- Sessile / pedunculated polyps
- Colon:
  - Stricture
  - Pseudotumor may be macroscopically indistinguishable from carcinoma
- Muscular and serosal layers may be involved with formation of pseudotubercles, and they may be associated with focal peritonitis and intestinal adhesions
- Granulomatous lesions may form in the mesenteric and retroperitoneal nodes → masses that may simulate a tumor (pseudotumor); intussusception, obstruction and rectal prolapse may supervene
- Perianal suppuration and fistulae
- Appendicitis
- Pyloric obstruction.

Filariasis

- The principle pathogenic agent in filarial infestation is the adult worm which affects the lymphatic system
- In an affected community, microfilariae are seldom seen in children below 3 years
- In some individuals showing microfilariae in their blood may not have any clinical disease
- The only clinical condition ascribed to microfilariae of *Wuchereia bancrofti* is 'tropical eosinophilia'.

The clinical manifestations of filariasis are almost exclusively produced by the adult worm which resides in the lymphatic vessels and lymph nodes. When alive, the adult worm does not seem to produce lymphatic obstruction, and any inflammatory reaction produced is mild. Antigenic substances related to the dead worm are responsible for widespread inflammatory changes.

The initial manifestations are inflammatory in nature and result from: 1. Toxic and antigenic products liberated by the female worm at the time of releasing the microfilariae, and 2. Superadded streptococcal and pyogenic infection.

Clinical features of the acute inflammatory response

- High grade fever with rigors
- Pain and swelling of the affected part.

The attack subsides in a few days and is accompanied with death of the worm—this event initiates a strong local inflammatory reaction consequently with obstruction of lymphatic channels.

Chronic stage of the disease represents the effect of chronic lymphatic obstruction of the target organs.

Effects of chronic lymphatic obstruction

1. Lymphedema occurs in those parts of the body where lymphatics are relatively fewer and set in denser tissues.
2. Lymphangiectasis occurs where lymphatics are plentiful and set in soft and loose tissues. The lymphatics tend to dilate and → soft compressible enlargement e.g. lymph varix.
3. Rupture of lymph vessels occurs when lymphatics in the vicinity of a cavity are involved. The dilated lymph vessels rupture and directly empty their contents into the cavity. Examples: Chylocele, chyluria, chylous ascites, chylothorax, chylous diarrhea, etc.

www.Ebook777.com

The spectrum of 'surgical filariasis'

The site	*Acute*	*Chronic*
Lymph nodes	Lymphadenitis	Lymphadenovarix
Lymphatic vessels	Lymphangitis	Lymphedema, elephantiasis
Genitals	Scrotal lymphangitis Funiculitis Epididymo-orchitis	Lymph varix of the cord Lymph scrotum Hydrocele Chylocele Lymphedema, elephantiasis of the penis and scrotum / vulva
Abdomen	Retroperitoneal lymphangitis	Chronic abdominal lesions Chyluria
Breast	-	Lymphedema, elephantiasis Nodular lesions
Joints	Arthritis	-
Serous cavities	Pericarditis	Chylothorax Chylous ascites

Abdominal lesions

Abdominal lesions are relatively rare.

1. Recurrent attacks of pain in abdomen. Exploratory laparotomy/laparoscopy reveals enlarged and congested lymph nodes, which on histopathological examination reveal filarial infestation.
2. Lymphatic and chylous cysts of the mesentery.
3. Lymphadenopathy (mesenteric/retroperitoneal) sometimes becomes big enough to present as a lump in abdomen.

Filarial arthritis

– Recurrent attacks of acute arthritis simulating septic arthritis found in endemic areas for filariasis
– Knee joint is most commonly affected
– Attack starts with a high-grade fever with rigors
– Within 2–3 days, the (knee) joint gets swollen and painful. 'Held' in semiflexed position
– Toxemia subsides in 4–5 days. Joint swelling persists for 8–10 days
– Neither microfilariae nor adult worms are recovered from the joint
– Difficult to explain the chylous nature of fluid in filarial arthritis.

Loa loa (Eye worm)

→ a puffy swelling often on the dorsum of the wrist—known as a Calabar swelling.
– May be misdiagnosed as a soft tissue infection. However, there is no abscess or septic arthritis
– The worm also has a propensity to migrate beneath the conjunctiva, causing local redness and swelling with puffiness of the eyelid. Subconjunctival worm can be visualized directly
– Occasionally, an adult *Loa loa* may be seen under the skin in the absence of a Calabar swelling
– *Confirmation of diagnosis*: 1. By examining a worm removed from under the conjunctiva, or occasionally, from under the skin. ± 2. By examining a peripheral blood smear for microfilariae in a sample of blood drawn between 12 noon and 2 PM.

Hydatid Disease

– The disease is caused by the larval form of the parasite—*Echinococcus granulosus/E. multilocularis/E. oligettas*

Free ebooks ==> www.Ebook777.com

- It is particularly prevalent in those regions where sheep and cattle are raised along with dogs
- The disease produces very few systemic manifestations. Its local effects remain dormant for several years before they manifest clinically
- Human infection is essentially a slow process and it is not possible to know the correct time of contract of infection. The growth of a hydatid cyst itself is so slow and mode of onset being insidious, that the patient does not become aware of its presence until a few years from its onset.

  Symptoms are produced by virtue of 3 mechanisms

  1. Space-occupying nature of the cyst.
  2. Allergic reactions secondary to absorption of the products from the parasite when the cyst bursts and fluid is absorbed by the adjacent tissues.
  3. Complications e.g. rupture, infection, etc.
- Hydatid disease is uncommon in pediatric age group
- Infection is usually acquired in the childhood. The interval between infection and manifestation of symptoms may exceed 30 years
- Cysts may be single (usual) or multiple
- Rupture of the cyst may result from secondary infection or trauma and may be accompanied by severe allergic manifestations that can be life-threatening
- Liver and lungs are the most commonly involved organs. Otherwise, any organ/celomic cavity in the body can be affected
- In clinical experience, hydatid disease of lungs seems to develop independent of disease in the liver. Indeed, concomitant involvement of liver is rare with pulmonary hydatid disease
- In about 20% of cases, a pulmonary hydatid cyst produces no symptoms. Such cases are detected incidentally. Clinically, there are very few physical signs specific for hydatid cyst. Rarely, patient may expectorate white fluid and typical hydatid membrane if the cyst ruptures into the bronchus. An average rate of growth of cyst is ~ 5 cm / year
- Alveolar cyst is most frequently found in the lungs in children. Infection of the cyst results in a chronic lung abscess. The cyst may rupture as a result of coughing or secondary infection
- The majority of hydatid cysts of the spleen are primary, there being no hydatid cysts in the liver or any other organ. Metastatic cysts may sometimes develop in the spleen
- It may be difficult to excise retroperitoneal hydatid cysts. They are densely adherent to adjacent structures and cannot be enucleated.

Dracontiasis (Guinea worm infestation)

- Although it is not fatal, morbidity produced by the disease in young agricultural laborers at the critical time of sowing of crops causes considerable loss of manpower
- The adult guinea worm seen in clinical lesions represents the gravid female
- For completion of its life cycle by discharging large number of developing embryos and juvenile worms in water, the female worm must migrate towards the subcutaneous tissue of such part of the body, which is frequently immersed in water. 80% of lesions are, therefore, open (sinus) located in the lower leg. In other instances, the worm fails to reach the surface, dies and gets calcified in the deeper tissues or produces closed clinical lesions (abscess/granuloma/cyst) in muscles, joints, extradural spaces, etc. Guinea worms may also be found in several other unusual sites—reported/unreported as yet in the medical literature e.g. upper limb, back, bulbar conjunctiva, heart, etc.
- The guinea worm pus is characteristically thick and straw colored. The dead worm is often calcified and lies in the midst of necrotic debris
- The disease is mainly found in rural population of farmers and laborers who show scant attention to personal hygiene. Recurrent and multiple lesions are frequently met with.

Clinical features of a typical guinea worm surface-lesion

- 2–3 days before the development of surface lesions, the patient suffers from prodromal symptoms—urticaria, pruritus, anorexia, vomiting, diarrhea, SOB, giddiness

www.Ebook777.com

– Some patients may even present with palpable worm under their skin
– Blister/bleb (size—pin-head to a walnut) forms near the ankle. It is surrounded by a zone of cellulitis. The blister contains serous/serosanguinous fluid. Rupturing of the blister uncovers a sinus through which the worm emerges
– The sinus leads to a long subcutaneous tract in which female worm lies.

Aftermath of open lesions

– Sinus/ulcer generally heal once the worm is fully extruded. Chronic ulcers may leave hypertrophied scars.

The spectrum of 'surgical dracontiasis'

– Ulcer
– Abscess, granuloma, cyst
– Osteomyelitis
– Arthritis and periarthritis
– Bursitis, tenosynovitis
– Pericarditis.

Gnathostomiasis

– Caused by a nematode *Gnathostoma spinigerum*
– Infestation usually follows ingestion of raw fish
– → single migratory subcutaneous swelling which may appear anywhere, including the abdominal wall, the chest, the face or hands
– Also, may → spontaneous perforation of the right side of the colon / hematuria / spontaneous pneumothorax
– ± peripheral blood eosinophilia
– Occasionally, the only findings are eosinophilia with nonspecific symptoms.

Malaria

a. *Plasmodium vivax*
   – Fever
      - The most constant sign after a brief period of remittent fever
      - Comes every second day
   – Anemia
   – Splenomegaly
   – Pain in abdomen is not a major feature.
b. *Plasmodium falciparum*
   – Fever—irregular and occurs daily
   – Malaise, nausea, vomiting and generalized joint pains may be the only symptoms
   – Acute pain in abdomen ±; may resemble acute appendicitis
   – Anemia
   – Hepatosplenomegaly.

Onchocerca volvulus

– Subcutaneous nodules may develop, especially over the iliac crests
– Cutaneous itching, excoriation, urticaria, depigmentation, lichenification, lymphadenopathy.

Cysticercosis

– Cysticerci may occur in any organ, but are found most commonly in skeletal muscle, subcutaneous tissue and CNS

Free ebooks ==> www.Ebook777.com

– Subcutaneous nodules—~ 1 cm in diameter. Cysticercotic nature may be unsuspected until their histology (on excision) is reported.

Sparganosis

– Painful subcutaneous swelling that may migrate. Inflammation becomes severe on death of the larva
– Diagnosis confirmed on biopsy reporting.

Myiasis

– The lesion begins as a papule, which enlarges to resemble a furuncle
– Careful inspection of the lesion will reveal spiracles (breathing tubes) of the larva.

Jiggers

– Elevated, pea-sized lesions on the feet though any part of the body may be affected. Secondary bacterial infection is quite common and tetanus may develop.

www.Ebook777.com

14 SKELETAL MUSCLES IN THE BODY AMENABLE TO PHYSICAL EXAMINATION (Applied Anatomy)

This chapter discusses in brief, those muscles in the body which may need to be, and can be tested particularly during physical examination of swelling / ulcer, etc.

HEAD

Masseter
Action: Closing the jaw.
Origin: Zygomatic arch.
Insertion: Lateral surface of the ramus of the mandible and coronoid process.

SUPERFICIAL MUSCLES IN THE NECK

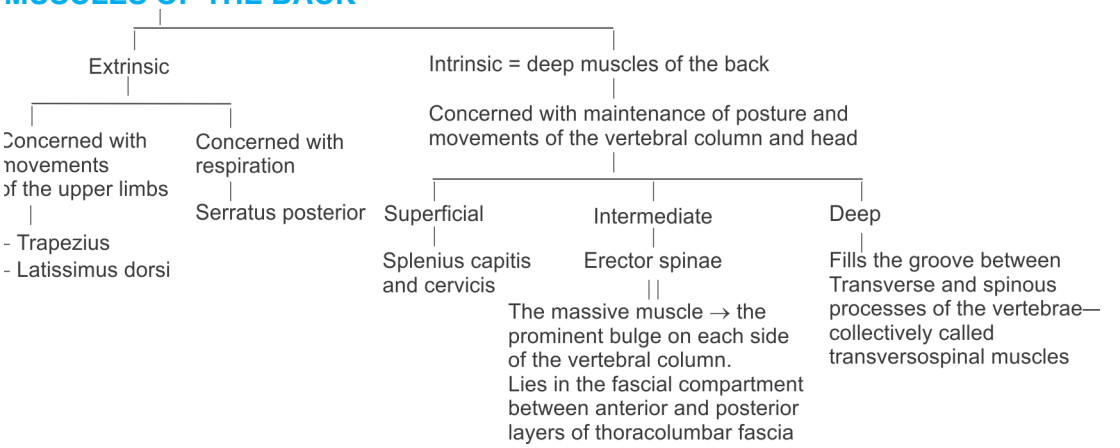
Platysma, SCM and Trapezius
Platysma = 'a flat plate'.
It covers upper part of the anterior triangle and inferior part of the posterior triangle.
Actions:
– Tensing the skin of the neck e.g. during shaving, easing out a tight neck-tie or collar
– When the entire muscle contracts → wrinkling of the skin in oblique direction, and widening of the mouth.
Superior attachment: Inferior border of the mandible and skin of the lower face.
Inferior attachment: Fascia and skin over the pectoralis major and deltoid.

SCM (Sterno Cleido Mastoid)—the key landmark in the neck.
Actions:
1. Acting unilaterally, it tilts the head to the same side + turns the face up and to the opposite side.
2. Acting bilaterally, → flexion of the neck e.g. when raising the head from a pillow.

Test: Ask the patient to turn the face to the opposite side against resistance and then tilt the head to the same side while maintaining the resistance.
Superior attachment: Mastoid process and superior nuchal line.
Inferior attachment:
– Sternal head—as a rounded tendon, to the manubrium
– Clavicular head—on the medial end of the clavicle.
Nerve supply: Spinal root of CN XI, and C_2, C_3 (proprioceptive).

Free ebooks ==> www.Ebook777.com

MUSCLES OF THE BACK



Trapezius

Muscles of both sides together form a trapezoid (a 4 sided figure).

Actions:

– Superior fibers—elevation of scapula
– Inferior fibers—depression of scapula

Test: Ask the patient to shrug the shoulder against resistance; the upper border of the muscle can be seen and felt.

Medial attachment: Superior nuchal line, external occipital protuberance, ligamentum nuchae, and spinous processes of $C_7 \leftrightarrow T_{12}$.

Lateral attachment: Lateral 3rd of the clavicle, acromion and spine of the scapula.

Nerve supply: Spinal root of CN XI, and C_3 and C_4.

www.Ebook777.com

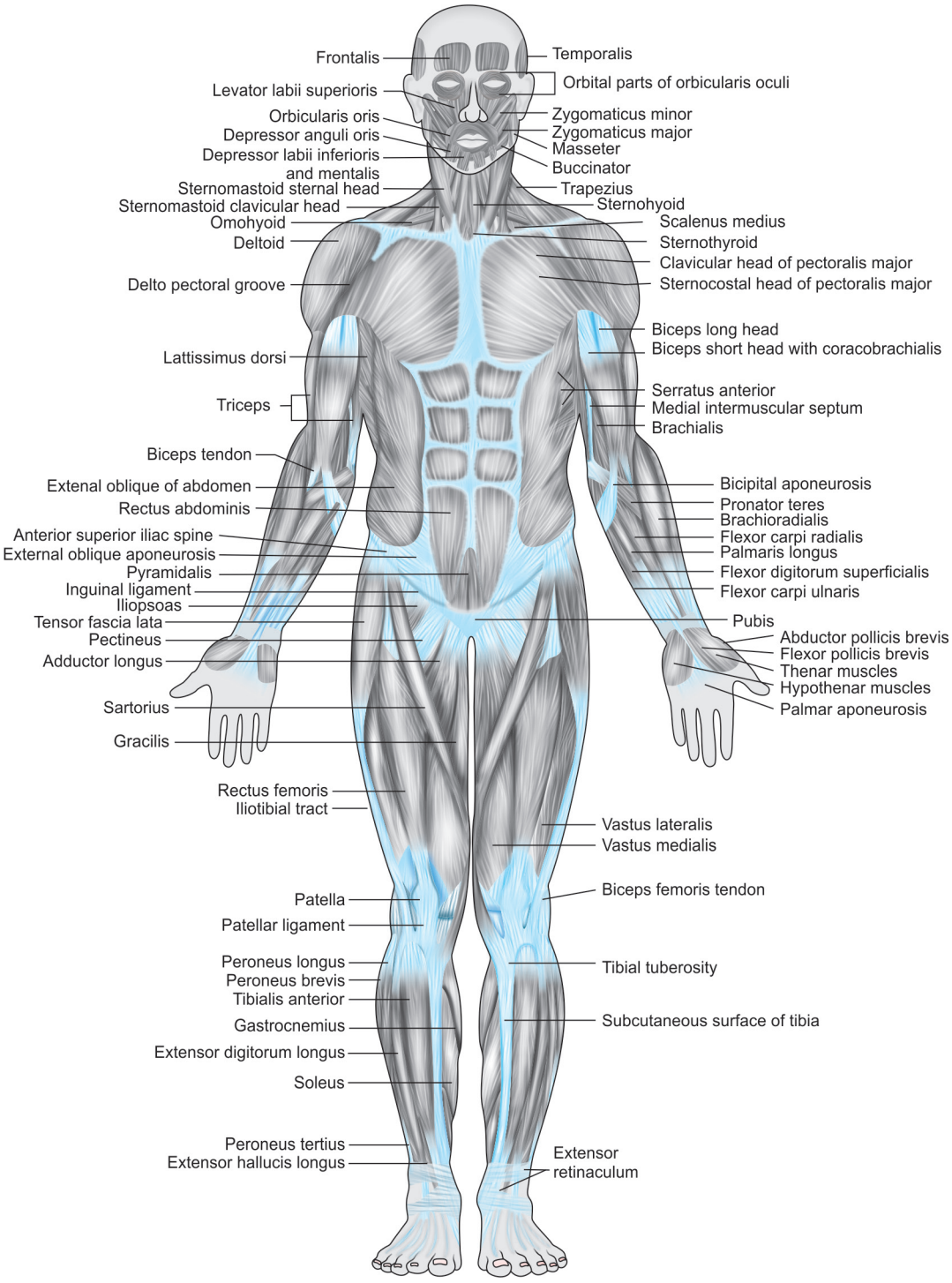


Fig 14-1 Front view

Free ebooks ==> www.Ebook777.com

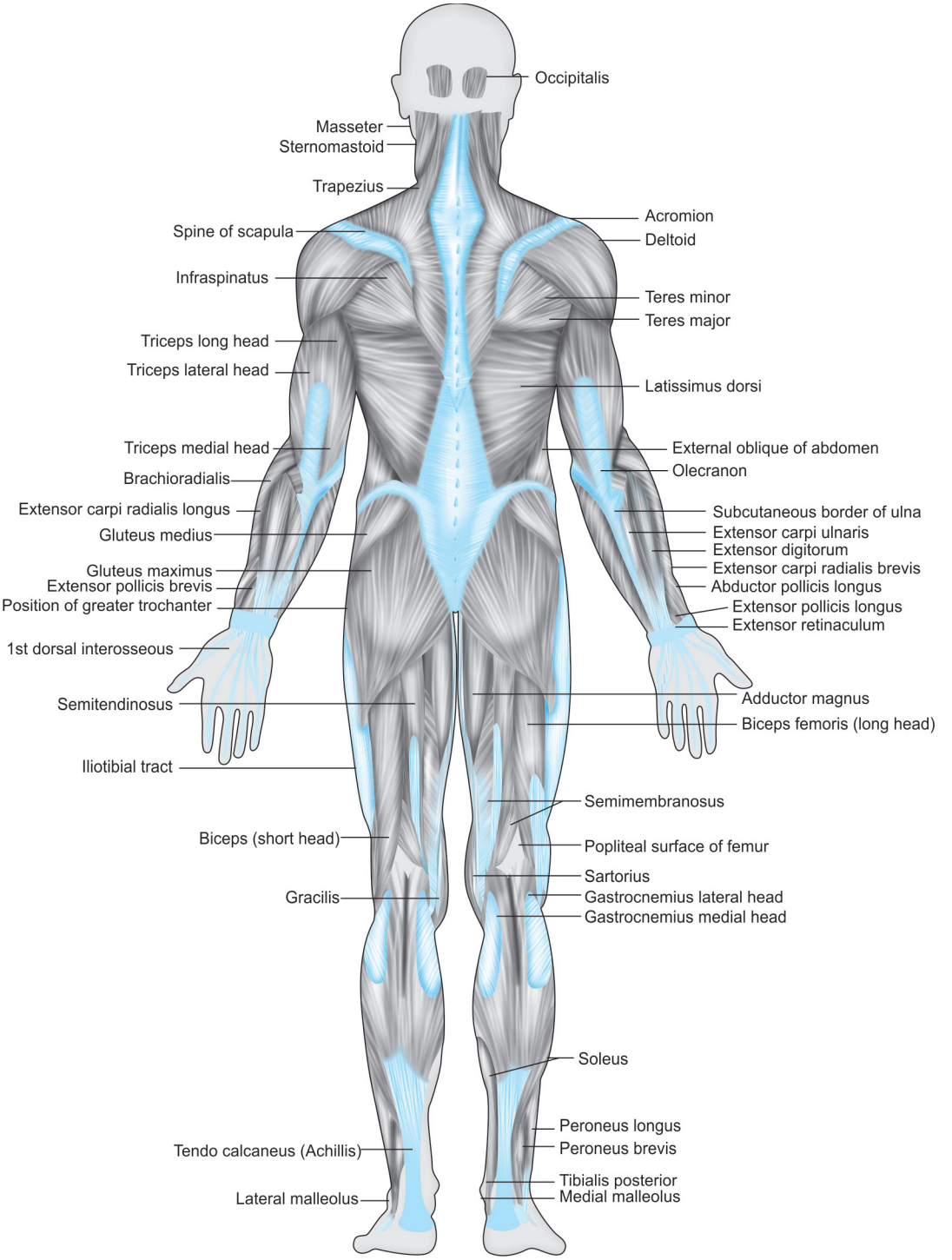


Fig 14-2 Back view

www.Ebook777.com

Latissimus dorsi = 'Widest of the back', so called as it covers the inferior half of the (entire) back.

Actions:

1. Extension, adduction and medial rotation of the arm (folding the arms behind the back, or scratching the opposite scapula).
2. Raising the body towards the arm during climbing.

   Examples: Chopping wood, paddling a canoe, swimming (particularly breast stroke), and climbing.

Test: Abduct the arm to a right angle, and then ask the patient to adduct it against resistance; the anterior border of the muscle can be seen and felt below the posterior axillary fold.

Medial attachments: Spinous processes of $C_7 \leftrightarrow T_{12}$, thoracolumbar fascia, iliac crest, and inferior 3 or 4 ribs.
Lateral attachment: Floor of intertubercular groove on the humerus.
Nerve supply: Thoracodorsal nerve.

Splenius muscles (capitis and cervicis)

Splenion = bandage. The muscle wraps the lateral and posterior aspects of the neck like a bandage; hence the name.

Actions:

– Acting unilaterally—lateral flexion and rotation of the head to the same side
– Acting together (i.e. bilaterally)—extension of the head.

Origin: Inferior half of ligamentum nuchae and spinous processes of $T_1 \leftrightarrow T_6$.
Insertion: – Of capitis—mastoid process and superior nuchal line (deep to SCM)
– Of cervicis—posterior tubercles of the transverse processes of $C_1 \leftrightarrow C_4$.

Erector spinae

Actions:

– Acting unilaterally—lateral flexion of head/vertebral column
– Acting bilaterally—extension of head/part or all of the vertebral column e.g. straightening of the flexed column, bending posteriorly, 'paying out' (i.e. release) during bending forwards so that the movement is controlled.

'Back strain' = some degree of stretching / microscopic tearing of muscles and / or ligaments of the back; muscles involved are especially parts of the erector spinae.

MUSCLES IN THE PECTORAL REGION

Pectoralis major

Action: Adduction (and medial rotation) of the arm.

Test:

– For the clavicular head: Abduct the arm to 90° and ask the patient to push the arm forwards against resistance
– For the sternocostal head: Ask the patient to keep and press the hands on the hips (iliac crests).

Origin:

– Clavicular head—from medial half of the clavicle
– Sternocostal head—from anterior surface of the sternum, upper 6 costal cartilages, and external oblique aponeurosis.

Insertion: Lateral lip of intertubercular groove on the humerus.

Serratus anterior

Actions: Protraction of scapula and holding it against the thoracic wall.

Test:

– Ask the patient to push against a wall by the outstretched hands
– Paralysis of the muscle results in 'winged scapula', where the vertebral border becomes prominently raised off the posterior chest wall.

Origin: External surfaces of the lateral parts of 1st ↔ 8th ribs.
Insertion: Medial border of scapula.

Free ebooks ==> www.Ebook777.com

Anterior axillary fold is formed by the lateral border of pectoralis major.
Posterior axillary fold is formed by teres major and latissimus dorsi.

TERES MAJOR

Action: Adduction and medial rotation of the arm.
Test: Abduct the arm and ask the patient to adduct it against resistance; the contracting muscle can be seen and felt from behind the posterior axillary fold.
Origin: Inferior angle of scapula.
Insertion: Medial lip of intertubercular groove on the humerus.

DELTOID

Actions:
– Anterior part—flexion and medial rotation of the arm
– Middle part—abduction of the arm
– Posterior part—extension and lateral rotation of the arm.

Test: Ask the patient to abduct the arm against resistance.
Origin: Lateral 3rd of the clavicle, acromion, and spine of scapula.
Insertion: Deltoid tuberosity on the humerus.

ARM

Brachialis

Action: Flexion of the forearm *in all positions*.
Origin: Distal half of anterior surface of the humerus.
Insertion: Coronoid process and tuberosity of the ulna.

Biceps brachii

Action: Flexion of the forearm *when it is supinated*.
Test: With the forearm supinated, ask the patient to flex the forearm against resistance; the contracted muscle in the arm, and the tendon and aponeurosis at the elbow are easily palpable.

Triceps brachii

Action: Extension of the forearm.
Test: Flex the forearm, and then ask the patient to extend it against resistance.

LATERAL ASPECT OF THE DISTAL PORTION OF THE ARM, AND THE ELBOW

Brachioradialis and extensor carpi radialis longus.
The brachioradialis lies on the anterior aspect of the extensor carpi radialis longus.

Brachioradialis

Action: Flexion of the forearm; acts most powerfully when the forearm is semipronated.
Test: With the forearm in midprone position, ask the patient to flex the forearm against resistance.
Origin: Supracondylar ridge on the humerus (proximal portion).
Insertion: Lateral aspect of distal end of the radius.
Nerve supply: Radial nerve.

Extensor carpi radialis longus

Action: Extension and abduction of the hand.
Test: With the forearm pronated, ask the patient to extend and abduct the hand against resistance; feel for the contracting muscle below and behind the lateral side of the elbow.
Origin: Supracondylar ridge on the humerus (distal portion)
Insertion: Base of the 2nd metacarpal.

www.Ebook777.com

FOREARM

Superficial and Intermediate Layer Muscles of the Anterior Compartment

Pronator teres

Action: Pronation, and weak flexion of the forearm.

Test: Ask the patient to pronate the forearm against resistance, from a supinated position; feel for the muscle at the medial margin of the cubital fossa.

Flexor carpi radialis

Action: Flexion and abduction of the hand.

Test: Ask the patient to flex and abduct the hand against resistance; the muscle/tendon is easily seen and felt.

Palmaris longus

Action: Flexion of the hand.

Test: Ask the patient to pinch the pads of the thumb and the little finger together, and then flex the hand against resistance; feel for the tendon.

Flexor carpi ulnaris

Action: Flexion and adduction of the hand.

Test: Ask the patient to flex and adduct the hand against resistance; feel for the tendon.

Flexor digitorum superficialis

Action:

– Flexion of the middle phalanges of the medial 4 fingers
– Acting more strongly, → flexion of the proximal phalanges + hand.

Test: Ask the patient to flex the fingers at the proximal interphalangeal joints against resistance being applied to the middle phalanges, while maintaining extension at the distal interphalangeal joints.

Deep Muscles of the Anterior Compartment: Flexor digitorum profundus, flexor pollicis longus, and pronator quadratus.

Flexor digitorum profundus

Action: Flexion of distal phalanges of the medial 4 fingers.

Test: With the fingers extended and the hand lying supine on the table, ask the patient to flex the distal phalanges against resistance while the middle phalanges are being held in extension.

Flexor pollicis longus

Action: Flexion of phalanges of the thumb.

Test: With the proximal phalanx of the thumb held steady, ask the patient to flex the distal phalanx against resistance.

Muscles (of Clinical Importance) of the Posterior Compartment

Extensor carpi radialis longus and brevis, extensor carpi ulnaris, extensor digitorum.

Extensor carpi ulnaris

Action: Extension and adduction of the hand.

Test: With the forearm pronated and the fingers extended, ask the patient to extend and adduct the hand against resistance; the contracting muscle can be seen and felt in the upper forearm and the tendon can be felt proximal to the head of the ulna.

Extensor digitorum

Action: Extension of the medial 4 fingers at metacarpophalangeal joints, and extension of the hand.

Test: With the forearm in pronation and the fingers extended, ask the patient to try to keep the fingers extended at the metacarpophalangeal joints while applying pressure (by you, the clinician) on the proximal phalanges and try to flex them at these joints.

Free ebooks ==> www.Ebook777.com

SUPERFICIAL MUSCLES IN THE THENAR EMINENCE

Abductor pollicis brevis

Action: Abduction of the thumb.

Test: Ask the patient to abduct the thumb (– in a plane at right angles to the palm) against resistance; the contracting muscle can be seen and felt.

Flexor pollicis brevis

The presence of the long flexor (and the variability of the nerve supply) makes testing flexor pollicis brevis of doubtful value.

GLUTEAL MUSCLES

Gluteus maximus

Action: Extension of the thigh.

Test: While lying face down with the leg straight, ask the patient to tighten the buttock and extend the thigh; the contracting muscle can be seen and felt.

Gluteus medius

Action: Abduction and medial rotation of the thigh.

Tests:
1. While lying face down with the leg flexed to a right angle, ask the patient to turn the foot outwards against resistance; feel for the contracting muscle below the iliac crest behind tensor fasciae latae.
2. Trendelenburg test: With the patient standing on one leg, the pelvis on the opposite side should rise slightly; if it falls due to loss of abductor power on the supporting side, the test is +ve.

THIGH

Anterior Group of Muscles

Iliopsoas, sartorius, tensor fasciae latae, and quadriceps femoris.

Overall actions:

– Flexion of the thigh—iliopsoas, sartorius, and tensor fasciae latae.
– Extension of the leg—quadriceps femoris
– Abduction—sartorius and tensor fasciae latae
– Lateral rotation—sartorius
– Medial rotation—tensor fascie latae

Superficial Muscles of the Medial Group (Adductors)

Pectineus, adductor longus, and gracilis.

Test: While lying on the back with the knee straight, ask the patient to adduct the thigh against resistance; feel for the upper ends of gracilis and adductor longus.

Posterior Group of Muscles

Medial to lateral—semimembranosus-semitendinosus, and biceps femoris.

Action: Extension of the thigh, and flexion of the leg.

Test: While lying face down with the limb straight, ask the patient to flex the leg against resistance; feel the tendons of semitendinosus and biceps above the knee on the medial and lateral sides respectively.

www.Ebook777.com

LEG

Muscles of the Anterior Compartment i.e. Dorsiflexors of the Foot

Tibialis anterior

Action: 1. Dorsiflexion and inversion of the foot.
2. Helps to maintain the medial longitudinal arch of the foot.

Test: Ask the patient to dorsiflex the foot against resistance; the tendon can be seen and felt.

Extensor hallucis longus

Action: Extension of the great toe, and dorsiflexion of the foot.

Test: Ask the patient to extend the great toe against resistance; the tendon can be seen and felt. This test is a useful index of the L5 anterior ramus and spinal nerve.

Extensor digitorum longus

Action: Extension of the lateral 4 toes, and dorsiflexion of the foot.

Test: Ask the patient to extend the lateral 4 toes against resistance; the tendons can be seen and felt.

Muscles of the Lateral Compartment

Peroneus longus and peroneus brevis

Actions: 1. Eversion of the foot + weak plantarflexion.
2. They are the plantarflexors when the tibial nerve is paralyzed.
3. Peroneus longus, in addition, maintains the lateral longitudinal and transverse arches of the foot.

Test: Ask the patient to evert the foot and the tendons can be seen and felt below the lateral malleolus.

Superficial Muscles of the Posterior Compartment

Gastrocnemius, soleus, and plantaris.

Action: Plantarflexion of the foot, and flexion of the leg.

Test: Ask the patient to plantarflex the foot against resistance; the tendo calcaneus and muscles above it can be seen contracting and can be felt.

A Deep Muscle of the Posterior Compartment

Tibialis posterior

Actions: 1. Inversion and adduction of the forefoot.
2. Plantarflexion of the foot.
3. Contributes in maintaining the medial longitudinal arch of the foot.

Test: With the foot in slight plantarflexion, ask the patient to invert it against resistance; the tendon can be felt behind the medial malleolus.

DORSUM OF THE FOOT

Extensor digitorum brevis

Action: Extension of the medial 4 toes. It is particularly of value when the long extensor is out of action, in the fully dorsiflexed foot.

Test: Ask the patient to dorsiflex the toes; the muscle belly towards the lateral side of the dorsum of the foot can be seen and felt.

ANTERIOR ABDOMINAL WALL

Rectus abdominis

Actions: 1. Compression of the abdominal viscera.
2. Flexion of the trunk.

Free ebooks ==> www.Ebook777.com

Test: While lying flat on the back, ask the patient to raise the head (without using the arms).
Origin: Pubic symphysis and pubic crest.
Insertion: Xiphoid process and 5th ↔ 7th costal cartilages.

There are no specific tests individually applicable for the other flat muscles viz. the obliques and the transversus. **However, straight leg raising test achieves an isometric contraction of all the flat muscles collectively**, and is a useful clinical test in physical examination of the abdomen e.g. in a c/o pain, lump.

External oblique

Actions: 1. Support to and compression of the abdominal viscera.
2. Flexion and rotation of the trunk.
Origin: External surfaces of 5th ↔ 12th ribs.
Insertion: Linea alba, pubic tubercle, and iliac crest.

Internal oblique

Actions: 1. Support to and compression of the abdominal viscera.
2. Flexion and rotation of the trunk.
Origin: Thoracolumbar fascia, iliac crest, and lateral half of the inguinal ligament.
Insertion: Inferior borders of 10th ↔ 12th ribs, linea alba, and the pubis via conjoint tendon.

Transversus abdominis

Action: Support to and compression of the abdominal viscera.
Origin: Internal surfaces of 7th ↔ 12th costal cartilages, thoracolumbar fascia, iliac crest, and lateral 3rd of the inguinal ligament.
Insertion: Linea alba, and the pubis via conjoint tendon.

www.Ebook777.com

15 SWELLING

Basic concepts, including differential diagnosis of acute swelling and chronic general swelling

Swelling is **defined** as an abnormal eminence or elevation.

GENERAL REMARKS

1. All swellings are not tumors, and all tumors do not present as swelling e.g. an 'antibioma' of the breast though presents as a swelling, is not a tumor, while a 'duct papilloma' of the breast though a tumor, does not present as a swelling.
2. 'Swelling' *v/s* 'edema': Edema leads to an abnormal eminence of the entire circumference of a region of the body say a limb for example. Swelling leads to an abnormal eminence of only a circumscribed area of a region but usually not the entire circumference.

HISTORY

Chief complaint

- Verify the *exact site* and *duration*.
- Is it *pulsatile*?
- Is it *reducible*?
- **R/o edema** (by a quick visual clue). If edema is found instead of a swelling, then proceed as in chapter 50.

Duration?

Mode of onset

- Sudden
- Insidious

OR

- Over and above a pre-existing condition?

How fast did it progress?

- Dramatically over minutes / hours
  OR
- Rapidly over days

⇒ Acute swelling

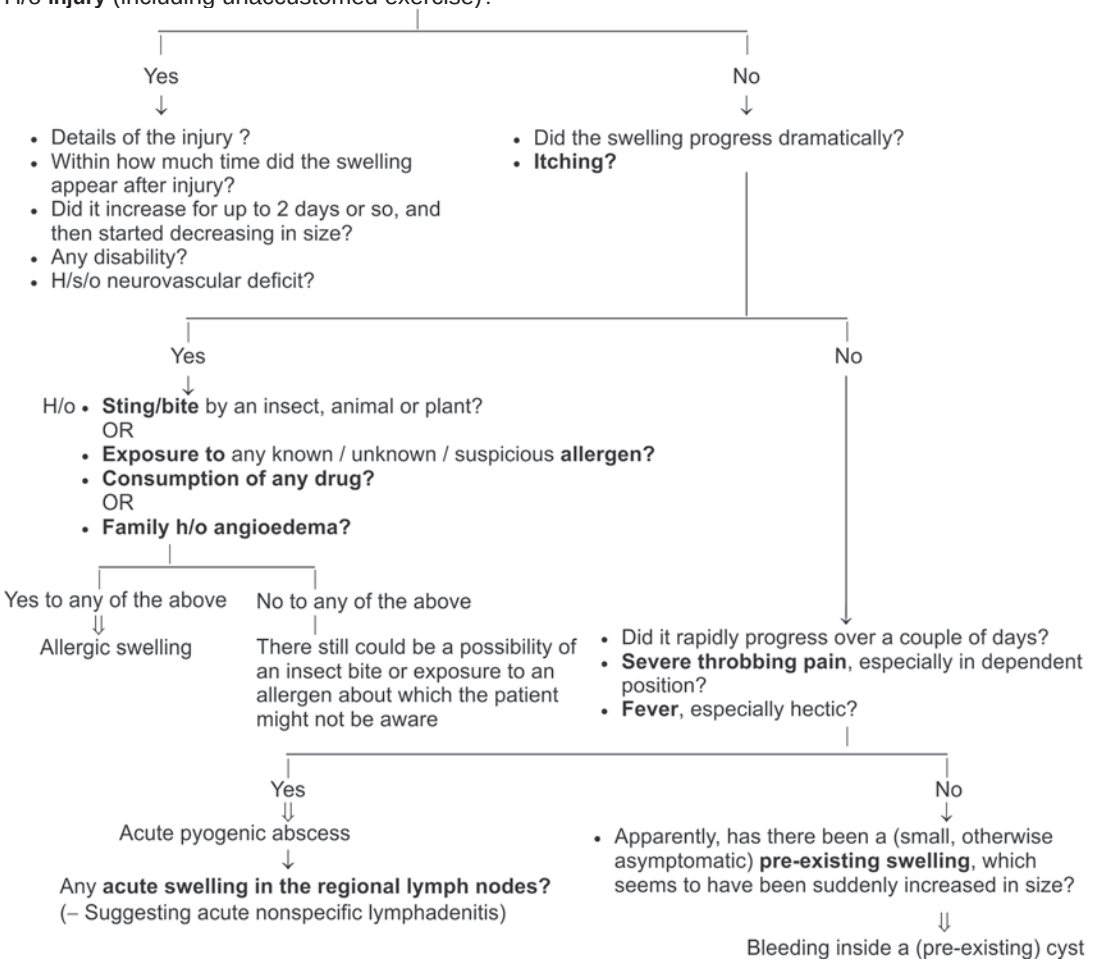
- Over weeks
  OR
- Slow over months / years
  OR
- Is it stationary ever since it was noticed or after initial growth?

⇒ Chronic swelling

Free ebooks ==> www.Ebook777.com

Further **HISTORY** to be asked in a **ACUTE SWELLING**

H/o **injury** (including unaccustomed exercise)?



H/s/o **sequelae and complications**? The details to be asked will vary according to the pathological nature of the swelling.

Treatment taken?: – Including—local applications of ointments / herbal medicines / fomentation, etc.
—Repeated manipulations.

Further **HISTORY** to be asked in a **CHRONIC SWELLING**

- **Any recent increase in size** of the swelling, which has been practically stationary for a long time?
- Did it **regress** any time during the course?
- Does it **fluctuate** in size?
- H/s/o **pressure effects**?
- H/o **disturbance of any (physiological) functions**?

www.Ebook777.com

Pain

If absent—'have you been taking any analgesic?'

When present,

|

- 'When did it start? Did it start before the appearance of the swelling, OR after the appearance of the swelling?'
- Exact site?
- Does it radiate?
- Intensity: Mild/moderate/severe?
- Nature: Throbbing/shooting, etc.?
- Precipitating-aggravating-relieving factors?
- **Fever**?
- **Any other swelling of similar nature elsewhere or in the same region**?
- **(Significant) Loss of weight**?

Additional points in history to determine etiology

- Repeated minor trauma/irritation?
- Physical/chemical trauma?, etc.

H/s/o **sequelae and complications**?

Treatment taken?: – Including—local applications of ointments/herbal medicines/fomentation, etc.
—Repeated manipulations.

H/o **recurrence**?

INTERPRETATION OF SYMPTOMS, AND DISCUSSION

Duration

– **Congenital** swellings are due to faulty formation i.e. dysmorphogenesis'. Examples: Meningocele, cystic hygroma, umbilical hernia, etc.
– Duration of the swelling of about a week ⇒ **Acute** swelling. Examples: Acute inflammatory swelling like acute pyogenic abscess, traumatic swelling like contusion, hematoma, etc.
– A swelling of > 3 week's duration ⇒ **Chronic** swelling. Examples: Tumor, chronic inflammatory swelling like tuberculous lymphadenitis, cold abscess, etc.

Mode of onset

– **Sudden**. Examples: Allergic conditions (like angioedema), traumatic swellings (hematoma, fracture/dislocation, etc.), acute inflammatory swellings (cellulitis, boil, abscess, etc.), bleeding in a cyst/joint, occasionally branchial cyst, descent of a hernia for the first time following severe strain in a preformed sac, etc.
– **Insidious**. Examples: Cysts, tumors, chronic inflammatory swellings.
– **Over and above a pre-existing condition**. Examples: Keloid from a scar, malignant melanoma from a mole, etc.

Progress = increase in size of the swelling and/or development of new symptoms/signs.

– **Dramatic** = over minutes or hours. Examples: Angioedema, traumatic swellings
– **Rapid** = over days or weeks. Examples:
  - Acute inflammatory swellings—progress over days
  - Malignant swellings—progress over weeks. *An exception—fibrosarcoma*
– **Slow** = over months or years. Examples: Cysts, benign tumors, chronic inflammatory swellings, **fibrosarcoma**
– **Stationary**: Example: Benign tumor like a lipoma
– *Causes of* '**recent increase in size of the swelling which has been practically stationary for a long time**'
  - Occasionally during pregnancy due to hormonal variations (Example: A fibroadenoma increasing in size during pregnancy)
  - Displacement of the swelling may lead to apparently recent increase in size of the swelling

Free ebooks ==> www.Ebook777.com

- Bleeding (→ dramatic increase)
- Acute inflammation (→ rapid increase in days)
- Edema
- Malignant transformation (→ rapid increase in weeks)
- Fungation due to infection.

– **(Development of) similar swellings** nearby/elsewhere. Examples: Lipomatosis, neurofibromatosis, pyemic abscesses, generalized lymphadenopathy (TB, AIDS, lymphoma, etc.), satellite nodules of a malignant melanoma
– **Development of swelling in the lymphatic drainage area** suggests lymphadenopathy
– **Development of secondary changes in the swelling**. Examples: – Softening may ⇒ TB
– Ulceration ⇒ TB, malignancy.

Tumors never ***regress*** in size; cysts and chronic inflammatory swellings may.
Fluctuation in size: Examples: – Sialolithiasis (submandibular salivary gland enlargement due to partial obstruction of its duct by a stone)
– Branchial cyst (in ~ 20% of cases).
Pressure effects *and* ***disturbance of (physiological) functions*** may help to determine the exact tissue/organ of origin and complications.

Pain

– The **pain** is absent in benign tumor, early stage of a malignant tumor, or if patient is consuming analgesic.
– Experience of pain before development of swelling ⇒ inflammation, osteosarcoma.
– Beginning of pain after development of swelling ⇒ (malignant) tumor. Exception—osteosarcoma.
– The presence of pain or unusual sensations in case of a malignant swelling may suggest perineural spread. Therapeutic implication: Involvement of a sensory nerve in the vicinity should be specifically looked for, and included in resection if found to be involved to prevent recurrence of the tumor.

Fever usually suggests infection.
Examples of ***multiple*** swellings: Pyemic abscesses, enlarged lymph nodes, sebaceous cysts, warts, lipomas, neurofibromas, etc.
Possible causes *of* ***significant loss of weight*** (applicable to a case of swelling): TB, malignancy, AIDS.
Recurrence of the swelling after apparently complete proper excision may suggest malignancy. A cyst, any benign tumor may recur if excised incompletely.

POSITION AND MANEUVER

During the physical examination, a patient is required to adopt some specific positions, and also some maneuvers need to be carried out. The aim of these is to make the swelling most prominent to its fullest extent. Examples:

1. In a c/o an inguinoscrotal swelling, begin the examination with the patient in standing position with legs apart, and also ask the patient to cough. Later the patient is examined also in lying down position.
2. A patient with a swelling in the neck should be examined in sitting position.

LOCAL EXAMINATION

INSPECTION

Number?
Common causes of multiple swellings—pyemic abscesses, enlarged lymph nodes, sebaceous cysts, warts, lipomas, neurofibromas, etc.

Site?
It is preferable to describe the site in proper anatomical terms and in relation to specific anatomical structures (-landmarks) e.g. relation of an inguinoscrotal swelling to pubic tubercle.

– Goiter: Anterior aspect of inferior portion of the neck
– Carbuncle: Frequently occurs on the nape of the neck, shoulder and back

www.Ebook777.com

– Cystic hygroma: Commonly occurs in the neck and axilla
– Sebaceous cyst: The commonest site is face; common sites for multiple sebaceous cysts—scalp and scrotum
– Congenital sequestration dermoid cyst: Occurs anywhere in the midline, at the outer/inner angle of the orbit, or behind the ear
– Basal celled CA: Most commonly occurs on the face, superior to an imaginary line joining angle of the mouth to tragus of the ear
– TB lymphadenitis: Cervical > axillary > inguinal.

Extent?
Note the extent in transverse and craniocaudal directions, OR in relation to certain anatomical landmarks in the vicinity of the swelling.

Margin?: Determine the margin on inspection with the help of the angle formed with the surrounding skin—as follows:



- Obtuse angle ⇒ Ill-defined margin



- Almost right angle ⇒ Well-defined swelling
- Acute angle = Pedunculated swelling



Note: Margin cannot be defined easily when a swelling is situated in deeper tissue planes.
– Examples of swelling with well-defined margin: Cystic swellings, tumors, chronic inflammatory swellings, keloid, etc.
– Examples of swelling whose margin is ill-defined: Cellulitis, abscess, lipoma (margin of a lipoma is not well-defined on inspection, but usually well-defined on palpation), malignant swellings, chronic inflammatory swellings.
Margin of a chronic inflammatory swelling can be well-defined or ill-defined.
– An example of swelling having 'pedunculated shape': Papilloma. *Pedunculated shape usually rules out malignancy.*

Shape?
Shape may help to determine the organ affected and/or pathological nature of the swelling.
How can it help in determining the organ affected?
A principle: *'Even when enlarged, organs usually retain their shape'; conversely, shape of the swelling may give an idea about the organ affected* e.g. thyroid, parotid, rib, liver, spleen, stomach, etc.
How can it help in determining the pathological nature?
– Swelling having regular shape viz. hemispherical/ovoid suggests cystic pathology, or benign nature if it is a tumor
– Examples of swelling having irregular shape: Malignant tumor, keloid
– 'Cold abscess' in a muscle takes the shape of the muscle.

While describing the shape of a swelling on inspection, better use the expression—'the swelling **looks** _____ (hemispherical / ovoid / irregular, etc.)', because palpation-finding may not correlate with the inspection finding.

Size?
In relation to duration, the size indicates progress of the swelling.
Inflammatory swellings usually do not attain large size because they soften and burst, while tumors may attain large size.

Pulsations?
The best way to notice pulsations is to observe the swelling tangentially.
Possible causes of pulsations:
– Aneurysm/AV fistula
– 'Transmitted' from an artery lying underneath the swelling
– Rapidly growing highly vascular sarcoma.

(Visible) Impulse-on-coughing?
This clinical test is applicable only to the swellings related to body cavities; hence, to the swellings related to head, neck and trunk (including groin).
An impulse-on-coughing is said to be present, when on coughing / sneezing / crying, the swelling expands momentarily, **OR** the swelling increases in size, **OR** a swelling appears where there was none.
The swelling **must expand**. **Slight movement-on-coughing should not be considered as +ve test.**

Free ebooks ==> www.Ebook777.com

Prerequisites

There must be 2 hollow cavities in communication with each other, such that a rise in pressure in the deeper and necessarily larger cavity must be freely transmitted to the superficial cavity.

Examples of swellings with visible expansile impulse-on-coughing

A. Clinically in the groin:
- Inguinal hernia (reducible and even irreducible)
- Saphena varix
- Iliopsoas abscess.

B. In other regions of the body:
- Encephalocele, meningocele, meningomyelocele
- Laryngocele
- Lumbar abscess, intercostal cold abscess, paravertebral cold abscess, empyema necessitans.

Surface?

Keep in mind: Surface of a swelling lying beneath the deep fascia or in deeper cavities appears smooth, even if it may be actually uneven, as for example in subfascial lipoma.

Surface (essentially of a superficial swelling i.e a swelling lying superficial to deep fascia)

Smooth (i.e. even/regular)	Uneven
Examples: Acute inlammatory swelling like cellulitis, acute pyogenic abscess; cystic swelling, benign tumor	– Lobular—an example: Lipoma – Nodular—examples: Multilocuted cyst, a mass of enlarged lymph nodes – Irregular ⇒ cancer – Cauliflower apperance ⇒ sqamous celled CA – Branching processes ⇒ papiloma

Skin

- Redness?

  Causes of redness: 1. Acute/chronic non-specific inflammation *even without skin involvement*.
  2. 'Cold abscess' on the verge of bursting.
  3. **A cancer infiltrating the skin.**
  4. Irritation / local applications of medicines or fomentation / repeated manipulations/any other cause of dermatitis.
- Red / purple color? ⇒ surface capillaries
- A dull blue hue? ⇒ **a large deep-seated vessel or vascular lesion**
- Edema?: Causes—inflammation, malignancy
- Multiple sieve-like openings showing beads of pus/underlying grayish-white necrotic mass? ⇒ carbuncle
- Ulceration (± fungation)?

  Causes:
  - Malignant tumor OR chronic inflammation (due to invasion and necrosis)
  - In huge benign swellings / tumors (due to stretching, thinning and breaking down of skin (there is no invasion)).

  The swelling is then called 'ulcerated swelling'.
- Sinus? - Commonly suggests tuberculosis
- Orange-peel appearance? = peculiar edema of the skin suggesting infiltration of the skin by a malignant tumor
- Loss of hair? ⇒ sebaceous cyst
- Punctum? ⇒ sebaceous cyst
- Pigmentation? ⇒ moles, tattoo marks, post-RT.



Fig 15-1 Congenital AVM

www.Ebook777.com



Fig 15-2 Orange-peel appearance

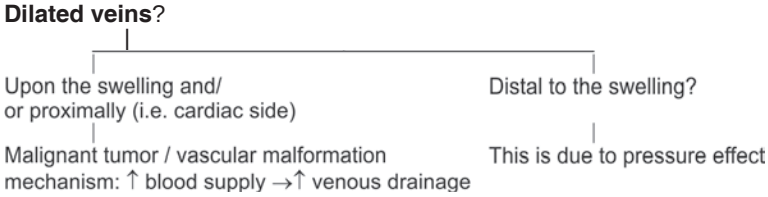


Fig 15-3 Dilated veins

Scar: Note the site, disposition, color, shape and size.

– Disposition i.e. whether in or across a skin-crease?
– Color?:
  - Red ⇒ recent scar
  - Pale/hyperpigmented ⇒ long-standing scar
– Shape: Regular OR irregular?
  - Regular e.g. straight / curvilinear / S-shaped, etc. ⇒ scar following a surgery
  - Irregular shape, generally ⇒ scar following an injury
– Size?:
  - A hair-line scar ⇒ 'healing by first intention'
  - Broad and puckered scar ⇒ 'healing by second intention' (the past e/o sepsis)
– Any crack in the scar? – This point is particularly important in a chronic scar of burns / radiation, as it may suggest development of cancer (SCC).

Dilated veins?



Color?

– Black ⇒ melanoma
– Red / purple ⇒ AV malformation, dermatofibroma, dermatofibrosarcoma protuberans
– Blue / bluish may be indicative of a deeper AVF; dermatofibroma, dermatofibrosarcoma protuberans, mucous cyst, ranula
– Yellowish-brown ⇒ dermatofibroma
– Reddish-brown: Could be due to rhabdomyosarcoma, which is basically a deep-seated tumor that often only presents when it projects into the skin as a reddish brown tumor.

Surrounding area?

– Cellulitis ⇒ infection
– 'Satellite nodules'/'Intransit' nodules may be seen in a case of malignant melanoma.

Closely related structures/organs?

Pressure effects?: Examples: Edema, wasting of muscles.

Free ebooks ==> www.Ebook777.com



Fig 15-4 Malignant melanoma—'Intransit' nodules

PALPATION

Temperature?
Normal / increased / decreased?
Acute inflammatory swelling, sarcoma → increased local temperature.

Tenderness?
Basically, tenderness ⇒ tissue injury, whether due to trauma or inflammation.

Number?
'A single swelling' on inspection may turnout to be actually 'many swellings' on palpation.

Site and extent?
Especially extent on palpation may differ from that on inspection.

Margin
- Well-defined OR ill-defined?
- Does it 'slip'?

Clinical method
Principle: Consistency of a swelling is different from that of the surrounding normal tissue.
Procedure for defining the margin: Start palpation from the normal tissue towards the swelling (from different directions), and note change in consistency as the swelling is reached.

Sudden and maximum change in consistency	Indefinite change in consistency
⇓	⇓
'Well-defined margin'	'Ill-defined margin'

Procedure for 'slipping margin': Slide the fingertip from the swelling towards the normal tissue at right angle to the margin. If the fingertip slips down, then the margin is said to be 'slipping margin'.
Slipping margin denotes solid benign tumor like a lipoma, fibroadenoma, etc. or encapsulated structure like a lymph node, rather than a cyst. Reason: Pressure of the fingertip tends to displace fluid from that pressure-point, bringing together the opposite walls of the cyst. Hence, a cyst does not usually slip, unless it is tense.
However, the margin of a diffuse lipoma and a solid benign tumor in deeper tissue planes will not 'slip'.

Shape?
What may have looked round on inspection may turnout to be ovoid on palpation.

Size?
Actual size on palpation may be more than that judged on inspection.

Vascular thrill = Vibrations felt on palpation. It is also defined as a palpable murmur.
Causes: A-V fistula, narrow segment of an artery, a narrow opening of the heart.

www.Ebook777.com

Pulsations?
Keep flat of fingers on the swelling for a few minutes to feel for pulsations.

Causes of pulsatile swelling

1. An aneurysm/AV fistula → expansile pulsations = 'true pulsations'.
2. A highly vascular swelling or tumor also → 'true pulsations'. Examples: Rapidly growing highly vascular sarcoma, osteosarcoma, encephalocele, pulsatile exophthalmos, toxic goiter, pulsatile liver in tricuspid incompetence.
3. A swelling overlying or underlying an artery → 'transmitted pulsations'.

Clinical criteria/tests differentiating aneurysm from other pulsatile swellings: Refer to chapter 18.

Impulse-on-coughing?
This clinical test is applicable only to the swellings related to body cavities; hence, to the swellings located in head, neck and trunk (including groin).

– Present OR absent?
– 'Expansile' OR 'thrill-like'?

Clinical method and inference

– Grasp the swelling firmly
– If the form of the swelling is such that it cannot be grasped, then place fingers upon the swelling firmly
– Ask the patient to cough
– ↑ in tension and expansion of the swelling ⇒ +ve result
– In a small swelling, expansion may not be appreciated. ↑ in tension must be felt at the least to qualify for +ve result. ↑ in tension will be appreciated only by the clinician performing the test
– A 'thrill' (more of thrill and less of expansion) suggests a vascular nature of the swelling. Examples: saphena varix, varicocele, lymph varix.

Fallacies:

1. A slight movement should not be considered as a +ve result.
2. Femoral hernia, even if uncomplicated, *may not* exhibit an impulse-on-coughing.

Examples of swellings with impulse-on-coughing.

A. Clinically in the groin:
   – Inguinal hernia (reducible and even irreducible)
   – Saphena varix
   – Iliopsoas abscess.
B. Clinically in the scrotum: Varicocele and lymph varix.
C. In other regions of the body:
   – Encephalocele, meningocele, meningomyelocele
   – Laryngocele
   – Lumbar abscess, intercostal cold abscess, paravertebral cold abscess, empyema necessitans.

Mobility

Principle

While eliciting mobility, the bone and occasionally some deeper soft but fixed tissues (like intercostal muscles, serratus anterior for example) are considered to be fixed points; so that when a swelling is mobile, usually it means that it is not fixed to bone or has not infiltrated the bone.

The clinical test of mobility may help to determine

a. Whether the swelling is bony (i.e. arising from bone) or otherwise.
b. If not bony, then the probable soft tissue/organ of origin.
c. Probable pathology. By virtue of other points in physical examination if a swelling is thought to arise from a particular tissue or organ, then one can also expect the range of mobility that the swelling can enjoy. Malignancy or chronic inflammation could be responsible if mobility is restricted to a degree that is much less than expected.

Free ebooks ==> www.Ebook777.com

Attributes of mobility tested

1. **Mobility in certain normal acts** like protruding the tongue, swallowing, deep breathing, etc.
   Examples: (a) Mobility 'during protrusion of tongue' is tested for a swelling thought to be attached to the thyroglossal tract e.g. thyroglossal cyst. (b) Mobility 'during swallowing' is tested for a swelling thought to be lying deep to the pretracheal layer of the deep cervical fascia e.g. a thyroid swelling. (c) Mobility 'during deep breathing' is tested for lumps related to the liver, gallbladder, stomach, spleen, colonic flexures, kidney.
2. **Extent of mobility**
   a. Freely mobile in all directions ⇒ – A lump arising from a freely mobile organ like ovary
      – A lump with a long pedicle e.g. ovarian cyst, floating kidney, floating spleen.
   b. Does the swelling appear to be more freely mobile in one axis than the other (at right angles)?
      Examples: A lump arising from the transverse colon/transverse mesocolon, greater omentum is more freely mobile in craniocaudal axis than any other axis.

Anatomical plane and fixity?

Every swelling lies in some or the other anatomical plane, and in that plane it may be attached to (= fixed to) some or the other structure depending upon the tissue of origin e.g. nerve (neurofibroma), artery (aneurysm), etc. In the course of time as the swelling enlarges, it may not, or it also may involve additional tissue plane or structure, depending on its pathological nature.

Cysts and benign tumors may remain confined to their original tissue plane, whereas malignant tumors and chronic inflammatory swellings usually involve > 1 anatomical plane due to infiltrative process or adhesion respectively.

There are various clinical methods/tests to determine the anatomical plane/s to which the swelling is confined, and structure/s to which it is attached (= fixed).

1. Skin / mucous membrane

Normally, skin overlying the scalp, palms and soles cannot be pinched or lifted; hence, determination of fixity of a swelling to the skin in these areas is practically not possible. Similarly, determination of fixity to mucous membrane overlying the hard palate and gums is not possible.

Clinical methods

a. *Pinch* the skin / mucous membrane in various areas overlying the swelling. If that is possible over the **whole** surface of the swelling then it suggests that the swelling is free from skin / mucous membrane.
b. *Glide* (i.e. roll) the skin with flat of fingers upon the swelling. If gliding is easily possible, **without causing dimpling anywhere on the whole surface** of the swelling, then it suggests that the swelling is free from skin.
c. *Lift* up the skin beyond the periphery of the swelling. If that leads to lifting up of the swelling as well, **without causing dimpling anywhere on the whole surface** of the swelling, then it suggests that the swelling lies in the plane of skin.

Development of dimpling while performing the above tests suggests attachment of the swelling to the skin by fibrous septa/duct/adhesions/infiltration.

2. Subcutaneous tissue

This is deduced by exclusion i.e.—a swelling can be considered to lie in the subcutaneous tissue plane if it does not seem to lie either in the skin or in the muscle.

3. Muscle and deep fascia

Principle: Put the concerned muscle in isometric contraction so as to make it *hard and immobile*, and re-examine the swelling for its prominence, definition of its margin, and mobility, to draw various conclusions.

Contraction of a muscle, when made to occur against resistance, is called 'isometric' contraction in which tone of the muscle increases making the muscle *hard and immobile*. The correct term is 'hard'. *'Taut' is incorrect term*. Passive stretching makes a muscle 'taut'—that will not make the muscle hard, making the test invalid. For example, active flexion of the hand against resistance → flexors of the forearm becoming 'hard/rigid' whereas passive extension of the hand → stretching of the flexors of the forearm, making them 'taut'.

www.Ebook777.com

Contraction of a muscle, made to occur without applying resistance is called 'isotonic' contraction in which only few fibers of the muscle contract leading to insignificant change in the tone of the muscle. The whole muscle-belly does not become hard and immobile, and makes the test invalid.

Clinical method

- Decide the muscle or the group of muscles to be tested
- Explain the steps to the patient. Make sure that the patient understands exactly what s/he has to do
- Practice the steps a couple of times. Practicing helps particularly when the action of a muscle is a bit complex e.g. action of SCM
- Put the appropriate muscle or the group of muscles in isometric contraction and observe any change in the attributes of the prominence, definition of the margin and mobility of the swelling (that was examined and made a note of earlier).

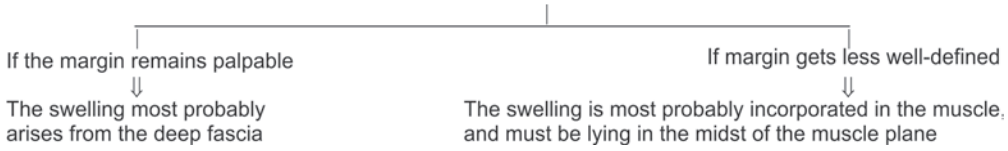
Inference

a. The swelling becoming more prominent suggests that the swelling lies in the plane superficial to the muscle.

b. The swelling becoming less prominent and/or margin of the swelling becoming less well-defined (i.e. gets difficult to feel) suggests that the swelling lies in the deeper layer of the muscle plane, or deeper to the entire muscle.

Swelling in the deeper layer of the muscle plane	v/s	*Swelling deeper to the muscle plane*
In relaxed state, mobility is more in transverse axis *cf* longitudinal axis.	|	In relaxed state, the degree of mobility may be equal in either direction.

c. If the size of the swelling remains unaltered, and mobility of the swelling is restricted to some extent, but



d. Restriction of mobility by and large suggests that the swelling is fixed to the muscle.

4. **Tendon**
   a. In relaxed state of the muscle concerned, the swelling is more mobile in the transverse axis than in the longitudinal axis of the muscle.
   b. On isometric contraction of the muscle concerned, mobility is restricted / abolished.

5. **Long structures like vessels, nerves, etc.**
   Swelling can be moved across (i.e. at right angles to their long axis) but not, or much less along the direction of their long axis.
   Swellings arising from nerves are more freely mobile than swellings arising from the vessels.

6. **Bone**
   The swelling arising from a bone has no independent mobility. In other words, a bony swelling does not move apart from the bone; it moves only along with the bone.

Surface?: Smooth looking surface on inspection may actually turnout to be bosselated on palpation.

Consistency?

- Soft/firm/hard/soft-to-firm/firm-to-hard/crepitus?
- Is it uniform all over the swelling OR is it variegated (different consistencies in different parts of the swelling)?

  Cysts and benign tumors usually have a **uniform** consistency.

  Inflammatory and malignant swellings may have a **variegated** consistency.

Out of the 3 forms of matter, 'solid' is the most consistent because it has fixed volume and fixed shape. 'Liquid' has fixed volume but variable shape. 'Gas' has variable volume as well as variable shape.

Free ebooks ==> www.Ebook777.com

While determining the consistency of a swelling, what one is actually determining is the amount of possible change in the shape of the swelling that can be brought about by applying pressure. The points to be noted are: 1. the amount of pressure required to produce the change, and 2. the amount of resistance felt by the fingers.

a. A marked change in the shape on applying just a slight pressure ⇒ '**soft**' consistency.

If only a part of an overall swelling exhibits softening, then note if the area of softening is at the center of the swelling or is it 'eccentric'?

- Normal anatomical structures having soft consistency: The female breast, and ear-lobule. They yield easily to pressure and do not show much recoil / rebound
- *Examples of swelling with soft consistency*: A-V malformations, cystic swellings containing fluid under minimal tension, lipoma (Fat is liquid at body temperature, hence fatty swellings are soft), and highly vascular sarcoma.

If collection of fluid is at one place within the swelling, then that spot feels soft in comparison to the rest of the swelling.

b. A slight change in the shape on applying moderate pressure ⇒ '**firm**' consistency.

- An example of a normal anatomical structure of firm consistency: tip of the nose
- *Examples of swelling with firm consistency*: Benign tumors like lipofibroma, cystic swellings containing fluid under moderate tension, (chronic) inflammatory swellings (as they consist of fluid exudates and a few cells).

c. No change in the shape even on applying maximum pressure ⇒ '**hard**' consistency.

Examples: Bony swelling (osteoma, etc.), carcinoma, calcification in a quiescent inflammatory lesion, cystic swelling containing fluid under (great) tension, etc.

Malignant tumors are mainly composed of cells; hence their consistency is firm or hard.

In carcinomas, marked fibrosis → hard consistency.

(In 'stony' hardness (seen in carcinomas), resistance offered by the swelling is > than that in 'bony' hardness.)

d. **Sometimes** the consistency may not fit exactly in the above-mentioned types. In that case, one may take liberty to describe the consistency as '**soft-to-firm**', '**firm-to-hard**'.

e. **Crepitus** ⇒ • Surgical emphysema (air) (causes: # ribs, rupture of esophagus, postoperative air in the wound)
• Gas gangrene (gas produced by infection).

Note

Although often mentioned, what exactly does **'cystic'** consistency mean? Ideally there is nothing as 'cystic' consistency. Consistency of a cystic swelling will vary according to the tension of fluid within the cyst. A swelling can be cystic, but its consistency should not be described as cystic.

Fluctuation?

This test determines the presence of fluid (or gas) in a swelling.

Clinical method

A. For *pendulous swellings like hydrocoele, > 2 cm in diameter*: Circumferential technique is used.

- Place the displacing and the perceiving hands at either pole of the swelling. Apply quick sharp and adequate pressure by the displacing fingers. Perceiving a pressure-wave at the opposite pole + spreading out of the perceiving fingers ⇒ +ve result for fluctuation.

B. For *swellings which are not pendulous, and > 2 cm in diameter*

- If the swelling is mobile, then ask an assistant to fix it
- Place the index fingers at 2 points near the periphery
- Give short, sharp taps by one index finger (the 'displacing finger')
- The other index finger (the 'perceiving finger') makes an attempt to feel the pressure-wave
- Repeat the same procedure in the axis at right angles to the previous one.

Fluctuation felt in both the axes only is considered +ve.

www.Ebook777.com

The purpose of carrying out the procedure in 2 axes at right angles to each other is to rule out the possibility of (a fleshy) muscle being tested. Normally, a fleshy muscle is fluctuant across but not in the direction of its muscle fibers.

C. For *swellings ≤ 2 cm in diameter*: Various tests are described in literature, but they are not reliable.

Inference: +ve test ⇒ cystic swelling containing fluid ↓ mild to moderate tension.

Fallacies

- False –ve result is obtained in a cystic swelling that is thick-walled or extremely tense or extremely soft (lax) or deep lying, or huge
- False +ve result may be obtained in swellings with soft consistency like lipoma, myxoma, a highly vascular sarcoma, etc.

Common examples of fluctuating swelling:

- Cyst
- Chronic abscess—nonspecific or tubercular*
- Degeneration in a solid tumor**.

'Cross fluctuation'

= transmission of fluid-pressure-wave in 2 cystic swellings communicating with each other.
Examples: Meningocele ↔ anterior fontanelle, 'bilocular' hydrocele.

Transillumination?

Purpose—to ascertain cystic nature of a swelling containing clear fluid (including serum and lymph and highly refractive fat).

Prerequisites

A bright pinpoint light source in dark room. It is better to perform the test in a dark room rather than performing it in the day-light using a paper tube. If anyway, the test must be performed during the daytime, then an X-ray roll applied in close contact with the swelling must be used.

Principle

- Light can pass through clear fluid, serum, lymph (and the skin and subcutaneous tissue)
- Red glow is due to RBCs in capillaries
- *In c/o swellings that are not brilliantly translucent, the disappearance of a faint red glow rather than its presence is more convincing proof of the presence of transillumination*
- *Precaution*: In c/o a scrotal swelling, the testis should never be pressed hard while making the swelling tense
- *Mistake to be avoided*: Not making the swelling tense.

Procedure

- Make the swelling tense by completely and tightly stretching the skin overlying the swelling
- (Gently) Press a small torch against one side of the swelling
- Observe from the opposite side of the swelling
- Switch-on the torch; switch-off the torch.

Inference

- Brilliant translucent swellings: Thin walled cysts containing clear fluid e.g. hydroceles, cysts of the epididymis, cystic hygroma, rannula, bursa, ganglion, meningocele
- Translucent swelling, but not 'brilliantly': An example—spermatocele
- Non-translucent swellings: Cystic swellings not containing clear fluid e.g. hematocele, pyocele, chylocele, dermoid cyst, sebaceous cyst, etc; and all other swellings of 'non-cystic' nature i.e. inflammatory, neoplastic, etc.

---

* Induration at the periphery ⇒ nonspecific abscess. In tuberculous ('cold') abscess there is no induration at the periphery of the swelling.

** Softening at the center goes in favor of a chronic abscess, whereas eccentric softening ± variegated consistency suggest degeneration in a solid tumor.

Free ebooks ==> www.Ebook777.com

Fallacies

– If wall of the sac/skin/subcutaneous tissue/all have become thick, transillumination will be falsely –ve. For example, long-standing chronicity leading to calcification of the wall, recurrent attacks of inflammation leading to fibrosis of the wall, etc.
– False +ve result: Hernias in neonates and infants will be translucent if the gut (which is thin-walled in these age groups) is distended with gas.

Moulding?
= Indentation.
On pressing with a fingertip (at least for about half a minute), the swelling gets moulded (indented); stays indented, and takes a long-time to return back to normal shape.
Examples: Cystic swellings containing pultaceous material e.g. sebaceous cyst, dermoid cyst, colon loaded with formed feces.

Compressibility?
– On compressing: Does the swelling diminish in size OR disappear?
– (And after removing the compression): Does it refill spontaneously slowly OR does it refill in series of throbs synchronous with heart beat?

Examples:
– A-V malformation, aneurysm
– A nevolipoma may be slightly compressible.

Fallacy: A saccular aneurysm filled with a thrombus may become incompressible.

Reducibility?
On gentle compression, contents are pushed back partially/completely into the adjacent deeper—essentially larger cavity, so that the swelling diminishes in size or completely disappears—and reappear only on an active maneuver like standing / coughing / sneezing / crying.
Examples: A reducible hernia, saphena varix, varicocele, lymph varix, meningocele.

Compressible swelling	v/s	*Reducible swelling*
On removing compression, the swelling reappears **spontaneously**		The swelling reappears **only on active maneuver** like standing/coughing/sneezing/crying, etc.

Surrounding area?

Closely related specific structures / organs?

Underlying bone and joint?

SPECIFIC CLINICAL TESTS

Various tests are carried out as applicable in the given case. They are discussed in relevant chapters.

PERCUSSION

– Resonant note ⇒ swelling filled with gas
– Dull note ⇒ swelling filled with solid/fluid matter.

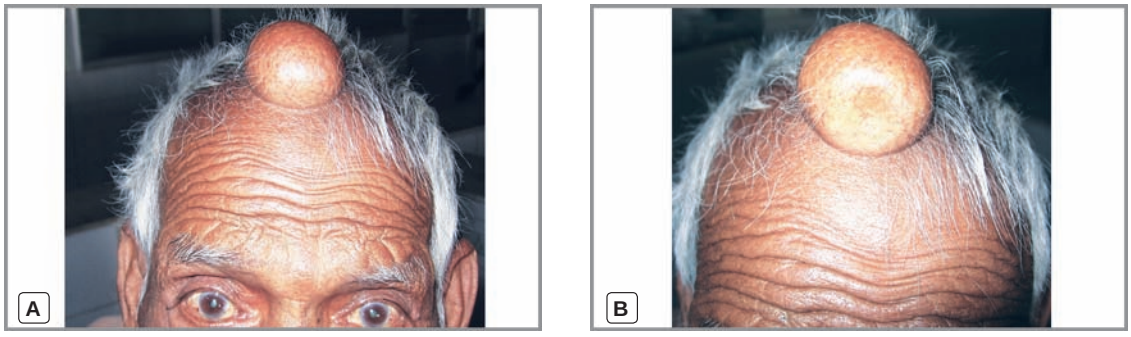
Clinical methods in a suspected case of hydatid cyst

a. 'Hydatid thrill'
Principle
A peculiar trembling or vibratory sensation is created due to floating daughter cysts striking against the endocyst.

www.Ebook777.com



Fig 15-5 A translucent swelling



Figs 15-6A and B (A) Sebaceous cyst; (B) Indentation on the anterior surface of the swelling after applying pressure

Procedure

- Place 3 fingers upon the swelling. The middle finger (plexiimeter) is placed firmly while the adjacent 2 fingers (the index and ring fingers—the watching fingers) are placed lightly to ascertain their sensitivity to vibrations
- Percuss over the plexiimeter
- Try to feel for a thrill. After feeling the thrill, do not lift the fingers off immediately instead, continue to place them on the swelling for a while for feeling 'after thrill'.

b. *'After thrill'* is felt due to continued strikes against the cyst wall by the freely-floating daughter cysts set in motion by percussion, even after percussion of the plexiimeter is stopped.

AUSCULTATION

Applicable to pulsatile and compressible swellings.

- *Bruit* (arterial) is an auscultatory sound produced by low frequency vibrations of the arterial wall caused by disturbances in blood flow
- *Murmur* is an auscultatory sound of vascular or cardiac origin. – It is a continuous buzzing sound which increases in loudness with each heart-beat
- *Hum* is an indistinct, low, prolonged sound. It is usually talked about in relation to a vein.

REGIONAL EXAMINATON

Lymph nodes

Occasionally, a band of muscle or an artery may be mistaken for enlarged lymph node. A lymph node can be rolled in 2 directions—up and down, and side-to-side. Neither a muscle nor an artery will pass this test.

Free ebooks ==> www.Ebook777.com

*Clinical method of palpation of **cervical lymph nodes***

- **Stand behind** the **seated patient**
- Palpate only **one side at a time** (to avoid cerebral ischemia)
- Flex the head slightly + slightly tilt it on the side to be palpated (to relax the muscles and deep fascia). This position is maintained throughout palpation of that side by the other hand resting on the head
- There is usual tendency of the patient to extend the head, which should be curbed
- Palpate first with **flat of the fingers** to find out the swelling, and then palpate with **fingertips** to find out the details.

Sequence: Submental, submandibular, preauricular, superior superficial cervical (= superficial to the upper part of the SCM), postauricular, occipital, deep cervical, supraclavicular, and unnamed scattered lymph nodes in the posterior triangle.

An additional useful technique of palpating supraclavicular lymph nodes from front

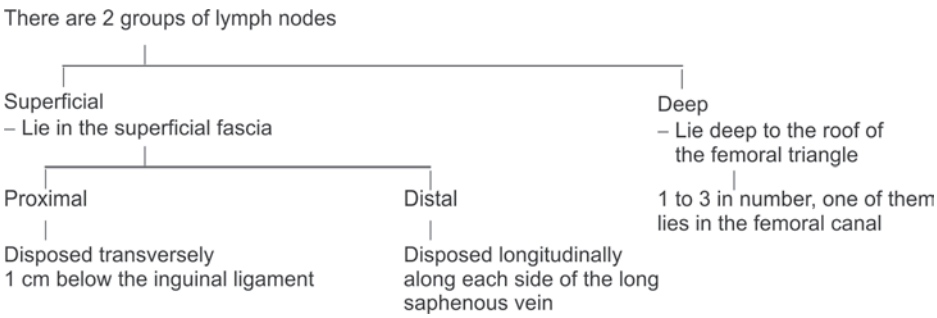
- Ask the patient to 'fold' the upper limbs (crossed forearms), and maintaining this folded position, ask the patient to flex the arms through 90° so that the forearms automatically move away from the trunk
- Now ask the patient to raise the assembly of the shoulders + folded arms up, and simultaneously protract the scapula
- These maneuvers deepen the supraclavicular fossa
- Palpate the fossae by gently poking the tips of fingers in, to detect any swelling/enlarged lymph nodes.

*Method of palpation of **axillary lymph nodes***

- **Stand facing** the **seated patient**
- Shoulder abducted fully. Insinuate fingers high in the axilla with flat of the fingers on the ribs. Bring down the abducted arm. Palpate the central group
- Insinuate the fingers as high as possible. Press the fingertips of the other hand in the supraclavicular fossa, and attempt to palpate the apical nodes bimanually (without hurting the patient), although it is difficult
- Grip the lower border of pectoralis major between fingers and thumb to palpate the pectoral (= interpectoral = anterior) group of lymph nodes
- **Change the hand** to palpate the lateral group against head of the humerus
- Palpate the posterior (= subscapular) group of lymph nodes against the posterior axillary fold **from behind.**
- For successful palpation, the musculofascial structures must be relaxed. Hence, if required, the examination may be carried out with the patient lying down in which position the muscles are completely relaxed.

*Palpation of **inguinal lymph nodes***

Abduct the thigh slightly to open out the groin, and then palpate the nodes.



Palpate THE OPPOSITE SIDE wherever applicable.

www.Ebook777.com

Stepwise Analysis of Data

Basically, is it **edema or a swelling**?

'Diffuse' eminence involving the entire circumference of a region of the body
↓
'Edema'
↓
Refer to the chapter 50

'Circumscribed' eminence = swelling.
A swelling usually does not involve the entire circumference of a region, although exceptions may be encountered.
↓
Step 1

OR

Acute?

- Duration: Few hours up to ~ a week *
- Progress: Dramatic over minutes / hours or 'rapid' over days
- Often associated with pain
- Patient may look distressed or sick
- Surface—smooth
- Pulsation ±: Example—false aneurysm
  (–Vascular thrill ⇒ A-V fistula)
- Consistency—soft or firm
- Fluctuation ±

Chronic?

- Duration > 3 weeks
- Progress:
  - Over weeks
    or slow over months / years
  - May remain stationary for a variable period (weeks / month / years) after initial growth.
- Disturbance of function is minimal unless the swelling is situated in a critical anatomical area, or is very large in size, or if pathology is advanced

* Some swellings that are basically acute may attain chronicity by virtue of natural history of the pathological process or inappropriate/inadequate treatment. Examples: Chronic nonspecific abscess, false aneurysm, traumatic A-V fistula, 'blood cyst', etc.

ARRIVING AT THE DIAGNOSIS OF **AN ACUTE SWELLING**

Step 2: **Physiological derangement**

- Degree of homeostatic imbalance?
  This may not be applicable in all cases, but will be applicable in anaphylactic reactions, and serious bacterial infections causing acute inflammatory swellings.

Step 3: **Tissue/organ of origin of the swelling**?

Clinical points which may give clues:

- Disturbance of function
- Exact site and extent
- Shape
- Mobility; anatomical plane and fixity.

Swelling may arise from a bone, a joint, or a soft tissue; and consequently labeled as a '**bony** swelling', '**articular** swelling', or a '**soft tissue** swelling'.

Step 4: **Etiopathology**?

Traumatic / allergic / acute infective inflammatory / acute non-infective inflammatory / Bleeding inside a joint or a pre-existing cyst.

Traumatic swelling (following soft-tissue injury, including unaccustomed exercise**)**

Criteria

- Appears immediately / within a few hours of trauma
- Increases in size for up to 48 hours and then gradually decreases
- Painful and tender
- Patient may look distressed due to pain and / or disability, but not sick

Free ebooks ==> www.Ebook777.com

– Usually disappears within 7 to 10 days
– Further increase in size + development of fever after a few days ⇒ infection. Then it can be called basically traumatic swelling complicated by sepsis.

Examples
– Skin and subcutaneous tissue: blebs (vesicles), contusion, hematoma, surgical emphysema, extravasation of body fluids like urine, etc; traumatic edema
– Muscles and tendons: Contusion, hematoma, complete rupture of belly of muscle or of tendon, rupture of muscle sheath producing 'hernia of muscle', traumatic myositis
– Bursa: Hematoma, traumatic bursitis (simple bursitis), rupture
– Joint: Traumatic synovitis and effusion, traumatic hemarthrosis.

Allergic swelling
(Due to immediate hypersensitivity reaction)

Criteria
– Occurs within seconds to minutes of exposure to an allergen (known / suspected / otherwise)
– Itching
– May be associated with other features of anaphylaxis viz.
  - Hoarseness of voice, stridor, wheezing (Respiratory features)
  - Tachycardia, hypotension (Cardiovascular features)
  - Nausea, vomiting, diarrhea (GIT features)
  - Rhinitis and edematous swelling of mucous membranes
– Duration up to ~ 24 hours.

- *Urticaria* = circumscribed wheals with raised serpiginous borders and blanched centers
- *Wheal* = a transient elevated lesion due to accumulation of fluid in the superficial dermis
- *Edema* – 'Angioedema' = massive edema in the dermis and subcutaneous tissue. Caused by vasculitis, or could be idiopathic.

Acute inflammatory swelling due to bacterial infection
Criteria
Basically an acute painful and a tender swelling of sudden mode of onset.
– Progresses 'rapidly' within days
– Regional lymphadenitis ±
– Restriction of movements/function of the part that is affected
– Fever ±
– Patient looks sick
– Ill-defined
– Redness of the overlying skin/mucosa
– Increased local temperature.

Acute non-infective inflammatory swelling
(= neither caused by trauma nor by infection)
It may be caused by immune (autoimmune, etc.), metabolic, hematological disorders, and a variety of other uncommon disorders.

Criteria
– More or less similar to those of acute infective inflammation except for the absence of regional lymphadenitis and the presence of additional clinical manifestations of a particular disease/disorder
– Fever and other constitutional symptoms may be present even in non-infective inflammatory disorders.

Step 5: **Associated disorder—**closely related OR otherwise?

www.Ebook777.com



Fig 15-7 Urticaria



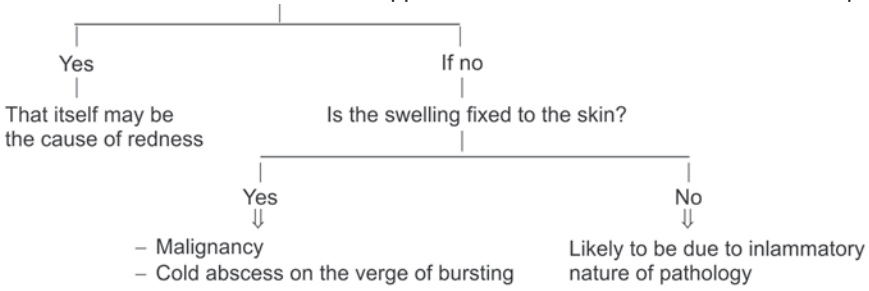


Figs 15-8A and B Examples of angioedema

ARRIVING AT THE DIAGNOSIS OF **A CHRONIC SWELLING**

PRINCIPLES AND PITFALLS

1. Although sarcomas occur frequently in the young and carcinomas in the older/elderly, yet instances of reverse occurrence are quite common.
2. Inflammatory swellings usually do not attain large size because they soften and burst, while tumors may attain large size.
3. Determination of the cause of redness of the skin overlying the swelling:
   Has there been a h/o irritation / local applications of medicines or fomentation / repeated manipulations?



4. Benign tumors and cysts are adherent to only one layer, while chronic inflammatory and malignant swellings are usually adherent to > one layer.
5. In a chronic non-specific abscess there is induration at the periphery, while in a tuberculous cold abscess there is no induration at the periphery.

Free ebooks ==> www.Ebook777.com

6. Cystic degeneration in malignancy v/s softening in a chronic granulomatous swelling: In c/o malignancy, softening is eccentric whereas in a granuloma, softening is at the center.
7. A swelling arising from skin / subcutaneous tissue will fit in one of the 3 categories on clinical grounds: (a) which can be confidently diagnosed, (b) which is most probably diagnosed, and (c) which cannot be diagnosed.

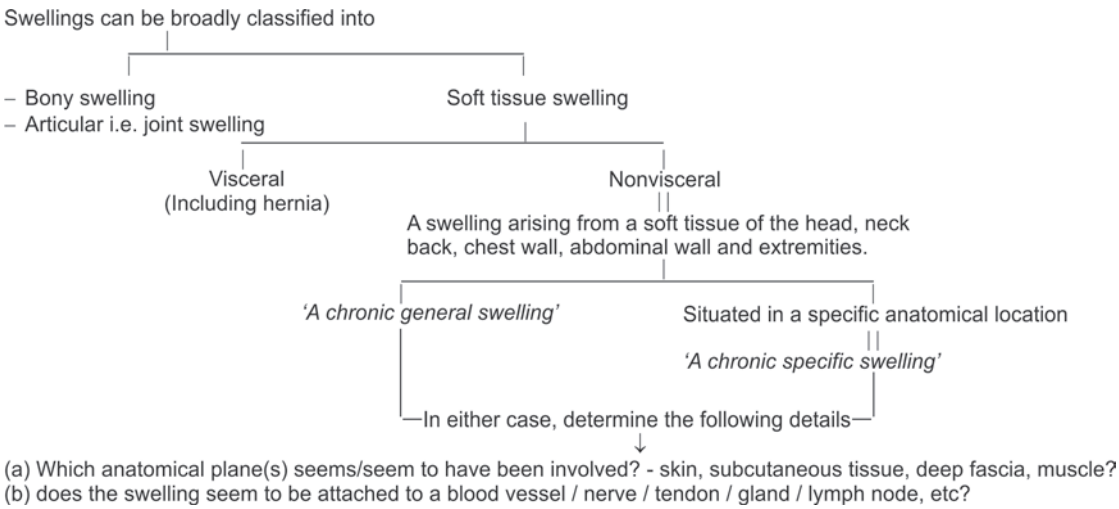
   For swellings in the 2nd category (i.e. b), a biopsy is mandatory.

   For swellings in the 3rd category (i.e. c), biopsy/excisional biopsy is a must. It is futile to give a long list of various possible clinical diagnoses.
   - *Skin-swellings which can be confidently diagnosed clinically*: Sebaceous cyst, papilloma, infective warts, condylomata, callosity, corn, keloid, hypertrophic scar
   - *Subcutaneous tissue swellings which can be confidently diagnosed clinically*: Sebaceous cyst, lipoma, neurofibroma and ganglion.
8. All *swellings deep to the deep fascia (practically muscle-swellings)* are impossible to diagnose clinically, and must be subjected to cytological/histological examination.

   The commonest swelling is probably an intramuscular lipoma but the characteristic signs seen in a subcutaneous-lipoma are absent in a intramuscular lipoma due to tough dense unyielding deep fascia overlying it.

Step 2: **Tissue/organ of origin** of the swelling?

Clinical points which may give clues:
- Disturbance of function
- Exact site and extent
- Shape
- Mobility; anatomical plane and fixity.



***Bony** swelling*:
- The swelling cannot be moved over the bone; it moves when the bone is moved
- Consistency is *usually but not necessarily* hard.

Conditions which may be mistaken for a bony swelling
- Normal bones e.g. prominent ribs in scoliosis, sacral promontory, tarsal bones in flat foot
- Hypertrophied bones due to overuse/in A-V fistula
- Calcification in soft tissue
- A tense cyst in close relation to bone

www.Ebook777.com

– Hot cross bun skull in rickets
– Osteitis deformans: Occurs in old age with chronic enlargement and bending of the bones of skull, spine, clavicle, pelvis and long bones of the limbs.

***Articular** swelling*: *Basically*, a swelling in the region of a joint.

Differentiating points
– Obliteration of fossae around the joint
– Swelling in the region of a joint corresponding to the extent of the capsule and synovial membrane of the joint.

Supporting points
– Pain in the region of a joint particularly on movements
– 'Night cries' due to relaxation of spasm, provided spasm is present otherwise through the day
– Stiffness and limitation of movements
– Deformity and fixed attitude.
– Involvement of multiple joint regions

When a joint is diseased, the patient may complain of pain in the region of the joint without any swelling (arthralgia), or may develop swelling in the region of the joint with or without other symptoms like pain, stiffness, etc.

A joint may swell due to any one of the following 4 conditions:
a. Synovitis: Inflammation of synovial membrane only.
b. Arthritis: Disease of articular cartilage mainly, but other structures of the joint also may be involved.
c. Hydrarthrosis: Collection of a large amount of fluid mainly.
d. Sympathetic effusion: Primary disease is not in the joint but in adjacent bone/soft tissue; the joint being swollen due to collection of clear fluid as a reaction. At first sight such a swelling may appear as a joint case. Differentiating points to suggest that joint is not the primary site of disease—(i) Swelling extends much beyond the extent of the capsule and synovial membrane. (ii) On detailed examination, evidence of the primary bone or soft tissue disease will be apparent.

The word 'arthritis' used in above-mentioned classification denotes synovitis or arthritis or hydrarthrosis. However, there is never a pure involvement of synovial membrane, cartilage or collection of fluid alone. Any one may be predominantly affected while remaining two are affected in either minute or dormant forms.

***Visceral** swelling*
= a swelling lying wholly or partly within a celomic cavity viz. cranial / thoracic / abdominal (intraperitoneal, retroperitoneal).

Clinical clues
– Disturbance of function/s of a specific organ/system related to a coelomic cavity
– Reducibility
– Impulse-on-coughing
– The swelling becoming less prominent/less palpable/impalpable, and/or becoming less mobile or immobile on putting the overlying parietal muscles into isometric contraction; e.g. on 'straight leg raising test' in case of an abdominal lump.

However, the absence of clinical clues does not necessarily rule out the possibility of a swelling being visceral.

Examples of visceral swelling
– Meningocele, encephalocele, a dumb-bell shaped congenital sequestration dermoid cyst (with an intracranial extension) and similar such swellings
– Empyema necessitans
– Hernia of the lung (in the root of the neck)
– Abdominal (including inguinal) hernia, etc.

Free ebooks ==> www.Ebook777.com

Chronic specific swelling

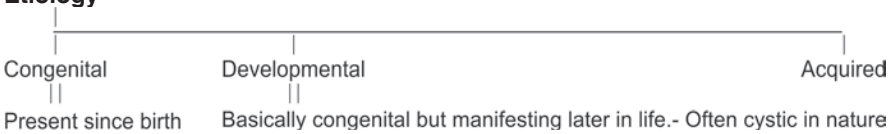
= a swelling situated in a specific anatomical area. A swelling arising in a specific anatomical area should be considered to be arising from the specific organ/tissue in that anatomical area as a rule; for example swelling of lymph nodes, thyroid, salivary gland, breast, etc.

Though it is possible for any swelling from a 'general' category to occur in a specific anatomical area, it is essential to make every clinical (and if required investigative) attempt to differentiate it from the specific swelling which is expected to occur in a specific anatomical area.

Specific swellings are discussed under separate headings (chapters) such as 'chronic lymph node swelling', 'swelling in the oral cavity', 'swelling in the region of the head/face', 'swelling in the region of the thyroid', 'swelling in parotid region', 'swelling in the midline of the neck', 'swelling in the region of the breast', 'inguinoscrotal swelling', etc.

Step 3: **Etiopathology**?

Etiology



Pathological nature?

The key clinical features to differentiate the pathological nature:

- Pulsatality, compressibility
- Reducibility, pseudoreducibility
- Duration, progress, regress, fever, loss of weight
- Shape, size, anatomical plane/s involved, uniformity of consistency, and regional lymphadenopathy.

Pathological nature of a swelling could be one of the following:

- Vascular
- Reducible
- Pseudoreducible
- Cyst / benign tumor
- Chronic inflammatory swelling / malignant tumor
- Miscellaneous e.g. keloid
- Specific, pertaining to a specific anatomical location e.g. multinodular goiter, cyclical nodularity in the breast, etc.

Cystic swelling and benign tumor clinically behave similarly in some respects. Chronic inflammatory swelling and malignant tumor also behave similarly in some respects.

Vascular swelling

It is safer to consider the possibility of a vascular swelling at the outset to avoid misdiagnosis and consequently wrong surgical treatment, which can be disastrous.

Differentiating criteria

a. Pulsatile OR compressible OR both pulsatile + compressible, and upon which a bruit / murmur / hum may be audible with a stethoscope. However, the absence of pulsations/compressibility does not rule out possibility of a swelling being vascular; an aneurysm may have been filled with a thrombus making it incompressible.
   A pitfall: Some nonvascular swellings also may be pulsatile e.g. rapidly growing highly vascular sarcoma, osteosarcoma, encephalocele, pulsatile exophthalmos, toxic goiter, pulsatile liver in tricuspid incompetence.
b. Palpable thrill. The absence of a thrill however, does not rule out the possibility of a vascular swelling.

Examples of vascular swelling: Aneurysm (both true and false), AV fistula.

Congenital AV fistulae may occur virtually anywhere. Those with a dermal component are easily visible *although it is important to remember that the clinically obvious cutaneous mark may represent only a small part of the total lesion (JUST THE TIP OF AN ICEBERG).*

www.Ebook777.com

***Reducible** swelling*

Examples:

a. In the groin: Inguinal hernia, femoral hernia, saphena varix.

b. In other regions:

- Meningocele, laryngocele, pharyngeal pouch
- Various abdominal hernias
- Hernia of hydrocele sac
- Hernia of muscle.

***Pseudoreducible** swelling*

On applying (gentle) pressure, the swelling *seems to* disappear mimicking reducibility. In fact, there is bodily displacement of the whole swelling under some organ or tough tissue plane.

Examples: 1. An encysted hydrocele of the cord located at the level of the root of the scrotum may disappear through the external inguinal ring into the inguinal canal under the cover of external oblique aponeurosis, 2. Partially descended (enlarged) testicle.

Clinical feature	*Cyst*	*Benign tumor*	*Chronic inflammatory swelling*	*Malignant tumor*
Duration	Months / years	Months / years	Weeks / months	Weeks / months
Progress	Slow. Later may become stationary	Slow. Later may become stationary	Initially may progress rapidly, f/b slow progress. Eventually may become stationary	Rapid persistent progress over weeks
Regress	±	No	±	No
Fever	Absent	Absent	±	±
Loss of weight	Absent	Absent	±	±
Shape	Regular	Regular	Regular OR Irregular	Irregular
Size, with respect to being large	May attain a large size	May attain a large size	Usually does not attain a large size; tends to breakdown before a large size could be attained	Less likely to attain a large size, although possible occasionally
Anatomical plane/s involved	Usually single	Usually single	> 1	> 1
Consistency	Uniform	Uniform	Variegated	Variegated
Regional lymphadenopathy	None	None	±	±

Differentiation between cyst and benign tumor

Clinical feature	*Cyst*	*Benign Tumor*
Regression	Possible	No
'Slipping margin'	Usually not	± Examples: A subcutaneous lipoma, fibroadenoma in the breast
Shape	Globular	Flattened
Moulding	± Examples: Sebaceous cyst, dermoid cyst	–

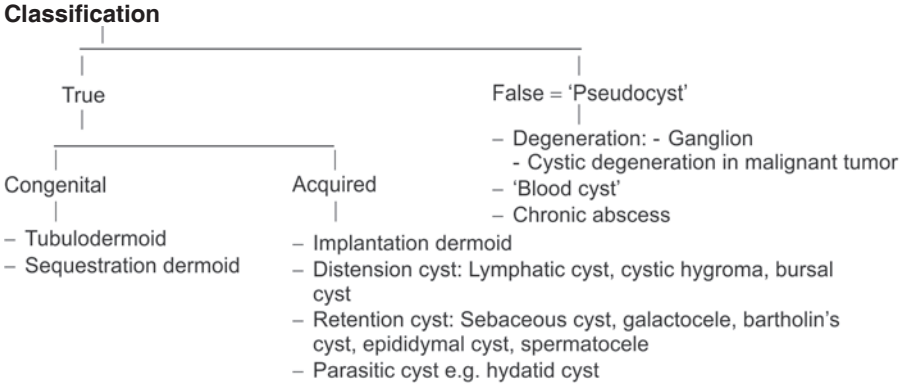
Free ebooks ==> www.Ebook777.com

Differentiation between chronic inflammatory swelling and malignant tumor

Clinical feature	*Chronic inflammatory swelling*	*Malignant tumor*
Duration	No specific diagnostic significance. However, could be > a year	Usually < a year
Progress	Initially rapid, f/b slow/stationary	Rapid and persistent
Pain	Local	Radiating
Number	May be multiple	Usually single
Dilated veins	–	+
Consistency	Soft / firm	Usually hard
Fluctuation	Usually present	Rarely present
Regional lymphadenopathy	Firm, and adherent to each other and to skin	Hard, and in later stages, adherent to deeper tissues and fixed

CYSTS

Classification



True cyst = a collection of fluid in a sac lined by epithelium.

Tubulodermoid
Arises because of the persistence of a segment of embryonic tubular structure. Examples: Thyroglossal cyst, urachal cyst, encysted hydrocele of the cord, hydatid of Morgagni, postanal dermoid.

Congenital sequestration dermoid
Arises from the persisting buried epithelium along the lines of embryonic fusion e.g. in the midline (anywhere), outer/inner canthus of the eye, anterior triangle of neck (branchial cyst).

Implantation dermoid
Due to (repeated) puncture wounds, a minute bit of epithelium is pushed in the subcutaneous tissue where it survives.

Distension cyst
Arises from distension of a pre-existing epithelium-lined space.

Retention cyst
Arises due to obstruction of the duct of the gland concerned.
Pseudocyst = collection of fluid in a closed space which is not lined by epithelium.

Ganglion
– Mucoid degeneration in a tendon sheath or synovial membrane of a joint
– Common sites—dorsum of the hand, knee and tarsal joints.
Cystic degeneration in a malignant tumor → development of softening that is *eccentric*.
Differentiating from granuloma: Softening in a granuloma occurs in the center.

www.Ebook777.com

'Blood cyst'

A hematoma may resolve into a cyst. Site where this is prone to happen—muscles in the loin, anterolateral aspect of the thigh and shin. Located between muscle, fascial or subcutaneous tissue planes and contains straw- or brown-colored fluid.

Complications of cysts

1. Bleeding → sudden increase in size; the cyst becomes painful due to sudden rise in tension within the cyst. It may also lead to significant pressure effects if located in a critical anatomical area; e.g. bleeding in a thyroid cyst leading to difficulty in breathing.
2. Rupture.
3. Torsion → sudden development of pain and tenderness.
4. Infection → acute cellulitis which may be followed by abscess formation.
5. Calcification.

CHRONIC INFLAMMATORY SWELLINGS

2 types of swelling occur under this heading: Chronically inflamed swelling, and chronic inflammatory swelling *de novo*.

Chronically inflamed swelling

– *Basically* an acute inflammatory swelling, which attained chronicity due to total neglect (lack of treatment)/ inadequate or inappropriate treatment
– Initially present features of acute inflammation subside; residual features in mild to moderate degree may persist
– An example—'antibioma'.

Chronic inflammatory swelling (Chronic inflammatory pathology *de novo*).

Criteria

– Mildly painful swelling with insidious mode of onset, gradually progressing
– Regional lymphadenitis ±
– Movements/function of the affected part may be restricted
– Fever ±
– Patient may or may not look sick
– Ill-defined swelling with redness of the overlying skin
– Slight increase in local temperature ±
– Tenderness—mild or absent.

Pathological classification of chronic inflammatory swellings: Nonspecific/specific [Tuberculosis (often leads to 'cold' abscess), actinomycosis, syphilis, etc.]

Chronic nonspecific cellulitis / pyogenic abscess

Differentiating points

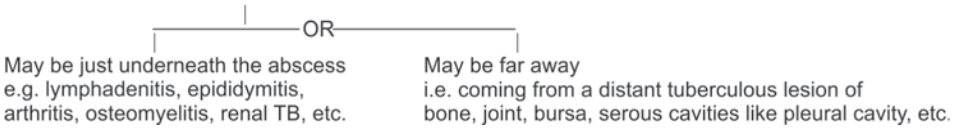
– Often occurs around a foreign body (including a worm like guinea worm)
– Softening at the center and induration at the periphery.

(Tuberculous) 'Cold' abscess

Differentiating points

(– If in a muscle, it takes the shape of the muscle in which it occurs e.g. psoas abscess.)

– Fluctuation +
– NO induration at the periphery
– A causative feeding lesion ±.



Incidentally, the feeding lesions of a tuberculous sinus also fall in the same categories.

Free ebooks ==> www.Ebook777.com

Actinomycosis

Differentiating points

– Prolonged course
– Site: Cervicofacial region / chest / abdomen (particularly RLQ)
– Essentially involves subcutaneous tissue
– Ill-defined, pigmented (bluish), firm consistency (indurated) swelling with areas of softening and multiples sinuses
– Openings of the sinuses are situated on top of the pustules
– Discharge from multiple sinuses contains yellow-colored granules of the size of a pinhead (Collect pus in a clear test tube and observe in a powerful light to see the granules)
– No regional lymphadenopathy.

Pathogenic agent

– actinomyces Israelii.

Mycetoma

Differentiating points

– Prolonged course
– Site: Foot
– Rest of the clinical features are same as in actinomycosis except for the type of granules.

Different types classified as per different pathogenic agents.

1. Actinomycotic mycetoma: Pathogenic agent—nocardia / streptomyces. Small / moderate sized yellow-red or yellowish white granules.
2. Mycotic mycetoma: Pathogenic agent—madurella group of fungi. Large sized dark or black granules.
3. Botryomycetoma: Pathogenic agent—mixed *bacterial* flora (Staphylococci, Streptococci, *Escherichia coli*, Pseudomonas, etc.); NOT fungi. NO granules.

Syphilis (Gumma)

Differentiating points

– Ill-defined margin
– Infiltration of the surrounding tissues e.g. gumma in the muscle will become adherent to periostium and subcutaneous tissue
– Firm consistency. Softening in the center.

HISTOLOGICAL CLASSIFICATION OF GENERAL SOFT TISSUE TUMORS

Tissue of origin ↓	*Benign*	*Aggressive*	*Malignant*
Adipose	Lipoma Angiolipoma	Atypical lipoma	Liposarcoma
Fibrous	Fibroma	Fibromatosis Example: Desmoid tumor	Fibrosarcoma
Fibrohistiocytic	Dermatofibroma	Dermatofibrosarcoma protuberans (*Syn*. Fibrous histiocytoma)	Malignant fibrous histiocytoma
Striated muscle	Rhabdomyoma	---	Rhabdomyosarcoma
Smooth muscle	Leiomyoma	---	Leiomyosarcoma
Synovium	Giant-cell tumor of tendon sheath	---	Synovial sarcoma
Mesothelium	Localized fibrous mesothelioma	---	Mesothelioma

Contd...

www.Ebook777.com

Contd...

Tissue of origin ↓	*Benign*	*Aggressive*	*Malignant*
Blood vessels	Hemangioma	Hemangioendothelioma	Hemangiosarcoma Hemangiopericytoma Kaposi's sarcoma
Lymph vessels	Cystic hygroma Lymphangioma	---	Lymphangiosarcoma
Lymph nodes	Virtually unknown	---	'Lymphoma' = Malignant lymphoma e.g. HL, NHL
Nerve sheath	Neurofibroma Neurilemoma	---	Malignant schwannoma
Uncertain	Myxoma	---	Alveolar soft-part sarcoma

Liposarcoma

– Usually arises from deeper tissues. Commonest sites—limbs and retroperitoneum
– Most frequently seen in older patients.

Fibroma

Considering the widespread distribution of fibrous tissue in the body, **true fibroma is of rare occurrence**. When it does occur however, it gives rise to a well-defined freely mobile swelling of firm consistency. According to consistency, it may be called hard fibroma or a soft fibroma. A hard fibroma may be difficult to differentiate from a fibrosarcoma. Fibroma more often occurs in combination with other tissue type e.g. fibroadenoma, fibrolipoma, neurofibroma, etc.

Fibrosarcoma

Differentiating points

– Usually a long h/o painless slow enlargement
– Irregular shape
– Fixed and adherent to skin and deeper structures
– Irregular surface
– Firm rubbery or firm-to-hard or hard consistency.

Supporting points

– Age 35–55
– Arm and thigh, though otherwise it can occur anywhere in the body
– Site—old scar, ulcer, area of previous irradiation, injection sites
– Regional metastatic lymphadenopathy is rare
– Metastatic spread suggests high-grade tumor.

Hemangioma

Majority of vascular tumors are benign (Hemangioma).

Differentiating clinical features

– Absent at birth. Appear 4 weeks after birth
– Tend to grow rapidly for the first year of life. During this time their growth is disproportionately high to the child's growth
– They then enter an involutional phase during which spontaneous regression occurs.
– In 90% of cases, hemangiomas disappear by 10 years of age.

Free ebooks ==> www.Ebook777.com

Superficial hemangioma (Strawberry nevus)
– Raised. Bright red. The color deepens in the first year of life
– Irregular bosselated surface
– Consistency—firm and rubbery
– NOT compressible
– Begins to involute generally in the second year of life
– Changes with involution:
  - Develops a pitted appearance. Wrinkling develops
  - Deepening of crimson color to purple with grayish or white flecks and patches appearing in the lesion
  - Consistency becomes softer.

Deeper hemangioma: May appear blue through the overlying skin.
– Commonest site—head and neck followed by trunk followed by extremities
– Ulceration and bleeding may occur during the proliferative phase.

Pyogenic granuloma
– Children/young adults
– Usual duration—3–4 weeks
– Stationary
– Pedunculated
– Often ulcerates. May become infected. May bleed
– Typical sites—face/limb
– H/o trauma ±. *Frequently arises spontaneously*
– *May involute*.

Complications of hemangioma: Ulceration, infection. Tissue destruction resulting from complications can be severe—such hemangioma is called wildfire hemangioma.
Cavernous hemangioma is a misnomer; it is actually a venous or arteriovascular malformation.

Cystic hygroma
– Age of occurrence: Early infancy, or from birth
– Typical sites: Cheek, neck, axilla, groin, mediastinum
– Diffuse (Ill-defined). A cervicofacial lump may extend into thorax/axilla
– May attain large size. Exceptionally may be so large as to obstruct labor.
– Visibly increases in size (only momentarily) when the child cries or coughs
– Objective relation to SCM cannot be defined
– Cystic hygroma does not respect tissue planes
– Surface—smooth or lobular
– Soft. Fluctuant



Fig 15-9 Cystic hygroma

– Compressible
– *Brilliantly translucent—the* characteristic that distinguishes it from all other cervical swellings.

Natural history is unpredictable.
1. May not enlarge; that means with growth of the child, the swelling becomes relatively smaller in size.
2. May grow with the child → increasing disfigurement and compromise of function.
3. Rarely, spontaneous regression may occur.
4. Complications: (a) Infection of or bleeding (spontaneous / traumatic) into a cyst may → rapid painful enlargement—may be life-threatening, (b) Pressure on vital structures viz. esophagus, trachea and great vessels, (c) Interference with speech.

www.Ebook777.com

Lymphangioma (Lymphangioma simplex)
– Occurs in skin / mucosa. Can affect tongue/lip
– Soft and fluctuant.

Cavernous lymphangioma
– More extensive than lymphangioma simplex
– Age of occurrence: At any time between birth and about 20 months
– Consistency:
  - Initially soft and fluctuant
  - Later on may become firm (due to fibrosis, after repeated attacks of infection)
– May resolve spontaneously by 8 years of age. Those persisting beyond that age are unlikely to resolve spontaneously.

Resemble venous malformation but lack the blue color seen with venous malformation.

- *Lymphangioma simplex* is composed of thin-walled capillary-sized lymphatic channels
- *Cavernous lymphangioma* is composed of dilated lymphatic spaces often with a fibrous adventitia
- *Cystic hygroma* is composed of cysts varying in size from a few millimeters to several centimeters.

Lymphangiosarcoma
– Arises in areas of chronic lymphedema
– Macules which may coalesce and ulcerate.

Neural tumors
– Basically they are derived from Schwann cells
– Benign tumor arising from a peripheral nerve is called neurofibroma—usually unencapsulated
– Benign tumor arising from proximal nerve / spinal nerve root/cranial nerve is called neurilemmoma—Capsulated.

Combining 'Anatomical' and 'Pathological' Inferences

The tissue plane ↓	*Cystic swelling*	*Benign tumor*	*Chronic inflammatory swelling*	*Malignant tumor*	*Miscellaneous*
SKIN	Sebaceous cyst	Hemangioma, lymphangioma, papilloma (wart), fibroma	'Pyogenic' granuloma, (infective) wart, molluscum contagiosum, condylomata, TB (scrofuloderma, lupus vulgaris), nodular leprosy	Squamous celled CA, basal celled CA, malignant melanoma, dermatofibrosarcoma protuberans, lymphangiosarcoma	'Colored lesions', callosity, corn, keloid, hypertrophic scar, keratoacanthoma, xanthoma, keratosis, calcinosis cutis
SC TISSUE	Dermoid cyst, sebaceous cyst, lymphatic cyst, chronic nonspecific abscess, cold abscess, hematoma cyst, hydatid cyst, other parasitic cysts	Hemangioma, lymphangioma, lipoma, neurofibroma, myxoma	Chronic nonspecific abscess, cold abscess, actinomycosis, granuloma (e.g. guinea worm)	Liposarcoma, fibrosarcoma, secondaries, lymphangiosarcoma	Organized thrombus, rheumatic nodule, a migratory swelling due to gnathostomiasis or other parasites
DEEP FASCIA/ SHEATH/ APONEUROSIS	None	Fibroma	Granuloma (e.g. guinea worm)	Fibrosarcoma, desmoid tumor	None

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

The tissue plane ↓	*Cystic swelling*	*Benign tumor*	*Chronic inflammatory swelling*	*Malignant tumor*	*Miscellaneous*
MUSCLE	Chronic nonspecific abscess, cold abscess, guinea worm abscess, hydatid cyst	Hemangioma, lymphangioma, lipoma, fibroma, rhabdomyoma, myxoma	Chronic nonspecific abscess (including guinea worm abscess), cold abscess, granuloma (e.g. guinea worm)	Rhabdomyosarcoma, sarcoma (fibrosarcoma), malignant synovioma	Myositis ossificans, bursal swelling
BONE (Including periosteum)	Solitary cyst, odontome cyst (in the jaw), cysts associated with osteitis fibrosa, hydatid cyst	Hemangioma, osteoma	Chronic osteomyelitis—nontubercular/ tubercular/ actinomycotic	Osteoclastoma, osteogenic sarcoma, Ewing's sarcoma, multiple myeloma, parosteal sarcoma, malignant synovioma, metastatic tumor	Post-traumatic. Examples: Malunited #/ dislocation, myositis ossificans traumatica, excessive callus, subperiosteal hematoma with / without its calcification

A malignant synovioma arising from a tendon sheath may behave like a muscle swelling whereas a malignant synovioma arising from synovial membrane of a joint will behave as a bone tumor.
Any **sequel/complication**?

Step 4: **Associated disorder**?—closely related OR otherwise.

DIFFERENTIAL DIAGNOSIS
of SELECTED **ACUTE INFLAMMATORY SWELLINGS** occurring in different regions of the body

(Although most of the conditions are caused by infection, there are exceptions e.g. mastitis of puberty, aseptic cavernous sinus thrombosis, etc.)

GENERAL REMARKS
The classical symptoms may be modified by antibiotic treatment e.g. fever may be low-grade grumbling, instead of swinging fever with rigors.

Pertaining abscesses

A. Pain and tenderness may be minimal in abscess developing in loose areolar tissue, and in pyemic abscesses.

B. Two criteria are required for fluctuation to develop **early**: 1. Abscess should be superficial (near the surface), and 2. Tension within the abscess cavity should be minimal.

Sites** where **fluctuation develops very late (i.e. the point of time until which patient's suffering has reached its zenith, and before which time the diagnosis should have been preferable made and abscess drained):

– Neck
– (Women's) breasts
– Pulp spaces of fingers and toes
– Fascial spaces of hands and feet
– Iliac region
– Ischiorectal fossa.

www.Ebook777.com

Specific acute inflammatory swellings	*Differentiating points*	*Supporting points*	*Comments*
Acute cellulitis	– Ill-defined swelling – Spreading brawny induration with surrounding edema – Fluctuation absent	Constitutional symptoms	– Constitutional symptoms are more marked – Cellulitis of the scrotum most frequently results from extravasation of urine – In children, cellulitis occurring near a joint should always be taken as acute osteomyelitis
Acute pyogenic abscess—superficial (= superficial to deep fascia)  **Fig 15-10**	– Throbbing pain, more in dependent position (often leading to sleepless night due to severe intensity) – Fluctuation	Constitutional symptoms	The common pyogenic abscess is frequently the end-result of cellulitis or acute lymphadenitis
Acute pyogenic abscess—deep (= deep to deep fascia)	Throbbing pain, more in dependent position (often leading to sleepless night due to severe intensity)	Increasing edema of the skin and subcutaneous tissue, which pits on pressure	Redness and warmth may be absent (at least initially)
Pyemic abscesses	– Multiple abscesses scattered in different regions – Pain and tenderness often minimal	Hectic fever with rigors and sweating	Several of them may develop simultaneously or a number of them crops up in succession
Boil  **Fig 15-11**	Localized painful swelling of skin, containing pus. On (spontaneous) bursting, greenish slough is disclosed at the depth. Separation of slough exposes a (deep) cavity lined by granulations	– May be multiple – Epidemic in the family/ community	Contagious pyogenic infection of hair follicle/sebaceous glands, usually caused by penicillin-resistant staphylococci

Free ebooks ==> www.Ebook777.com

Carbuncle  **Fig 15-12**	– Begins as a painful hard infiltration of the subcutaneous tissue. The overlying skin looks red and dusky – The swelling enlarges assuming quite a big size – Ill-defined, indurated. Softening in multiple areas leading to discharge of pus and exhibition of grayish necrotic slough → the pathognomonic appearance viz. sieve-like = cribriform	– Age > 40 – On the back of the neck / trunk / fingers – DM – Size—large	– Result of neglected skin infection (contagious pyogenic infection of hair follicles/sebaceous glands, usually caused by penicillin-resistant staphylococci) in a dirty/malnourished / underprivileged patient, particularly diabetic – Instead of true diabetes, a state of toxic glycosuria is often present – The pathological process is usually confined to the subcutaneous tissue plane. If resistance is poor the process may extend to any depth even down to the level of bone

ACUTE LYMPHADENITIS

Causes: Nonspecific bacterial, TB (in children under 5 years of age), brucellosis, filariasis, viral (infectious mononucleiosis, rubella, etc.), blastomycosis, plague, etc.

Signs of acute cellulitis of the overlying skin and subcutaneous tissue will be pronounced if periadenitis is significant e.g. in bacterial infections (staphylococcal, streptococcal, etc.)

Acute generalized lymphadenitis associated with fever is often caused by 'medical' illnesses e.g. acute leptospirosis, typhoid, paratyphoid, toxoplasmosis, plague, etc.

Acute nonspecific septic lymphadenitis	– Acute painful and tender swelling/s at the specific known anatomical areas of lymph nodes viz. neck/ axilla/groin, etc. – When superficial, the overlying skin is red, hot and brawny	Primary focus of sepsis	Possible outcome: 1. May subside with the primary focus. OR 2. End in suppuration (clinical manifestations of acute pyogenic abscess develop). Though commonly nonspecific, in infants and children < 5 years of age, keep in mind a possibility of acute tuberculous lymphadenitis
Plague	– Acute painful enlargement of lymph nodes affecting multiple groups – Headache, fever, prostration	– Epidemic – Pneumonia with multilobar involvement	–

HEAD and NECK (excluding intracranial abscess)

– **ORBIT**: Periorbital cellulitis, orbital cellulitis
– Orbital septum (a sheet of fibrous tissue which stretches from the edges of the orbital margin into the eyelids) divides the periorbital region from the orbit

www.Ebook777.com

Periorbital cellulitis = infection is confined to lids and periorbital structures anterior to the orbital septum  **Fig 15-13**	– Edema of the lids – Periorbital swelling – Absence of proptosis – Occular movements are not restricted	– Secondary to local injury/skin sepsis/a recent URTI. Sinusitis is the most common cause in children – Begins with erythema and edema of the eyelids – Induration and tenderness of lids	– More common than orbital cellulitis Examples of periorbital cellulitis: Dacryoadenitis, dacryocystitis, sty, acute infection of chalazion, infection of tarsal cyst, frontal and anterior ethmoidal sinusitis
Dacryoadenitis	Acute inflammatory swelling in the upper outer part of orbit, involving outer third of the upper lid	–	This is an example of periorbital cellulitis
Dacryocystitis  **Fig 15-14**	– An acute inflammatory swelling below the medial aspect of the lower lid – Extrusion of pus through the punctum on pressing the lacrimal gland	–	This is an example of periorbital cellulitis
Sty	Looks like a pimple or boil pointing on the lid margin	–	This is an example of periorbital cellulitis
Acute infection of a chalazion	– A h/s/o a pre-existing uncomplicated chalazion ± – The swelling points inside the lid	–	This is an example of periorbital cellulitis
Orbital cellulitis = suppurative inflammation of the soft tissues of the orbit – Caused by infection behind the orbital septum	– Proptosis – Chemosis (subconjunctival edema) – Limitation of ocular movements – Loss of visual acuity	– Secondary to paranasal sinusitis, commonly frontal/ethmoidal – Begins with erythema and edema of the eyelids – Induration and tenderness of lids	– Less common, but more serious than periorbital cellulitis – Gently separate the eyelids to examine the eye

Free ebooks ==> www.Ebook777.com

<table>
<tr><td>2 examples of orbital cellulitis →</td><td> 
Fig 15-15</td><td colspan="2"> 
 
Figs 15-16A and B Dramatic progress. Patient lost the eye</td></tr>
<tr><td>Subperiosteal orbital abscess</td><td>Displacement of the eye (upwards or downwards depending upon the location of abscess) in addition to the features of orbital cellulitis</td><td>–</td><td>– Periostitis usually affects the orbital margin
– Etiology: TB, syphilis, trauma
– Marginal periostitis → painful swelling intimately connected with the bone. It tends to form orbital abscess or progresses to orbital cellulitis
– Periostitis of the deeper part of the orbit → less-defined symptoms and signs, and mimics orbital cellulitis. It may → orbital apex syndrome</td></tr>
<tr><td colspan="4">FACIAL INFECTIONS OTHER THAN ORBITAL, and INFECTIONS IN THE NECK
Causes of acute cellulitis in the parotid region
– Acute bacterial parotitis / parotid abscess
– Mumps parotitis
– Preauricular lymphadenitis
– Cervical lymphadenitis
– Alveolar abscess.</td></tr>
<tr><td>Acute bacterial parotitis / parotid abscess</td><td>– Shape and extent of the swelling conforms to that of parotid gland
– Facial nerve paresis
– ‘Moderate’ trismus
– Pus extrudes from the parotid duct opening (intraorally, in the buccal sulcus)</td><td>– Predisposing factor is basically a debilitated status (debilitated patient / after major surgery) when mouth care has been neglected
– Unilateral</td><td>Can occur without any predisposing cause</td></tr>
</table>

www.Ebook777.com

Mumps parotitis	– Bilateral – Skin overlying the swelling—less shiny	– Child – No pus seen at the parotid duct opening – Contact with a c/o mumps in the previous 3 weeks time	– Unilateral parotitis can be probably mumps as well – Both sides may be affected at slightly different times, and with very different grades of severity – Epidemic ± – Orchitis/pancreatitis rare in a prepubertal child – Recurrent attack of mumps is rare although possible. Nevertheless, the possibility must be considered particularly unlikely if the gap in time between the 2 attacks is only weeks or months rather than years
Mumps submandibular sialoadenitis	– Bilateral – Skin overlying the swelling—less shiny	– Child – No pus seen at the submandibular duct opening – Contact with a c/o mumps in the previous 3 weeks time	– Unilateral sialoadenitis can be probably mumps as well – Both sides may be affected at slightly different times, and with very different grades of severity – Epidemic ± – Associated mumps parotitis ± – Recurrent attack of mumps is rare although possible
Preauricular cellulitis / abscess	– Swelling in front of the tragus – NO trismus	Congenital preauricular sinus	Possible sites of infection: 1. Preauricular lymph nodes i.e. lymphadenitis. 2. Infection of a congenital preauricular sinus, if present In case of an abscess, search for a punctum (= blocked opening of preauricular sinus). It may be hidden by hair
Deep neck infection – Parapharyngeal abscess – Retropharyngeal (retrovisceral) abscess – Prevertebral space abscess – Submandibular abscess	– Difficulty in, and pain on swallowing – Limitation of neck movements – Torticollis – Trismus	– Sick with fever with chills, malaise, loss of appetite – Airway may be compromised	– If airway is compromised, then immediate steps are essential to secure it – An external neck swelling, swelling in the floor of the mouth or bulging of the pharyngeal wall may be visible depending on the site of the abscess – Physical examination of pharynx and larynx may be difficult because of trismus. **Flexible fiberoptic nasopharyngoscopy** is useful in this situation – Great vessels may be involved in infection of a few days' duration

Free ebooks ==> www.Ebook777.com

Acute cervical lymphadenitis (Nonsuppurative/ suppurative)	Basically, it is an example of deep neck sepsis, in the region of the cervical lymph nodes	–	– Especially deep cervical – Common in children – Particularly in children, a thick overlying muscle like SCM provides effective camouflage for even a large abscess – In small infants (abscesses are common) aspiration (of pus) will confirm the diagnosis, and on occasions may alleviate the need for any surgical intervention – **Beware of abnormal overlying vessels or discoloration of skin** – Though commonly nonspecific, in infants and children ↓ 5 years of age, keep in mind a possibility of acute tuberculous lymphadenitis
Acute bacterial thyroiditis Thyroid abscess	Wide swelling of the neck, which is maximal over the thyroid	–	– Fluctuation absent as pus is deep – Complication: Ludwig's angina
Laryngopyocele	– K/c/o external laryngocele – Pain in the throat – (Rapidly) Enlarging (pre-existing) swelling essentially in the carotid triangle – Firm – Becomes less compressible, or not compressible at all	–	–

ORAL CAVITY
- DENTAL ABSCESSES
- PERITONSILLAR (PARATONSILLAR) ABSCESS
- RETROPHARYNGEAL ABSCESS
- PREVERTEBRAL ABSCESS

DENTAL (VIRTUALLY PERIODONTAL) ABSCESSES: Pericoronal, periodontal, alveolar.
Basic clinical features of dental abscess:
- Painful (usually throbbing) ill-defined swelling of the jaw × a week
- Trismus
- Past h/o toothache
- Sick-looking, in distress
- 'Fat face'—unilateral, diffuse, shiny red, warm, tender, indurated swelling overlying the respective jaw.

Other causes of similar clinical features (DD): Acute sialoadenitis, mumps, lymphadenitis, lymphoma, glandular fever.

www.Ebook777.com

Pus from any type of dental abscess (in particular alveolar abscess) **can track in any direction**: Laterally towards the cheek / medially towards the tongue / superiorly towards the palate / inferiorly i.e. downward into the neck. And consequently, pus can collect:

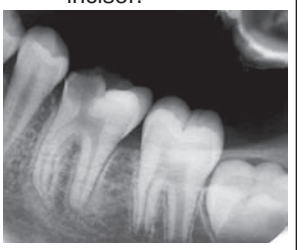
– On any of the surfaces of the gum (gumboil)
– In the buccal sulcus of either jaw
– On the surface of the face, superficial to the attachment of buccinator
– In the submasseteric space (between the masseter and ascending ramus of mandible)
– In the lateral pharyngeal space (→ difficulty in speaking and swallowing)
– In the pterygomandibular space (between the medial pterygoid and ascending ramus of the mandible)
– In the sublingual space above or below the myelohyoid muscle
– In the submandibular space superficial to the myelohyoid
– In the submental space (under the jaw)
– On the palate (less common).

Life-threatening spread

1. From the upper jaw → **Septic cavernous sinus thrombosis.**
2. From the lateral pharyngeal space → Up towards the base of the skull / down into the glottis (causing Ludwig's angina) or mediastinum (**mediastenitis**).
3. From the lower jaw → **Ludwig's angina.**

Pericoronal cellulitis	Infection of the gum over the crown of an unerupted (/ partially erupted) impacted tooth	Usually a lower 3rd molar i.e. 'wisdom tooth'	Usually cellulitis. Often, an abscess does not form
Periodontal abscess	– Halitosis – Obvious periodontal disease – Several loose teeth	Degree of tenderness on percussing (tapping with a metallic object) the tooth—much less (*cf* alveolar abscess)	At the side of a tooth, caused by spread from an infected gum
Alveolar abscess = infection in the bone around a nonvital infected tooth  **Fig 15-17**	– Toothache (presently/in the recent past). Pain on chewing – The site of the swelling will depend on the culprit tooth (Refer to the column of comments) – Severe degree of trismus – Halitosis – Caried tooth—obvious cavitation within a tooth often colored brown, or around an existing restoration (filling) – Tenderness on pressing (with a gloved finger)/percussing (tapping with a metallic object) on the culprit tooth	The culprit tooth may be slightly raised in its socket	– Pain on chewing suggests infection spreading beyond the root into the bone and leading to an abscess – Site of swelling: 1. Around the eye—from maxillary canine 2. Facial swelling in the region of the mandible—from mandibular molar

Free ebooks ==> www.Ebook777.com

	– **No fluctuation** during the initial 48 hours		3. Palatal swelling—from maxillary lateral incisor.  **Fig 15-18** X-ray of the same patient (Fig 15-17)
Acute peritonsillar (paratonsillar) abscess (Quinsy)	– Tense swelling above and behind the tonsil, displacing it downward and forward	– Child – Following an attack of tonsillitis	Much of the swelling is inflammatory edema rather than an abscess. However, it needs to be distinguished from paratonsillar cellulitis. In both, constitutional symptoms and leukocytosis are present, and there is often a preceding h/o tonsillitis As the diagnosis is uncertain, admission to hospital is necessary; commence IV antibiotics. Cellulitis normally responds rapidly, within 12–24 hours. A paratonsillar abscess does not respond to antibiotics alone
Acute parapharyngeal abscess (*Syn*. Lateral pharyngeal abscess) – An example of deep neck infection	– **Considerably more sick** than a c/o quinsy – The head is held immobile in moderate extension – Ill-defined swelling of woody consistency in the neck beneath the angle of the mandible/deep to the mid portion of SCM – **Tenderness on moving the head from side-to-side** – Trismus – Tonsil and pharyngeal wall displaced medially – Involvement of cranial nerves and Horner's syndrome ⇒ abscess involving the posterior compartment of the parapharyngeal space	–	– Features differentiating from quinsy: 1. Considerably more sick than a c/o quinsy 2. Tenderness on moving the head from side-to-side – **Complications**: **1**. Thrombosis of IJV. Complications of IJV thrombosis—thrombosis of the lateral and cavernous sinuses, septic pulmonary embolism, metastatic abscess formation

www.Ebook777.com

			2. Involvement of carotid artery → torrential bleeding in the external auditory canal, or asphyxiation due to its rupture into the pharynx
Acute retropharyngeal (retrovisceral) abscess – An example of deep neck infection	– If it occurs apparently spontaneously, then it does so in children ↓ 5 years – Other causes: Injury e.g. instrumentation (passage of NGT, esophagoscopy, ET intubation), noniatrogenic penetrating / blunt trauma, extension of infection from adjacent area – A reddened, rounded fluctuant mass on the posterior pharyngeal wall to one side of the midline—in c/o an abscess below the level of palate – Obstruction of the posterior nares and inferior displacement of the soft palate—in c/o an abscess above the level of the soft palate – 'Hot potato' voice—in c/o an inferior abscess (supraglottic level)	– Age < 5 years – Sick with fever and refusal to eat – Associated cervical lymphadenitis – Nuchal rigidity ±	This condition arises from prevertebral lymph nodes in the midline – May be difficult to diagnose in a young child – Complications: 1. Large abscess → asphyxia 2. Bursting may → aspiration pneumonia – Infection from here can pass to anterior mediastinum → breathlessness – **Major DD**: Subacute/ chronic tuberculous retropharyngeal abscess
Acute prevertebral space abscess – An example of deep neck infection	Presenting symptoms—vague – Pain in the neck and shoulder – Dysphagia ± – **Midline** bulge of the posterior wall of the pharynx	–	– Classical infection of this space results from tubercular osteomyelitis of vertebral body – Other causes: Surgery, injury, extension of retropharyngeal abscess

ACUTE LIFE-THREATENING COMPLICATIONS OF HEAD AND NECK INFECTIONS

– Ludwig's angina
– Cavernous sinus thrombosis

Free ebooks ==> www.Ebook777.com

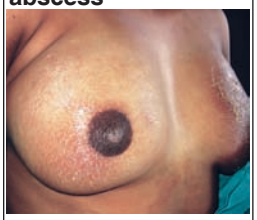
LUDWIG'S 'ANGINA'	– Saliva dribbling out because of odynophagia – A high-pitched inspiratory and expiratory stridor due to upper airway obstruction caused by the displaced tongue – Severe bilateral brawny cellulitis of sublingual and subnmandibular regions (may extend as far down as clavicles) – **Trismus is not common** **Trismus ⇒ extension of infection to parapharyngeal space** – Tongue pushed up (against the roof of the mouth) and backwards, due to induration and edema of floor of the mouth	– Antecedent mandibular tooth root abscess	The basic pathology is a median cellulitis or abscess situated between the genioglossus and the geniohyoid muscles behind the mandible which provides a firm base for retropulsion of the whole tongue – Portal of infection may not be obvious, though 80% of infections arise from infection of teeth or mandible – A complete clinical examination of the mouth is impossible anyway – External swelling is limited because of the strength of the deep fascia forming the inferior boundary of this space – Inspiratory difficulties may worsen alarmingly, and death can occur, not from edema of the glottis, but from strangulation by the posteriorly displaced tongue GA poses formidable problems and LA is difficult in restless, anxious patient particularly if the patient is a young child; the choice depends on the skilled assistance available. Submental surgical drainage can produce dramatic relief following the evacuation of an abscess, but in cellulitis the symptoms may not be relieved immediately because the infection is more diffuse. If exploration is not followed by immediate improvement, tracheostomy may be necessary

CAVERNOUS SINUS THROMBOSIS

– Can be **septic** (more common) or **aseptic**
– Causes of aseptic thrombosis: Marasmus / tumor / trauma
– Causes of septic thrombosis: Spread from practically any primary source of sepsis in the head/neck
– However, the most common sources are the **nose and upper lip**

Septic cavernous sinus thrombosis  **Fig 15-19**	– On ~ 5th/6th day after commencement of facial cellulitis – High-grade fever, severe headache, nausea, vomiting, delirium, semi-consciousness (Severe toxemia). O/E of the eyes: - Edema of the eyelids and conjunctivae – Movements of the eyeball reduced/ absent – Pupil dilated and fixed – Cornea becomes anesthetic – Exophthalmos later on	–	The eye signs may occur on the opposite side in varying degree due to spread of infection

www.Ebook777.com

THORAX – BREAST: Breast abscess, retromammary abscess, milk congestion during lactation, mastitis of puberty – AXILLARY lymphadenitis (suppurative/nonsuppurative), abscess (single episode/recurrent), hidradenitis suppurativa – EMPYEMA NECESSITANS			
Acute pyogenic lactational breast abscess  **Fig 15-20**	– During the span of breast feeding: 1. At the time of commencement of breast feeding period 2. Again after 6 months when the child's first teeth erupt 3. At weaning. Incomplete drainage of breasts—residual milk—may get infected – Tense induration (after emptying the breast)	– During early stage (tense induration) before fluctuation develops (which is a late sign): Pitting-on-pressure'	An obvious fluctuating swelling is a late sign. At the early ('prefluctuation') stage, the diagnosis is strongly suggested by 'pitting-on-pressure', and it may be confirmed by diagnostic aspiration *An important DD to be kept in mind, although rare*: Inflammatory carcinoma of the breast
Nonlactational breast abscess(es) most probably secondary to streptococcal and anaerobic infection  **Fig 15-21**	– Periareloar – Systemic upset is less pronounced than in lactational abscess – Scarring and distortion – (Other) Clinical manifestations of duct ectasia / periductal mastitis may be present [Discharge from nipple, (transverse slit-like) retracted nipple, etc.]	Frequently recur	Pathology is entirely different from lactational abscess. It is a manifestation of duct ectasia/periductal mastitis
Peripherally situated nonlactational abscess(es)	–	May be associated with DM, RA or steroid treatment	–
Retromammary abscess	– On tangential inspection, the breast appears to have been pushed forwards; consequently, both nipple and breast appear prominent – On palpation: The whole breast appears to float on the underlying fluid (the retromammary abscess)	–	Originates from infected hematoma, chronic empyema thoracis, or tuberculous osteomyelitis of rib

Free ebooks ==> www.Ebook777.com

Milk congestion during lactation	– Slight pain (due to distension,) in both breasts – No redness of the skin – Tenderness absent/minimal	No regional lymphadenitis	– Usually occurs at the time of weaning – Fever **may be present**. In fact it is due to fever, that the condition may have to be differentiated from acute bacterial mastitis/ abscess – Causes of milk congestion: 1. Inadequate drainage with residual milk 2. Blockage of lactiferous ducts by epithelial debris
Mastitis of puberty	– Occurs only in males – A button-like painful and tender swelling – Tenderness subsides within 2 weeks – Induration takes many months to subside	–	– Does not require active treatment – Reassurance
Axillary Single episode of single abscess	–	–	Forms: 1. Infection of apocrine glands. Superficial. The usual form 2. Deep seated—in the lymph nodes (e.g. under pectoralis major)
Axillary Multiple recurrent small abscesses	–	–	Causes: – Tuberculous – Fungal – Hidradenitis suppurativa—postpubertal only
Hidradenitis (suppurativa)  **Fig 15-22**	– Postpubertal only – The affected area becomes indurated; subsequently forms sinuses with discharge of small amounts of pus	–	Other regions which can be similarly affected: The areas containing apocrine glands—breasts, inguinoscrotal region, perianal region

www.Ebook777.com

Empyema necessitans = A collar-stud abscess in relation to an area of penetration of the chest wall by the neurovascular supply, in a patient suffering from empyema thoracis	– Fever from a respiratory tract infection which has failed to respond or had relapsed during or after treatment – Recent thoracic / abdominal surgery, or trauma or liver disease or pulmonary TB – Gnawing chest pain – Pleurisy – Impulse-on-coughing +	– Swinging fever with rigors, malaise, anorexia, loss of weight	– Cough: unusual. Cough will be present in bronchopleural fistula expectorating large quantities of pus – Dyspnea may be due to lung compression. Both these symptoms may be quite indolent – Often caused by TB

ANTERIOR ABDOMINAL WALL SEPSIS

– In the umbilicus proper = omphalitis
– Subumbilical cellulitis/abscess: Infected enterocyst or urachal cyst
– At any other place: *de novo*
– Superficial extravasation of urine with cellulitis

For chronic discharge from the umbilicus, refer to chapter 44.

Omphalitis in the neonate	Cellulitis/suppuration	–	– Cause: Faulty (not maintaining aseptic precautions) technique of dealing with the umbilical cord – An important possible complication: Portal vein thrombosis leading to portal HT
Omphalitis in later life (older children, adults)	– Seropurulent, often foul-smelling discharge – Granuloma – 'Umbilical stone'	H/s/o a pilonidal sinus ±	– A more chronic condition – Causes: 1. Lack of hygiene 2. Foreign body including 'umbilical stone' (concretion due to desquamated dead superficial epidermal cells) 3. Secondary infection of a pilonidal sinus
Subumbilical cellulitis / abscess	The swelling moves anteroposteriorly with respiratory excursions	Pre-existing subumbilical chronic swelling may have known to be present	Cause: Pyogenic infection of an enterocyst or a urachal cyst
Superficial extravasation of urine with cellulitis	Refer to chapters 25 and 54		

Free ebooks ==> www.Ebook777.com

GROIN

– Acute inguinal/femoral lymphadenitis (Nonsuppurative / suppurative)
– Iliac abscess, psoas abscess: Refer to chapter 34
– Superficial extravasation of urine with cellulitis: Refer to chapters 25 and 54

SCROTUM

Refer to the chapters 25 and 85

HAND SEPSIS

GENERAL REMARKS

– Some infections arise spontaneously; others follow quite minor injuries, or even a seemingly trivial scratch
– There are many spaces in the hand where pus can collect. These spaces are not rigidly defined; some run into one another, and more than one may be infected at the same time
– Fluctuation is a late sign. Severe throbbing pain leading to sleepless night is important evidence of an abscess
– To localize the exact site of pus, feel for the point of greatest tenderness by probing with a matchstick
– Fever, lymphangitis and lymphadenitis suggest spreading sepsis

Skin and subcutaneous tissue can be infected anywhere in the hand. However, don't be misled by a collar-stud abscess.

Infection almost anywhere in the hand makes its back (i.e. dorsum) swell, but pus seldom collects there. On rare occasions if abscess does form on the dorsum, it does so usually in subcutaneous tissue plane and only occasionally in subaponeurotic space deep to extensor tendons.

Carbuncles may form in the hair follicles on the back of the fingers and hand.

Causes of persistent pain and discharge:

1. Inadequate drainage and debridement.
2. Spread of a more superficial infection to a tendon sheath or another fascial space.
3. Sloughing off of a tendon.
4. Osteomyelitis.
5. Presence of a foreign body.

WHITLOW = Acute pyogenic abscess of **fingers** (/toes)

Subcuticular	Purulent blister	–	Strip off the cuticular layer to open the blister. Look for a hole leading to extension into deeper layer—'collar-stud' abscess
Subcutaneous	Abscess superficial to palmar aponeurosis	–	After draining the abscess look for a hole leading to extension into deeper layer—'collar-stud' abscess
Paronychia	Acute cellulitis or a small abscess at the side or base of a nail	–	–
Onychia	Pus seen through the nail	Severe pain	Onychia = pus in the nail-bed

www.Ebook777.com

Apical space abscess	– H/o a splinter having run under the nail – Little swellin – Maximum tenderness at or just under the free edge of the nail	–	Apical space = the space between the distal part of the nail and the bone (distal phalanx)
Terminal pulp-space abscess	Throbbing pain occurs early	–	– The most common whitlow due to frequency of puncture wound in the tip of finger – For anatomical reasons, throbbing pain occurs early
Abscess in the volar spaces of the middle/proximal phalanx	– Swollen, tender and indurated finger held semiflexed – Passive extension severely tender	–	– Abscess superficial to the flexor tendon sheath – **Differentiation from suppurative tenosynovitis may not be possible clinically**
Thecal—suppurative tenosynovitis  **Fig 15-23**	– The whole finger swollen —sausage shaped, tender **without becoming red** – The swelling extends into the distal palm – The finger is held semiflexed and rigid – Tenderness along middle of front (i.e. anterior aspect) of the whole finger – Active flexion not possible. Passive extension severely tender	The whole hand swollen like an inflated rubber glove	– Usually caused by streptococci – In c/o little finger and thumb, swelling and tenderness of flexor tendon sheath at wrist will be present
Web-space infection = Subcutaneous infection of the web space  **Fig 15-24**	– Separation of adjacent fingers (i.e. fingers on either side of the infected web space) – Point of maximum tenderness on the palmar surface of the web; may extend a short way into the palm	–	– Pus mostly gathers near the palmar surface – **Pain and swelling may be so great that patient often comes to seek medical advice before much pus has formed** – **Sometimes it may be difficult to clinically differentiate it from suppurative tenosynovitis** – If not drained, pus may track: 1. Posteriorly towards the dorsal aspect of the web-space 2. Proximally into the middle palmar space 3. Across the front of the finger into the neighboring web space 4. Distally into the finger

Free ebooks ==> www.Ebook777.com

Septic arthritis	– Exquisitely painful and tender joint. Moving the joint **even little in any direction** → severe tenderness – Swelling of the joint	–	– Source: Open injury/spread from nearby infection – Inevitably ends up with a stiff joint
Osteomyelitis of phalanx	Exquisite pain and tenderness	–	Common cause: Neglected pulp space infection
SUPPURATION IN THE PALM			
Subcuticular abscess	Purulent blister	–	Strip off the cuticular layer to open the blister. Look for a hole leading to extension into deeper layer—'collar-stud' abscess
Subcutaneous abscess	Abscess superficial to palmar aponeurosis	–	After draining the abscess look for a hole leading to extension into deeper layer—'collar-stud' abscess
Superficial palmar space abscess	Deep to palmar fascia. May track superficially to form a collar-stud abscess, the patient presenting with subcutaneous component of the abscess	–	–
Mid (middle) palmar space abscess  **Fig 15-25**	– Normal hollow of the palm obliterated – Induration beneath the medial half of central part of palmer aponeurosis i.e. just lateral to hypothenar eminence – Movements of the ring and middle fingers not possible – Will not be able to hold a piece of paper between extended fingers – Cross fluctuation between the swelling in the flexor aspect of the distal forearm and the swelling in the palm suggests extension of pus into the space of Parona	Marked edema and redness (without pus), on the dorsum of the hand—the whole hand is swollen like an inflated rubber glove	– Frequently occurs in leprosy patients – Source: From a lumbrical canal or from an infected tendon sheath – The middle palmar space communicates through the carpal tunnel with a space deep to the flexor tendons in the forearm (the space of Parona)
Thenar space abscess	– Gross swelling of the thenar eminence – Maximum swelling over the web of the thumb – Thumb held in abduction – Index finger held flexed – Cannot move the index finger or thumb	The whole hand is swollen like an inflated rubber glove	–

www.Ebook777.com

Infections of the flexor tendon sheaths

– Sources of infection:
  1. Commonest portal of infection is puncture wound in the distal flexor crease. Anatomical basis—the flexor tendon sheaths come nearest to the skin as they pass under the flexor creases of the fingers
  2. Spread from pulp space infection
– If an infected sheath bursts, it does so into the middle palmar space through one of the lumbrical canals.
– Tendon sheath infections are a common complication of the anesthetic hands of leprosy. Infection is neglected until it is so advanced that it has destroyed the tendon sheaths

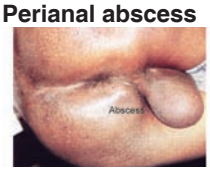
Localized infection of flexor tendon sheath	Only one segment of the finger is swollen	–	– Usually caused by staphylococci – Differentiation from middle plamar space/thenar space infection can be difficult
Fulminant infection of flexor tendon sheath	Refer above to 'suppurative tenosynovitis'	–	–
Infection of the ulnar bursa	– The whole hand—edematous – Palm—moderately swollen—all the fingers especially the 5th, held semiflexed and rigid – Fullness immediately above the flexor retinaculum ± – Active flexion not possible. Passive extension severely tender, particularly of the little finger; index finger least affected	–	– The most serious hand infection, because it contains all the flexor tendons – The ulnar and radial bursa sometimes communicate, so infection of one may follow in the other a day or two later
Infection of the radial bursa	– Distal phalanx of the thumb semiflexed and rigid – Passive extension severely tender – Extension of other fingers normal – May feel a swelling above the flexor retinaculum – Tenderness overlying the sheath of FPL	–	Radial bursa is continuation of the tendon sheath of the FPL

FOOT SEPSIS

GENERAL REMARKS

– Leprosy patients are particularly liable to foot sepsis, and have their own special problems
– Fine movements are not as important in the foot as they are in the hand, so infection of the tendon sheaths of the foot is less of a disaster
– Clinical features and principles of management of subcuticular/subcutaneous infections, paronychia, apical toe-space abscess, pulp-space abscess and web-space abscess are similar to those in hand sepsis
– Deep infection of the plantar aspect occurs usually due to an injury such as a thorn-prick, which has penetrated deeply

Free ebooks ==> www.Ebook777.com

PERINEAL ABSCESSES			
Acute penile periurethral abscess = an infection of one of the urethral glands, usually occurring as a complication of acute gonococcal urethritis	A tender induration on the undersurface of the penis	Preceded by acute gonococcal urethritis	Natural history: Bursts externally → fistula
Acute bulbar periurethral abscess *Syn.* Acute periurethral phlegmon	– Pain in the perineum – High-grade fever with rigors – Initially, tenderness and swelling are limited to the perineum – Later, **often in a matter of hours**, cellulitis spreads to the scrotum, penis and the abdominal wall – Septicemia	– Urinary symptoms – Retention of urine due to inflamed stricture ±	– Commonly arises in bulbar urethra – In about half of the cases, there is no stricture of the urethra. In the remaining, stricture of the urethra predisposes the condition – Complication: Extensive cellulitis in the same plane in which superficial extravasation of urine occurs viz. into the scrotum, beneath the superficial fascia of the penis, and up the abdominal wall beneath the deep layer of the superficial fascia – Urine is (already) infected in patients suffering from stricture. Cellulitis resulting from infected urine is more dangerous than that following traumatic rupture of urethra
Inflammation of the bulbourethral gland (Cowperitis)	Least pressure on the bulbourethral gland causes excruciating tenderness. This way it is differentiated from periurethral and ischiorectal abscess	–	With index finger in the rectum and thumb first on one side and then on the other of the median raphe of the perineum, bulbourethral glands can be palpated
Perianal abscess  **Fig 15-26**	– Severe perianal pain – Acute inflammatory swelling at the anal margin – On DRE: No or little tenderness, induration or bulging in the anal canal	–	Abscess may discharge spontaneously, but because of severity of symptoms patients often present before this has occurred

www.Ebook777.com

Ischiorectal abscess	– Severe perianal pain – At an early stage: Acute inflammatory swelling may not be evident. Pressure on the ischioanal fossa → tenderness ± pitting edema – In an advanced abscess: A large area of tender induration overlying ischioanal fossa. (larger and further from perianal abscess) On DRE: Tender induration bulging into anal canal	–	–
Gluteal abscess	**> 7 cm beyond the anal margin**	–	–
Pilonidal [sinus(es) and abscess(es)]	– When sinus is infected: Pain and discharge in the natal cleft – Variable number of pits in the upper natal cleft—may be seen to exude pus during active inflammation – Acute inflammatory swelling just to one side of the midline in the natal cleft	–	– Pilonidal sinus may be asymptomatic – **May be mistaken for an anal fistula and vice-versa**
Perianal hidradenitis suppurativa	– Postpubertal only – The affected area becomes indurated; subsequently forms sinuses with discharge of small amounts of pus	–	Other regions which can be similarly affected: The areas containing apocrine glands—axillae, breasts, inguinoscrotal region
Bartholin's gland infection / abscess	Acute inflammatory swelling in the region of the vaginal introitus	Pus coming out of the duct or erythema around the duct opening	–

ACUTE PYOMYOSITIS = Pus in the muscle.
Acute pyomyositis may have to be differentiated from various other disorders, some acute inflammatory and some other than inflammatory, in different regions of the body.

A. *In the limbs in general*: Acute septic arthritis, acute osteomyelitis.
   – *Particularly in the thigh*: Acute osteomyelitis, hematoma, sarcoma, guinea worm infection
   – *Particularly in the calf*: Acute DVT, sickle cell crisis with bone infarction.

B. *In the anterolateral abdominal wall*: Acute intra-abdominal inflammatory disorders.

C. *Particularly in the loin*: Perinephric abscess, pyonephrosis.

Free ebooks ==> www.Ebook777.com

Classical pyomyositis	– Site: Larger muscles more commonly affected like in thighs, buttocks, shoulders, back and abdominal wall – Exquisite pain, ill-defined swelling and tenderness – Restriction of movements at the joints nearby – Fever with rigors, and patient quite sick if abscess is large – Overlying skin—smooth and shiny. Pitting on pressure during early stages – Affected muscle—tender, indurated. Fluctuation is a late sign – Lymphadenitis NOT conspicuous	–	– Common between 5 and 25 years of age – A single muscle may be involved or a group of them, or there may be several abscesses in different parts of the body – Large abscesses may form in deeper muscles with few localizing signs – **Keep in mind a possibility of acute osteomyelitis** and try to differentiate accordingly
Pyemia associated with pyomyositis	Sequence of abscesses in one muscle after another	– H/o a trivial skin laceration/ blister/a small sore ±	–
Septicemia associated with pyomyositis	– Multiple tender areas in muscles – Rapid progress – Septic shock	–	May be fatal and often not diagnosed
ACUTE INFECTIVE BURSAL SWELLING			
Acute suppurative bursitis	Penetrating injury to the bursa	Spread of local cellulitis	–
ACUTE INFECTIONS OF BONES AND JOINTS			
Acute osteomyelitis	– A child – Exquisitely painful and tender bone near a joint Unwilling to move the limb particularly at the concerned joint. → a limp in c/o lower limb – Classical site (Sites in the descending order of frequency: Proximal tibia, distal femur (i.e. around the knee), proximal femur, proximal humerus, distal radius / ulna, distal tibia, calcaneus) – Fever. Constitutional symptoms	– Poor socio-economic status living under very unhygienic conditions – A focus of septic lesion somewhere in the body such as the skin, middle ear, etc. from where the infection may have spread to the bone	– Apart from the 'classical site', any other bone can be infected; sometimes multiple bones may be infected simultaneously – **During earlier stage swelling may not be present.** Soft tissue swelling is a late feature which suggests that pus has already started to spread out of the bone – Before the age of 6 months an epiphysis offers no barrier to the spread of infection, so that pus in a metaphysis rapidly spreads to the joint

www.Ebook777.com

	– Causes: Hematogenous, traumatic, following unskilled orthopedic procedures in unsterile theaters		– **Atypical presentations**: 1. In a very sick child, fever may be absent and few general signs of infection may be present 2. General signs of severe sepsis may be present, but local signs may be few; ∴ **think of osteomyelitis as a possibility in any child who is ill and reluctant to move one of her/his limbs**
Acute septic arthritis	– Exquisitely painful and tender joint. Moving the joint **even little in any direction** → severe tendernes – 'Night cries' – Fever. Constitutional symptoms – Swelling of the joint – In a child, with one large joint affection, think of acute pyogenic arthritis – In a adult, with multiple large joint affection without symmetry, think of acute gonococcal arthritis – Causes of acute septic arthritis: 1. Before 6 months of age, from spread of osteomyelitis in the metaphysis of a long bone 2. At any age, it may affect the hip for anatomical reason 3. Hematogenous. → knee/ hip/shoulder/ankle arthritis in the descending order of frequency 4. Penetrating injury of the joint	Risk factors: 1. Infancy and old age 2. Poor socio-economic status living under very unhygienic conditions 3. Local joint damage due to OA/previous surgery	– **Atypical presentations**: 1. In very young or very old, constitutional symptoms may be few 2. Pain may be the only presenting symptom in c/o arthritis of spine, SI joint and hip joint 3. Fever / leukocytosis present in only half of the patients 4. It may be difficult to differentiate tuberculous arthritis from subacute suppurative arthritis – Each risk-group (refer to the previous column) has its own characteristic infective organisms, patterns of joint involvement and clinical response

Retropharyngeal acute pyogenic abscess v/s tuberculous abscess

Tuberculous abscess is usually subacute or chronic and follow infection of the body of a vertebra.

Acute osteomyelitis v/s other acute conditions

1. **Poliomyelitis**: No swelling. No bony tenderness.

Free ebooks ==> www.Ebook777.com

2. **Acute septic arthritis**:
   – Maximal tenderness overlying a joint; NOT overlying the adjacent bone
   – ALL the movements of the joint are exquisitely tender.
3. **Acute cellulitis / pyomyositis**: Tenderness in the soft tissues rather than overlying the bone.
4. **Rheumatic fever**:
   – Pain flits from joint to joint
   – Tenderness at the joint line and may extend both above and below the joint.
5. **Sarcoma**: Much swelling but not much fever.
6. **Acute subperiosteal hematoma due to scurvy in children**:
   – Bleeding swollen gums
   – Marked anemia.
7. **Infarction of bone in sickle-cell disease**:
   – Multiple bones involved
   – Unusual bones involved like skull, small bones of hands/feet.

DIFFERENTIAL DIAGNOSIS of SELECTED CHRONIC CIRCUMSCRIBED SWELLINGS

Chronic Circumscribed General Swellings Arising from the Skin

Sebaceous cyst
Basically a cystic swelling.
Differentiating points
– Adherent to the skin at the punctum (seen as a black dot)
– Soft, indentable
– Nontranslucent
– Yellowish-white cheesy pultaceous material occasionally apparent if a cyst ruptures/has ruptured.

Supporting point: Multiple in one region like scalp/scrotum/labia.
Atypical points
– Punctum may be absent / invisible
– A cyst, particularly of very long duration and large size may be completely free from the skin.

Notes
– Sebaceous cyst can occur anywhere in the body except for the palm and sole
– As the duct of a sebaceous gland is attached at one point in the skin, *it is a cyst in connection with the skin but the cyst proper is situated in the subcutaneous* tissue. Hence, a 'small' sebaceous cyst (< 1 cm) may get lifted up wholly on lifting up the adjacent skin. As a cyst enlarges over a long period of time, eventually it may come to lie completely in the subcutaneous tissue plane leaving all its attachment to the skin.
– Complications: Infection, ulceration (may → Cock's peculiar tumor), sebaceous horn, sebaceous adenoma, sebaceous adenocarcinoma.



Fig 15-27 Sebaceous cyst of the scalp



Fig 15-28 A sebaceous cyst showing the punctum

www.Ebook777.com



Fig 15-29 Multiple sebaceous cysts of the scrotum



Fig 15-30 Cock's peculiar tumor on the scalp



Fig 15-31 Sebaceous horn



Fig 15-32 A papilloma

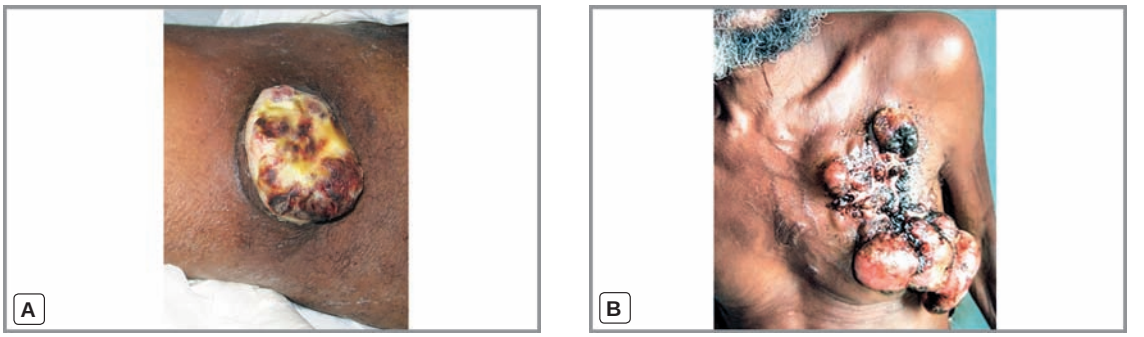
Superficial hemangioma (Strawberry nevus)—refer to page 156.
Pyogenic granuloma—refer to page 156.

Papilloma (Noninfective 'wart').
Sites—anywhere on the skin, tougue, lip. (Deeper sites—vocal cords, renal pelvis, urinary bladder, rectum, colon).
Pedunculated outgrowth. Branched processes.
No induration at the base.
Notes
– Basically, wart means a hyperplastic epidermal lesion (i.e. epidermal proliferation) of either a non-viral origin, or caused by a human papilloma virus
– A wart of non-viral origin is called 'papilloma'. It has a smooth surface
– A wart of viral origin is called 'infective wart'. It has a dry, rough-granular surface with branching processes.
– Sometimes the surface of a papilloma is very hard—called **'horny papilloma'**.

Fibroma
– Pedunculated
– Smooth surface
– Consistency—soft or firm
– Frequently multiple
– May be associated with neurofibromatosis
– A fibroma producing large pendulous folds is called 'patchydermatocele'.

Dermatofibroma (*Syn.* Sclerosing angioma, subepidermal nodular fibrosis)
– Single or multiple.

Free ebooks ==> www.Ebook777.com



Figs 15-33A and B Examples of dermatofibrosarcoma protuberans

Differentiating points
- Indolent
- Only a few millimeters in diameter (seldom > 1 cm).

Supporting points
- Most common site: Extremities
- Reddish / yellowish brown (due to lipid) / blue-black (due to hemosiderin. DD—melanoma).

Dermatofibrosarcoma protuberans
('Aggressive' tumor—probably a low-grade fibrosarcoma)
- One or more red or bluish dermal nodule/s
- Grow slowly
- May suddenly start to enlarge and ulcerate. Persistent outward growth to attain large size.

Squamous-celled carcinoma-protuberant type
Basically 2 patterns of growth are noted, although these can overlap-protuberant type and infiltrative type.

Differentiating points of protuberant type
- Offensive discharge
- Dirty necrotic floor which is raised above the level of the surrounding skin to form a cauliflower-like appearance
- Raised everted edge
- Stony hard induration of the edge and at the base
- May bleed easily on touch
- Regional metastatic lymphadenopathy after about three months.

Supporting points
- Middle age
- Site:
  - Lip / tongue / vulva / penis / anus.
  - Old scar / area of radiation burns / sun-exposed areas in fare-skinned patients (including sites of actinic keratosis) / site of the bite by an elapid snake may be years earlier.

Stage - ...?
Notes
- When SCC develops in an old scar or area of burns, hardening and cracking of skin are usually the first indications of malignant change
- Shape of a Marjolin's ulcer (a squamous celled carcinoma developing in an old scar) may be regular.

www.Ebook777.com



Fig 15-34 SCC-protuberant type



Fig 15-35 Marjolin's ulcer in an old burn scar on the scalp

Basal-celled carcinoma

1. *Noduloulcerative (Syn. ulcerative BCC, 'rodent ulcer')*
   - Slow growing
   - Nodule with a waxy or pearly appearance associated with telangiectasia
   - Later, the skin breaks down to form a typical 'rodent ulcer' (irregular shape with raised and beaded edge)
   - Typical site: On the face—superior to an imaginary line joining angle of the mouth to tragus of the ear.
2. *Pigmented*: Similar to noduloulcerative type but with a pearly black sheen.
3. *Superficial spreading (Syn. 'field fire' BCC)*
   - Scaling erythematous lesion
   - Does not ulcerate. Does not get indurated
   - Spreads radially while appearing to heal in the center. The central scar area—characteristically hairless (due to destruction of hair follicles)
   - Typical site—trunk
   - Frequently multifocal. Commonly advanced at the time of presentation due to insidious mode of onset and slow progress.
4. *Morphoeic*
   - Dense grayish yellow plaque
   - Gradual increase in size
   - May ulcerate.
5. *Fibroepithelioma of Pinkus*: Presents as papilloma without scaling or ulceration.



Fig 15-36 BCC

Stage - .....?

Notes

- The term 'rodent ulcer' should be avoided because ulceration may appear late
- If left untreated, it can produce a horrible mutilation as it erodes cartilage and bone
- Occasionally a lesion diagnosed clinically as basal cell CA will prove on histological examination to be a squamous cell CA or a melanoma, or occasionally even a non-neoplastic condition.

Malignant melanoma

Basically, 2 modes of presentation:

- Malignant transformation of a pre-existing nevus
- Development *de novo* in normal skin.

Basic morphology of the lesion: Pigmented—flat lesion, ulcer, nodule, or ulcerated nodule.

Free ebooks ==> www.Ebook777.com

Differentiating points
- Itching
- Nonhomogeneous pigmentation, especially mixtures of black, blue and red
- Irregular margin
- Size > 6 mm in diameter
- Satellite nodules.

Clinical features to suggest malignant transformation of a pre-existing nevus: One or more of the following:
- Development of itching / burning
- (Rapid) Increase in size
- Development of nodularity or ulceration, and / or bleeding
- Loss of homogeneity with development of areas of darker or lighter pigmentation
- Irregular margin
- Development of satellite lesions.

Types: Superficial spreading, lentigo maligna, nodular, and acral.

1. *Superficial spreading malignant melanoma*
   - Flat
   - Age—between 40 and 50
   - Typically develops in a pre-existing nevus
   - This is the commonest type of malignant melanoma.
2. *Lentigo malignant melanoma*
   - Slow growing
   - Flat
   - Typically develops in some damaged skin usually on the face in elderly individuals.
3. *Nodular malignant melanoma*
   - Nodule
   - Age—between 50 and 70
   - Typically develops in a pre-existing nevus.
4. *Acral malignant melanoma*
   - Arise in the nail beds or on the palmar or plantar skin
   - Slow growing.



Fig 15-37 MM of the sole

Stage - ...?

Notes
- A large variety of red, brown or blue pigmented lesions may be mistaken for a malignant melanoma (and vice-versa)

  The best mimic is the juvenile melanoma which may for a time grow quite fast; nevertheless, before puberty such lesions are nearly always benign in their ultimate behavior
- Malignant melanoma has a very bad prognosis. Hence, once this possibility is raised, there can be no other management than 'urgent' biopsy in an appropriate manner.

Gumma (Nonulcerated)

Time of development—from 6 months upto many years after inoculation of the infection.
- Adult
- Duration—in months
- Progress—slow
- Regression ±
- Painless
- Single/multiple

www.Ebook777.com

– Site—overlying subcutaneous / submucous bones (skull, palate, sternum, clavicle, scapula, ulna, tibia), SCM muscle, testis
– Margin—well-defined
– Shape—hemispherical / ovoid / serpentine
– Surrounding area—coppery red pigmentation Slightly edematous
– Base—indurated
– Regional lymphadenitis absent.



Fig 15-38 Infective wart on the sole

Infective wart
– Site—hands / feet
– Dry, rough—granular surface with branching processes
– May be giving a cauliflower appearance.

In the sole, a wart becomes buried under a thick layer of keratin, and presents as a pale yellow or light tan, characteristically translucent nodule that may be exquisitely tender.

Notes
- Natural history of (infective) warts: Varies between 2 extremes. Some of them may regress spontaneously. Others may be extremely resistant to treatment
- Immunocompromized patients tend to get widespread resistant warts.

Molluscum contagiosum
– Multiple small (up to 1 cm in diameter) globular nodules with glistening white color
– Look like pearls umbilicated in the center
– Children and young adults
– Caused by viral infection. It is contagious.

Condylomata
– Situated at mucocutaneous junctions e.g. vulva, penis, anus, mouth
– Fungating sessile growth with moist sodden surface and typical (offensive) smell
– Usually due to syphilis.

Condyloma acuminatum = venereal wart
– Situated around the genitalia
– A single, or multiple cauliflower-like dry masses
– Variable size
– Usually due to gonorrhea.

Mycobacterial infection
Two forms of ulcerated swelling due to mycobacterial infection are seen: Scrofuloderma and lupus vulgaris.

Scrofuloderma
= tuberculous or non-tuberculous mycobacterial infection of the skin caused by direct extension from an underlying infected structure, most commonly a lymph node or a bone. The lesion first manifests as a painless, bluish-red swelling that breaks open and then forms an ulcer with irregular undermined blue edge. Neck and groin are the common sites affected.

Lupus vulgaris
Differentiating point: Soft nodules—appear brownish and semitranslucent on pressure with a glass slide. It starts as superficial ulceration that tends to heal in the center but remains active at the periphery.

Free ebooks ==> www.Ebook777.com



Fig 15-39 Molluscum



Fig 15-40 Vulval condylomata



Fig 15-41 Penile warts



Fig 15-42 Perianal warts



Fig 15-43 Scrofuloderma





Figs 15-44A and B Examples of lupus vulgaris

www.Ebook777.com

Supporting points
- Children and young adults
- Site—face, arm
- Ulceration
- Regional lymphadenopathy.

Notes
It is a form of cutaneous tuberculosis
Lupus = wolf. Owing to its spreading nature, the ulcer is called lupus.

Nodular leprosy
- Multiple small nodules in the skin, especially of the face and arm
- Smaller nodules coalesce to form large plaques
- Other symptoms and signs of leprosy present.

Callosity (Callus)
= a diffuse area of relatively even thickness.

Corn
= an area of callosity which has become moulded into a nucleus.
The size and shape of the nuclei vary according to their location and prevailing stresses.
Basically, callosity and corn are hyperkeratotic lesions caused due to recurrent friction and pressure.
Common site: Hand / foot at the points/s of friction/ pressure.



Fig 15-45 Callosity and corn

Keloid
Differentiating points
- Occurring in the scar of operative or accidental wound particularly disposed across the skin creases and situated on the neck / back / deltoid region / sternum; scar of vaccination, piercing of the ear
- Well-defined
- Shape—irregular. Claw-like processes extend into the surrounding area
- Transgresses the boundaries of the scar-bed
- Raised, flattened. Uneven surface
- Color—reddish or dark brown
- Firm-to-hard.

Supporting points
- Adult age
- Itching
- Previous suturing seems to have been done under tension and/or in the presence of infection.

Notes
- A keloid may be spreading, or stabilized. Criteria of spreading keloid: Edge looks pink with capillaries over it, and tender
- May have to be differentiated from a hypertrophic scar, however that may not be possible in the initial period of 6 months since injury.

Hypertrophic scar
Differentiating points
- Starts regressing after about 6 months from the time of injury
- Itching if was present, may disappear
- Does not transgress beyond the boundaries of the scar-bed.

Free ebooks ==> www.Ebook777.com

Keratoacanthoma

Differentiating points

– A self-limiting lesion
– A rapidly growing smooth shiny pink nodule with a central crater containing a keratin plug
– Grows to its maximum size over 6 weeks; then begins to shrink over the next 6 weeks leaving a depressed scar at the site of the lesion
– Maximum diameter 2.5 cm.

Actinic keratosis (*Syn.* Solar keratosis, senile keratosis)

– Red / pink / brown / tan / grayish scaly patch or roughened area with papilliform surface on the (sun) exposed skin of elderly individuals
– Often multiple.

  Clinical course: Slow, with regression of some lesions and progression of others to malignancy. Beginning of ulceration or bleeding suggests possibility of malignant transformation.

Notes

– No particular significance in elderly
– In young people or the lesion occurring in old scar should be watched closely because a malignant transformation is quite common.

Seborrheic keratoses

– Yellowish to brown, typically multiple lesions often symmetrically distributed on the trunk, face or other areas
– Feel slightly greasy and velvety or warty
– Common in old age. Benign. Not a premalignant condition.

Xanthomas

– Multiple diffuse lesions which may occur in various parts of the body
– Size—variable
– Tuberous xanthomas = yellow colored nodules occurring on the extensor surface of limbs and buttocks
– They are usually associated with abnormality of lipid metabolism.

Calcinosis cutis (= dystrophic calcification in the skin and subcutaneous tissue.)

– Circumscribed hard consistency nodules, adherent to the skin.

AV malformations

– Some of them can be brown colored
– Blanch on pressure with a transparent plate like a microscope glass slide.



Fig 15-46 Keratoacanthoma





Figs 15-47A and B Examples of seborrheic keratosis



Fig 15-48 Xanthoma

www.Ebook777.com



Fig 15-49 Calcinosis cutis

Benign pigmented nevus (Mole)

– Extremely common. Usually appear in the first few decades of life.

Typical clinical features

– Well-defined. Round or oval. Flat or raised
– Size < 6 mm in diameter
– Uniform color—usually tan or brown.

Clinical significance

1. Cosmetic concern.
2. May mimic and hence a need may arise to differentiate from malignant melanoma.
3. A small proportion of nevi may have malignant potential e.g. junctional nevus in palm / sole in adults.

Junctional nevus:

– Macule or papule
– Brown
– Smooth. Hairless
– Site—anywhere in the body.

Intradermal type of nevus:

– Well-defined papule
– Brown / flesh colored
– Often hairy
– Common sites—face and neck.

Bowen's disease (Carcinoma *in situ*)

– Slowly growing pink thickened plaque with scaling and crusting of its surface
– When it occurs on the prepuce or glans it is called erythroplasia
– It is a premalignant condition.

Free ebooks ==> www.Ebook777.com

Colored 'lesions' of skin (at a glance)

Flat or only slightly raised	Elevated / nodular (forming a swelling)
– Vascular - AVM (port wine stain, spider angioma, angiokeratoma) - Cherry angioma, spider vein – Purpuric: - Petechiae / purpura - Ecchymosis – Mole – Actinic keratosis – Seborrheic keratosis – Basal celled carcinoma – Malignant melanoma – Dermatofibroma – Bowen's disease	– AVM (venous malformation, capillary lymphatic malformation) – Hemangioma (strawberry nevus, pyogenic granuloma) – Keloid – Keratoacanthoma – Basal celled carcinoma – Malignant melanoma – Dermatofibrosarcoma protuberans – TB (lupus vulgaris) – Xanthoma

Chronic Circumscribed General Swellings Arising from Subcutaneous Tissue

Sebaceous cyst

Refer to page 180

Sebaceous cyst lies mostly in the subcutaneous tissue but can almost always (exception: 'very' long duration) be demonstrated to have an attachment to the skin.

Congenital sequestration dermoid

– *Basically*, a cystic swelling
– *Differentiating point*: Situated at the specific site—anywhere in the midline / root of the nose / external or internal angle of the eye / behind the ear.

Additional differentiating points specifically in favor of a subcutaneous dermoid

– Relatively more freely mobile
– Consistency—soft (/firm)
– Fluctuation ±
– Moulding ±
– Depression of the underlying bone may be very shallow.

Supporting point: Present practically since birth.

Notes

– Dermoid cyst = any cystic swelling lined by squamous epithelium.
– The term 'dermoid' is incorporated in 3 different distinct swellings viz. epidermoid cyst, dermoid cyst, and teratomatous dermoid
– 'Epidermoid cyst', often called sebaceous cyst, is lined by squamous epithelium, and its wall consists of fibrous tissue without any epidermal appendages
– 'Dermoid cyst' proper is also lined by squamous epithelium but its wall contains epidermal appendages like hair, sebaceous glands and sweat glands the fact which differentiates it from an epidermoid (sebaceous) cyst
– 'Teratomatous dermoid' is basically a teratoma. There are 3 types of teratoma—cystic, differentiated solid and undifferentiated solid. The cystic form most commonly occurs in the ovary; it differentiates mainly along the ectodermal lines with formation of a cyst lined by normal-appearing skin, and hence the designation—'teratomatous dermoid'. It is almost always benign. In rest of the places (i.e. other than ovary), teratomas usually do not take a dermoid cystic form and consequently they are not called teratomatous dermoid, but only teratoma e.g. testicular teratoma, sacrococcygeal teratoma, mediastinal teratoma, etc.

www.Ebook777.com

Implantation dermoid
= an acquired type of dermoid cyst.

Results from implantation of epidermal cells into deeper tissues due to puncture injury of repetitive nature. The implanted squamous epithelial cells continue to grow in the depth and form a cyst. Commonly occurs in fingers of tailors.



Fig 15-50 Implantation dermoid

Lymphatic cyst
A (large) translucent cyst.

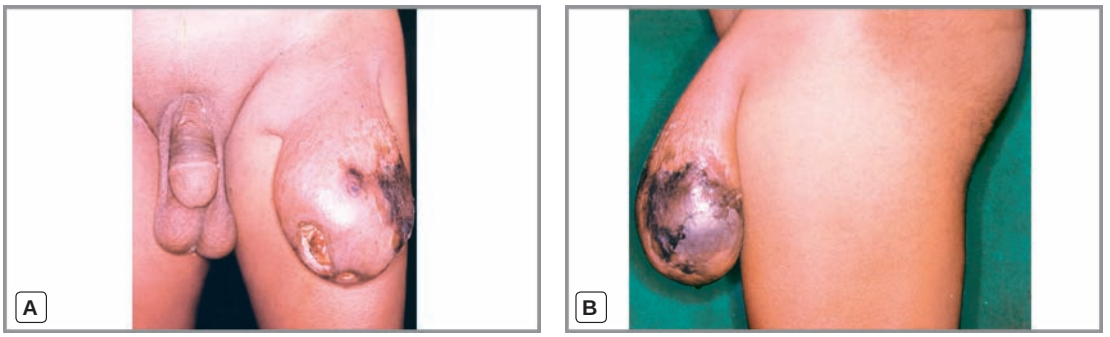
Hydatid cyst
Rare in the subcutaneous tissue.

Gnathostomiasis
– An infestation in the tropics caused by a nematode *Gnathostoma spinigerum*. Infestation usually follows ingestion of raw fish
– Single migratory subcutaneous swelling which may appear anywhere, including the abdominal wall, the chest, the face or hands
– It may also cause hematuria, spontaneous pneumothorax, and spontaneous perforation of the right side of the colon

(Encapsulated) **Lipoma** (in subcutaneous tissue plane)
– *Basically*, a benign tumor.
Differentiating points
– Almost stationary
– 'Slipping margin'
– Ovoid. Flattened
– Mobile. Produces dimpling of the overlying skin at the attachments of fibrous septa, when the swelling is moved
– Lobular surface
– ('Sense of) fluctuation' +.
This is the commonest benign tumor in subcutaneous tissue plane.
Supporting points
– Middle age onwards
– Multiple
– Painless
Possible causes of pain when present: Angiolipoma, adiposis dolorosa.
Variations
1. *Nevolipoma*: **Slight reduction in size on pressure ±**. Common on the face. Danger of excessive bleeding if the surgeon is not aware of this condition
2. Margin not so well-defined and does not slip on pressure ('slipping sign' absent) ⇒ *'diffuse lipoma'*: Does not have a definite capsule.
3. A lipoma may become *pedunculated*.
4. Softer consistency in a long-standing lipoma ⇒ *myxomatous degeneration.*
5. Firm consistency ⇒ *fibrolipoma*.
6. Firm-to-hard consistency in a long-standing lipoma without other symptoms and signs indicative of malignancy ⇒ *calcification*.
7. Painful and tender multiple lipomata especially around elbows, knees and ankles in menopausal women ⇒ *adiposis dolorosa*.

Lobulation and 'slipping sign' may not be demonstrable if the lipoma is situated in deeper planes.
A lipoma situated in the midline of the back may be overlying a spina bifida.

Free ebooks ==> www.Ebook777.com



Figs 15-51A and B Pedunculated lipoma of the thigh complicated by ulceration, infection and necrosis

Possible complications:
– Ulceration and subsequently infection of the skin particularly in pedunculated lipomas
– Repeated trauma may → fat necrosis and calcification
– Malignant transformation especially in those situated on the back / thigh.

Neurofibroma
Basically, a benign tumor.

Differentiating points
– Ovoid with its long axis oriented along the direction of a nerve
– Degree of mobility is more in transverse direction (i.e. in the direction that is across the long axis of the nerve)
– Soft or soft-to-firm.

May be multiple.

Variations:
1. Pain, tenderness and free mobility in practically all directions when they arise from cutaneous nerves.
2. May attain large size when arising from bigger nerves.

Distinct varieties
1. Von Recklinghauson's disease: Multiple subcutaneous neurofibromatosis all over the body; size varying from a pea to a cricket ball. Patchy pigmentation of the skin. Cystic degeneration and sarcomatous change may occur.
2. Plexiform neurofibromatosis: Usually multiple in one region of nerve plexuses or its branches where folds of tissue along with the skin hang down loosely. – Common in the region of the branches of trigeminal nerve. May be associated with generalized neurofibromatosis.
3. Elephantiasis neuromatosa: Skin and subcutaneous tissue become thickened and coarse like elephant skin. Rare. Congenital.
4. Pachydermatocele: Rare. Coils of soft tissue hang around the root of the neck.

Adenoma (Fibroadenoma)
– *Basically*, a benign tumor
– Adenoma is usually combined with fibrous tissue to form fibroadenoma
– Typical example—fibroadenoma in the breast
– Deeper sites—thyroid, parathyroid, pancreas, rectum (presents as a pedunculated rectal polyp).

Myxoma
Basically, a benign tumor.
– Slightly lobular surface
– Soft consistency.

www.Ebook777.com

Rare. Appears at any age. Common sites apart from subcutaneous tissue—jaw and other bones, spermatic cord, urinary bladder, heart.

Lymphangioma
Basically, a benign swelling.
Differentiating points:
– Soft. Fluctuating
– Brilliantly translucent.
Common types:
– Capillary lymphangioma in the skin
– Cavernous lymphangioma in the cheek / neck / axilla / groin
– In the tongue (→ macroglossia), in the lip (→ macrochelia).

Secondary malignant deposit/s
Basically, a malignant tumor.
Usually present around the umbilicus and in the operation scar.
– Restricted mobility
– Firm / hard consistency
– Previous case history about the 'primary' and its management is usually available / known, though may not be always available.

Organized thrombus
– An elongated nodule along the course of a vein
– Mobile in transverse axis
– Firm consistency
– H/o precipitating injury to the vein usually in the form of intravenous injection / infusion may be available.

Rheumatic nodules
Differentiating points
– Site—near a joint or overlying a subcutaneous bone
– Tender
– Young patient with present or past involvement of joint with fever.
Supporting point: Rheumatic heart disease.

Chronic Circumscribed General Swellings Arising from Deep Fascia / Sheath / Aponeurosis

Fibroma: Refer to page 155
Fibrosarcoma: Refer to page 155

Desmoid tumor
– An 'aggressive' tumor of fibrous tissue
– Common sites—head and neck, back, chest wall, abdominal wall particularly at the site of previous scar
– May be associated with Gardner's syndrome.

Chronic Circumscribed General Swellings Arising from Muscle

All swellings deep to the deep fascia (practically muscle-swellings) are impossible to diagnose clinically, and must be subjected to appropriate investigation.

Hydatid cyst
– Minimal disturbance of function
– May be huge
– Tense
– Characteristic thrill—'hydatid thrill' and 'after thrill'—may be present.

Free ebooks ==> www.Ebook777.com



Fig 15-52 An adventitious bursa



Fig 15-53 Olecranon bursitis

Myositis ossificans
- Appears weeks or months after injury
- Gradually increasing pain and decreasing movements
- Ill-defined
- Takes the shape of the muscle.

Bursal swelling
- Behaves as a muscle swelling if the bursa is situated between the muscles or deep to the muscle.

Notes
- Bursa is defined as a fluid-filled cavity lined by flattened endothelium similar to synovium, which develops between tendons / bones / skin to allow easier movement between them
- Types—(a) Natural = normal anatomical bursae e.g. subscapsular, olecranon, prepatellar, etc. (b) Adventitious = develop secondarily as a result of prolonged pressure over bony prominences e.g. bunion of hallux valgus, Billingsgate hump
- Inflammation of a bursa is called bursitis
- Types of bursitis—simple, acute suppurative, and chronic.

Chronic bursitis follows repeated pressure or slight injuries to the natural bursae e.g. 'student's elbow' (olecranon bursitis), 'housemaid's knee' (prepatellar bursitis), 'clergyman's knee' (infrapatellar bursitis), 'weaver's bottom' (ischial tuberosity bursitis), subsartorial bursitis, etc.

Physical findings in a bursal swelling in general
- Hemispherical or ovoid
- Surface—smooth
- Overlying skin: Normal / hyperkeratotic / red and shiny
- Consistency variable depending on the tension of fluid within the swelling
- Fluctuation +
- Transillumination ±.

Hernia of the muscle
- Appears on contraction of the muscle, and disappears (reduces) as the muscle relaxes
- Small soft protrusion of the muscle through a defect in the deep fascia. The swelling hardens on isometric contraction of the muscle.

Rhabdomyosarcoma
Basically, a malignant tumor.

In children:
- 2 peaks in age distribution are seen
- Soon after birth: Head, neck and genitourinary sites are commonest
- During adolescence: Paratesticular tumor is commonest.

www.Ebook777.com



Fig 15-54 Rhabdomyosarcoma

In adults:
- Most common sites—shoulder, upper arm and thigh
- A deep-seated tumor that often only presents when it projects into the skin as a reddish brown tumor
- Restricted movements
- Firm-hard consistency.

Chronic Circumscribed Swellings Related to Tendon

Ganglion

Basically, a benign swelling in the vicinity of wrist/ankle joint.
- Commonest site: Dorsum of wrist, ankle
- Painless or painful
- Usually non-tender
- Pearl ganglia are tender. ('Pearl ganglia' = very small ganglia developing in relation to flexor tendons of fingers. They are exquisitely painful and tender.)
- Well-defined. Hemispherical
- Size—1 to 2 cm in diameter
- Consistency—firm or hard. 'Tense'.

A note

Ganglion = cystic swelling containing clear gelatinous fluid. Formed probably due to myxoid degeneration of fibrous capsule of joints/tendons. Often communicates with joint cavity.

Tuberculous tenosynovitis: 2 types—'dry' and 'wet'.

Dry type (Dry tuberculous tenosynovitis): → Soft elastic boggy swelling.
Later, pus may track into neighboring sheaths or joints.

Wet type (Tuberculous tenosynovitis with effusion (effusion in tendon sheaths)**)**: → Soft fluctuant swelling.
- Crepitus on pressure—due to the presence of a large number of melon-seed bodies (composed of cellular debris bound together by fibrin) floating freely in the effusion fluid
- '*Compound palmar ganglion*': This term is applied when the condition (i.e. tuberculous tenosynovitis with effusion) occurs in connection with the flexor tendons of the fingers. → bilocular swelling across the wrist joint i.e. there is a swelling in the wrist and a swelling in the hand; cross fluctuation between the two can be demonstrated.

Free ebooks ==> www.Ebook777.com

16 CHRONIC LYMPH NODE SWELLING

WHY LYMPHADENOPATHY?

Justification: Usually multiple swellings situated in the specific anatomical areas known to be lymph node stations (e.g. neck / axilla / groin), and usually ovoid in shape.

Single swelling suggests that the diagnosis of lymphadenopathy is highly improbable. However, rarely, a secondary deposit may result in single swelling.

DEFINITIONS AND GENERAL REMARKS

1. **Lymphadenopathy** is defined as inflammatory or noninflammatory enlargement of lymph nodes, however, inflammatory enlargement is often specifically called **lymphadenitis**.

   Enlargement may be purely reactive (sinus histiocytosis) or the nodes may be diseased (by infection or tumor).

   Agreement on the criterion of enlargement is elusive, but palpably enlarged lymph nodes should be considered diseased until proved otherwise.

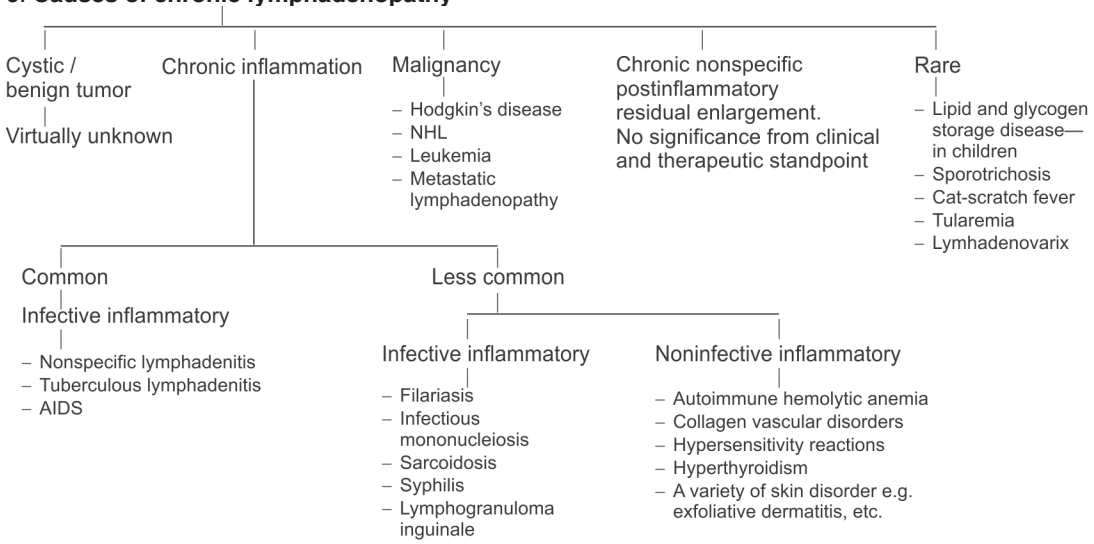
   However, in older children and adults, enlarged lymph nodes up to 1 cm in diameter, which are otherwise asymptomatic, and situated in the groins or the neck (only occipital / submandibular / upper deep cervical) have no clinical and therapeutic significance. Such enlargement is specified as chronic nonspecific post-inflammatory residual enlargement.
2. **Regional** lymphadenopathy = enlargement of lymph nodes in a specific anatomical region usually with a causative lesion in the drainage area. A clinical exception: Metastatic lymphadenopathy with occult primary.
3. Criteria of **localized** lymphadenopathy: Enlargement of lymph nodes in one region or 2 contiguous regions.

   Criteria of **generalized** lymphadenopathy: Enlargement of lymph nodes in at least 2 noncontiguous, or 3 contiguous regions.
4. **Acute generalized lymphadenopathy associated with fever is often caused by 'medical' illnesses** (that is to say a lymph node biopsy is not indicated) e.g. acute leptospirosis, typhoid, paratyphoid, toxoplasmosis, etc.
5. **Causes of acute lymphadenitis**
   - Nonspecific bacterial infection
   - Tuberculosis—in children under 5 years of age
   - Brucellosis
   - Filariasis
   - Viral e.g. infectious mononucleiosis, rubella, etc.
   - Blastomycosis
   - Plague, etc.

   Signs of acute cellulitis of the overlying skin and subcutaneous tissue will be pronounced if periadenitis is significant e.g. in bacterial infections (staphylococcal, streptococcal, etc.).

www.Ebook777.com

6. Causes of chronic lymphadenopathy



7. *Factors taken into consideration to decide if lymphadenopathy needs to be investigated*

- Age
- Site
- Physical characteristics
- Clinical setting / background.

Examples which do not warrant investigation

a. The postauricular lymph node enlarged in the 1st 2 years of life (situated anywhere in a fair range of territory from the tip of the mastoid process to the superior nuchal line). Enlargement is often bilateral.
b. Chronic nonspecific postinflammatory residual enlargement in the groins or the neck (only occipital / submandibular/ upper deep cervical).

An example which warrants investigation: Axillary lymphadenopathy must be investigated irrespective of age and physical characteristics, unless the cause is obvious such as an overt septic focus in the drainage area e.g. the hand.

HISTORY

- **Occupation**?
- In case if multiple regions / groups are involved—**which region / group was the first to be affected**?
- (If applicable,) **When did the swelling burst** (forming an ulcer or a sinus)?
- **Fever**? Details.
- **Loss of weight**?—how much, and over how much period?
- Symptoms of **anemia**?
- **Other swellings of similar nature** in the same region and / or elsewhere?
- Lump in the abdomen (s/o **hepatosplenomegaly**)?
- H/o red eye (**conjunctivitis**)?
- **Pain in the joints**?
- **Skin rash**?
- Any **lesion in the drainage area**?
- Do you do **gardening**?
- Do you get **contact with pets like rabbits / squirrels,** etc.?
- **Past h/o TB**?
- **Family h/o TB**?

Free ebooks ==> www.Ebook777.com

- **Pressure symptoms**?
- **Treatment** taken, and response to it?

INTERPRETATION OF SYMPTOMS, AND DISCUSSION

Age

In children, think of leukemia first, then nonspecific lymphadenitis, f/b lipid and glycogen storage diseases.

Occupation

– Medical / paramedical—possibility of AIDS
– Farming / gardening—possibility of sporotrichosis
– Veterinary particularly dealing with animals like rabbits, squirrels, etc.—possibility of tularemia.

Fever

– Evening low-grade fever may suggest TB
– Intermittent bouts of remittent fever with rapid enlargement of nodes during that period occurs in Hodgkin's disease. Such episodes should not be mistaken for acute pyogenic lymphadenitis
– Periodic fever with chills may suggest filariasis.

Gardening may predispose to sporotrichosis.

Contact with rabbits / squirrels may → tularemia.

Possible causes of progressive enlargement of tuberculous lymph nodes inspite of the patient being on ATT

1. Secondary infection.
2. Formation of cold abscess.
3. Resistant organisms.
4. Diagnosis may be wrong.

In GENERAL EXAMINATION

Specifically look for

– Anemia
– Conjunctivitis
– Skin rash
– Arthritis
– Loss of weight
– Examine all lymph node regions in the body.

In SYSTEMIC EXAMINATION

Specifically look for hepatosplenomegaly.

LOCO-REGIONAL EXAMINATION

- **Region/s** affected?
  In each region—**exact site/group** affected, **and extent**?
  Examples:
  – In the neck—deep cervical/supraclavicular/posterior triangle, etc?
  – In the axilla—anterior group/central group, etc?
  Certain diseases have predilection for specific region(s) / group / site(s).
  Yet, instances of atypical involvements are not infrequent.
- **Ulceration/sinus formation**?
  – Occurs in TB / lymphogranuloma inguinale/metastatic lymphadenopathy.
- **Number**?
  – A single swelling implies that the diagnosis of lymphadenopathy is highly improbable
  – A single swelling may imply however rarely, a secondary deposit
  – The apparent single swelling *may actually be a mass of 3 or 4 nodes matted together* (due to an advanced pathology).

www.Ebook777.com

- **Shape**?:
  - Ovoid
  - Hemipherical ⇒ softening ⇒ collection of pus
  - Irregular—may suggest malignancy.
- **Size**?:
  - Up to 1 cm (= small)
  - 1–2.5 cm (= moderate)
  - > 2.5 cm (= large)
  - Size of a normal lymph node = that of a pin-head (i.e. just about a millimeter or two in diameter)
  - Usual size of an individual enlarged lymph node is 0.5–1 cm in diameter or slightly larger
  - > 2.5 cm diameter of an individual enlarged lymph node ⇒ - Malignant lymphadenopathy
    - Rarely, TB (hyperplastic variety OR formation of a cold abscess).
- **Matting**? = adherence of adjacent lymph nodes to each other forming a mass with nodular surface.

  Clinical method: Try to move adjacent enlarged lymph nodes from each other.

  Inability to move them from each other = 'matting'.
  - In TB—matting occurs due to periadenitis
  - In malignant growth—matting occurs due to infiltration.

  When adjacent lymph nodes are enlarged to large size it may not be possible to elicit mobility between them even though they are free from each other → a false idea of matting.
- **Mobility and adherence to deeper tissues**?
- **Consistency**?

  Consistency of chronically enlarged lymph node is usually firm. When it is so (i.e. firm) it cannot really help to differentiate various disorders. However, sometimes consistency may be other than firm.
  - Soft consistency ⇒ development of abscess. Examples: 'Cold' (i.e tuberculous), sporotrichosis, tularemia.
  - 'Rubbery' (practically a variant of firm and rather subjective, nevertheless) consistency ⇒ Hodgkin's disease
  - Hard consistency ⇒ metastatic malignant lymphadenopathy (secondaries).
- **Pressure effect/s**:
  - Due to pressure on adjacent anatomical structures?

EXAMINATION OF THE DRAINAGE AREA

1. Look for the primary focus of infection or (malignant) tumor.
2. Path of the lymphatic vessels intervening between the primary malignant tumor and the lymph nodes: Multiple hard subcutaneous nodules may be palpable in carcinoma and malignant melanoma. In malignant melanoma these nodules are bluish-colored.
3. Lymphedema.

ARRIVING AT THE DIAGNOSIS

STEPWISE ANALYSIS OF DATA

1. Does the lymphadenopathy appear to be **localized** or **generalized**?

Causes of localized lymphadenopathy:
- Nonspecific bacterial sepsis, tuberculosis, filariasis, lymphogranuloma inguinale, sporotrichosis, cat-scratch fever, mycosis fungoides, etc.
- Any disease leading to generalized lymphadenopathy will begin at some or the other region i.e. will appear localized to start with. Examples: Tuberculosis, infectious mononucleosis (cervical to start with), etc.

Free ebooks ==> www.Ebook777.com

Causes of generalized lymphadenopathy

- Primary disorder of lymph nodes
  - Leukemia
  - Lymphoma
- Infection / infestation
  - Examples
    - AIDS
    - Tuberculosis, syphilis
    - Infectious mononucleosis,dengue
    - Filariasis, brucellosis, toxoplasmosis
    - Diffuse skin infection / inflamation, etc.
- Hypersensitivity disorders, connective tissue disorders
  - Drug reactions
  - Serum sickness
  - SLE
  - RA
- Miscellaneous
  - Sarcoidosis
  - Kikuchi / Fujimoto disease

2. Could it have been caused by a **systemic disease**?

(Irrespective of whether the lymphadenopathy is localized or generalized at the time of clinical presentation) The presence of one or more of the following clinical features suggest systemic disease. However, their absence may not rule out the possibility of systemic disease.

- Conjunctivitis and skin rash ⇒ collagen vascular disease / Kawasaki disease / histiocytosis / leptospirosis
- Skin rash ⇒ leukemia / AIDS / infectious mononucleiosis / RA / SLE / Kawasaki disease / histiocytosis
- Arthritis ⇒ collagen vascular disease
- Supraclavicular lymphadenopathy ⇒ a serious underlying disease—lymphoma / metastatic lymphadenopathy / tuberculosis / sarcoidosis / histoplasmosis
- Hepatosplenomegaly, ⇒ cancer/infection (e.g. miliary tuberculosis) / storage disease (Gaucher, Niemann-Pick, etc.).

3. **Differentiation between chronic inflammatory and neoplastic lymphadenopathy**, which are the most common causes of lymphadenopathy

Differentiating clinical features	*Chronic inflammation*	*Malignancy*
Duration	Months	Weeks
Progress	Slow/almost stationary	Rapid, in weeks
Loss of weight	Absent / slight. Rarely marked (in advanced systemic disease)	Marked
Anemia	Absent. Exception: TB	Present. May be marked in some cases
Size of the enlarged nodes	Small to moderate	Moderate or large
Hepatosplenomegaly	Absent	±

Chronic lymphadenitis

Chronic lymphadenitis could be caused by infective or non-infective pathology.

Factors to be considered in getting a preliminary judgment:

- Region / site / group
- Whether lymphadenitis is localized or generalized.

Significance of region / site / group

- Occipital: Scalp infection, syphilis, sarcoidosis
- Preauricular: Most often bacterial, beginning in eyelids / conjunctivae; sarcoidosis
- Upper deep cervical: TB
- Epitrochlear: Childhood viral illness, secondary syphilis, rarely sarcoidosis
- Inguinal: Nonspecific postinflammatory residual enlarged lymph nodes, filariasis, lymphogranuloma inguinale, lymphadenovarix, etc.

Yet, instances of atypical involvements are not infrequent.

www.Ebook777.com

4.

Differentiating clinical features	*Nonspecific lymphadenitis*	*Tuberculous lymphadenitis*	*Syphilitic lymphadenitis*
Age	Common in children	Nothing specific	Young adults
Progress	Often stationary	Slow	Slow
Number of groups	Single	Single to start with	Multiple
Ulcer/sinus	Absent	±	Absent
Size of each enlarged node	Small	Variable	Moderate
Matting	Absent	Usually present	Absent
Deeper tissue involvement	Absent	Rarely	Absent
Lesion (primary focus of infection) in drainage area	Present	±	Absent
Typical region/s involved	Nothing specific	Upper deep cervical group first; then other	Posterior cervical and supratrochlear

Enlarged lymph nodes due to syphilis are often described as 'shotty'. Shotty = small and hard like lead pellets used in firearms

Differential diagnosis of other causes of infective lymphadenitis is discussed later in the section of 'differential diagnoses'.

5.

Differentiating clinical features	*Leukemia*	*'Lymphoma'* (= Lymphosarcoma: Hodgkin's / non-Hodgkin's)	*'Secondaries'* i.e. regional metastatic lymphadenopathy
Age	Children and young adults	Adults	Usually 'old' age
Pain	Absent	Absent	Present in later stages; of variable intensity. May be severe in late stage of the disease
(Degree of) Anemia	Marked	Present	Present
Number of groups	Multiple	Multiple	Single
Typical region/s involved	Many	Neck	Any
Ulcer	Absent	Absent	±
Size	Moderate	May be large	Moderate or large
Matting	–	±	±
Involvement of deeper tissues	Absent	Absent	Usually present
Consistency	Firm	Firm	Hard
A lesion in the drainage area	Absent	Absent	Usually present Absence ⇒ occult primary OR a primary lesion which has been previously treated
Hepatosplenomegaly	±	±	Hepatomegaly may be present due to distant metastatic spread

Free ebooks ==> www.Ebook777.com

6.

Differentiating clinical features	*Hodgkin's disease*	*Non-Hodgkin's lymphosarcoma*
Age	Young and adults	Adults
(Preferential) Regions involved	Bilateral supraclavicular first	Supraclavicular, mediastinal
Size of the enlarged nodes	May be large and equal	Large and unequal
Consistency	'Elastic'	Firm

7. **Think of less common and rare causes** if necessary.

DIFFERENTIAL DIAGNOSIS of some other disorders (which are not covered in the tables above)

Metastatic lymphadenopathy

- Persistent and progressive enlargement. Progress—rapid
- Painless to begin with; (severely) painful later on
- Fixed to deeper structures and surrounding tissues, and later on to the skin with subsequent softening and ulceration
- Evidence of primary malignant tumor in the drainage area ±

↓

  - Usually a carcinoma
  - Only few sarcomas give early lymph node metastases: Malignant melanoma* (correct term—melanosarcoma), malignant neurofibroma, lymphosarcoma, reticulosarcoma and Ewing's tumor of bone.



Fig 16-1 Metastatic lymphadenopathy

Evidence of primary cancer may be absent if the primary cancer is occult, or if it has been previously treated.
Stage - .....?

Metastatic lymphadenopathy with occult primary (clinically)

- = malignant lymphadenopathy without the knowledge of the primary site of cancer
- In about 1/3rd of patients, the primary site can be found on investigation
- The primary site found in the descending order of frequency: Nasopharynx, tonsil, base of the tongue, thyroid, supraglottic larynx, floor of the mouth, palate, pyriform fossa, bronchus, esophagus, breast, and stomach.

Chronic nonspecific postinflammatory residual lymphadenitis—of no clinical and therapeutic significance

- Almost always palpable in groins and neck. The individuals are otherwise asymptomatic. Reason for it being common in groin / neck: Feet, scalp and mouth are the most frequent sites of recurrent minor injuries and portal of infection
- Nontender
- Small (i.e. up to 1 cm in diameter) and 'shotty'
- Consistency—firm.

Notes

- Residual enlargement occurs following recurrent attacks of nonspecific lymphadenitis through childhood and young adulthood

* Progress—much rapid. Consistency—less hard than due to secondary carcinoma. Black pigmentation is evident when the lymph node ulcerates.

www.Ebook777.com

– This condition is of no clinical concern provided it is confined to groins or neck (only occipital/ submandibular/upper deep cervical lymph nodes) regions and if clinical diagnosis is confident.



Fig 16-2 Tuberculous caseating lymphadenitis

Tuberculous lymphadenitis: There are 4 distinct clinical types:

1. ***Acute***
   – Infants and children < 5 years of age
   – Painful and tender lymphadenitis
   – Moderate fever
   – Clinical picture resembles acute septic lymphadenitis.
2. ***Caseating***
   – The most common type seen in young adults
   – Constitutional symptoms may be present
   – Lymph nodes *moderately* enlarged
   – Caseation → softening, formation of cold abscess and subsequently sinuses / ulcers.
3. ***Hyperplastic***
   – Very little constitutional disturbance
   – Lymph nodes—*large*, freely mobile, and feel fleshy/elastic—resemble those due to lymphoma.
4. ***Atrophic***
   – Seen in elderly individuals in whom lymphoid tissue undergoes natural involution
   – Lymph nodes—comparatively small and soon burst with the onset of caseation

Clinical diagnosis of tuberculous lymphadenitis is not difficult in a classical case.

Pitfalls

– In children under 5 years of age, tuberculous lymphadenitis develops as an acute process and consequently may be mistaken for septic lymphadenitis
– Hyperplastic variety is easily mistaken for lymphoma.

Filarial lymphadenopathy

Basically, chronically enlarged lymph nodes most commonly inguinal, although no site is immune. Filarial lymphadenopathy may affect axillary and even cervical lymph nodes.

Differentiating points

– Lymphedema in the drainage area such as the limb, penis and scrotum, vulva, breast, etc.
– Past h/o recurrent attacks of high-grade fever with rigors + acute lymphadenitis ± acute funiculitis (in c/o inguinal lymphadenitis) ± acute lymphangitis
– The patient residing in an area endemic for filariasis.

Infectious mononucleiosis

– Young adults between 15 and 30. Rate is higher in college students than in general population
– Anorexia, fatigue, malaise, headache
– The conventional triad of fever, sore throat and rash immediately f/b cervical lymphadenopathy—may last for several weeks
– Subsequently other regions are affected
– Hepatosplenomegaly in half of the cases
– Tonsillitis ± difficulty in swallowing. Rarely, tonsillar enlargement → airway obstruction.

Notes

– High grade fever late during the illness suggests bacterial superinfection.
– Mode of transmission—direct contact with virus-infected saliva, such as through kissing.
– A patient is infectious for several days prior to getting symptomatic

Free ebooks ==> www.Ebook777.com

- The virus may continue to be excreted in the saliva for years after infection
- Complications: Splenic rupture, autoimmune hemolytic anemia, thrombocytopenia, pericarditis, encephalitis, cranial nerve palsies.

Sarcoidosis

- Region/site/group affected: Submental, preauricular, occipital, epitrochlear
- Painless small discrete enlargement
- Generalized lesions in the
  - Skin: Papules/nodules
  - Bones: Cysts
  - Various glands → enlargement of lacrimal gland, parotid gland, etc.
  - Lungs: Peribronchial fibrosis (X-ray finding).

Lymphogranuloma venerium / inguinale

It is a venereal infection (sexually transmitted tropical disease caused by *Chlamydia trachomatis*) affecting inguinal lymph nodes. (In females it usually produces a rectal stricture).

- The primary lesion consists of a fleeting painless genital papule or ulcer often unnoticed by the patient
- Incubation period of the secondary lesions involving the inguinal lymph nodes is 2–8 weeks, occasionally as long as 4 months
- Lymph nodes both above and below the inguinal ligament are enlarged, painful and matted → the 'sign of the groove'
- Characteristic abscess formation
- In later stages, lymphatic spread and blockage → edema of the genitals, vulval elephantiasis, vaginal stricture, urethritis and urethral stricture.

Lipid and glycogen storage diseases

- Children
- Hepatosplenomegaly
- Generalized lymphadenopathy is a minor part of the clinical picture.

Sporotrichosis

- A fungal infection
- Starts with a wound on the hand or face in farmers and gardeners
- Regional lymphadenopathy with softening and ulceration.

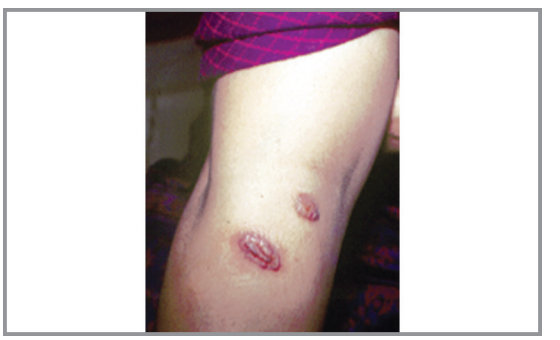


Fig 16-3 Primary lesion of sporotrichosis

Cat Scratch fever

- Primary lesion at the site of inoculation:
  - 3–5 mm macule develops 3–10 days after animal contact. – Evolves in a papule / vesicle / pustule.
  - Inoculation pustules ±
- Regional lymphadenitis:
  - 1–8 weeks later (i.e. 1–8 weeks after the primary lesion appears)
  - Suppuration ±
  - Spontaneous resolution in 2–4 months.
- Toxemia ± (malaise, headache, myalgia)
- Systemic involvement ±: Hepatosplenomegaly, thrombocytopenic purpura, pneumonia, osteomyelitis, encephalitis.

Tularemia

- This disease is contracted through rabbits and squirrels
- Lymphadenopathy with abscess formation.

www.Ebook777.com

Lymphadenovarix: Refer to page 309, in chapter 24.

***BASELINE* INVESTIGATIONS** in a case of lymphadenopathy

All the investigations listed below are not indicated in every case; a selective approach is recommended. For example, tuberculin test is of value in children but not in adults.

- **Full blood count**
  - Anemia
  - ↑ lymphocytic count

  } ⇒ Leukemia / infectious mononucleiosis.
- **Serological tests**—for evidence of infection with HIV, EBV, CMV, etc.
- **Tuberculin test**—for tuberculosis
- **S. LDH (serum lactate dehydrogenage)**: ↑ values ⇒ leukemia / lymphoma
- **Serum bilirubin and transaminase levels** (ALT, AST) for the evidence of involvement of liver in miliary tuberculosis, leptospirosis, HIV, EBV, CMV, etc.
- **X-ray chest**
- **Bone marrow aspiration** if hematological condition is suspected (hepatosplenomegaly, pancytopenia, lymphocytosis,↑ LDH)
- **FNAC / biopsy** of enlarged lymph node—for infection / tumor.

Free ebooks ==> www.Ebook777.com

17 CHRONIC BONE SWELLING

- Diseases of the intracapsular part of bone present as a 'joint case'
- Diseases of the remaining part of the bone are considered in this chapter as 'chronic bone swelling'.

Causes of bone swelling

Common

- Post-traumatic.
- Developmental. Example —metaphyseal aclasis (Many bones are invloved)
- Deficiency conditions. Examples —rickets, scurvy (Many bones are invloved)
- Cyst—solitary
- Benign tumor
- Malignant tumor
- Chronic inflammatory swelling

Rare

- Nonossifying fibroma (monostotic fibrous dysplasia)
- Polyostotic fibrous dysplasia
- Hydatid cyst
- Lipoma, fibroma
- Osteoid osteoma
- Benign chodroblastoma
- Adamantinoma
- Hemangioma
- Reticulosarcoma
- Periosteal sarcoma
- Malignant osteoclastoma
- Soft tissue malignancy invading the bone
- Eosinophilic granuloma

HISTORY

- **Duration**? • **Mode of onset**? • **Progress**?
- In case of sinus(es) being present—do you find extrusion of bone chips through the opening (mouth) of the sinus(es)?
- Any recent or remote past h/o **injury**?
- **Any other swelling of similar nature elsewhere or in the same region**?
- **Pain**? – Details.
- **Fever**? – Details.
- Did pain precede the appearance of fever? – If so, how long?
- **(Significant) Loss of weight**?
- Any **pressure effects**?
- H/s/o fracture?
- **Past h/o** any skin infection, otitis media, measles, pneumonia or typhoid fever? - This is frequently obtained in osteomyelitis.

INTERPRETATION OF SYMPTOMS, AND DISCUSSION

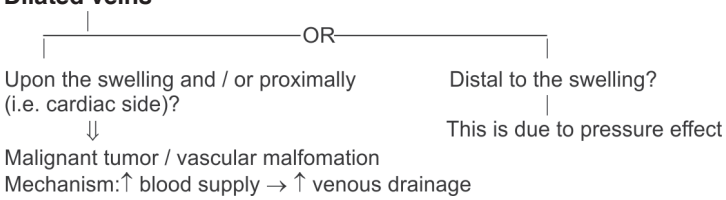
- *H/o **injury*** is useful only if the swelling has appeared immediately after a severe injury
- If the swelling appears about a week after injury, then most probably it could be acute osteomyelitis
- If the swelling follows months after injury, it has no diagnostic significance except that it may be an excessive callus or a malunited fracture in a case where diagnosis of fracture was missed or the fracture neglected
- ***Severe** pain **with high grade fever** for a few **days preceding** the appearance of the swelling or, both appearing more or less simultaneously* ⇒ acute osteomyelitis
- ***(Severe)** pain **with/without fever** for a few **weeks preceding** the appearance of the swelling* ⇒ osteogenic sarcoma / Ewing's sarcoma.

www.Ebook777.com

LOCAL EXAMINATION

INSPECTION

- **Number**?
- **Site**:
  - Part of the body?
  - Which bone/s:
    - Region of the (long) bone—epiphysis / metaphysis / diaphysis?
    - Circumscribed to only one surface (= surface swelling) OR circumferential i.e. all around the bone (= central swelling)?
- **Extent**?
- **Shape**?:
  - Globular shape ⇒ 'central swelling'
  - 'Surface swelling' tends to produce pedunculated shape as for example in cancellous osteoma.
- **Size**?
- **Skin**:
  - Redness?
  - Edema?
  - Ulceration (± fungation)?
  - Sinus(es)?
    - Sprouting granulation tissue at the mouth of the sinus ⇒ not only osteomyelitis, but the presence of dead tissue (sequestrum) at the depth
    - Extrusion of bone chips—strongly suggests osteomyelitis
    - Discharge from multiple sinuses containing yellow-colored granules of the size of a pinhead (Pus is collected in a clear test tube and observed in a powerful light to see these granules) ⇒ actinomycosis.
- **Scar**. Depressed? A depressed scar suggests fibrosis resulting from previous suppuration.
- **Dilated veins**



- **Pulsations**: A bony swelling may be rarely pulsatile to some extent.
  Examples: Rapidly growing highly vascular sarcoma, hemangioma if the overlying bone is thinned out.
- **Local wasting**?
- Evidence of **pressure effects distal to the swelling**: Edema, ischemic changes, paresis / paralysis.

DISCUSSION

- Bony swelling is usually circumferential as bone is the central structure in the limb
- 'Surface swellings' usually arise from the outer part of the compact bone or periosteum or soft tissue growth invading the bone
- Clinically, it may be difficult to decide about the site of origin in the long bone i.e. whether the swelling is in the epiphysis or metaphysis or diaphysis.

PALPATION

- Temperature?
- Tenderness? - Inflammatory swellings are tenderer than malignant swellings.
- Site and extent?
- Shape?
- Size?
- **Mobility** of **soft tissue overlying the bone swelling**?

Free ebooks ==> www.Ebook777.com

Clinical method: Grasp a wide area of soft tissues between fingers and thumb so as to include skin, subcutaneous tissue and muscles, and move it over the swelling.

Observation: Mobility present or absent?

Inference:

– Fixity ⇒ malignant tumor (exception—multiple myeloma) / osteomyelitis
– Mobility ⇒ cyst / benign tumor.

- **Surface**:
  – Irregularity?
  – Rounding or blunting of the sharp margin?
  – Increase in the transverse diameter?

  (Drawing an inference is facilitated by comparison with the opposite side.)

  The presence of one or more of these abnormalities ⇒ Thickening of the bone.
- **Pulsations**? A bony swelling may be rarely pulsatile to some extent.

  Examples: Rapidly growing highly vascular sarcoma, hemangioma if the overlying bone is thinned out.
- **Consistency**?
  – Soft / firm—osteosarcoma
  – Hard in rest of the diseases.
- **Egg-shell crackling** i.e. does the swelling yield or break on pressing? ⇒ expansion of the bone.

Enlargement of a bone could occur due to either thickening or expansion.

Thickening *of bone*	***Expansion*** *of bone*
→ Broadening of the wall and narrowing of the cavity	→ Thinning of the wall and broadening of the cavity
– New bone is laid down by periosteum on the surface and in the interlamellar spaces (sclerosis) and there may be even narrowing of the medullary cavity	– Compact bone gets thinned out and medullary cavity or the central part of the bone gets greatly expanded, giving the overall increased diameter
– Thickening of the end of a (long) bone enlargement in anteroposterior and mediolateral diameters but *not* towards the joint as there is no periosteum where the bone is covered by articular cartilage	– Expansion of the end of a (long) bone → enlargement in not only anteroposterior and mediolateral diameters, but also towards the joint → disturbance in the joint axis especially if the disease producing expansion is eccentrically situated causing varus/valgus deformity beyond the joint – Expansion occurs in centrally placed benign tumors and cysts

MEASUREMENTS

1. Length of the limb?
   – Shortening may be due to pathological fracture or result of acute osteomyelitis (leading to destruction of epiphyseal cartilage)
   – Lengthening may be seen in chronic nonspecific osteomyelitis. Mechanism: Increased vascularity in the region of metaphysis → increased growth rate.
2. Circumference of the limb?

   This may reduce due to muscular wasting.

REGIONAL EXAMINATON

- Examination of the nearby **joints**
  – Any swelling?

    Swelling of joint could be either due to diseased joint or sympathetic effusion secondary to bone disease (such as osteomyelitis)

www.Ebook777.com

  - Does the swelling involve both bones on either side of the joint?—if so, then it may be most probably a joint case rather than a bone disorder
  - Disturbance of the axis of the joint?—valgus / varus deformity ⇒ expansion of the bone.
- **Oral cavity** in c/o a jaw swelling
  - Oblique position / dislocation/shedding of any tooth / teeth? ⇒ expansion of the bone (jaw).
- **Pressure effects distal to the swelling**?
- **Regional lymph nodes**?

THE OPPOSITE SIDE

SYSTEMIC EXAMINATION

Examine other bones in the body, in addition to routine aspects of systemic examination.

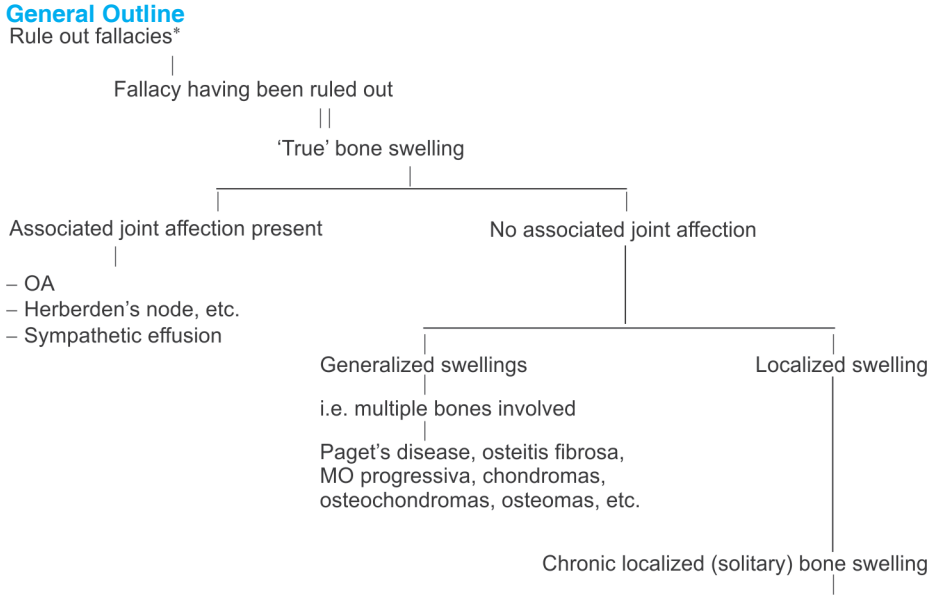
ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. Some of the otherwise common physical signs like margin, consistency are not very useful while attempting to make diagnosis in case of a bony swelling. Hence, additional data from X-ray films of the bone is necessary before giving a clinical diagnosis.
2. Rare causes should not be considered in a routine case but should only be discussed when the case does not fit in any common condition.
3. Due to sympathetic effusion if present, a case may be mistaken as a joint case.
4. Rarely, malignant synovioma and synovial chondroma of a joint may be mistaken for a bony tumor.

STEPWISE ANALYSIS OF DATA

General Outline

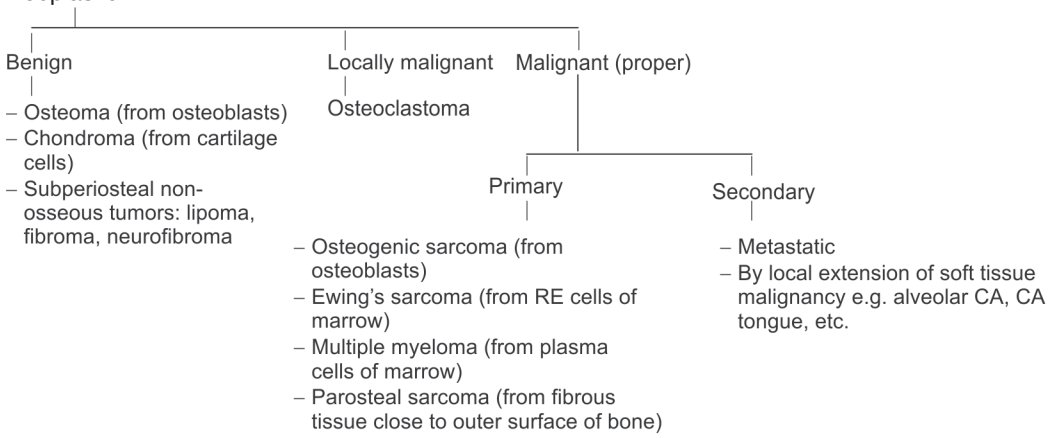


Contd...

***Fallacies**: – Normal bones e.g. prominent ribs in scoliosis, sacral promontory, tarsal bones in flat foot
– Hypertrophied bones due to overuse and in A-V fistula
– Calcification in soft tissue
– A tense cyst in close relation to bone.

Free ebooks ==> www.Ebook777.com

1. Post-traumatic: Malunited fracture / dislocation, myositis ossificans traumatica, excessive callus, subperiosteal hematoma with / without its calcification.
2. Cystic: Solitary cyst, aneurysmal bone cyst, odontome-cyst (in the jaw), cyst associated with osteitis fibrosa, hydatid cyst.
3. Inflammatory: Chronic osteomyelitis—nonspecific, tuberculous, actinomycotic, syphilitic.
4. Neoplastic



Easily diagnosed amongst the common conditions

- In infants – scurvy
- In children – rickets
- Post-traumatic conditions
- Multiple chondromata
- Osteoma

For differential diagnosis, refer to pages 215 and 216

If these (above-mentioned common conditions) are ruled out, especially a solitary bone swelling then needs to be differentiated **broadly** between the following 4 groups viz. cyst / benign tumor, chronic osteomyelitis and malignant tumor.

Differentiating clinical features	**Cyst / benign tumor**	**Chronic osteomyelitis** *in general*	**Osteoclastoma** *Locally malignant tumor*	**Malignant tumor**
Duration	Usually > a year	≥ 6 months	< a year. Usual history 3–6 months	Usually < a year
Progress	Slow or 'very slow'	–	Fairly rapid	Rapid and persistent
Pain	Absent	±	Absent	Present. Pain is a presenting symptom in osteosarcoma
Fever	Absent	±	Absent	Present. Exception: Multiple myeloma
Number	May be multiple	Single	–	Usually single. Exception: Multiple myeloma and secondary deposits
Loss of weight	Absent	±	Absent	+
Signs of inflammation viz. redness and edema	Absent	+	Absent	+

Contd...

www.Ebook777.com

Contd...

Differentiating clinical features	**Cyst / benign tumor**	**Chronic osteomyelitis** *in general*	**Osteoclastoma** *Locally malignant tumor*	**Malignant tumor**
Ulcer/sinus	Absent	±	Absent	±
Dilated veins	Absent	Absent	+	±
Fixity of soft tissue to the swelling	Absent	+	Absent	+. Exception: Multiple myeloma
Egg-shell crackling	±	Absent	+	Absent
Regional lymphadenopathy	Absent	±	Absent	±

Further differentiation between various types in each of the above broad groups

Differentiating clinical features	**Solitary cyst**	**Single (solitary) chondroma**	**Osteoclastoma** *– locally malignant tumor*
Bones commonly affected	Long	Long / short / flat. Most frequent sites: Phalanges, metacarpals, metatarsals	Long. – Epiphysis; jaw, etc.
Size	'Moderate'	May be 'large'	May be 'large'
Warmth	-	-	+. (Other signs of inflammation viz. redness and edema absent)
Surface	Smooth	Lobulated	Smooth
X-ray findings			
Soft tissue shadow	-	+	Absent
Expansion	+	Only in central tumor	++
Loss of bone shadow/ cavity	+	+	+. Well-defined with septa → 'soap-bubble' appearance
Rarefaction (Decalcification)	-	+	Absent
Sclerosis	Absent	Absent	Absent
Periosteal new bone formation	Absent	Absent	Absent
Sequestrum	Absent	Absent	Absent
	 **Fig 17-1**	 **Fig 17-2**	 **Fig 17-3**

Free ebooks ==> www.Ebook777.com

Solitary cyst (= Localized osteitis fibrosa)

– Adolescents
– Common sites—proximal metaphysis of humerus and femur
– Usually not recognized until spontaneous fracture has occurred.

Recurrence of the cyst after grafting is fairly common when the grafting is performed under the age of 10 years.

Aneurysmal bone cyst

– An uncommon lesion occurring in vertebrae, flat bones as well as long bones
– There is a formation of numerous vascular spaces so that the area affected is transformed into an expanded blood-filled sponge
– The swelling is progressive and destructive
– Patient presents with a swelling or a fracture
– X-ray findings: Expanded cyst of honeycomb texture.

Differentiation between a bone cyst and osteoclastoma on an X-ray film: Bone cyst expands along the long axis of the bone, whereas osteoclastoma expands transversely.

Multiple bone cysts = Generalized osteitis fibrosa: Result of pararthyroid adenoma.

– Several bones affected in adult
– Pain or fracture draws the attention.



Fig 17-4 Aneurysmal bone cyst

Differentiating clinical features	**Nonspecific osteomyelitis**	**Tuberculous osteomyelitis**	**Actinomycotic osteomyelitis**	**Syphilitic osteomyelitis**
Duration	≥ 6 months	≥ 6 months	Often > a year	≥ 6 months
Pain	+	±	Absent	Deep boring pain particularly when the part is warm
Fever	+	Low-grade in the evening	Absent	Absent
Loss of weight	Slight, in the late stage	+	Absent	Absent
Bones commonly affected	Long. – Metaphysis extending to diaphysis	Short long, flat and irregular – Diaphysis. Predilection for spine, ribs, and joints with extensive synovial membranes e.g. hip and knee	Foot, leg, hand and jaw. – All parts of the bones involved	Long bones, skull, ribs, sternum, hand and foot. – Diaphysis
Extension into joint space	Usually not	+	–	–
Size	Moderate. Never large	Small	'Moderate'	'Moderate'
Sinus / ulcer	+	+	Many sinuses	±
Regional lymphadenitis	+	+	Absent	Absent
Remarks	Usually a successor process following acute osteomyelitis, although it may result from other causes (Refer to the flow-chart below)	Chronic *de novo*. ∴ mode of onset is insidious, and the diagnosis is delayed until widespread destruction has already occurred	–	–

Contd...

www.Ebook777.com

Contd...

X-ray findings	Nonspecific osteomyelitis	Tuberculous osteomyelitis	Actinomycotic osteomyelitis	Syphilitic osteomyelitis
Soft tissue shadow	Absent	Absent	Absent	Absent
Expansion	Absent	Absent	Absent	Absent
Loss of bone shadow/cavity	One or more. 'Small to moderate' size	One or more. 'Small to moderate' size	Many small cavities on the surface	One or more. 'Small to moderate' size
Rarefaction (Decalcification)	+	++	+	Absent
Sclerosis	++	Absent	+	+++
Periosteal new bone formation	Parallel type	Absent	Absent	Parallel type
Sequestrum	++. Sequestrum is ensheathed by involucrum. Sequestrum is frequently more dense than involucrum	Absent	Absent	Absent
	 **Fig 17-5**	 **Fig 17-6**	 **Fig 17-7**	 **Fig 17-8**

Causes of chronic (nonspecific) osteomyelitis

A. Following inadequate treatment of acute pyogenic osteomyelitis
Causes of acute osteomyelitis:
Hematogenous / traumatic / iatrogenic (e.g. postoperative) spread / introduction of infection to the bone

C. Spread from a neighboring focus of sepsis for example: Perforating neuropathic ulcer, complicated dental infection, etc.

B. Chronic osteomyelitis *de novo*—Brodie's abscess. Usually not associated with sinus formation.

Free ebooks ==> www.Ebook777.com

Differentiating clinical features	**Osteosarcoma**	**Chondrosarcoma**	**Ewing's sarcoma**	**Multiple myeloma**	**'Secondaries'**
Age (in years)	10–20	20–30 and 50–60	5–15	40–60	Usually > 45
Bones commonly affected	Long – Metaphysis	Long and flat – Metaphysis	Long and flat – Diaphysis	Bones with red marrow – Usually their ends	Bones with red marrow – Usually their ends
Size	May be 'large'	May be 'large'	'Moderate'	Small	Small
Ulcer/fungation	+	+	Absent	Absent	+
Dilated veins	++	+	+	Absent	Absent
Surface	Irregular	Lobulated	Irregular	Smooth	Smooth
Local wasting	++	+	Absent	Absent	+
Regional lymphadenopathy	+	Absent	+	Absent	Absent
Pathological fracture	Likely	Likely	No	Likely	Likely
X-ray findings					
Soft tissue shadow	+	+	+	Absent	Absent
Expansion	Absent	Absent	Absent	Absent	Absent
Loss of bone shadow/cavity	++	++	+	++. Multiple punched out areas from the size of a pea to that of an orange	++
Rarefaction (Decalcification)	+	+	+	+	+
Sclerosis	++	Calcified flakes	Absent	Absent	Absent. Exception: CA prostate
Periosteal new bone formation	1. Sunray type —may appear as irregularly scattered spicules 2. Codman's triangle at periphery	±	→ 'Onion layers' appearance	Absent	Absent
Sequestrum	Absent	Absent	Absent	Absent	Absent

Contd...

www.Ebook777.com

Contd...

X-ray findings	Osteosarcoma	Chondrosarcoma	Ewing's sarcoma	Multiple myeloma	'Secondaries'
	 Fig 17-9	 Fig 17-10	 Fig 17-11	 Fig 17-12	 **Fig 17-13** Lytic mets  **Fig 17-14** Blastic mets

Osteosarcoma

- Average duration of osteosarcoma is about 6 months. If a patient presents within one month or after one year from the onset of symptoms, the case is probably not one of osteosarcoma
- **Severe** pain **with / without fever precedes** the appearance of the swelling by a few **weeks** / months
- Consistency varies between soft and hard. A highly vascular tumor feels soft, may pulsate and even a murmur may be heard.

Secondaries

- Deep seated pain in the affected bone is often the chief presenting complaint
- Spontaneous fracture may be the first indication of the disease.

DIFFERENTIAL DIAGNOSIS of scurvy, rickets, post-traumatic conditions, multiple chondromata, osteoma, and some other disorders (which are not covered in the tables above)

Scurvy

Differentiating points

- Acute swelling due to subperiosteal hematoma
- Immobility due to severe pain and tenderness
- Bleeding swollen gums
- Marked anemia.

DD: Paralysis, acute pyogenic abscess, acute osteomyelitis.

Free ebooks ==> www.Ebook777.com

Rickets

Differentiating points

– Bossing of the skull → 'hot cross bun' appearance to the skull
– Bending of ribs and rosary formation at costochondral junctions
– Enlarged epiphyses
– Saber shaped tibia.



Fig 17-15 Rickets

Post-traumatic conditions

– H/o sever trauma usually available, **but may be forgotten**
– Static mass.

1. Malunited fracture: Painless and without tenderness.
2. Dislocated bone ends.
3. Excessive callus → usually a fusiform swelling.
4. Myositis ossificans:
   – Appears weeks or months after injury
   – Gradually increasing pain and decreasing movements
   – Ill-defined
   – Takes the shape of the muscle.
5. Localized periostitis → periosteal nodule.

Multiple chondromata

– Young age
– Common sites: small bones of hand and foot
– Multiple painless swellings of hard or elastic consistency
– Centrally placed swellings → expansion of the bone with globular shape and smooth surface
– The disease tends to be unilateral and the affected limb is short.

*Complication*s:

– Pathological fracture
– Malignant transformation can occur.

Compact (Ivory) osteoma

Sessile mass of compact bone usually arising from the bones of the face and skull, especially frontal and mastoid regions.

Cancellous osteoma

– Starts in the adolescent age group and grows till the bone grows
– Common site—long bones, in the region of metaphysis growing towards the middle of the bone
– Pedunculated
– Lobulated
– Familial.

Complication: Malignant transformation possible. Exostosis becoming painful or continued growth after the completion of skeletal growth should raise the suspicion of malignant transformation.

Multiple exostoses (= Multiple cancellous osteomas = Metaphyseal aclasis = Diaphyseal aclasis)
= Widespread affection of cancellous osteoma.

– Often a dwarf with or without articular deformities
– Common sites—around wrist, shoulder and knee joints
– May be asymptomatic; even the patient may be unaware of their existence
– Attention is drawn due to pressure symptoms upon nerves / muscles / tendons.

www.Ebook777.com

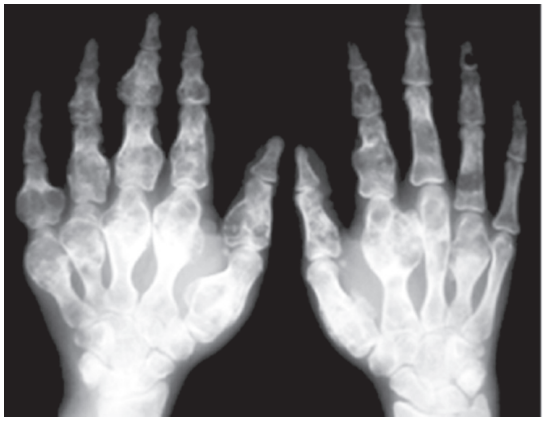


Fig 17-16 Multiple chondromata

Subungual exostosis
= cancellous osteoma occurring on the dorsum of the terminal phalanx of the great toe.
Forms a granulating mass (due to infection) projecting from under the side of the nail. Consistency—hard.

Osteoid osteoma
Differentiating points
- Age: 10–30 years
- Persistent severe pain unrelieved by rest, more at night; relieved significantly by aspirin
- Predilection for long bones
- The only physical sign is bone tenderness; even this is often absent; so the patient may be regarded as hysterical.

X-ray findings
- A small cavity of < 1 cm diameter surrounded by sclerosis. Towards the center of the sclerotic area and often obscured by it, there is a radiopaque 'nidus' in a radiolucent zone, which represents the tumor itself
- In case of a surface tumor, there may be overlying subperiosteal new bone formation.

Eosinophilic granuloma
- Age: 10–20 years
- Painful and tender swelling. Fever, loss of weight
- Skull, jaw, ribs and femur.

X-ray findings
- A punched-out area without any surrounding reaction
- The cortex is not expanded but may be destroyed.

Basically, the disease ranges in severity from a solitary lytic lesion in the bone to a fatal leukemia-like disorder.

Adamantinoma
- 20–40 years
- Long bones
- Slow progress
- X-ray: Multilocular appearance.

Soft tissue malignancy invading the bone e.g. malignant synovioma
The tumor invades both bones on either side of the joint which is rare in primary bone tumors.

Free ebooks ==> www.Ebook777.com



Fig 17-17 Osteoid osteoma



Fig 17-18 Eosinophilic granuloma



Fig 17-19 Adamantinoma

Osteitis deformans (Paget's disese)

- Can be monosteotic or generalized
- > 40 years
- Slowly progressive
- Pain
- Deformity resulting from bending of bones (bowing of legs, kyphosis, etc.)
- Thickening of bones especially tibia, skull (patient progressively requires hats of larger size).

Complications:

- Pathological fracture
- Development of sarcoma.

Myositis ossificans progressiva

- Congenital
- Formation of masses of bone in the course of muscles especially of the neck and trunk → progressive restriction of movements of the spine and ribs.

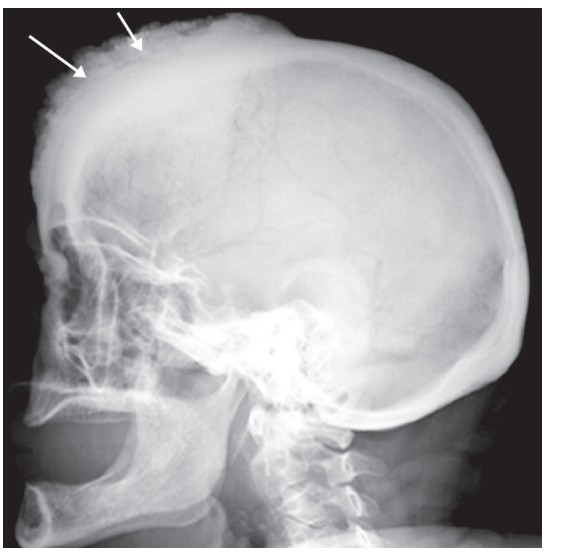


Fig 17-20 Osteitis deformans

www.Ebook777.com

18 CHRONIC VASCULAR SWELLING

Criteria of a vascular swelling: Pulsatile OR compressible OR both pulsatile + compressible.
However, the absence of pulsations/compressibility does not rule out the possibility of a swelling being vascular in nature.

HISTORY

- **Duration**?
  Present since birth OR Did it appear later?
- **Progress**, **regress**, **fluctuation**?
- **Any other swelling of similar nature elsewhere**?
- H/o **injury**?
  H/o injury **may be forgotten in long-standing fistulae.** Hence, the absence of h/o injury should not rule out the possibility of (an acquired) fistula
- **Past h/o any vascular surgery** (particularly anastomosis)?
  Desirable details to look from medical record if available
  - Duration between the vascular surgery and the onset of the present vascular swelling (false aneurysm)?
  - Was an endarterectomy performed simultaneously?
  - Suture material used?
  - Was an anastomosis performed? Does the anastomosis seem to have been performed under great tension?
- Do you feel a **vibrating sensation OR buzzing sound** over the swelling?
- **Paresthesia/pain** in distal part of the limb?
  - Intermittent 'pain / limp'/rest pain?

  OR
  - Severe agonizing continuous bursting pain, radiating down the course of the artery?
- **Fatigue/weakness** in the limb?

- Raynaud's phenomenon?
- Chronic indolent ulcer?
- Blackening of one/more digits or more extensive area (suggesting gangrene)?
- Paresis/paralysis?

} **Symptoms of chronic arterial insufficiency** (other than pain and fatigue)

- Coldness and deadness of the limb?
- Acute spreading ulcer with cellulitis?
- Blackening of one/more digits or more extensive area (suggesting gangrene)?

} **Symptoms** (other than pain and paresthesia) **of acute arterial insufficiency** leading to critical limb ischemia

Free ebooks ==> www.Ebook777.com

- **Edema** of the affected limb?
  - Itching, scaling and weeping [due to dermatitis (eczema)]?
  - Discoloration of skin in the gaiter area?
  - Active/healed/repeatedly recurring ulcer?
  - 'Venous limp': A bursting discomfort in the leg following use/exercise?
  - Vague cramps?
  - Aching on dependency/before periods?
  - 'Heaviness' in the legs?

  } **Symptoms of chronic venous insufficiency**
- Any associate illness—particularly HT, DM, IHD, CVD?

INTERPRETATION OF SYMPTOMS, AND DISCUSSION

Duration

- The point of time of onset is the single most important question to differentiate AVM from hemangiomata
- The presence of swelling since birth definitely means an AVM
- Presentation later in life suggests any vascular lesion, though differentiation is possible with the help of additional points in history and physical examination
- Age at the time of first clinical presentation of an AVM is inversely related to the severity of the lesion.

Progress, regress, fluctuation:

- Hemangiomata grow in the early life, and then tend to regress
- AV malformations usually grow with age
- AV malformations may enlarge dramatically during puberty and pregnancy.

Multiple true aneurysms beginning to develop at a young age suggests some connective tissue disorder as an etiological factor.

Anastomotic false aneurysm can arise many years after arterial surgery.
A false aneurysm occurring at an early stage after vascular surgery suggests graft infection as a possible cause.

- *Causes of anastomotic (false) aneurysm*
  - Infection—the commonest cause
  - Simultaneous performance of endarterectomy
  - Anastomosis performed under great tension
  - Use of absorbable suture material or silk for anastomosis
- Possible causes of ***paresthesia / pain*** in distal part of the limb
  - Ischemia
  - Pressure on sensory nerves
  - Intermittent 'pain/limp'/rest pain suggests chronic arterial insufficiency
  - Severe agonizing continuous bursting pain, radiating down the course of the artery suggests acute arterial insufficiency
- Possible causes of ***fatigue/weakness*** in the limb: Ischemia / pressure on motor nerves
- Possible causes of ***edema*** of the affected limb: Due to pressure on veins by an aneurysm / AVF.

LOCO-REGIONAL EXAMINATION

- **Inspect the swelling in the most dependant position and then in elevated position** (–the positions with respect to the heart)
  - If the swelling decreases in size or disappears in elevated position ⇒ predominantly venous malformation. On complete disappearance of the swelling, the part may look completely normal (except for a dermal component if present)
  - Subsequently, examine the swelling with the part in the most dependant position to get maximum details of the swelling that is apparent to its fullest extent.
- **Skin overlying the swelling**?
  - Redness
  - A dull blue hue ⇒ a large deep-seated vessel or vascular lesion.

  A cutaneous mark may represent only a small part of the total lesion (just the tip of MAY BE A VERY BIG iceberg).

www.Ebook777.com

- Any **scar**?
  - Scar may implicate accidental injury or iatrogenic cause in the development of a false aneurysm or acquired AVF
  - Anastomotic false aneurysm can arise many years after arterial surgery
- **Color of the swelling itself, and color in the distal portion of the limb**?
  - Red/purple ⇒ AV malformation
  - Pallor/cyanosis in the limb distal to the swelling ⇒ ischemia
- **Temperature overlying the swelling and in the distal portion of the limb**?
  - Raised overlying an AV fistula
  - Reduced temperature in the limb distal to the swelling ⇒ ischemia
- **Simultaneous examination of pulses on both sides**
  - Compare their volume
  - Is there any delay between the 2 sides?

  Low volume and delay ⇒ ischemia.
- **Margin**?
- **Shape**? The shape of a true aneurysm may be fusiform or saccular.
- **Size**?
- **Thrill**?
  - Causes—AV fistula, narrow segment of an artery (a narrow opening of the heart).
- **Pulsations**?

  Keep flat of the fingers on the swelling for a few minutes, and try to perceive pulsations carefully.

  Causes of pulsatile swelling

  1. An aneurysm/AV fistula → expansile pulsations = 'true pulsations'.
  2. A highly vascular swelling or tumor → true pulsations. Examples: Rapidly growing highly vascular sarcoma, osteosarcoma, encephalocele, pulsatile exophthalmos, toxic goiter, pulsatile liver in tricuspid incompetence.
  3. A swelling overlying or underlying an artery → 'transmitted pulsations'.

  Clinical criteria / tests differentiating aneurysm from other pulsatile swellings

  1. A swelling lying in line of a 'main' (large / medium-sized) artery is more likely to be an aneurysm.

  2. Expansile pulsations ⇒ aneurysm or AVF, whereas transmitted pulsations ⇒ a swelling overlying an artery.

  Clinical method: Place two index fingers on 2 sides of the swelling as far apart as possible, and observe.

  Fingers raising and separating from each other ⇓ Expansile pulsations

  Mere raising of the fingers ⇓ Transmitted pulsations

  3. – Press the main artery proximal to the swelling

  ↓ in size of the swelling ⇒ aneurysm

  – Then, press the swelling itself gently: The swelling getting compressed i.e. yielding to pressure ⇒ aneurysm.

  Exception: Aneurysm filled with a thrombus will not yield to pressure

  – Observe on removal of pressure (from both sites i.e. from over the swelling and from over the artery proximally): Return to the former size in series of throbs synchronous with heart beat ⇒ aneurysm.

  4. Apply pressure on the main artery distal to the swelling.

  ↑ in size and tension of the swelling ⇒ aneurysm.

  5. Compare the pulse (with opposite normal side) beyond the swelling for force and timing: Often feeble and delayed.

  6. Mobility: Mobility of an aneurysm is more in transverse axis than that in the longitudinal axis.

Free ebooks ==> www.Ebook777.com

- **Compressibility**
  - On compressing, does it diminish in size OR disappear completely?
  - (And after removing the compression,) Does it refill spontaneously slowly OR in series of throbs synchronous with heart beat?
    - Completely compressible by applying gentle pressure and refilling slowly after removing the compression suggests predominantly a venous lesion
    - Minimally compressible or compressible with difficulty (needs a great pressure), and will fill up in series of throbs synchronous with heart beat suggests an aneurysm or a high-flow AV fistula
    - Incompressible swellings: Hemangioma, saccular aneurysm filled with a thrombus
    - A low-flow AV fistula will fill-up slowly.
- Erosion of the **underlying bone**?

  The presence of erosion ⇒ very long-standing swelling and hence probably congenital.
- **Distal part of the limb**
  - Edema (due to pressure on veins)?
  - Hypertrophy of the limb? - The limb is longer and also larger in diameter than the normal counterpart in congenital AV fistula
  - Veins: Distended / varicose? ⇒ AV fistula
  - Chronic venous HT? (– Skin changes characteristic of postthrombotic syndrome) ⇒ long-standing fistula
  - State of the circulation? (including gangrene) –

    ↓

  a. **Signs of acute ischemia**
    - Discoloration: Serial color changes with advancing ischemia—marked pallor, mottling, cyanosis, and black discoloration
    - Trophic changes if present, suggest a pre-existing chronic arterial insufficiency
    - Venous guttering
    - Reduced temperature
    - Tenderness:
      - Of the (apparently) affected muscles ⇒ necrosis
      - Along the artery ⇒ recent thrombosis
    - Stiff consistency
    - Sensation:
      - 'Partial' ('inconstant') loss of sensation (grade 2 ischemia)
      - Profound anesthesia (grade 3 ischemia)
    - Function (of muscles):
      - Paresis (grade 2 ischemia)
      - Paralysis (grade 3 ischemia)

      Another possible cause of weakness: pressure on motor nerves.
    - Pitting edema ⇒ thrombophlebitis
    - Pulses: The level of block lies proximal to the highest impalpable pulse and distal to the lowest palpable pulse
    - Longer (> 3 seconds) capillary refilling time.

  b. **Signs of chronic ischemia**
    - Discoloration: Pallor/cyanosis/black discoloration
    - Trophic changes:
      - Wasting of the part
      - Skin in general looks thin and shiny
      - Hairs—sparse
      - Digits—shrunken due to loss of pulp
      - Nails—deformed, and show transverse ridges
      - A chronic indolent ulcer.

www.Ebook777.com

  - Venous guttering
  - Reduced temperature
  - Tenderness:
    - Of the (apparently) affected muscles ⇒ necrosis
    - Along the artery ⇒ recent thrombosis
  - Loss of sensation ⇒ gangrene
  - Paralysis ⇒ gangrene

    However, the absence of muscle weakness does not rule out the presence of gangrene. Intact tendon attachment may produce movement of the dead toe.
  - Pitting edema ⇒ thrombophlebitis
  - Pulses: The level of block lies proximal to the highest impalpable pulse and distal to the lowest palpable pulse
  - Longer (> 3 seconds) capillary refilling time
- **Measurement**
  - Objective assessment of
    - Wasting due to chronic ischemia
    - Hypertrophy associated with AVM
- **Recording serial measurements** of hypertrophied limb may be helpful in determining the rate of growth and the need for intervention as timing of intervention may be critical
- **Auscultation**
  - Overlying the swelling, for murmur:
    - Systolic murmur ⇒ aneurysm
    - Continuous machinery murmur ⇒ AVM
  - Along the course of the artery, for bruit.

  [*Murmur* = a continuous buzzing sound which increases in loudness with each heartbeat. *Bruit* (arterial) = an auscultatory sound produced by low frequency vibrations of the arterial wall caused by disturbances in blood flow.]

Branham's sign

- *Purpose*: Demonstration of the presence of central effect produced by a fistula.
- *Prerequisite*:
  - Tachycardia (which has been produced as a central effect by the fistula)
  - Proximal artery of the fistula should be accessible
- *Procedure*:
  - Record BP and pulse rate
  - Apply pressure on the proximal artery of the fistula
  - Record BP and pulse rate again
- *Intended observations*:
  - Temporary increase in BP
  - Decrease in pulse rate by more than 4/min
- *Inference*: Obtaining intended observations ⇒ Branham's sign +ve.

Examine all pulses

- In **abdominal examination**, specifically look for a AAA.

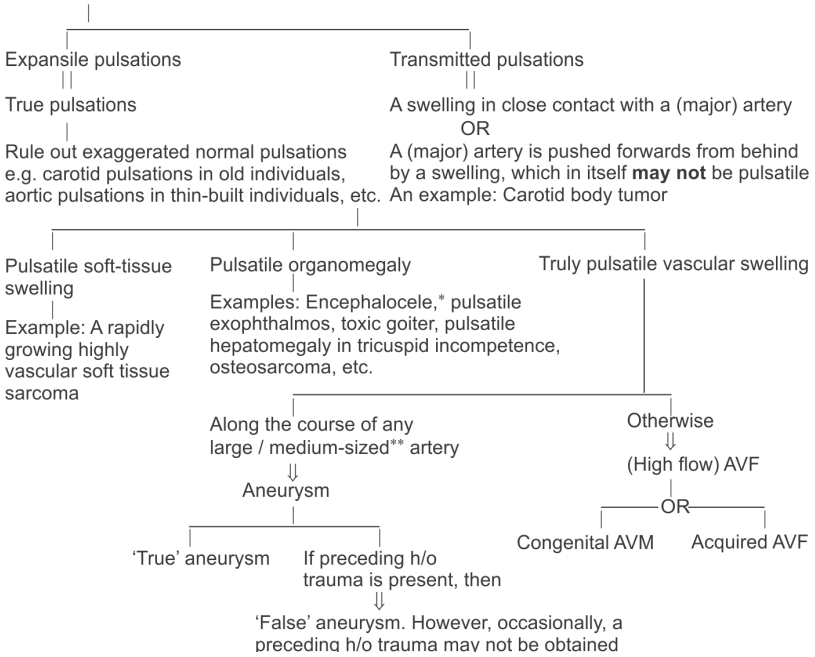
ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. – A truly pulsatile vascular swelling may or may not be compressible
   – And a compressible swelling may or may not be pulsatile.
2. Giant hypertrophy of one limb can occur without an underlying AVM.

Free ebooks ==> www.Ebook777.com

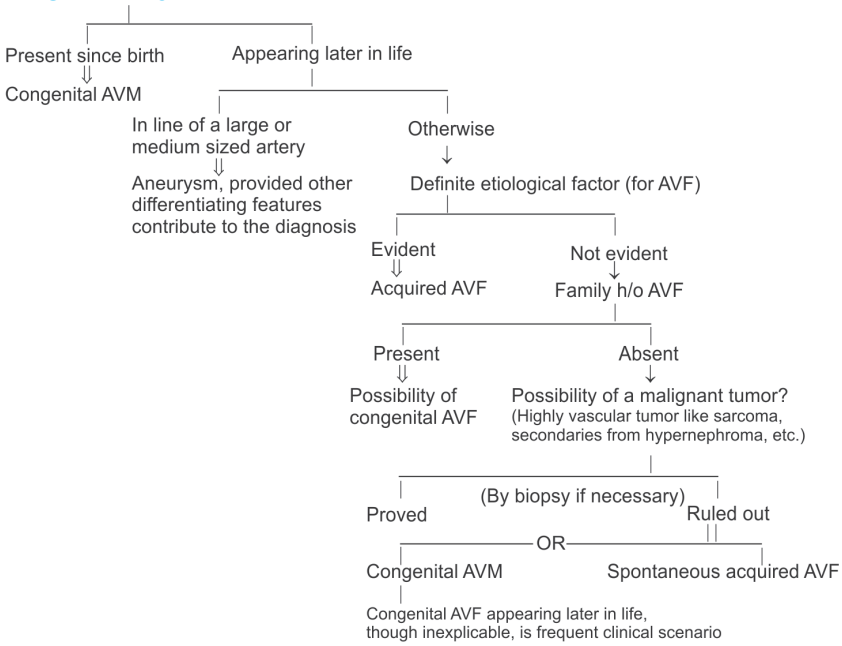
A PULSATILE SWELLING



* Encephalocele/hydroencephalocele is virtually an example of congenital herniation rather than organomegaly.

** Aorta and its major branches are called 'large' arteries.
Arteries with distributive function—'muscular' arteries—these are 'medium' and 'small' arteries. Objective distinction between medium and small arteries is not very clear-cut.

A COMPRESSIBLE* SWELLING



*** Partially compressible:**
- An aneurysm that may have been filled with a thrombus
- Cavernous lymphangioma.

www.Ebook777.com

AVF

Low-flow OR high-flow?

Following are the *clinical manifestations suggesting a high-flow fistula*.

- H/o spontaneous bleeding, or bleeding resulting from a relatively minor injury
- Gigantism of digit / limb
- Raised and warm lesion
- Palpable thrill. Systolic murmur on auscultation
- Erosion of underlying bony structures (due to pulsatile flow)
- Cardiac failure.

Loco-regional sequelae/complications?

- Arterial aneurysm
- Varicose veins. CVI
- Erosion of underlying bony structures
- Gigantism of digit / limb
- Bleeding.

ANEURYSM

Complications?

- Pressure effects on adjacent anatomical structures
- Bleeding
- Acute limb ischemia
- Chronic arterial insufficiency.

DIFFERENTIAL DIAGNOSIS

'True' aneurysm

Differentiating points

- In line of a large or medium-sized artery
- Fusiform OR globular (saccular)
- Expansile pulsations
- Thrill ±
- Easily compressible if the artery proximal to the aneurysm is compressed (occluded by pressure), but otherwise only slightly compressible requiring great pressure.
  After pressure is released, the swelling refills rapidly throb-by-throb synchronous with heart beat
- Distal pulse weak and delayed as compared to opposite (normal) side
- Systolic murmur overlying the swelling.

Additional clinical features which may be present by virtue of the following:

- Involvement of other arteries
- Complications
  - Compression of neighboring structures
  - Acute or chronic ischemia in the region distal to the location of the aneurysm.

Note: The absence of compressibility, thrill and murmur ⇒ the aneurysm must have been filled with a thrombus.

False aneurysm

Differentiating points

- H/o injury **involving an artery alone.** Hence, situated in line of an artery
- Diffuse (Ill-defined)
- Usually globular
- Expansile pulsations

Free ebooks ==> www.Ebook777.com

– Thrill ±
– Distal pulse weak and delayed as compared to opposite (normal) side
– Systolic murmur.

Possible complications of large (> 2 cm in diameter) false aneurysms (unlikely to thrombose spontaneously)

- Bleeding
- Breakdown of the overlying skin (ulceration, which in turn may get secondarily infected)
- Compression of neighboring structures
- Distal embolization.

Acquired AVF

Differentiating points

– H/o injury
  - Penetrating injuries from needle / knife / bullet, etc.
  - Blunt injury causing fracture or joint dislocation.
– H/s/o etiological factor other than injury ±
– Vibrating sensation or a buzzing sound over the swelling
– Temperature
  - Overlying the swelling—raised
  - Distal to the swelling—reduced
– Expansile pulsations
– Thrill
– Machinery murmur.

Supporting points

– Gigantism of digit / limb
– Erosion of underlying bony structures (due to pulsatile flow)
– Distal ischemia
– Cardiac failure
– Branham's sign +ve.

Additional clinical features, which may be present

– Distended or varicose veins
– Chronic venous HT—skin changes characteristic of post-thrombotic syndrome—in long-standing fistula
– **> 50% of traumatic fistulae have an associated false aneurysm.**

Causes of acquired AVF

1. Trauma is the most common cause.
   - Penetrating injuries from needles, knives, bullets, catheters, etc.
   - Blunt injuries causing fracture or joint dislocation. Examples: Carotid-cavernous fistula from facial injury, hypogastric artery fistula from pelvic fracture, etc.
   - Examples of iatrogenic injuries: Arterial access (commonly femoral), lumbar discectomy, mass ligation of major arteries and veins (such as during splenectomy, nephrectomy, etc.), needle biopsy of vascular organs like kidney
     H/o injury may be forgotten in long-standing chronic fistulae.
   - Erosion of intravascular appliances such as IVC filters.
2. As a complication of an aneurysm: Example: Rupture of an aortic aneurysm into IVC.
3. Sepsis of a false aneurysm.
4. Spontaneous.
5. Therapeutic. Examples: Hemodialysis, surgical treatment of portal HT, to maintain graft patency, to enhance limb growth.

AVM (Arteriovenous malformation)
= a type of congenital AVF.

www.Ebook777.com

The most common presenting symptoms in young children
- Birth mark (Discoloration)
- Obvious swelling
- Disfigurement
- Limb enlargement.

The most common presenting symptoms in adults
- Obvious swelling
- Varicose veins
- Limb length discrepancies
- Aching and heavy sensation in the affected limb (and in the pelvis in case of pelvic lesions).

Other symptoms
- Pain—can be severe—often the reason for the request for treatment.
  Occasionally, pain is due to pressure on the local structures like brachial plexus
- Ulceration (This is a late presentation. Cause of ulcer—venous HT)
- Bleeding (This also is a late presentation)
- Asymmetric limb length.

Differentiating physical findings
- Skin discoloration
- Firm **spongy** consistency.

Supporting points
- Enlarged draining veins—may or may not be pulsatile
- Varicose veins suggest long-standing lesion
- Expansile pulsations ±
- Thrill ±
- Compressibility ±. Predominantly venous lesions are compressible
- Continuous or systolic murmur on auscultation. (Systolic murmur is one of the criteria of high flow fistula).

Notes
- Venous HT of AVM is never totally relieved by elevation of the part, and healing may be further impaired by a degree of superimposed ischemia.
- Bleeding, fortunately rare, can be severe if there is a major arterial component to AVM
- Bleeding can also occur from low-flow lesions but this is virtually never life-threatening and is easily controlled by elevation or by direct pressure
- Many lesions are silent on auscultation. In some cases a fistula can be detected using handheld Doppler
- *An AVM situated in the midline of lumbar level of the back* may indicate spina bifida occulta.

Port-wine stain

= a low-flow capillary malformation
- Presents at or soon after birth
- Flat area of discoloration. With increasing age it may become macular, giving a cobblestone appearance
- Initially pink / scarlet, but with age it deepens to purple
- Grows parallel with the patient's growth
- Port wine stain on the face + focal seizures ⇒ associated abnormalities of the meningeal and choroid plexus vasculature
- Port wine stain on the trunk and limbs may be associated with venous or lymphatic abnormalities.

Capillary lymphatic malformations
- Present at birth
- Raised well-defined areas
- Red-colored
- With age, become hyperkeratotic → a warty appearance.

Free ebooks ==> www.Ebook777.com

Angiokeratoma

An abnormality of microvasculature

- Papules up to 1 cm in diameter
- Dark red colored
- Sites—hands and feet or on abdomen and thigh
- Patient may also develop renal failure and hypertensive cardiovascular disease.

Spider nevi (= Telangiectasia)

= Dilated vessel with radiating fine blood vessels resembling legs of a spider.

- Common sites—face, chest and arms
- Blanches on pressure (with a pinhead) over the feeding vessel
- Found in pregnancy and liver cirrhosis
- Hereditary hemorrhagic spider nevi = Multiple spider nevi + widespread bleeding and late development of arteriovenous pulmonary malformations.

Venous malformation

- Present at any site. However, common sites are face (including lips) and brain
- Presence of pain suggests phlebothrombosis
- Color:
  - Often dark blue when superficial or of the one having a superficial component
  - Dark red when combined with a capillary malformation
- Non-pulsatile
- Consistency:
  - Soft
  - Induration suggets phlebothrombosis
- Compressible
- Adjacent bone—distorted or hypertrophied.

Minor injury may → rapid enlargement of the vessels or the formation of AV fistula.

Congenital arteriovenous fistula

High-flow congenital AVFs are rare. They tend to occur on the head and neck and limbs and may arise from an innocent low-flow malformation. Injury or the hormonal changes of puberty and pregnancy may precipitate the formation of arteriovenous communications in such lesions.

Differentiating points

- Pre-existence of a low-flow (congenital) AVM
- Development of the following clinical features following injury or during puberty/pregnancy
- Vibrating sensation or a buzzing sound over the swelling
- Raised lesion
- Temperature
  - Overlying the swelling—raised
  - Distal to the swelling—reduced
- Expansile pulsations
- Thrill
- Machinery murmur.

Supporting points

- Gigantism of digit / limb
- Erosion of underlying bony structures (due to pulsatile flow)
- Distal ischemia
- Cardiac failure
- Branham's sign +ve.

The overlying skin may become ischemic.

The lesion may bleed either spontaneously or as a result of relatively minor trauma.

www.Ebook777.com

Cirsoid aneurysm = Congenital AVF specifically occurring on the scalp / hand.

Additional specific features

– Diffuse, red, hot
– Tenderness ±
– Usually affects children / young adults
– Intracranial extension or connection may be present in case of an aneurysm on the scalp.

Hemangioma

Differentiating clinical features

– Absent at birth. Appear 4 weeks after birth
– Tend to grow rapidly for the first year of life. During this time their growth is disproportionately high to the child's growth
– They then enter an involutional phase during which spontaneous regression occurs
– In 90% of cases, hemangiomas disappear by 10 years of age.

Superficial hemangioma (Strawberry nevus)

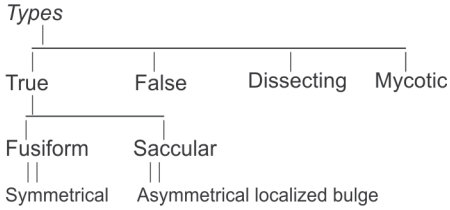
– Raised
– Bright red. The color deepens in the first year of life
– Irregular bosselated surface
– Consistency—firm and rubbery
– NOT compressible
– Begins to involute generally in the second year of life
– Changes with involution: Develops a pitted appearance. Wrinkling develops
  - Deepening of crimson color to purple with grayish or white flecks and patches appearing in the lesion.
  - Consistency becomes softer.

Deep hemangioma: May appear blue through the overlying skin.

– Commonest site—head and neck followed by trunk followed by extremities
– Ulceration and bleeding may occur during the proliferative phase
– In the case of multiple cutaneous, GIT, hepatic or pulmonary hemangiomas, infants of 2–8 weeks may present with anemia and congestive heart failure.

A note on aneurysm

Aneurysm is *defined as* an abnormal dilatation of a blood vessel (the term 'blood vessel' includes vein).



- **'True' aneurysm** = the wall of the aneurysm comprises of all layers of the normal arterial wall
- **'False' aneurysm** results from a defect in the arterial wall due to trauma, or arises at the site of an anastomosis (i.e. arterial anastomosis). The wall of a false aneurysm comprises of adventitia or fibrous tissue
- **'Dissecting' aneurysms** arise from a defect in the intima with formation of a false channel tracking in the media which may re-enter the true lumen more distally
- **'Mycotic' aneurysms** are caused by embolism of infective material which lodges in an artery. The term is also widely used to describe any infection associated with the presence of an aneurysm
- The stage at which the increase in diameter of a blood vessel becomes abnormal to qualify for aneurysm is debatable

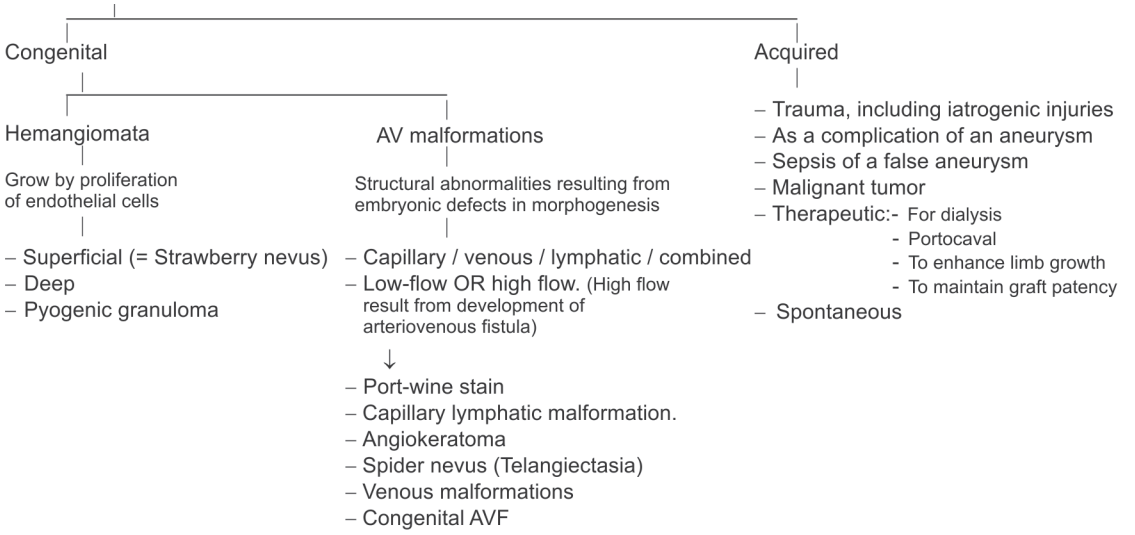
Free ebooks ==> www.Ebook777.com

- In practice, an aorta > 30 mm in diameter or double the diameter of the adjacent aorta is considered to be aneuresmal. Similar argument ('double the diameter of the adjacent segment of the artery') can be applied to aneurysms at other sites
- Risk factors for the development of aneurysm: Family history, smoking, HT
- Femoral artery is the commonest site for false aneurysm. True aneurysm in femoral artery occurs if at all in conjunction with true aneurysm at multiple other sites
- Popliteal artery is the commonest site for true aneurysm apart from the aorta. There is an association with AAA. Bilateral in 50% of cases. As with AAA the diameter at which the popliteal artery is considered aneurysmal is debatable but a popliteal artery > 2 cm is accepted to be an aneurysm.

A note on AV fistula

- AV fistula is *defined as* an abnormal communication between the arterial and venous systems.
- The congenital AVF comprises a spectrum of developmental abnormalities that may involve all components of the peripheral circulation i.e. arteries, veins, capillaries and lymphatics.

Classification of AVFs



- *Hemangiomas* demonstrate endothelial hyperplasia. NOT visible at birth. Present a few weeks after birth and have a far greater likelihood of spontaneous regression. Grow in the early life, and then tend to regress and in 90% of cases involute naturally by the age of 5–10 years
- *Malformations* have normal endothelial turnover. They are structural abnormalities. They are invariably present at birth. Usually grow with age. In women, they may enlarge dramatically during puberty or pregnancy. Some congenital AV malformations may present for the first time later in life—an inexplicable, but frequent clinical scenario
- *The severity of pathophysiological effects of AVF depends upon* duration, location, and the magnitude of the leak. Those closer to the heart are more likely to produce central effect, whereas those located in the extremities are more likely to cause ischemia
- Because of their complexity and low-flow, congenital AV malformations produce more local effects than the high-flow acquired AV fistulae
- The age at the time of first clinical presentation of an AVM is inversely related to the severity of the lesion.

Loco-regional effects of AVF:

– Development of arterial aneurysms
– Development of varicose veins
– Gigantism of digit / limb

www.Ebook777.com

– Erosion of underlying bony structures (due to pulsatile flow)
– Distal ischemia.

Systemic (central) effect of AVF: Cardiac failure.

The clinical presentation of AVF is extremely variable and depends upon the anatomical location, tissues involved, contiguous structure/s and the amount of blood shunted from the arterial to the venous circulation. Fistulae within the brain, chest or abdomen are only detected when complications arise.







Figs 18-1A to C Examples of congenital AVM



Fig 18-2 Port-wine stain

Free ebooks ==> www.Ebook777.com

19 CHRONIC CIRCUMSCRIBED SWELLING IN THE REGION OF THE HEAD AND FACE

(Excluding swelling of the jaw and oral cavity)

CHRONIC CIRCUMSCRIBED SWELLING IN THE REGION OF THE HEAD (Including SCALP)

The following two factors may be considered for arriving at the diagnosis:

1. Whether congenital or acquired
   Examples of congenital/developmental swellings:
   - Meningocele / encephalocele / hydroencephalocele
   - AV malformation
   - Dermoid cyst.
2. Whether bony or soft tissue

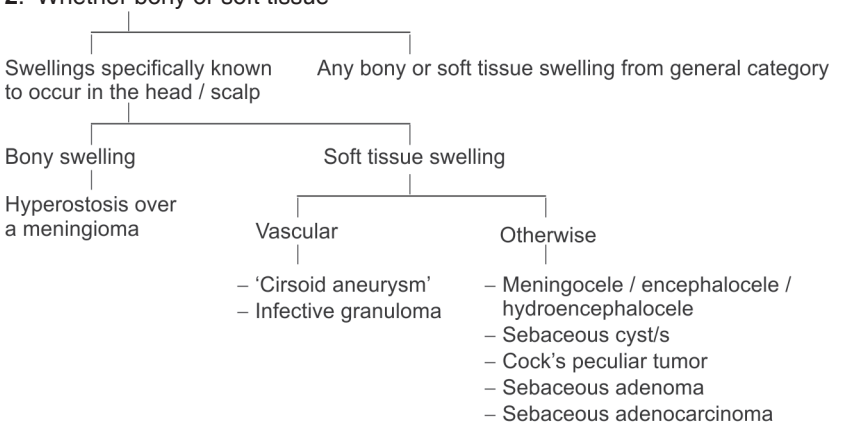
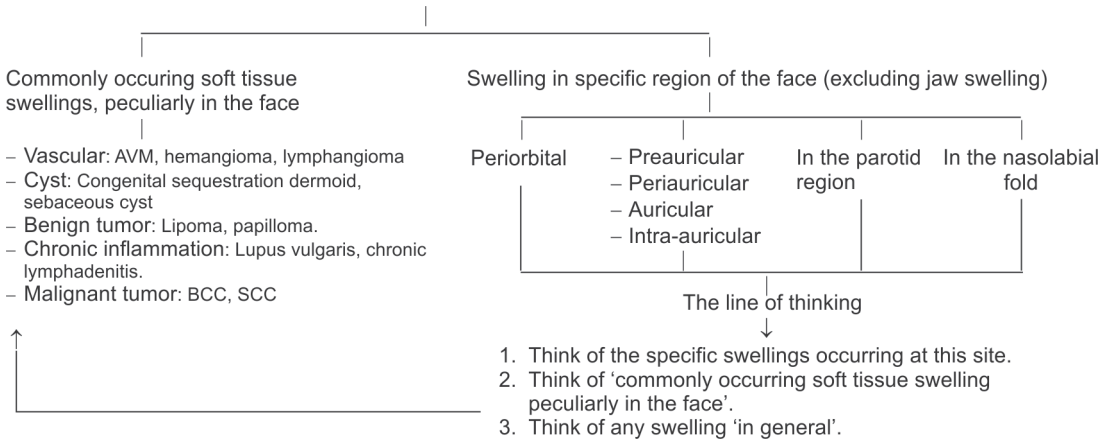




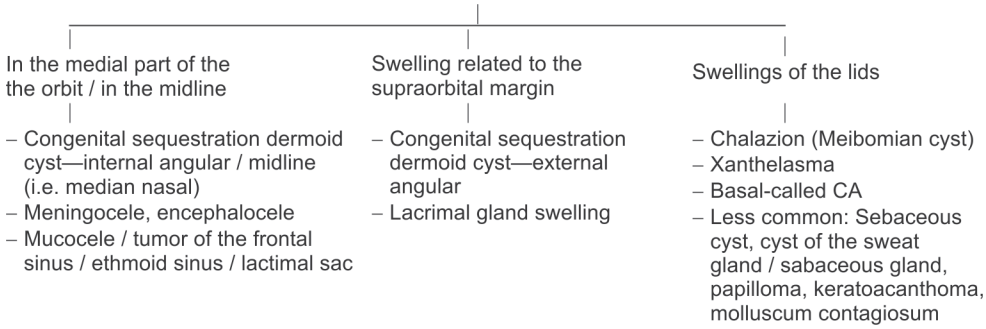
Fig 19-1 Occipital encephalocele + cervical meningocele

www.Ebook777.com

CHRONIC SWELLING IN THE REGION OF THE FACE



CHRONIC PERIORBITAL SWELLING



Congenital sequestration dermoid

Basically a cystic swelling.

Differentiating point: Situated at the specific site viz. at the root of the nose, or external or internal angle of the eye.

Additional differentiating points specifically in favor of a subperiosteal dermoid

– Relatively immobile
– Firm (or almost hard) in consistency
– Saucer-shaped depression of the underlying bone (i.e. the outer table. The outer table is attenuated or almost absent).

Additional differentiating points specifically in favor of a subcutaneous dermoid

– Relatively more freely mobile
– Softer in consistency
– Fluctuation ±
– Moulding ±
– Depression of the underlying bone may be very shallow.

Notes

– The swelling may be present practically since birth
– A dermoid cyst may lie in one of the 3 planes; in the descending order of frequency: Subperiosteal (the commonest), subcutaneous, and a cyst with an intraorbital / intracranial extension i.e. a dumb-bell cyst (rare)

Free ebooks ==> www.Ebook777.com

– A dumb-bell cyst may not or may cause symptoms / signs s/o intraorbital / intracranial SOL. In c/o intracranial extension, impulse-on-coughing may be present (only) if communication between the superficial and deep elements is wide

Otherwise, the only way to know about it is imaging investigation. Plain X-ray may show bone defect with a sclerotic margin. A CT/MRI may have to be requested.

Meningocele / encephalocele

– Tense swelling
– (Arterial) Pulsations from subarachnoid space ±
– Expansile impulse-on-coughing ±. In older children however, impulse-on-coughing may be absent especially in a small-sized meningocele (as growth of the skull may occlude the neck of the sac)
– Translucent
– A defect in the frontal bones may be palpable at the base of the swelling.

Mucocele of the frontal sinus

– H/o years of chronic frontal sinusitis
– Defect in the frontal bone palpable

Mucocele of the lacrimal sac

– H/o epiphora
– H/o one or more attacks of dacryocystitis ±
– A fluctuant swelling that bulges out just below the medial canthus
– This condition occurs from obstruction of the lacrimal duct beyond the sac
– Infection of the mucocele → a painful tense swelling—acute dacryocystitis
– If untreated, it may give rise to a fistula.

Lacrimal gland swelling (e.g. a tumor)

– Location—inferior to the eyebrow (raising the lateral part of the eyebrow), within the limit of the bony orbit. Differentiating feature of external angular dermoid: The dermoid often underlies the lateral portion of eyebrow, and lies outside the limit of the bony orbit
– Eventually leads to impairment of ocular movements and displacement of the globe forwards, downwards and inwards
– Pathologically the lacrimal gland tumors resemble parotid tumors.

Chalazion

A subacute, painless, beady nodule occurring in an otherwise normal eyelid. May get complicated by acute inflammation.

Xanthelasma

– Slightly raised, well-defined, yellowish plaques in the skin, situated along the nasal portions of one or both eyelids
– May accompany lipid disorders, or occur independently.



Fig 19-2 Median nasal dermoid

CHRONIC PREAURICULAR SWELLING

A skin tag = a floppy tag with a narrow stalk without any cartilage within.

Accessory pinna = a wide-based skin tag with cartilage within.
It is very important to identify a palpable cartilage as an accessory pinna needs a more formal surgical excision.

www.Ebook777.com



Fig 19-3 Turned out to be ethmoidal sinus tumor on imaging investigation



Fig 19-4 External angular dermoid cyst



Fig 19-5 Lacrimal gland swelling (tumor)



Fig 19-6 Xanthelasma

CHRONIC PERIAURICULAR SWELLING

Periauricular cysts
They result from abnormalities of 1st and 2nd branchial arches.
Apparently simple cysts (and sinuses) may have extensive and deep branching ramifications which pass in close proximity to the facial nerve.

Postauricular dermoid cyst results from inclusion of epithelium as 2 adjacent auricular tubercles merge.
A *DD* of postauricular dermoid cyst: Sebaceous cyst.

Postauricular 'pseudodermoid'
The postauricular lymph node is often enlarged in the 1st 2 years of life and may be situated anywhere in a fair range of territory from the tip of the mastoid process to the superior nuchal line. Enlargement is often bilateral, but when one is particularly large, it can be misdiagnosed as a dermoid cyst.

(Postauricular) **Cutaneous cyst**
– Mobile over underlying tissues, but attached to the epidermis
– A blackhead may be visible on the surface.

Histologically, it may be a sebaceous cyst or a pilar cyst.

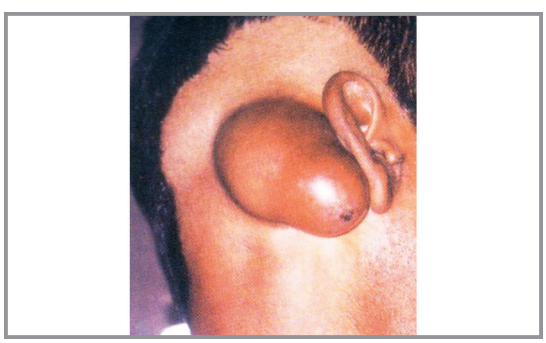


Fig 19-7 Postauricular dermoid cyst

Free ebooks ==> www.Ebook777.com

CHRONIC AURICULAR SWELLING

Tophi

– Hard nodules in the helix or antihelix and may discharge their chalky white crystals through the skin
– Other sites where tophi may occur: Near the joints, as in the hands, feet and other areas.

A tophus is a deposit of uric acid crystals characteristic of chronic tophaceous gout. Tophi usually develop only after years of sustained blood levels of uric acid.

Rheumatoid nodules on helix / antihelix in a patient suffering from chronic (rheumatoid) arthritis.
Other sites: Hands, along the surface of the ulna distal to the elbow, knees, heels.
May ulcerate due to repeated minor trauma.
Rheumatoid nodules may antedate arthritis.
DD: Tophi.

Keloid

On the ear, it usually develops on the earlobe that was pierced for earrings.
It is troublesome especially because of cosmetic concern.

Lepromatous leprosy

→ multiple papules and nodules on the auricle.

Chondrodermatitis helicis

– Chronic inflammatory lesion starts as a painful, tender papule on the helix / antihelix
– Typically single
– Usually older men are affected.

Further course: Ulceration and crusting. Reddening may occur.
DD: CA. A biopsy is needed for differentiation.

INTRA-AURICULAR SWELLING

Chronic nodular nontender swelling/s covered by normal skin deep in the ear canal suggest exostosis (/exostoses).

CHRONIC SWELLING IN THE PAROTID REGION

Causes of swelling in the parotid region

Swelling arising from a tissue other than the parotid gland

– Temporal artery aneurysm
– Winged mandible
– Bony swelling of the mandible
– Mastoiditis
– Hypertrophy/myxoma of masseter muscle
– BCC
– Preauricular lymphadenopathy
– Lymphangioma
– Facial vein thrombosis
– Facial nerve neuroma

Swelling arising from the parotid gland

– Bilateral cystic enlargement—may be a presenting feature of HIV infection
– Cyst/s—rare
– Benign tumor
– Malignant tumor
– Tumor of variable malignant potential
– Rare tumor: Basal cell adenoma, lipoma
– Chronic or recurrent acute parotitis
– Sialectasis (obstructive, allergic idiopathic, secondary to medical disorders)
– Sjogren's syndrome
– Mikulicz disease
– Parotid lymphadenopathy (clinically difficult to differentiate form a protid gland swelling proper)
– Drug-induced: For example. antithyroid drugs. Many other drugs → dryness of mouth → ascending infection of the glands
– Metabolic causes: Overweight / obesity, DM, acromegaly, patients sticking ot bizarre diets

www.Ebook777.com

HISTORY

- **Age**?
- **Side affected**: Single OR both?
  If bilateral
  ↓
  - Have you been strictly consuming a 'rather unusual' diet? If so, how long?
  - High-estrogen containing contraceptive pill?
  - Hypersensitivity?
  - Alcoholic?
  - Have you been on any (long-term) medication? For example, antithyroid drugs, phenylbutazone, isoprenaline, dextropropoxyphene, etc.
  - Have you been suffering from any systemic medical disorder? For example, DM, hepatic cirrhosis, myxoedema, Cushing's disease, gout, TB, actinomycosis, sarcoidosis
  - [(HIV +ve?)]
- **Progress**?
  - Long periods of remissions and relapses are a feature of obstructive sialectasis most commonly due to calculous disease
  - Slow progress with long quiescent period, and short periods of rapid growth ⇒ pleomorphic adenoma (mixed tumor)
  - Rapid progress over weeks ⇒ malignant tumor. *However, a slow progress does not necessarily rule out malignancy*
  - A dramatic sudden recent increase in size of the swelling which has been practically stationary ⇒ bleeding inside a cyst or a cystic tumor.
- **Pain**?
  - Constant OR intermittent?
  - When intermittent:
    - Does pain come on soon on eating or even with a thought of (a tasty) food?
    - Is it associated with swelling of the gland? How long does it persist?
    - Duration of each attack of pain?
    - Does the pain get relieved rapidly following sudden spurt of saliva or foul-tasting fluid into the mouth?
    - Long periods of remissions and relapses?
  - In between attacks, does the swelling subside or persist to be continuous (pain subsiding)?

INTERPRETATION

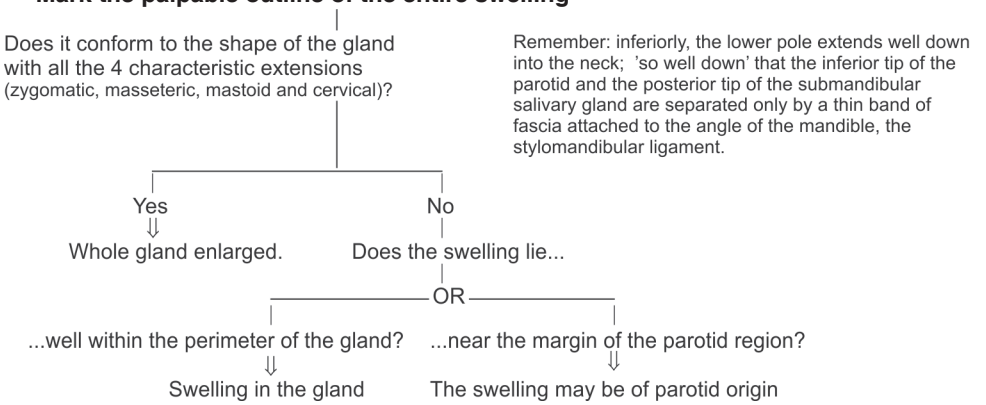
- Painful conditions: adenoid cystic carcinoma, inflammatory conditions, obstructive sialectasis most commonly due to calculous disease
- Causes of constant pain—inflammation, malignancy. Pain due to malignant tumor may radiate to ear and over the temporal region
- Causes of intermittent (recurrent) pain: Recurrent subacute / chronic parotitis, obstructive sialectasis, Sjögren's syndrome
- Painful enlargement of the affected gland soon after eating, duration of pain during each attack being a few hours. Swelling may persist for less than an hour to several days. Long periods of remissions and relapses ⇒ obstructive sialectasis most commonly due to calculous disease.

- **Injury from a denture** at the site of the opening of the duct?
- **Sharp tooth causing irritation** at the site of the opening of the duct?
- **(Repeated) cheek bite** involving the site of the opening of the duct?
- **Dryness of mouth, eyes**?
- **Rheumatic manifestations**?
- **Anesthesia** of skin / mucous membrane?
- **Symptoms of facial nerve paralysis**?
- **Regional and generalized lymphadenopathy**?

Free ebooks ==> www.Ebook777.com

LOCO-REGIONAL EXAMINATION

- **Exact site and extent**:
  - Relation of the swelling to the lobule of the ear?
  - Is the normal furrow behind the angle of the mandible—obliterated?
- **Mark the palpable outline of the entire swelling**



- **'Diffuse'** i.e. involving the whole gland **OR 'localized'**?
  Localized swelling goes more in favor of a tumor. Exception—pleomorphic adenoma (mixed tumor) which usually affects the whole gland
- **Exact site of the fistula**, if present?:
  - Overlying the masseter ⇒ fistula of the gland
  - Anterior to the masseter ⇒ fistula of the duct
- **Mobility**?: Restricted mobility, or absence of mobility (i.e. fixed) ⇒ malignant tumor.
  The muscles in relation: **Masseter** and partly SCM.

Surface of the swelling is of little help in arriving at the diagnosis.

- **Consistency**?:
  - Hard ⇒ malignant tumor, mucoepidermoid carcinoma
  - Soft ⇒ papillary cystadenoma lymphomatosum, acinic cell tumor (variable biological behavior)
  - Consistency of a pleomorphic adenoma (mixed tumor) is firm or hard
- **Fluctuation**?: Papillary cystadenoma lymphomatosum is soft, often fluctuant
- **Facial nerve paresis / paralysis**? ⇒ malignant tumor.
  Signs of facial nerve paralysis: Basically, lower motor neuron type.
  - Forehead does not wrinkle
  - Eye:
    - Palpabral fissure becomes broad
    - Tear does not fall because it fails to enter the lacrimal punctum as the lower lid is not in contact with the conjunctiva
    - Lids do not close, and give rise to a blind man's appearance
  - Nasolabial fold is vanished
  - Ala of the nose does not move
  - Mouth:
    - Lips do not part, pout or purse
    - Angle will not rise
    - Teeth will not show
    - Cheeks will not puff
    - Food accumulates in the mouth
- **Difficulty in opening the mouth**? ⇒ invasion of jaw or muscles of mastication by malignant tumor

www.Ebook777.com

- **Intraoral examination**
  - Tonsil and palate—Is one or both of them pushed medially? ⇒ swelling arising from the deep portion of the gland. (Swelling arising from the whole gland → dumb-bell shaped swelling.)
  - Orifice of the parotid duct (opposite the crown of the second molar tooth)
    - Any stone visible?
    - Does it look gaping with surrounding edema and inflammation? ⇒ Recently, a stone might have been passed spontaneously
  - On pressing the gland gently, does pus / blood extrude?
    - Pus ⇒ suppurative parotitis
    - Blood ⇒ malignant tumor
  - Does the oral cavity appear dry?
  - Do the lips, tongue and palate appear to stick together?
- **Examine all other salivary glands and lacrimal glands**
- **Cervical lymphadenopathy**?

  Adenocarcinoma and squamous-cell carcinoma spread early to cervical lymph nodes.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. The normal gland is impalpable, except occasionally in thin-built individuals.
2. The gland affected by inflammation or by mechanical obstruction becomes 'hardened' and therefore palpable even if it is not greatly enlarged.
3. Even when the gland enlarges considerably, its characteristic shape (with all the 4 extensions) remains unchanged.
4. Be a little cautious about accepting a h/o recurrent parotitis in the absence of concrete evidence. Swelling and discomfort in parotid region/s and the related salivary symptoms of dryness of mouth or excessive salivation are notorious as favorite complaints of neurotic subjects.
5. Benign tumors never cause facial nerve paralysis.
6. The pathological nature of a parotid tumor cannot be ascertained on clinical examination in large number of patients. In other words, most swellings in the parotid region cannot be diagnosed clinically with any accuracy.
7. Adenoid cystic carcinoma is difficult to differentiate clinically from the benign tumors and the tumors of variable biological behavior.
8. • 75% of salivary gland tumors occur in parotid
   • 75% of them are benign
   • 75% of benign are pleomorphic adenoma

   A pleomorphic adenoma may be as soft as a lipoma or as hard as bone. It may grow as rapidly, and be as painful as the most malignant carcinoma, or it may remain indolent and otherwise asymptomatic for 50 years. It is usually unilateral but bilateral tumors can occur.
9. The commonest presenting feature of adenoid cystic carcinoma is pain and the tumor may be present for **some years** before diagnosis. It is often sometime before mass becomes palpable or evident and the patient may spend some years visiting different specialists with the complaint of facial pain.
10. Every swelling in the parotid region should be considered malignant until proved otherwise. This statement applies even if the swelling has been present for years without altering in size, even if it is otherwise asymptomatic, even if the patient says s/he thinks it is getting smaller, and even if the swelling on physical examination appears to be lying in the skin overlying the parotid region rather than in the parotid gland itself.
11. Apart from the common condition known as mumps, parotid swellings are not frequently encountered by most clinicians.
12. The vast majority of cases can be treated logically, though not necessarily successfully.

Free ebooks ==> www.Ebook777.com

STEPWISE ANALYSIS OF DATA

Is the parotid swelling **apparent or true**?

'Apparent' parotid swelling

A bump in the parotid region, which gives a primary impression of being a 'true' parotid swlling

- Swelling arising in the skin/subcutaneous tissue overlying the parotid gland
- Winged mandible
- Bony swelling of the mandible
- Mastoiditis
- Hypertrophy / tumor of masseter muscle
- Branchial cyst

'True' parotid swelling

Appearing below, in front of and behind the lobule of the ear often obliterating the normal furrow behind the ramus of the mandible

Clinical points which help in taking a preliminary judgment :

- Age?
  Swellings occurring particularly during childhood: Recurrent subacute parotitis of childhood, mucoepidermoid carcinoma, adenocarcinoma
- Unilateral OR bilateral?
- Diffuse enlargement OR circumscribed swelling?

↓

Unilateral true parotid swelling

- Diffuse organomegaly i.e. enlargement of the whole gland
- A swelling in the gland i.e. a circumscribed swelling lying within the perimeter of the parotid gland
- A swelling lying near the margin of the parotid region
  ⇓
  The swelling may be of parotid origin

Diffuse organomegaly

Symptomatic ⇓

- Recurrent acute or chronic parotitis due to ductal obstruction
- Sialectasis—affecting only one side, or symptomatic only on one side

Otherwise asymptomatic ⇓

Could be pleomorphic adenoma (mixed tumor)

A circumscribed swelling in the gland

Inflammatory

- A tumor undergoing inflammatory degeneration—more likely with papillary cystadenoma lymphomatosum rather than with other tumors
- An abscess with a stone lying within it
- Inflammation (lymphadenitis) affecting the lymph node/s lying in the gland substance (deep to the fascia of the gland)

Noninflammatory

Arising from the gland itself

Tumor

From nonglandular tissue

- AVF, aneurysm
- Lymphadenopathy
- Limpoma
- Fibroma, etc.

Clinically, it is impossible to differentiate between the 2 groups

www.Ebook777.com

A swelling lying near the margin of the parotid region ⇒ the swelling may be of parotid origin

- The swelling lying immediately in front of the ear
  ⇓
  Possibility of parotid tumor—obvious
- The swelling overlying the mastoid process OR lying rather 'low' in the neck behind the submandibular gland
  ⇓
  The possibility of the swelling being a parotid tumor—not as strong

Bilateral true parotid swellings

- Diffuse organomegaly i.e. enlargement of the whole gland
  ⇓
  – Patients sticking to bizarre diets
  – High estrogen-containing oc pill
  – Hypersensitivity
  – Alcoholism
  – Drug-induced
  – Systemic medical illness
  – AIDS → bilateral cystic enlargement
  – Idiopathic
- Circumscribed swelling in the glands
  ⇓
  Following possibilities
  – Papillary cystadenoma lymphomatosum—bilateral, but rarely synchronous
  – Acinic cell carcinoma
- Commonly unilaterally occuring disorders may occur on both sides

Causes of cystic swelling
(The diagnosis of cystic swelling is rather retrospective on investigation.)
Papillary cystadenoma lymphomatosum, cystic pleomorphic adenoma, branchial cyst, parasitic cyst, AIDS.

DIFFERENTIAL DIAGNOSIS of selected parotid swellings

Recurrent subacute parotitis of childhood (associated with sialectasis)
– 3 months to 10 years
– Pain and constitutional symptoms are mild
– One or more previous episodes affecting either gland, each lasting 10–14 days ±
– The first attack may have been mistaken for mumps
– Purulent fluid issues from the orifice when the duct is compressed

- *Sialogram* shows no obstruction of the duct or orifice, but a snowstorm of sacculations 2–4 mm in diameter along the radicles of the gland
- Spontaneous remission occurs by 10–15 years of age

Recurrent subacute OR chronic parotitis
– Recurrent attacks of pain and swelling accompanied by discharge of flecks of pus in the saliva
– Always secondary to obstruction of duct or autoimmune disease
– In Sjögren's syndrome, attacks of pain and swelling may occur only on one side.

Notes
– Criteria of acute recurrent (subacute) parotitis: Genuine h/o recurrent (> 1) attacks + collateral evidence such as a convincing description of swelling from a third party + positive physical findings during the attacks
– Chronic parotitis = continuous enlargement of one or both parotids (although possibly variably).

Obstructive sialectasis
– Painful enlargement of the affected gland soon after eating
– Duration of pain during each attack—a few hours
– Pain may subside rapidly following sudden spurt of saliva or foul-tasting fluid into the mouth
– Long-periods of remissions and relapses.

Free ebooks ==> www.Ebook777.com

Causes: Blockage of the main duct by stone(s) / stricture of the duct wall / edema or fibrosis of papilla (due to trauma from a denture / irritation from a sharp tooth/bite of the cheek) / pressure on the duct due to an adjacent mass / invasion of the duct by a malignant tumor.

(Nonobstructive) ***Sialectasis***: = the salivary gland analogue of bronchiectasis with areas of stenosis and dilatation of the ductal system.

– Congenital or follows infective or inflammatory changes in the duct system
– Painful enlargement of one salivary gland shortly after eating. Takes a few hours to regress and is made worse by eating again
– Attacks come in runs lasting for days or weeks with long-periods of remission.

The attacks are thought to be due to blockage of the main ducts by either epithelial debris or calculi.

Malignant tumor

– Rapid progress. However, **slow progress does not rule out possibility of malignancy**
– Early development of pain, anesthesia of skin or mucous membrane and paralysis of muscles
– Facial nerve paresis / paralysis
– Difficulty in opening the mouth.

Sjögren's syndrome

– Triad of dry eyes, dry mouth and rheumatoid arthritis
– Progressively more severe dryness of mouth → rampant uncontrollable caries of teeth
  - Lips, tongue and palate stick together
  - After the loss of natural teeth, dentures are almost unwearable
  - Intermittent, unilateral or bilateral diffuse enlargement of salivary gland or formation of localized nodules.

Mikulicz disease: Triad of symmetrical enlargement of all the salivary glands, narrowing of palpabral fissure due to enlargement of the lacrimal glands, and parchment-like dryness of the mouth.



Figs 19-8A to C Examples of parotid gland swelling. Note that even if small, the swelling appears below, in front of and behind the lobule of the ear, obliterating the normal furrow behind the ramus of the mandible.

www.Ebook777.com

CHRONIC SWELLING IN THE NASOLABIAL FOLD

Nasolabial cyst

Basically, a benign swelling in the upper lip in the region of nasal ala.

– The nasal ala is lifted up and the nasolabial fold is flattened
– Soft. Fluctuant.

A note

The cyst lies outside the maxilla. It arises from epithelial rests trapped between lateral nasal and maxillary embryonic processes or lower end of nasolacrimal duct.

Free ebooks ==> www.Ebook777.com

20 CHRONIC SWELLING OF THE JAW

GENERAL REMARKS

1. **Causes** of swelling of the jaw

Epulis i.e. arising from the gum (mucoperiosteum)	Odontome i.e. arising from the tooth germs	Osseous swelling i.e. arising from the bone	And in c/o upper jaw, additional specific swellings arising from the maxilla
– Different type of benign epulides – Alveolar SCC	– Dental cyst – Dentigerous cyst – Adamantinoma		– Empyema – CA maxilla (from columnar cells of the antrum or squamous cells of the hard palate)

2. **Causes of jaw swelling in infancy and childhood**
   - Infantile cortical hyperostosis: Hyperplasia of subperiosteal bone with soft tissue swelling in mandible, skull, clavicle, scapula, ribs and long bones
   - Chronic osteomyelitis
   - Reticuloendotheliosis
   - Ossifying fibroma
   - Cysts
   - Burkitt's sarcoma, osteosarcoma, lymphosarcoma.
3. A facial swelling of hard consistency that is otherwise asymptomatic is usually benign (although it is important to exclude early malignancy).
4. Malignant facial swellings invariably → characteristic X-ray findings: Tooth-root resorption, with poorly defined 'moth eaten' boundaries.
5. Any lesion in the oral cavity that has failed to resolve or improve within 2–3 weeks should be biopsied to exclude malignancy.
6. Oral cancers are usually locally invasive. The **extent of induration** should be included in estimating the size of the lesion.
7. Pain is a late symptom of oral cancer.

HISTORY

(Points specific / relevant to jaw swelling only are discussed below.)

In the case of UPPER JAW SWELLING

- Diplopia? Diplopia may be due to upward displacement of the eyeball
- Epiphora? Epiphora suggests blockage of the lacrimonasal duct
- Nasal blockage?
- Discharge from the nose?—purulent OR bloody?

In the case of LOWER JAW SWELLING: Past h/o a fracture?

www.Ebook777.com

LOCAL EXAMINATION

Inspection of the swelling

- Number?
- Site and extent?
- Shape: Globular OR pedunculated?
- Size?
- Skin:
  - Redness?
  - Edema?
  - Ulceration (± fungation)?
  - Sinus(es)? - Basically, number and exact site?
    - A single sinus situated a little below the jaw ⇒ nonspecific osteomyelitis
    - Multiple sinuses situated overlying the jaw (in addition to other suggestive features) ⇒ actinomycosis of the jaw.
  - Sprouting granulation tissue at the mouth of the sinus ⇒ not only osteomyelitis, but the presence of dead tissue (sequestrum) at the depth
  - Extrusion of bone chips strongly suggests osteomyelitis
  - Discharge from multiple sinuses contains yellow-colored granules of the size of a pinhead (Pus is collected in a clear test tube and observed in a powerful light) ⇒ actinomycosis
- Scar. Does it look depressed?
- Dilated veins upon the swelling?
- Pulsations? Possible causes of pulsations—highly vascular sarcoma, rarely hemangioma.

Palpation of the swelling

A jaw-swelling is also palpated bimanually with one hand on the external aspect of the face and a gloved finger in the oral cavity.

- Temperature?
- Tenderness? Inflammatory swellings are more tender than malignant swellings
- Site and extent?
- Shape? size?
- Pulsations? Possible causes of pulsations—highly vascular sarcoma, rarely hemangioma
- Mobility of the soft tissue overlying the bony swelling?
- Surface?
  - Irregularity?
  - Rounding or blunting of the sharp margin?

  The presence of one or both of these abnormalities suggest thickening of the bone.
- Consistency?:—soft / firm OR hard?
- Egg-shell crackling?

EXAMINATION OF THE UPPER JAW

- **Eye**:
  - Epiphora?
  - Proptosis?
- **Inferior orbital margin**: Palpate for sharpness and the level (Compare with the opposite side).
- **Nose**
  - Occlude the apparently normal side and ask to breath with the mouth closed. Difficulty / inability ⇒ nasal obstruction
  - Examine internal surface of the vestibule with a nasal speculum. Look for—blocking, purulent / blood-stained discharge.

Free ebooks ==> www.Ebook777.com

- **Temporal region**: Fullness?
  Fullness ⇒ extension of a growth from the posterior surface of maxilla. Basically, extension of a growth from the posterior surface of maxilla first occurs into the infratemporal region and thence to the temporal fossa.
- **Outer surface of the jaw**
  – Overlying skin?
  Evert the upper lip and cheek.
  – Inspect and palpate the external surface in the vestibule with a gloved finger.
  Tenderness on the antrum without (expanded) enlargement may suggest empyema.
- Ask the patient to open the mouth. – **Trismus**?
- **Palatine surface**: Any bulging? – if so, is it confined to one-half of the palate OR does it extend beyond the midline?
- **Teeth**:
  – Number?
  – Noneruption?
  – Caries?
  – Oblique disposition / dislocation / shedding of any tooth / teeth? ⇒ exapansion of the bone (jaw).
- **Trigeminal nerve**: Involvement (infiltration) of branches of trigeminal nerve ⇒ backward and upward extension of a growth.

EXAMINATION OF THE LOWER JAW

- **Outer surface of the lower jaw**
  – Overlying skin?
  Evert the lower lip and cheek.
  – Inspect and palpate the outer surface of the body and as much as possible of the ramus in the vestibule, with a gloved finger
- Ask the patient to open the mouth. – **Trismus**?
- Inspect and palpate the **inner surface** of the body and as much as possible of the ramus of the mandible. Body of the mandible can be readily palpated but the ramus and the coronoid and condylar processes are not so easily accessible. An attempt should however be made to palpate the 'difficult to assess' portions bimanually
- **Teeth**:
  – Number?
  – Noneruption?
  – Caries?
  – Oblique disposition/dislocation/shedding of any tooth/teeth? ⇒ exapnsion of the bone (jaw).

REGIONAL EXAMINATON

Cervical lymph nodes. Metastatic lymphadenopathy is more suggestive of the primary being carcinoma than sarcoma.

SYSTEMIC EXAMINATION

Examine other bones in the body, in addition to routine aspects of systemic examination.

ARRIVING AT THE DIAGNOSIS

POST-TRAUMATIC CONDITIONS

– H/o severe trauma usually available, **but may be forgotten**
– Static mass.

www.Ebook777.com

Examples:
1. Malunited fracture: Painless and without tenderness.
2. Dislocated bone ends.
3. Excessive callus: Usually a fusiform swelling.
4. Localized periostitis: Periosteal nodule.

CHRONIC OSTEOMYELITIS

Nonspecific / actinomycotic / tuberculous / syphilitic.

OTHER than the above-mentioned

- Arising from mucoperiosteum i.e alveolar margin of the jaw i.e. from the gum
  - Various (benign) 'epulides'
  - Polypus
  - Squamous-cell carcinoma
  - Fibrosarcoma
  - Localized hypertrophy of the gum due to irritation—rare
- Arising from odontome / bone / paranasal sinus
  - Jaw enlarged in all diameters i.e. expanded
    - Odontomes
    - Tumors including CA antrum (maxilla)
    - Empyema of the antrum
    - Granuloma in the lower jaw
    - Osteitis deformans
    - Hemangioma
  - Surface swelling
    - Ossifying fibroma
    - Localized fibrous osteoma in adults
    - Osteoma
    - Creeping diffuse osteitis
    - Melanoma of the palate

Epulis = 'Upon the gum'.
- Types of epulides: Fibrous, giant cell, granulomatous, sarcomatous
- Pyogenic granuloma, pregnancy, drugs like phenytoin and calcium channel blockers may all cause gingival swelling
- Swollen bleeding gums, although usually the result of poor oral hygiene, may occasionally be an early sign of leukemia.

Odontomes: Dental cyst, dentigerous cyst, adamantinoma.

Tumors
- Locally invasive—osteoclastoma
- Malignant—sarcoma, CA maxilla (antrum / hard palate)
- Rare—solitary cyst, osteitis fibrosa cystica, myelomatosis, xanthomatosis, ossifying fibroma which is rarely central, Burkitt's lymphoma.

DIFFERENTIAL DIAGNOSIS

Fibrous epulis
- Slow progress
- Pedunculated
- Dark red in color
- Firm consistency
- Common site—lower incisors.

Giant cell—myeloid epulis = Periosteal osteoclastoma.
- Sessile
- Lobular surface
- Purple intact mucous membrane.

Granulomatous epulis = a mass of granulations around a carious tooth.



Fig 20-1 Granulomatous epulis

Free ebooks ==> www.Ebook777.com

Sarcomatous epulis

– Rapid progress
– Softer consistency
– Bleeds easily on touch.

Alveolar carcinoma (= squamous-celled CA): Refer to chapter 51.



Fig 20-2 Advanced alveolar CA infiltrating through the skin → orocutaneous fistula

Dental cyst

Form at the site of a carious tooth or a retained root.

– Middle age
– 2–3 cm in diameter but may become larger
– Mucosa overlying the swelling is stretched but mobile
– Consistency—initially hard. However subsequently, expansion of the bone → egg-shell crackling
– Fluctuation, if the bone is completely destroyed.

X-ray finding: A cyst at the root of an erupted tooth.

Dentigerous cyst

Form at the site of an unerupted permanent tooth usually a third molar/premolar/canine.

– Age: 10–30 years
– Spherical, ovoid or irregular
– 2–3 cm in diameter, but can involve the entire ramus (when affects the mandible)
– Consistency—initially hard. However subsequently, expansion of the bone → egg-shell crackling
– Mucosa overlying the swelling is stretched but mobile
– A tooth is missing
– Can be correctly diagnosed by X-ray alone. *X-ray finding*: A cyst containing the missing unerupted tooth.

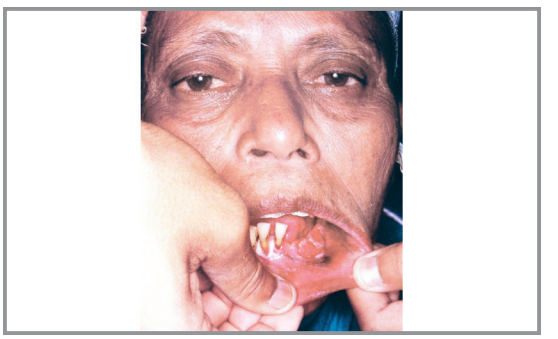


Fig 20-3 Early alveolar CA

A note

A dentigerous cyst is potentially capable of becoming an 'aggressive' swelling. It may hollow out the entire ramus, displace several tooth roots. Apart from recurrence due to incomplete surgical excision of the lining of the cyst wall, its potential complications include development of ameloblastoma, epidermoid carcinoma.

Ameloblastoma (Adamantinoma)

– Age: 20–40 years
– Common site—near the angle of the mandible
– Obvious facial disfigurement and derangement of occlusion
– May attain huge proportions
– Well-defined. Shape—irregular
– Surface—multinodular
– Consistency—initially hard. However subsequently, expansion of the bone → egg-shell crackling
– Invasion of the surrounding soft tissue ±.

X-ray findings: Unilocular or multilocular radiolucency and thus may give the appearance of a cyst.

– Differentiation from osteoclastoma is difficult even on X-ray
– Ameloblastoma is more slow-growing (compared to osteoclastoma)
– May burst into the mouth and become infected.

Osteoclastoma

– Usual sites—ramus of the mandible anteriorly, maxilla
– Rapid expansion as compared to odontomes.

www.Ebook777.com

Osteosarcoma

– Younger age
– Radiating pain
– Loosening and extrusion of teeth
– Paresthesia of the lip in case of mandibular tumor.

X-ray findings:

– Loss of the lamina dura around the tooth root
– Resorption of tooth root
– Irregular destruction simulating poorly defined 'moth-eaten' boundaries or 'sunray spicules'.

CA maxillary antrum

Initial clinical features

– Asymptomatic, before obstruction of the osteum develops
– Obstruction of the ostium and infection of the secretions, or ulceration of the tumor → clinical manifestations s/o chronic sinusitis
– Epistaxis ±.

X-ray at this juncture: Thickening of the lining. Often no bone destruction and the seriousness of the disease not appreciated.

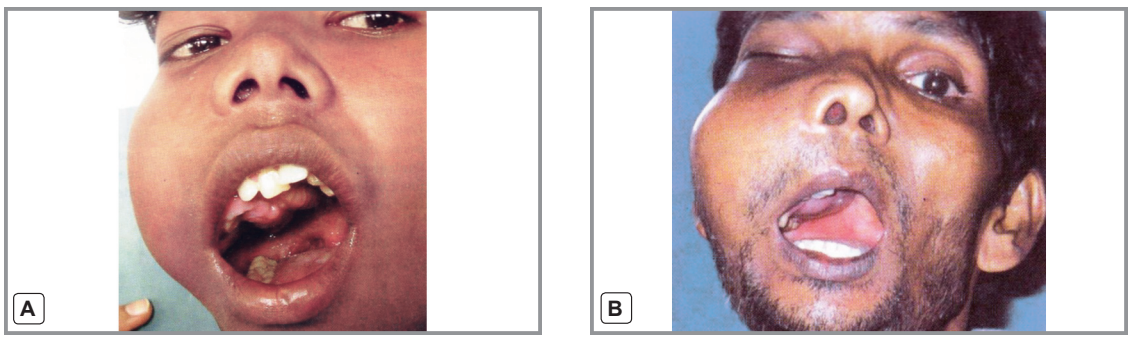
The clinical features after destruction of bony wall vary according to the direction in which the tumor spreads.

– Pain/anesthesia in the cheek and anterior teeth and gums, obvious swelling of the face, ultimate fungation and ulceration ⇒ involvement of the *anterolateral wall*
– Diplopia, proptosis ⇒ invasion of the *roof*
– Epistaxis (practically blood-stained mucoid or foul-smelling purulent discharge), epiphora, nasal obstruction, unilateral pansinusitis, a growth into the nose ⇒ invasion of the *medial wall*
– Difficulty in opening the mouth, pain / anesthesia of cheek, tongue and lower lip, bloody postnasal discharge, fullness in the temporal fossa ⇒ extension of growth from the *posterior surface of the maxilla*
– Pain in related teeth, loosening of related teeth, upsetting of the fitting of dentures, expansion of the alveolar process, bulging of the hard palate, ulceration and large sloughing antro-oral fistula ⇒ invasion of the *floor*.

In the early stage, it is extremely difficult to distinguish a sarcoma from carcinoma of the antrum.
Burrowing through one of the walls and ulceration and/or cervical metastatic lymphadenopathy suggest carcinoma.

Empyema of the maxillary antrum

– Unilateral intermittent discharge and nasal obstruction
– Tenderness
– On anterior rhinoscopy, pus will be seen to flow over the inferior turbinal
– Transillumination, posture tests and X-ray will be of great value to arrive at the diagnosis.



Figs 20-4A and B Examples of CA maxillary antrum

Free ebooks ==> www.Ebook777.com

Osteitis deformans

– Age: > 40 years
– More common in the maxilla. Deformity
– Progressive facial anesthesia, deafness and blindness secondary to nerve compression ±.

X-ray finding: 'Cotton-wool' appearance.
Avoid surgery of the jaw or tooth extraction if possible, as profuse bleeding or infection may occur.

Hemangioma

– Progressive painless swelling
– Pulsatile if the overlying bone is thinned out.

X-ray finding: A radiolucent lesion.
Death from exanguination following tooth extraction may occur.

Eosinophilic granuloma

– Age: 10–20 years
– Usually the mandible is affected
– Painful and tender swelling
– Loss or loosening of teeth
– Fever, loss of weight.

X-ray finding: Teeth appear to be 'floating in air'.
Basically, the disease ranges in severity from a solitary lytic lesion in the bone to a fatal leukemia-like disorder.

www.Ebook777.com

21 CHRONIC CIRCUMSCRIBED SWELLING IN THE NECK

POINTS IN PHYSICAL EXAMINATION specifically applicable to a swelling in the neck only are discussed below.
(Other points in routine case-taking must be asked/examined as usual.)
Position of the patient: **Sitting**.

PALPATION

Palpate the neck by standing behind the seated patient (Reason: Concavity of the hands conforms to the convexity of the neck). Slightly flex the head anteriorly + slightly tilt it on the side to be palpated (to relax the muscles and deep fascia). This position is maintained throughout the palpation of that side by the other hand resting on the head.
There is usual tendency of the patient to extend the head, which should be curbed.

Mobility

a. **Mobility on swallowing**?
This clinical test should always be done even if the swelling appears to be located far from the anticipated normal location of thyroid.
Purpose: To support the diagnosis of goiter. 'The swelling moving up during swallowing' is one of the most important signs of goiter.
Principle
During the 2nd phase of swallowing, the larynx moves up, and along with it, the superior tracheal rings. Any swelling attached to the larynx by virtue of a direct ligamentous attachment, or by virtue of being in the same visceral compartment as that of the larynx viz. the one deep to pretracheal layer of deep cervical fascia, would also move up, during swallowing.
Procedure
1. Ask the patient to act as swallowing, and observe towards any upward movement of the swelling.
2. Hold the swelling at its upper pole between fingers and thumb. Ask the patient to swallow again and try to feel for a 'tug'.

Inference: Upward movement during swallowing OR feeling of a 'tug' suggests swelling attached to the larynx.
Movement is better felt than seen.
Examples of swelling moving up with swallowing: Goiter, thyroglossal cyst, subhyoid bursa/bursitis, pretracheal enlarged lymph nodes, and a swelling of the larynx itself including laryngocele.
Fallacy
Mobility of thyroid swelling/any other swelling in the region may be restricted due to malignant infiltration or postinflammatory adhesions.

b. **Mobility on protrusion of tongue**?
Purpose: Essentially, to support the diagnosis of thyroglossal cyst.
Principle: Anatomical. A thyroglossal cyst originates from a segment of thyroglossal duct, which extends from foramen cacum of tongue to isthmus of thyroid, and on its way, is attached to hyoid bone while curving around it in a peculiar way. Protrusion of tongue is brought about by contraction of myelohyoid + genioglossus muscles. The myelohyoid muscle (which forms floor of the mouth) rises up during its contraction, along with the hyoid bone to which it is attached. Any swelling attached to hyoid bone directly or indirectly will also move up along with it, during swallowing.
Procedure
Extend the head slightly, so that anterior aspect of the neck is fully visible. Ask the patient to open her/his mouth, and instruct to maintain the same degree of opening throughout the test.

Free ebooks ==> www.Ebook777.com

– Reason: Movement of the mandible during opening and closing of the mouth may lead to some unintended movement of the swelling, making the test invalid.
  1. Ask the patient to protrude and retract the tongue alternately, a couple of times. Observe for upward-downward movement of the swelling.
  2. Hold the swelling at its upper pole between fingers and thumb. Ask the patient again to protrude and retract the tongue alternately and try to feel for a 'tug'.

Inference: Upward and downward movements of the swelling OR feeling of a 'tug' suggests thyroglossal cyst. Movement is better felt than seen.

Example of a swelling other than thyroglossal cyst, which moves on protrusion of the tongue: A swelling very closely related to the hyoid bone e.g. a subhyoid bursa / bursitis.

A point to remember: The absence of such a movement does not rule out the possibility of thyroglossal cyst.

c. Extent of mobility?
d. Axes in which mobility is free or restricted?

Anatomical plane and fixity

SCM is the key muscle in the neck. It is repeatedly required to demonstrate its relation to various swellings in the neck.

On isometric contraction of the muscle:
– A swelling superficial to it becomes more prominent
– A swelling partly deep to it often becomes more prominent
– A swelling deep to it becomes less prominent.

Closely related specific structures/organs/bone/joint: Example: Cervical spine for TB, which may → cold abscess in the neck.

ARRIVING AT A PRELIMINARY IMPRESSION

PRINCIPLES AND PITFALLS

1. Sometimes a transverse linear scar looks exactly like a surgical incisional scar; it may actually result from healing of several (tuberculous) sinuses or ulcers.
2. Swellings in the neck which exhibit impulse-on-coughing: Pharyngocele, laryngocele.

Arriving at a preliminary impression of a swelling in the neck

Irrespective of specific area / site in the neck

Consider the following possibilities first

– Bony
– Vascular
– Reducible:
  - Pharyngeal diverticulum (in the lower parts of the anterior and posterior triangle)
  - External laryngocele (in carotid triangle)
– Lymphadenopathy

In the midline*

Roughly from above downwards

– Ranula
– Subhyoid bursa / bursitis
– CA—larynx
– Thyroglossal cyst
– Goiter affecting isthmus or pyramidal lobe

Other:
– Midline / paramedian dermoid
– Hydatid cyst
– Teratoid cyst
– A thoracic swelling coming up in the neck
– Choristoma

Away from the midline

– In submandibular Δ
  - Submandibular sialomegaly
  - Plunging ranula
  - Lateral sublingual dermoid
  - Lymphadenopathy
  - Cystic hygroma
  - Glomus vagale tumor
  - Teratoid cyst
– In carotid Δ
  - Carotid body tumor
  - Laryngocele
  - Lymphadenopathy / cold abscess.
  - Goiter
  - Branchial cyst
  - A large lateral sublingual dermoid
  - SCM tumor
  - Chondroma in branchial remnant
  - Branchial (Branchiogenic) carcinoma
  - Ganglioneuroma
– In supraclavicular Δ
  - Cervical rib
  - Pharyngeal diverticulum (pouch)

Overlapping both the areas (i.e. midline and lateral)

Basically, any large swelling OR a swelling originating from a tissue lying roughly between the midline and lateral area
Examples: Cystic hygroma hydatid cyst, SCM 'tumor', etc.

* The exact breadth of the midline area has not been anatomically defined/specified.

www.Ebook777.com

Lymphadenopathy

Criteria: Usually multiple swellings situated in the specific anatomical areas known to be lymph node stations (e.g. neck / axilla / groin), and usually ovoid in shape. Refer to chapter 16.

Swellings which move on swallowing *(They are essentially midline swellings)*: Refer to page 251.

A chronic circumscribed swelling situated in the region of the thyroid: Refer to chapter 22.

Submandibular swelling

- Bilateral
  - Acute ⇓ Mumps
  - Chronic / recurrent* ⇓
    - Sjögren's syndrome
    - Sarcoidosis
- Unilateral
  - Acute
    - Mumps
    - Sialoadenitis due to stone
    - (Nonspecific) Lymphadenitis
  - Chronic / recurrent acute*

Underlying cause demonstrable

1. Stone in the submandibular duct

3 practical situations may be defined from point of view of planning the most appropriate surgical approach

(a) Stone readily palpable in the anterior 2 cm of the duct intraorally, with minor attacks of sialoadenitis
(b) Stone readily accessible in the floor of the mouth, but associated with much trouble from severe attacks of sialoadenitis
(c) Stone back in the hilum of the gland i.e. not easily accessible in the floor of the mouth

2. Ranula with more of a cervical component and inconspicuous intraoral component i.e. plunging ranula.
3. Lymphadenopathy.
4. Sjögren's syndrome affecting only one side, or symptomatic only on one side.
5. Rare conditions e.g. glomus vagale tumor, teratoid cyst, etc.

If possibility of a specific swelling is ruled out, then think about any swelling from 'general category'.

It will usually be necessary to take history again to ask for specific details, and repeat loco-regional examination depending on the 'preliminary impression' arrived at.

Underlying cause not demonstrable

Histological diagnosis must be sought

Salivary gland tumor?
Considerably more chance of being malignant unlike in case of parotid tumor



Fig 21-1 A submandibular swelling

* Chronic = continuous enlargement of the gland.
Recurrent acute = the presence of swelling only during the inflammatory attack. The swelling subsides considerably or almost completely after subsidence of inflammatory attacks.

SUBMANDIBULAR SIALOMEGALY

- **Age**?
  - Disorders in childhood: Mucoepidermoid carcinoma, adenocarcinoma.
- **Side affected**: Single OR both?
- **Progress**?
  - Long-periods of remissions and relapses: Obstructive sialectasis most commonly due to calculous disease
  - Slow with long quiescent period, and short-periods of rapid growth: Pleomorphic adenoma (mixed tumor)
  - Rapid progress over weeks ⇒ malignant tumor.

Free ebooks ==> www.Ebook777.com

However, a slow progress does not r/o malignancy.

– A dramatic sudden recent increase in size of the swelling which has been practically stationary ⇒ bleeding inside a cyst or cystic tumor.

- **Pain**
  - Constant OR intermittent?
  - When intermittent:
    - Does pain come on soon on eating or even with a thought of food?
    - Is it associated with swelling of the gland? How long does it persist?
    - Duration of each attack of pain?
    - Does pain get relieved rapidly following sudden spurt of saliva or foul-tasting fluid into the mouth?
    - Long periods of remissions and relapses?
  - In between attacks, does the swelling subside or does it persist (pain subsiding)?

INTERPRETATION of pain

- Painful conditions: Adenoid cystic carcinoma, inflammatory conditions, obstructive sialectasis most commonly due to calculous disease
- Causes of constant pain: Inflammation, malignancy
- Causes of intermittent—recurrent pain: Recurrent subacute / chronic sialoadenitis, obstructive sialectasis, Sjögren's syndrome
- Painful enlargement of the affected gland soon after eating
- Duration of pain during each attack—a few hours
- Swelling may persist for less than an hour to several days
- Long periods of remissions and relapses

} ⇒ Obstructive sialectasis most commonly due to calculous disease

- **Injury from a denture** at the site of opening of the duct?
- **Dryness of mouth, eyes**?
- **Rheumatic manifestations**?
- **Symptom s/o regional and generalized lymphadenopathy**?
- **Bimanual palpation of the floor of the mouth**
  - Insert a gloved finger into the mouth along the groove between the alveolus and the tongue as far back as possible and then press on the floor of the mouth
  - Place the fingers of the other hand under the jaw
  - The actual process of palpation involves a gentle attempt to make fingers of both the hands meet. Fingers will not meet but in the attempt, details of the tissues/structures in the floor of the mouth can be evaluated. Start palpating from behind forwards up to midline.
  - Normal and abnormal structures which can be thus palpated—submandibular salivary gland, duct of the gland, the presence of any stone, etc.
  - Press upon the gland gently (bidigitally) and look for extrusion of pus from the opening of the duct
  - Bimanual palpation enables to differentiate between the swellings of (/ arising from) the structures deep to the myelohyoid muscle and the swellings of (/ arising from) the structures superficial to myelohyoid muscle. Example: A submandibular salivary gland is felt bimanually whereas an enlarged submandibular lymph node can be felt only by the finger outside but not by the intraoral finger.

IN REST OF THE CONDITIONS ask for specific symptoms and look for signs, by referring to 'differential diagnosis'.

DIFFERENTIAL DIAGNOSIS

DD of extracranial carotid artery aneurysm

1. ***Kinked / coiled carotid artery***
   - Usually the common carotid artery is affected, producing a pulsatile mass at the base of the neck typically on the right side in obese, hypertensive, older women
   - Neurological manifestations are similar to those produced by atherosclerotic disease of the carotid bifurcation—TIA / stroke / amaurosis fugax
   - The symptoms may be typically precipitated by turning / moving the head.

www.Ebook777.com

Differentiating points
- Characteristic location
- The nature of pulsation—parallel to the long-axis of the vessel, in contrast to the pulsation of an aneurysm, which is typically expansile at right angles to the axis of the parent vessel
- Occasionally, the kink can be palpated intraorally in the tonsillar fossa
- A bruit may be heard, particularly when the patient turns the head.

2. ***Prominent carotid bifurcation*** in a person with a thin neck.
3. ***Any nonarterial swelling in close proximity to the artery*** e.g. carotid body tumor, enlarged lymph node, etc.

Cervical rib
- Tenderness in the supraclavicular fossa
- A lump in the lower part of the neck—may or may not be visible
- Bony hard and totally fixed
- Vascular symptoms, if the rib is 'complete'.

Pharyngeal pouch

Usually seen in elderly.

Symptom of stage-1: A sensation of foreign body in the throat. But otherwise, usually asymptomatic.

C/Fs of stage-2
- Regurgitation of undigested food at an unpredictable time after a meal, during the swallowing of the next meal, or after turning from one side to the other at night
- Awakening from sleep by a feeling of suffocation, f/b a violent fit of coughing.

C/Fs of stage-3

They are in addition to the clinical manifestations of stage-2, which persist.
- Gurgling noises in the neck, especially when the patient swallows
- A visible swelling in the neck
  - Ill-defined. 3–4 cm in diameter
  - Palpation may cause bout of coughing due to spilling of the contents into larynx
  - Palpation also → gurgling sound
  - Impulse-on-coughing +
  - Sometimes it can be seen to enlarge when the patient drinks
  - Reducible.
- Increasing dysphagia.
- Progressive loss of weight or even extreme cachexia due to starvation.

External laryngocele

Differentiating points

The symptoms come in attacks.
- The swelling often appears when the patient blows his nose; the swelling persists completely or partially for hours or days. The attack often terminates with a gurgling noise and discharge of mucus into the pharynx.

Characteristics of the swelling: Ill-defined. 2–3 cm in diameter. Impulse-on-coughing +. Moves on deglutition. Soft boggy. Translucent. Reducible.

Supporting points
- Hoarseness of voice
- Resonant / tympanic note on percussion.

Additional clinical features which may be seen
- Very rapid enlargement leading to stridor
- If sufficiently big, then it may cause discomfort in the throat, noisy respiration, and dysphagia.

Notes
- Common age at presentation—6th decade
- About half of the patients have both external and internal components. External component → swelling in the neck

Free ebooks ==> www.Ebook777.com

Internal component → swelling in the region of the vallecula.
- It is essential to exclude a coexisting carcinoma or papilloma of the larynx. DL scopy is recommended to exclude this possibility
- Complication: Laryngopyocele.

Subhyoid bursa / bursitis
- Recurrent attacks of painful swelling in the region of the hyoid (bursitis)
- Well-defined. Ovoid, transversely disposed
- 1–2 cm in diameter
- Moves on deglutition as well as on protrusion of the tongue
- Usually translucent.

A note: It is rare. Clinically, it cannot be distinguished from a thyroglossal cyst in subhyoid location.

Thyroglossal cyst
- Usual age at presentation—childhood and young adulthood
- Site—at any point between foramen cecum to manubrium and the anterior borders of SCMs laterally. Thus, a little lateral placement from the midline does not rule out the possibility of a thyroglossal cyst
  - The most common site—just in front of or below the hyoid
  - Very occasional site—base of the tongue
- Shape—hemispherical or ovoid. Occasionally, dumb-bell-shaped (on both sides of hyoid)
- 1–2 cm in diameter
- Moves up both on swallowing and on protrusion of the tongue
- Moves from side-to-side but not in craniocaudal direction
- Smooth but may be nodular if it contains thyroid tissue
- Transillumination usually absent.



Fig 21-2 Thyroglossal cyst

Complications
1. Recurrent infection.
2. Pressure symptoms.
3. Development of carcinoma within the cyst or tract, but this is usually only detected histologically.

Notes

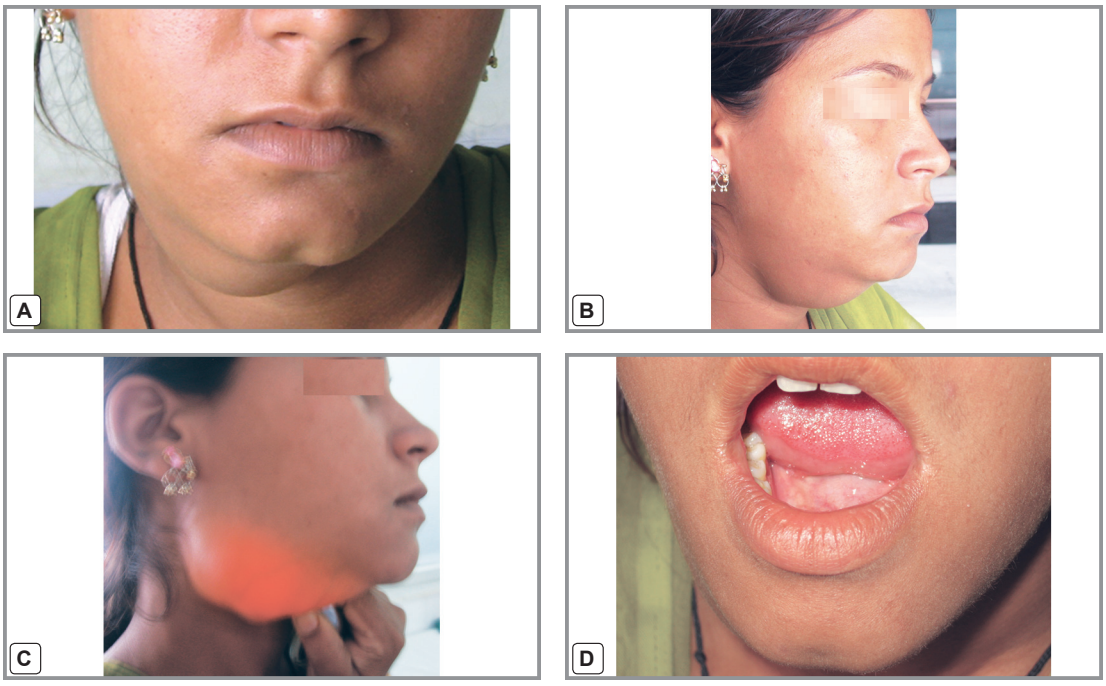
Sometimes it may be difficult to differentiate thyroglossal cyst from other midline cervical swellings pre-operatively and even intraoperatively. For example, it may not be possible to clearly distinguish even at operation between a thyroglossal cyst and a dermoid cyst.

Teratoid cyst

= teratoma-like tumor that lacks a trigerminal or complex histological appearance.
- Present at birth
- It is situated in or adjacent to the midline, anywhere between the submental region and sternal notch.

Sialoadenitis due to stone in the submandibular duct
- Pain comes on soon on eating or even with a thought of food
  - Associated with swelling of the gland
  - The attacks of painful swelling persist for variable period of time to recur with the next meal
- (Gentle) Pressure (bidigitally) upon the gland may lead to extrusion of pus from the opening of the duct
- Occasionally, a h/o of such recurrent minor symptoms is complicated by an acute attack of sustained pain and swelling for several days, possibly with constitutional symptoms. This results from ascending infection.

www.Ebook777.com



Figs 21-3A to D Ranula

Ranula with more of a cervical component and inconspicuous intraoral component i.e. 'plunging ranula'

- The cervical component: Soft. Fluctuation ±. Translucent
- The intraoral component: Dome-shaped swelling in part of the floor of the mouth. Bluish
- Pressure upwards on the cervical swelling makes the oral component more prominent
- There may be a h/o repeated bursting of the swelling into the mouth, with discharge of a little watery fluid.

Lateral sublingual dermoid cyst: Bidigitally palpable. Refer to in chapter 51.

Carotid body tumor

Basically, a chronic circumscribed swelling in the carotid triangle.

Differentiating points

- Ovoid or irregular shape
- 2–3 cm in diameter
- Moves from side-to-side but not in craniocaudal direction
- Surface—nodular
- Transmitted pulsations
- Thrill—often present
- Hard consistency.

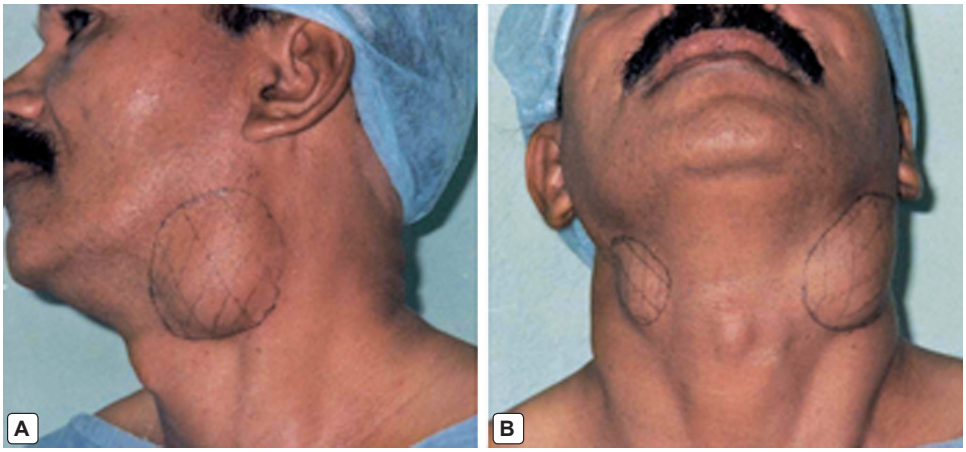
Supporting points

- Highest incidence—5th decade
- Bruit on auscultation.

Notes

- Situated at the bifurcation of common carotid artery
- Basically it is a paraganglioma. Paragangliomas arise from aggregations of nonchromaffin paraganglionic cells adjacent to the carotid bulb, jugular bulb, in the cavity of the middle ear, in the ganglion nodosum of the vagus and elsewhere in the body
- A rare tumor. Relatively high incidence in populations living at high altitudes.

Free ebooks ==> www.Ebook777.com



Figs 21-4A and B Branchial cyst

- The tumor most commonly arises from the inner aspect of the bifurcation of the carotid artery and may present as a pharyngeal mass displacing the tonsil
- **Open biopsy is contraindicated in view of the extremely vascular nature of the tumor.** Imaging investigations are appropriate
- A tortuous carotid artery may sometimes be confused with a carotid body tumor.

Branchial cyst

Basically, a chronic circumscribed swelling in the carotid triangle.

Differentiating points

- Classic position—anterior border of SCM at the junction of its upper one-third and lower two-thirds
- Intermittent presence of the swelling (in ~ 20% of cases)
- Well-defined. Hemispherical / ovoid
- 3–4 cm in diameter
- Limited mobility
- Relation to SCM
  - Posterior half of the swelling lies under the cover of SCM
  - The swelling becomes prominent on isometric contraction of the muscle
- Transmitted pulsations ±
- Fluctuation ±. Transillumination ±.

Notes

- Peak age incidence—3rd decade
- 2% bilateral
- If infection has occurred, it may be difficult to differentiate from lymphadenitis / cold abscess. Cholesterol crystals in aspirated fluid ⇒ branchial cyst.

Branchial (Branchiogenic) carcinoma

- Very rare
- Clinical manifestation is similar to 'malignant lymphadenopathy with occult primary'. However, this is a diagnosis of exclusion. This diagnosis may be considered only after every possible source of a primary growth in the mouth, pharynx and external auditory meatus has been excluded.

Cystic hygroma

- Age of occurrence: Early infancy or from birth
- Typical sites: Neck, cheek, axilla, groin, mediastinum
- Diffuse (Ill-defined). A cervicofacial lump may extend into thorax / axilla

www.Ebook777.com







Figs 21-5A to C Cystic hygroma

- May attain large size. Exceptionally may be so large as to obstruct labor
- Visibly increases in size (only momentarily) when the child cries or coughs
- Objective relation to SCM cannot be defined. Cystic hygroma does not respect tissue planes
- Surface—smooth or lobular
- Soft. Fluctuant
- Compressible
- *Brilliantly translucent*—the characteristic that distinguishes it from all other cervical swellings.

Natural history: Unpredictable.

1. May not enlarge; that means with growth of the child, the swelling becomes relatively smaller in size.
2. May grow with the child → increasing disfigurement and compromise of function.
3. Rarely, spontaneous regression may occur.
4. Complications:
   a. Infection of or bleeding (spontaneous / traumatic) into the cyst may → rapid painful enlargement—may be life-threatening.
   b. Pressure on vital structures viz. esophagus, trachea and great vessels.
   c. Interference with speech.

GENERAL REMARK with reference to investigation of a swelling in the neck

Open biopsy is contraindicated unless and until possibility of the swelling being a paraganglioma (glomus vagale in submandibular triangle and carotid body tumor in carotid triangle) can be ruled out with certainty clinically, in view of the extremely vascular nature of the tumor. Imaging investigations are appropriate in such a situation. FNAC is controversial, although one school of thought says that a carefully performed aspiration with a 25# needle should not prove invasive.

Free ebooks ==> www.Ebook777.com

22 GOITER AND THYROID CANCER

Goiter is **defined** as benign enlargement of the thyroid.

GENERAL REMARKS

1. Thyroid disorders are more common in women than in men.
2. Classification of thyroid diseases:
   - Congenital
   - Disorders of function
   - Disorders of structure → goiter, cancer
   - 'Clinically solitary nodule'—a special clinical entity
   - Autoimmune disorders.
3. Patients may present with
   a. Only hormonal dysfunction without a goiter.
   OR b. Only swelling (a goiter) without hormonal dysfunction.
   OR c. Combination of both hormonal dysfunction and a goiter.

   Obviously, the most difficult to diagnose at an early stage is '(a)' e.g. nongoitrous cretinism (where early diagnosis and replacement therapy are crucial to prevent permanent damage to the CNS), primary myxedema.
4. Thyrotoxicosis = hypermetabolic state induced by an oversupply of thyroid hormones to the tissues of the body.

   The oversupply can arise in 2 ways: (a) Hyperfunction of the thyroid referred to as hyperthyroidism, or (b) Excessive release of preformed hormones from a gland that is damaged often from inflammation. Thus, hyperthyroidism is only one of the causes of thyrotoxicosis. Despite these different implications, the two terms are often used interchangeably.
5. Any dominant nodule within a multinodular goiter (MNG) should be essentially investigated as a solitary thyroid nodule (STN), as there is ~ 10% incidence of malignancy occurring in the dominant nodule.
6. 'Dominant nodule' is a retrospective term—retrospective, after ultrasound scan examination of a clinically solitary nodule has been done. In practice, a clinical diagnosis of STN in fact turns out to be a dominant nodule in a MNG in 50% of the cases as shown on subsequent investigation (ultrasound scan).

HISTORY

- **Residence**?
  - Residence at high altitude (→ Iodine deficiency)
  - Residence in lowland areas (→ Iodine deficiency)
  - Do many people in your village suffer from goiter? ⇒ endemic goiter.
- **Diet**?
  - Does your diet contain excess of cauliflower, cabbage, soybean, mustard oil?
  - These 'goitrogens' predispose to endemic goiter.
- **Source of routine potable water**?
  - Deep tube-well water is deficient in iodine content.

www.Ebook777.com

Symptoms produced by thyroid disease can be grouped as under:

1. Swelling and related symptoms.
2. Symptoms produced by thyrotoxicosis ± autoimmune disorder.
3. Symptoms due to hypothyroidism.

SWELLING

- **Mode of onset**?
  - Almost always insidious
  - The so-called sudden m/o onset is always apparent. It virtually suggests bleeding in a pre-existing cyst / nodule, which was never noticed by the patient earlier.
- **Pain**?
  - Pain may be present in acute / subacute thyroiditis, and in later stages of malignancy
  - Otherwise, thyroid disorders are painless by and large.
- **Dragging sensation / discomfort during swallowing**?

  This symptom is present especially with large goiters. The large goiter, by virtue of its size and weight resists upwardly movement of larynx during swallowing leading to sensation of drag / discomfort.
- **Dysphagia**?

  Causes: Large / retrosternal goiter.
- **Dyspnea**?
  - Serial degrees of severity: Stridor, actual difficulty in breathing, eventually cyanosis
  - Usually caused by a large goiter, and a retrosternal goiter
  - Nocturnal dyspnea when sleeping on a side may ⇒ a retrosternal goiter. Mechanism: Stretching and angulation of trachea over the goiter causes dyspnea.
- **Hoarseness** of voice (Dysphonia)?

  Causes:
  - A malignant tumor infiltrating the recurrent laryngeal nerve
  - Occasionally a large goiter causing pressure damage of the recurrent laryngeal nerve
  - Hypothyroidism (hoarseness is not related to recurrent laryngeal nerve palsy).
- H/s/o **SVC syndrome** (Compression of SVC)?
  - Headcahe?
  - Visual disturbances?
  - Facial and conjunctival edema?
  - Dilatation of veins of upper thorax and neck, and plethora?
  - Reduced state of consciousness?

Symptoms produced by **THYROTOXICOSIS**

- *CVS*

  SOB, chest pain, palpitations?
- *Nervous and musculoskeletal systems*

  Nervousness, irritability, lability of mood, tremors, weakness of muscles producing tiredness in general, tiredness of the eyes on reading, difficulty in writing / grasping, difficulty in climbing down the staircases (due to quadriceps weakness)?
- *GIT and metabolic*

  Increase in appetite, loss of weight, diarrhea, excessive sweating, and intolerance to heat?
- *Reproductive system*

  Oligomenorrhea / amenorrhoa?
  - Rapid progress, and exacerbations and remissions of the above-mentioned symptoms are seen in Grave's disease
  - Many of the symptoms and signs of thyrotoxicosis are present in anxiety state. The most useful differentiating feature of thyrotoxicosis is combination of weight loss with the fact that food intake has not decreased (in fact often increased).

Free ebooks ==> www.Ebook777.com

Symptoms produced by **AUTOIMMUNE DISORDER**, seen in Grave's disease

- Exophthalmos: This is due to infiltrative ophthalmopathy. It may be bilateral or unilateral
  Complications of exophthalmos
  - Redness of the eyes
  - Conjunctival edema
  - Diplopia (due to weakness of extraoccular muscles particularly the elevators)
  - Corneal ulceration.
- Hyperpigmentation, thickening of skin of leg and dorsum of foot?: This occurs due to infiltrative dermopathy.

Symptoms produced by **HYPOTHYROIDISM**

In infants

- Delay in the appearance of normal milestones of development?
- Has the mother been taking antithyroid medicines during pregnancy?
  The clinical manifestations of cretinism usually do not become apparent for several months—hence, a high index of suspicion is required for early diagnosis.

In adults

- Nervous system: Lethargy, and tiredness?
- GIT and metabolic: Constipation, weight-gain, intolerance to cold, dry skin, puffiness in periorbital area, supraclavicular area and hands and feet?
- Reproductive system: Profuse menstrual flow?
- Hoarseness of voice?
- Carpal tunnel syndrome?
  M/o onset of these symptoms is *insidious, in fact often subtle*.

Have you been on **any long-term thyroid medication** for a thyroid hormonal dysfunction? How long? Response to the treatment?
Is the treatment being monitored in appropriate manner?

- Patient who has been on thyroxine replacement treatment may suffer from thyrotoxicosis due to over dosage
- Patient on antithyroid medication may suffer from hypothyroidism due to over dosage.

Past history of RT to the neck?

Family history h/o thyroid cancer?

In **GENERAL EXAMINATION**, the points specifically applicable to a thyroid swelling are as follows:

Level of consciousness?
Causes of reduced state of consciousness:

- May be due to SVC syndrome caused by retrosternal goiter.
- Myxedema.

In severe cases of thyrotoxicosis, the whole body becomes shaky and trembling.

Facial appearance?

- Staring looks (due to lid-retraction) and expression of fear (due to exophthalmos) = *hyperthyroid facies* (Hyperthyroidism due to Grave's disease only)
- Dull look, malar flush, periorbital puffiness and supraclavicular puffiness ⇒ *severe degree of hypothyroidism* (myxedema)
- In a child: Pale puffy face and protruding tongue may suggest cretinism
- Facial edema, dilatation of veins of the upper thorax and neck and plethora – Could be due to *SVC syndrome*.

Compare the facial appearance with a previous photograph if available.

www.Ebook777.com



Fig 22-1 SVC syndrome

Eyes

- Exophthalmos and consequences
  - Deep injection of the conjunctiva i.e. dilatation and engorgement of conjunctival vessels
  - Chemosis i.e. conjunctival edema
  - Conjunctival ulceration
- – Slight drooping of the upper lid
  – Slight sinking of the eyeball into the orbit
  – Myosis (Contraction of pupil).
  ⇒ Horner's syndrome—involvement of the sympathetic trunk

Extremities

- Look for
  - Hyperpigmentation
  - 'Orange peel' appearance
  - Coarse hair
  - Palmar erythema
  - Clubbing of fingers and toes
- Palpate the skin and hands
  - Hyperpigmentation
  - Coarse hair
  - 'Orange peel' appearance ±
  - Thickening of skin

  ⇒ Pretibial myxedema—the dermopathy of Grave's disease
  - Palmar erythema, and warm and wet hands ⇒ thyrotoxicosis
  - Cold, rough, thick and dry skin is seen in hypothyroidism.

Pulse

- Tachycardia ⇒ thyrotoxicosis
  - Tachycardia can result from anxiety. The best clinical way to differentiate between thyrotoxicosis and anxiety is to check pulse rate (quietly) when the patient is sleeping
  - Tachycardia persisting during sleep ⇒ thyrotoxicosis
  - Degree of thyrotoxicosis on the basis of sleeping pulse rate:
    - < 90/min = mild thyrotoxicosis
    - 90–110/min = moderate thyrotoxicosis
    - > 110/min = severe thyrotoxicosis
- Bradycardia ⇒ hypothyroidism
- Water hammer pulse ⇒ thyrotoxicosis.

Height

Short stature is seen in cretinism.

Free ebooks ==> www.Ebook777.com

Weight

- Underweight suggests thyrotoxicosis
- Overweight / obesity could be associated with hypothyroidism.

LOCAL EXAMINATION

POSITION of the patient: Sitting.

– Inspection, and palpation by Crile's method are carried out from front (with the head slightly extended)
– Rest of the examination (palpation) is carried out from behind.

INSPECTION

Inspection is carried out from front. Head of the patient is slightly extended so that anterior portion of neck is fully visible.

- **Site**?
  - A thyroid swelling is located in the anterior aspect of inferior portion of the neck, i.e. 'in the region of thyroid'
  - The middle (central) portion of the swelling always lies inferior to Adam's apple, though a large goiter may mask it
  - Anatomical explanation for this: Thyroid lies deeper to sternothyroid muscle. Superior attachment of sternothyroid is on the oblique line of thyroid cartilage
  - This attachment restricts thyroid enlargement in upward direction
- **Margin**?

  As far as inferior border is concerned, in case of a goiter with retrosternal extension, it becomes visible only when the patient swallows
- **Size**?

  Endemic goiters can attain huge size.
- **Surface**

  Smooth / nodular / irregular?
- **Skin**?
  - Red, tense and glossy: May be due to rapidly growing malignant tumor, or acute bacterial thyroiditis
  - 'Orange-peel appearance' (due to peculiar edema of the skin) ⇒ infiltration of the skin by a malignant tumor
  - Dilated veins upon the swelling and/or proximally (i.e. on the cardiac side) ⇒ malignant tumor
  - Sinus ⇒ TB [i.e. pretracheal chronic tuberculous lymphadenitis, or tuberculous thyroiditis (rare)]
  - Ulcer ⇒ TB (associated with pretracheal chronic tuberculous lymphadenitis, or tuberculous thyroiditis (rare)), or malignancy
    - The lesion is clinically called 'ulcerated swelling'
- **Pulsations**?

  May be seen in Grave's disease
- **Impulse-on-coughing**?

  Seen in laryngocele
- **Mobility**: Refer to page 251.
- **Pressure effects**?

  Nonpulsating venous congestion over neck and chest occurs in retrosternal goiter.

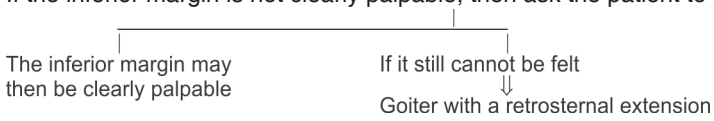
PALPATION

While still standing in front of the patient, place the thumb on the thyroid and ask the patient to swallow. During the upward movement of the gland, try to appreciate small nodules within the substance of the gland. – This is called Crile's method of palpation. Crile's method of palpation is useful for detection of nodules within the substance of the gland i.e. those nodules, which cannot be appreciated as easily as surface nodules and thereby standing a chance of getting missed. It is also useful for detection of a slight enlargement of the gland, otherwise not easily appreciated.

www.Ebook777.com

- Further palpation should be carried out by standing behind the patient
- Place the thumbs on the nape of the neck and use them gently to maintain slight flexion of the head to relax the visceral compartment of the neck
- Palpation is done initially with **flat of the fingers** to find out the swelling, and later with **fingertips** to find out the details of the swelling
- The lobe being palpated is made prominent by gently pushing on the contralateral lobe

- **Margin**?
  - Try to 'get below' the inferior margin of the swelling
  - If the inferior margin is not clearly palpable, then ask the patient to swallow



- **Impulse-on-coughing**?: Expansile impulse-on-coughing is present in a laryngocele
- **Mobility**
  a. **Mobility on swallowing**?
    - Hold the swelling at its upper pole between fingers and thumb. Ask the patient to swallow and try to feel for a 'tug'
    - *Inference*: Feeling of a 'tug' ⇒ swelling attached to the larynx. Movement is better felt than seen.

  b. **Mobility on protrusion of the tongue.**
    - Ask the patient to open her / his mouth, and instruct to maintain the same degree of opening throughout the test. Reason: Movement of the mandible during opening and closing of the mouth may lead to some unintended movement of the swelling making the test invalid
    - Hold the swelling at its upper pole between fingers and thumb. Ask the patient to protrude and retract the tongue alternately and try to feel for a 'tug'
    - *Inference*: Feeling of a 'tug' ⇒ thyroglossal cyst. Movement is better felt than seen.

  c. **Mobility on holding and moving the gland in all directions**
    - **Extent** of mobility is normally more in transverse direction than craniocaudal direction
    - **Restriction** of mobility ⇒ cancer / chronic thyroiditis
- **Surface**: Smooth / nodular / irregular? Size of individual nodules?
- **Thrill**?
  - A thrill may be palpable overlying a primary toxic diffuse goiter of Grave's disease.
- **Pulsations**?
  - Keep flat of the fingers on the swelling for a few minutes
  - Pulsations may be felt overlying a primary toxic diffuse goiter of Grave's disease.
- **Closely related specific structures / organs**
  1. Pulsations of carotid artery?
    - *The artery should be palpated just behind the enlarged lobe and not in the upper part of the neck above the swelling*
    - Palpable pulsations rule out possibility of an advanced malignant tumor. Impalpable pulsations ⇒ malignant tumor of the thyroid
    - Generally, carotid artery pulsations are palpable in a c/o goiter, however big the goiter may be. – This is because the goiter merely pushes the carotid sheath posteriorly but does not engulf it; whereas, in a c/o cancer, the swelling engulfs the carotid sheath and hence pulsations cannot be felt.
  2. Position of the trachea: Central, OR shifted to one side?
  3. *Kocher's test*
    - *Purpose*: Demonstration of tracheal compression from both sides giving rise to what is called 'scabbard trachea'
    - *Procedure*: Gently press both lobes of the gland simultaneously
    - *Observe* for stridor
    - Development of stridor suggests that trachea has been compressed.

Free ebooks ==> www.Ebook777.com

PEMBERTON'S SIGN

- *Purpose*: Demonstration of retrosternal goiter.
- *Principle*
  Superior mediastinum is already a narrow anatomical space. Raising the arms above the head makes it further narrow with development of symptoms and signs suggestive of visceral compression, which were otherwise absent.
- *Procedure*
  Ask the patient to raise the arms above the head. Observe for induction of pressure symptoms and signs.
- *Inference*
  Induction of obstructive symptoms and signs e.g. dyspnea, distress, venous congestion over anterior chest-wall and neck, and cyanosis suggest the presence of a retrosternal goiter.

EYE SIGNS

Normal appearance of the eyes

– Upper lid covers the pupil as it crosses it at ~ 11 and 1 o'clock positions. Hence, the upper limbus is not visible
– Lower lid passes just below the lower limbus which is barely visible along with a strip of sclera.

Eye signs in Grave's disease

1. *Exophthalmos*
   As the eyeball is pushed forwards, sclera is visible both above and below the superior and inferior limbus.
   Complications of long-standing exophthalmos
   a. Dilatation and engorgement of conjunctival vessels.
   b. Conjunctival edema.
   c. Corneal ulceration.
2. *'Lack of forehead wrinkling' while looking upwards.*
   a. Ask the patient to look upwards towards your finger without moving the head. Normally, the skin on the forehead wrinkles in an attempt to do so. Wrinkling does not occur if exophthalmos is present.
3. *Weakness in convergence for near objects.*
   a. Hold your finger at a distance of about 1 meter from the patient's eyes.
   b. Ask the patient to follow the moving finger while bringing it nearer the patient.
   c. The inward convergence movement of the eyes is slow, jerky and unequal on two sides.

Eye signs due to thyrotoxicity

Basic mechanism: Thyrotoxicosis → contraction of the involuntary part of levator palpabrae supirioris under sympathetic over activity.

1. *Staring / frightened look.*
   This characteristic look is produced due to involuntary retraction of the upper lid leading to exposure of a strip of sclera at the upper limbus.



Fig 22-2 Exophthalmos



Fig 22-3 Malignant exophthalmos



Fig 22-4 The staring looks

www.Ebook777.com

2. *Infrequent blinking.*
3. *Lid-lag*
   - Hold your finger at the level of the patient's eyes, at a distance of about 2', and ask the patient to follow the movement of the finger strictly only by moving the eye keeping the head still
   - Move the finger down fairly quickly
   - Observe at the movement of the upper lid while the patient's eye is following the moving finger.

   The upper lid failing to keep pace with movement of the eyeball is the criterion of +ve sign.
4. *Globe lag*
   - This test is done in continuity with lid-lag test
   - Move the finger up, back to the previous level with the patient's eyes
   - Observe the movement of the eyeball relative to the movement of the upper lid.

   Criterion of +ve sign: The upper lid which was lagging behind the globe while looking down, races ahead of it while looking up; whereas the globe lags.

(Other) SIGNS OF THYROTOXICOSIS

1. **Tremors in the tongue**

   Ask the patient to put out and hold the tongue and look for tremors.
2. **Tremors in the hands**
   - Switch off the fan in the room, if it was on
   - Ask the patient to stretch out both the upper limbs fully to level with the shoulders, and spread out the fingers, palms facing downwards
   - 'Course' tremors are easily visible
   - To demonstrate 'fine' tremors, keep a piece of paper on the dorsum of the hand, and observe the paper.
3. **Examination of shoulder girdle muscles for weakness**
   - Test the deltoid, latissimus dorsi, pectoralis major and trapezius
   - Ask the patient to contract these muscles against resistance, and judge the grade of power.
4. **Examination of pelvic girdle muscles**

   Ask the patient to sit on the floor and then get up. Patient finds it difficult to get up if pelvic girdle muscles are weak.
5. **Deep reflexes**: Brisk.

(Other) SIGN OF HYPOTHYROIDISM

Ankle jerk: Prolonged and sluggish.

MEASUREMENT

Circumference of the neck at the most prominent part of the swelling taken at intervals is of great clinical value in determining whether the swelling is increasing or decreasing in size.

AUSCULTATION

- Bruit may be heard over a vascular thyroid swelling e.g. in Grave's disease
- The best site to hear bruit: Superior pole of the thyroid.

Free ebooks ==> www.Ebook777.com

In **SYSTEMIC EXAMINATION**, look specifically for the following:

- Signs of atrial fibrillation and heart failure—in secondary thyrotoxicosis
- Protuberant abdomen—in hypothyroidism
- 'Pot belly'—in cretinism
- Hepatomegaly, splenomegaly—in Hashimoto's disease.

IDL (Indirect laryngoscopy)

Purpose: Diagnosis of recurrent laryngeal nerve palsy.

Possible causes:

- Involvement of recurrent laryngeal nerve by thyroid tumor
- Pre-existing laryngeal nerve palsy unrelated to the present goiter.

Medicolegal significance

It is important to inform the patient about the presence of recurrent laryngeal nerve palsy if present, and document the fact before a thyroidectomy.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. The Normal thyroid gland is neither visible nor palpable, even when pushed towards the examining fingers from the opposite side.
   Dimensions of each lobe of a normal gland: 5 × 3 × 2 cm.
2. If a **smooth** enlargement of only one side of the thyroid is felt, the goiter must be classified as a single nodule. One cannot speak about diffuse enlargement of one lobe, because one can never be sure that even the largest unilateral goiter is the whole of one lobe; the rest of the lobe may be lying posteriorly, completely overshadowed by the large anterior single nodule.
3. A solitary thyroid nodule may be as small as 1 cm in diameter or assume a huge size and displace the trachea well over to the opposite side.
4. The sign of fluctuation is unreliable in thyroid swellings. It is unwise to clinically diagnose a swelling in the thyroid as either cystic or solid.
5. A swelling situated in the region of the thyroid, and which moves on swallowing should be clinically considered as a thyroid swelling unless proved otherwise (on investigation) though other possibilities should not be forgotten.

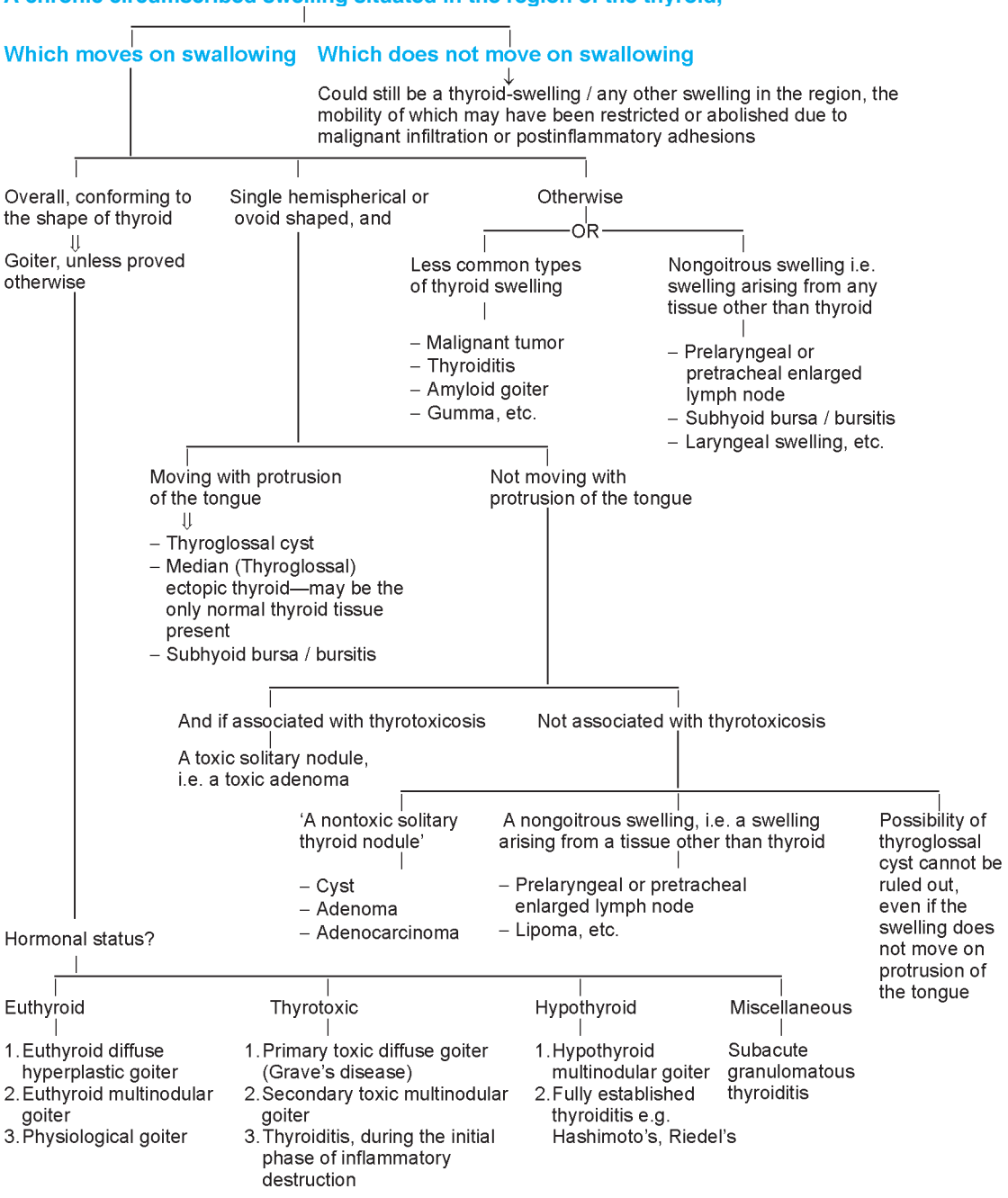


Fig 22-5 Clinically, a solitary thyroid nodule

www.Ebook777.com

STEPWISE ANALYSIS OF DATA

A chronic circumscribed swelling situated in the region of the thyroid,



Chronic thyroiditis is rare and should only be considered after common conditions are ruled out.

Free ebooks ==> www.Ebook777.com

Differentiating and supporting clinical features	*Benign enlargement (goiter)*	*Thyroid cancer*
Age	Any	Usually > 40
Duration	'Long'	'Short'
Pain	–	+
Hoarseness of voice	Absent. Exception—hypothyroid goiter	+
Loss of weight	Absent. Exception—toxic goiter	'Marked'
Dilated veins	Absent. Exception—toxic goiter	+
Mobility	+	Restricted
Consistency	Firm to soft	Hard
Carotid pulse at the level of thyroid	Palpable	Not palpable
Regional lymphadenopathy	–	+

DIFFERENTIAL DIAGNOSIS

Euthyroid diffuse hyperplastic goiter
Without / with pressure symptoms
Probable cause: Endemic (due to deficiency of iodine) **OR sporadic** (due to mild to moderate degree of dyshormonogenesis) **OR** due to **goitrogen**

- Thyroid hormonal status—normal
- Age 15–25 years
- Shape—conforming to the shape of thyroid
- Size—'moderate'
- Surface—smooth
- Consistency—soft or firm.

Euthyroid multinodular goiter
Affecting the entire gland, or right / left lobe
Without / with pressure symptoms
Probable cause: Endemic (severe deficiency of iodine) **OR sporadic** (severe degree of dyshormonogenesis)

- Thyroid hormonal status—normal.
- 20–30 years of age suggests endemic goiter
- 25–40 years of age suggests sporadic goiter
- Shape—irregular, though the overall shape conforms to that of the thyroid
- Size—'moderate' to 'large'
- Surface—nodular
- Consistency—firm. In later stages, may be hard if calcification has taken place
- Trachea may be deviated
- Kocher's sign may be +ve.

Complications

- Difficulty in breathing due to tracheal displacement / compression
- Bleeding in a cyst / necrotic nodule → dramatic increase in size of the pre-existing swelling, acute severe pain ± sudden severe breathlessness
- Toxic change in a very long-standing goiter—called secondary toxicity
- Rarely, malignant transformation.

Physiological goiter—associated with menarche / pregnancy / lactation

- Thyroid hormonal status—normal
- Shape—conforming to the shape of thyroid; symmetrical
- Usually only 3–4 times the size of a normal gland

www.Ebook777.com

– Surface—smooth
– Consistency—firm
– Usually self-limiting. Subsides by itself or with medical treatment (usually only iodine supplement).

Note: Marked or asymmetrical enlargement is NOT physiological.

Primary toxic diffuse goiter (*Syns.* Exophthalmic goiter, Grave's disease)

– Symptoms and signs of thyrotoxicosis
– Exacerbations and remissions of symptoms ±
– Usually affects women between 20 and 40 years of age
– Characteristics of the goiter:
  - Rarely more than 3 time the size of a normal gland
  - Surface—smooth
  - Soft / firm
  - Thrill and bruit may be present, usually at the superior pole
– Eye signs of Grave's disease
– Pretibial 'myxedema'.

Notes

– Grave's disease = a triad of hyperthyroidism, infiltrative ophthalmopathy and infiltrative dermopathy. Usually 2 out of the 3 disorders are present; rarely only one is present
– *Natural history of untreated disease (Complications)*
  1. Complications of untreated exophthalmos: Ophthalmoplegia, conjunctival edema, corneal ulceration.
  2. Cardiac complications: Failure, arrhythmias.
  3. Thyrotoxic crisis.

Secondary toxic (multinodular) goiter

– Recent development of thyrotoxicosis over a pre-existing euthyroid multinodular goiter that has been present for a long time (usually > 10 years)
– Other features are described in the table below.

Differentiating and supporting clinical features	*Primary toxic diffuse goiter (Grave's disease)*	*Secondary toxic multinodular goiter*
Age	Young	Adult
Progress	Rapid	Slow
Perspiration	Marked	Slight
Loss of weight	Marked	Slight
CNS manifestations	More marked	Less marked
Eye signs	Usually present	Rare
CVS manifestations	Less marked	More marked
Characteristics of the goiter	Smooth, soft, vascular	Multinodular, firm

Hypothyroid multinodular goiter affecting the entire gland, or right / left lobe, without / with pressure symptoms

Probable cause: Endemic (severe deficiency of iodine) **OR sporadic** (severe degree of dyshormonogenesis).

– Symptoms and signs of hypothyroidism are present
– Characteristics of the goiter—same as those of euthyroid multinodular goiter.

'Clinically solitary thyroid nodule'

– Reason for specifying as 'clinically' is that on imaging investigation, the nodule may turn out to be a 'dominant' nodule of a multinodular goiter
– Any dominant nodule within a MNG should be essentially investigated as a solitary thyroid nodule, as there is ~ 10% incidence of malignancy

Free ebooks ==> www.Ebook777.com

– The diagnostic dilemma revolves around the anxiety induced by the fear of malignancy in the nodule, against a background of common benign nodular disease
– A nodule could be euthyroid / hyperthyroid / hypothyroid

↓

An example of hypothyroid nodule: Nodular Hashimoto's disease possibly with lymphomatous change.

Risk factors with respect to cancer

- Age < 20 / > 50
- Male gender
- Rapid increase in size. **However, a cancer can be extremely slow growing**
- H/o irradiation to head / neck
- Family h/o thyroid cancer
- Clinical features
  – Size > 4 cm in diameter
  – Hard consistency
  – Restricted mobility
- Recurrent laryngeal nerve palsy
- Cervical lymphadenopathy.

On investigation, a 'clinically solitary nodule' will turn out to be one of the following, on the basis of which further line of management may be planned.

– Cyst—benign OR malignant
– Nontoxic STN (i.e. hormonal status normal)—benign (colloid adenoma / simple adenoma / papillary adenoma/thyroiditis, etc.) OR malignant (DTC / MTC)
– Toxic STN (i.e. a STN associated with thyrotoxicity) = Toxic (thyroid) adenoma—usually not malignant
– Hypothyroid solitary nodule (rare)—benign (adenoma) OR nodular Hashimoto's disease possibly with lymphomatous change
– (Usually euthyroid) Multinodular goiter with a dominant nodule—adenoma OR adenocarcinoma.

Hashimoto's disease = Autoimmune goitrous thyroiditis

– Characteristically affects women only. 40–50 years of age
– Mode of onset:
  - Insidious and asymptomatic
    OR
  - Sudden and painful resembling acute form of granulomatous thyroiditis
– Goiter:
  - Diffuse, or localized to one lobe
  - Slightly lobular
  - Firm
– Thyroid hormonal status:
  - Initially, mild thyrotoxicosis (during the phase of inflammatory destruction)
  - Eventually, hypothyroidism when the disease is fully developed
– ± Hepatosplenomegaly
– Family history ±.

Riedel's thyroiditis

– Diffuse, or localized to one lobe. If unilateral, the other lobe is usually involved later
– 'Moderate' enlargement
– Fixed (i.e. restricted mobility or, immobile)
– Hard (described as 'woody' or 'stony' hard) consistency
– Early and marked pressure effects (due to extension of sclerosing process beyond the gland)
  Clinically, it may not be possible to differentiate it from undifferentiated thyroid cancer.

www.Ebook777.com

– Thyroid hormonal status:
  - Initially, mild thyrotoxicosis (during the phase of inflammatory destruction)
  - Eventually, hypothyroidism when the disease is fully developed
– May be associated with retroperitoneal and mediastinal fibrosis.

Lymphadenoid goiter

Similar to Riedel's thyroiditis except for:

1. Most commonly affects young girls.
2. Inflammatory process does not extend beyond the gland.

Subacute granulomatous thyroiditis

– Pain in the neck, fever, malaise
– Goiter:
  - One or both lobes
  - Irregular and firm
– Self-limiting. The goiter subsides in a few months time.

Gumma of the thyroid

– Unilateral
– Painless
– Hard
– Later, fixity and pressure effects.

A note on retrosternal goiter

Literally, goiter posterior to the sternum can be called a 'retrosternal goiter'.
It can arise in 2 ways. (1) From the inferior pole of a basically cervical goiter, which means—a goiter with a retrosternal extension. This type is more common. (2) Arising from an ectopic thyroid tissue, posterior to the sternum. There are 2 subtypes of this: (a) (Fully) Intrathoracic. Many of these patients may initially attend a chest clinic for a mistaken diagnosis of asthma before the true problem is revealed. (b) Plunging: Basically an intrathoracic goiter;—occasionally forced into the neck by increased intrathoracic pressure.

Short notes on selected clinical conditions

Causes of thyrotoxicosis



– Diffuse toxic hyperplasia (i.e. Graves' disease)
– Diffuse multinodular goiter
– Toxic solitary nodule

– Acute / subacute thyroiditis. Mild thyrotoxicosis may occur in the early stages due to liberation of thyroid hormones from damaged gland
– Over dosage of l-thyroxine (> 0.2–0.3 mg/day)
– Jod-Basedow phenomenon: Excessive release of thyroxine in an iodine-deficient patient on resumption of dietary iodine intake, or administration of intravenous contrast
– A large mass of secondary carcinoma may rarely induce mild hyperthyroidism
– Neonatal thyrotoxicosis: Babies born to hyperthyroid mothers or to euthyroid mothers who have had thyrotoxicosis
– Struma ovarii (ectopic thyroid), choriocarcinoma
– Primary malignant tumor of the liver

Thyrotoxic crisis

= a manifestation of extreme hypermetabolism in a thyrotoxic patient.

Precipitating factors

1. 'Surgical storm'—after a thyroidectomy in an improperly / inadequately prepared patient.
2. 'Medical storm'—infection, trauma or any surgical procedure other than thyroidectomy.

Free ebooks ==> www.Ebook777.com

C/Ms
- Extreme irritability, delirium
- Hyperpyrexia
- Marked tachycardia
- ± Vomiting, diarrhea.

Aggressive treatment should be started immediately without waiting for the reports of serum hormone levels.

Cretinism

= Fetal or infantile hypothyroidism

Nongoitrous	Goitrous
Due to agenesis or hypoplasia of thyroid = 'Sporadic cretinism' Parents and siblings may be perfectly normal	Due to – Maternal and fetal iodine deficiency = 'Endemic cretinism' – Mother on antithyroid drug

C/Ms of cretinism

Usually do not become apparent for several months—hence, a high index of suspicion is required for early diagnosis

Basically, comprise of retardation of mental and physical development i.e. it → delay in the appearance of normal milestones of infant development
- Pale puffy face, protruding tongue
- Short stature due to slowed epiphyseal bone growth
- Delayed dentition
- Dry skin
- 'Pot belly' i.e. protuberant abdomen.

Mental retardation can become irreversible if treatment of cretinism is delayed for up to the age of 2 years. Hence, the importance of early diagnosis.

Adult hypothyroidism

Myxedema = a very advanced form of adult hypothyroidism and the term should never be applied to milder forms seen much more frequently in practice.

Causes of hypothyroidism

Nongoitrous	Goitrous
More common in practice – Surgical / radiation ablation of toxic goiter or thyroid cancer – Primary—idiopathic: Probably of autoimmune etiology—'autoimmune thyroiditis' *Less common causes* – Therapeutic ablation of pituitary, vascular damage to anterior pituitary – Hypothalamic disorders	– Endemic Iodine deficiency – Hashimoto's disease = autoimmune goitrous thyroiditis – Riedel's thyroiditis – Goitrogens – Severe dyshormonogenesis

C/Ms of mild to moderate degree of hypothyroidism
- Insidious and often subtle
- Requires a high-index of suspicion to make an early diagnosis. Comparison of the facial appearance with a previous photograph is helpful
- Lethargy, tiredness, intolerance to cold, constipation, weight-gain, profuse menstrual flow
- Slowing of mentation, speech and movements; hoarseness of voice, a dull look, periorbital puffiness, slow pulse rate, cold, rough, thick and dry skin, slow relaxation of ankle-jerk
- Carpal tunnel syndrome ±

www.Ebook777.com

C/Ms of severe degree of hypothyroidism i.e. myxedema

– Previously described c/ms of mild to moderate degree of hypothyroidism are accentuated + malar flush, supraclavicular puffiness, yellow tinge to the skin
– Coma in undiagnosed and neglected cases;—high-mortality
– Myxedema is so called because of edematous, doughy thickening of the skin owing to accumulation of mucopolysaccharide in connective tissues throughout the body.

Myxedema coma

– *Precipitating factors*: Inadequate treatment of hypothyroidism, infection, exposure to cold, trauma. It is most likely to occur in elderly in cold season
– *C/Ms*: Coma, hypothermia, marked bradycardia, hypoventilation, hypotension
– *Associated abnormalities*: Hypoglycemia, dilutional hyponatremia, lactic acidosis
– *Diagnosis: Essentially clinical.*

Treatment should be started without waiting for the reports of serum hormone levels, as the condition is acutely life-threatening.

A note on thyroid cancers

Primary thyroid cancers →	*DTC*	*MTC*	*UTC*	*Lymphoma*
Cell of origin	Thyroid follicular cell	'C' cell	?	MALT i.e. Mucosa-associated lymphoid tissue
Modes of diagnosis	1. FNAC 2. After a previous thyroid operation	1. FNAC 2. After a previous thyroid operation	1. Core biopsy ↓ LA 2. FNAC	1. FNAC 2. Core biopsy ↓ LA – Allows immunochemical subtyping
Modes of presentation	A STN/a 'dominant' nodule within a MNG – The nodule of 'large' size and hard consistency ± Restricted mobility ± Regional lymphadenopathy ± Recurrent laryngeal nerve palsy	1. Sporadic 2. Familial MEN associated 3. Familial non-MEN associated A thyroid nodule and / or regional lymphadenopathy – Pain – Diarrhea – Dyspnea / dysphagia due to compression	1. *De Novo* 2. In a long-standing benign goiter 3. Recurrence in the form of a transformation of a previously treated well-differentiated thyroid cancer, which may have been in remission for a long time – 70% patients have mets at the time of presentation – A large diffuse swelling of hard consistency, usually invading adjacent structures ± Restricted mobility ± Regional lymphadenopathy ± Recurrent laryngeal nerve palsy	Usually upon a preceding long-standing goiter or hypothyroidism, in 80% of cases which is due to autoimmune disease e.g. Hashimoto's

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

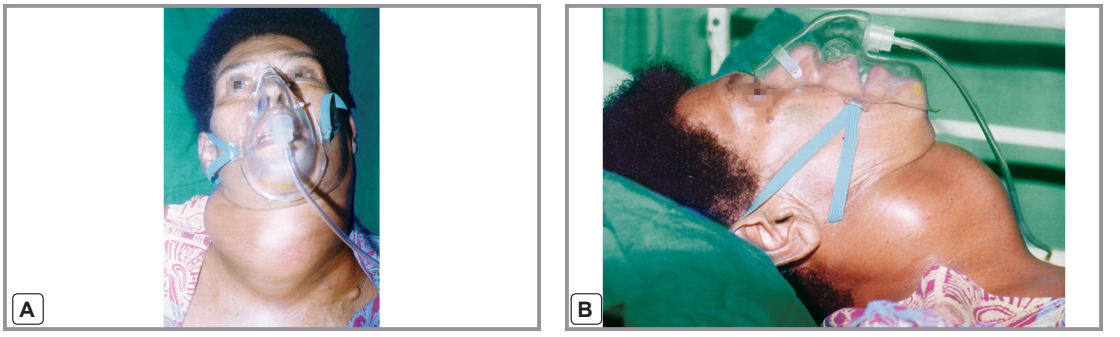
Primary thyroid cancers →	*DTC*	*MTC*	*UTC*	*Lymphoma*
Specific clinical background, supporting the diagnosis	– Age < 20/> 50. – Male gender. – A h/o RT to the neck. – A family h/o thyroid cancer	– Family h/o thyroid cancer/pheochromocytoma – Rarely, Cushing's syndrome due to gut peptide or ACTH release by the tumor	Age 60–70.	– Age > 60 – Preceding autoimmune thyroid disease in ~80% of cases

– DTC = Differentiated thyroid carcinoma. Examples: papillary, follicular
– MTC = Medullary thyroid carcinoma
– UTC = Undifferentiated thyroid carcinoma.

Secondary thyroid cancers are rare. Blood-borne metastases occur from primary carcinomas of breast, colon, kidneys and from malignant melanomas.

Goiters simulating anaplastic carcinoma:

– Riedel's thyroiditis
– (Localized forms of) Granulomatous thyroiditis
– Lymphadenoid goiter.



Figs 22-6A and B Anaplastic cancer. Terminal stage. Patient had refused surgical treatment at an earlier manageable stage

www.Ebook777.com

23 A REDUCIBLE (IRREDUCIBLE) SWELLING IN THE GROIN

DEFINITIONS

1. **Groin** = an oblique strip of area 3 cm above + 3 cm below, and parallel to the inguinal ligament.
2. '**Reducible swelling**': On gentle compression, the contents of the swelling are pushed back completely into the adjacent deeper (- essentially larger) cavity viz. the abdomen, so that the swelling completely disappears; and reappears only on an active maneuver like standing (at the least) or coughing or sneezing or crying.
3. '**Irreducible swelling**' = basically a swelling which has been reducible, which fails to reduce completely or even partially.
4. '**Nonreducible swelling**' = a swelling which never disappeared (or got reduced) ever since it appeared.
5. **Hernia** = an abnormal protrusion of a part or the whole of a viscus, through a (anatomically) normal or abnormal opening in the wall of its containing cavity.
6. **Saphena varix** = a soft compressible dilatation at the termination of long saphenous vein.

GENERAL REMARKS

1. Before declaring a swelling as 'non-reducible', make sure you are not dealing with an 'irreducible swelling'. Occasionally, patients do present with h/o an acute / subacute nonreducible swelling when they are actually having an irreducible hernia which they had never noticed earlier, or disregarded it in its reducible state.

2. Most cases of chronic pain in athletes are due to soft tissue injuries but in a small number, undeclared hernia is the cause.

3. If a patient says that he has been wearing a truss, do not take for granted that he is suffering from a hernia. Sometimes, a patient with a hydrocele uses a truss under the wrong impression of having a hernia!

4. Pubic tubercle is such an important landmark that the clinician must satisfy herself / himself by finding it rapidly and accurately, even in a fat or uncooperative patient.

Clinical methods

a. If a case is suspected to be an inguinal/inguinoscrotal/inguinolabial swelling: Ask the patient to adduct the (straight) lower limb against resistance. Follow the adductor tendon upwards until its attachment on the pubic bone is reached. Follow the bone slightly upwards to reach the pubic crest. Follow the pubic crest laterally until the prominence of the pubic tubercle is felt.

b. If a case is suspected to be a femoral swelling: Follow the midline from the umbilicus downwards until the pubic symphysis is felt. Follow the pubic crest laterally until the prominence of the pubic tubercle is felt.

5. Importance of distinguishing between the oblique and the (common type of) direct inguinal hernias: The common type of direct hernia is less likely to strangulate because the defect is a diffuse area without deeply defined edges. Therefore, it is safe to keep a direct inguinal hernia waitlisted. It is also safe to treat a (common type of) direct inguinal hernia with a truss. It is not safe to treat an oblique inguinal hernia with a truss; instead it is advisable to operate.

6. A direct inguinal hernia does not lie within the coverings of the spermatic cord and therefore does not show a great tendency to follow the cord into the scrotum unlike an oblique hernia.

Free ebooks ==> www.Ebook777.com

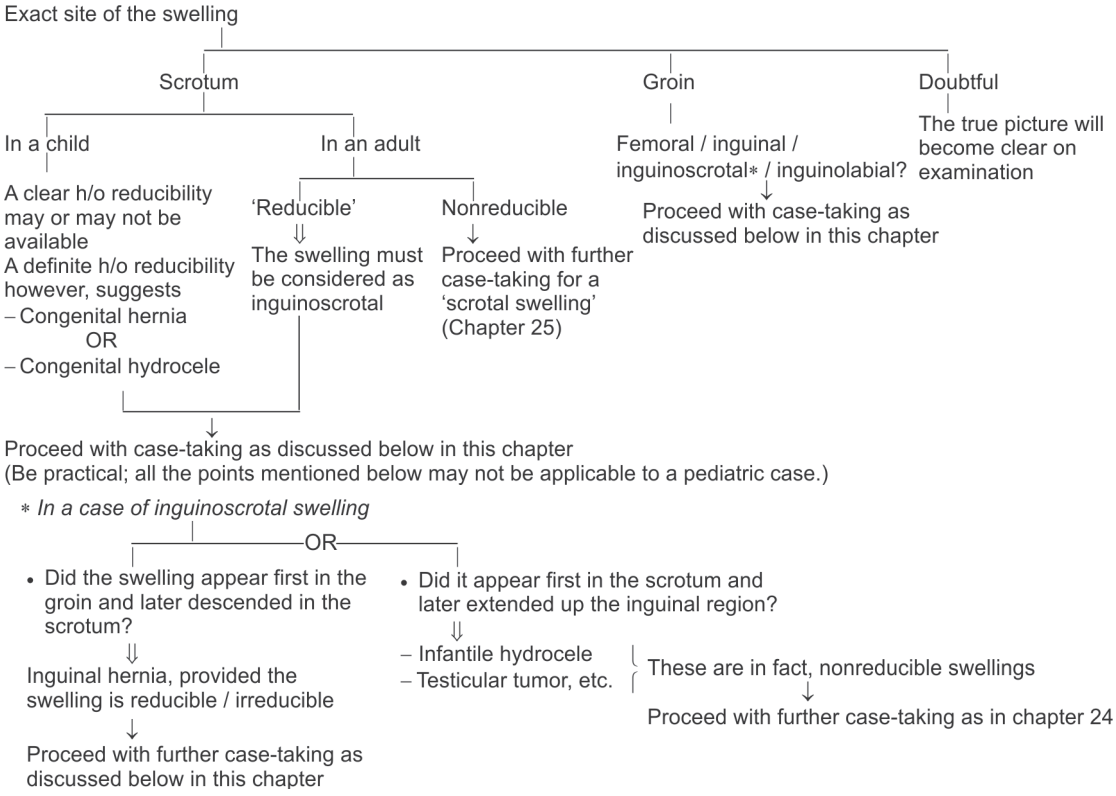
7. Busoga (an area in Uganda) type is a rare type of direct inguinal hernia. It arises through a narrow defect in the conjoint tendon or transversalis fascia. It may have a tendency to descend into the scrotum as against with the common type of direct hernia. As the neck of the Busoga type of direct hernia is narrow, it is prone to develop complications.
8. The symptoms to which a femoral hernia gives rise are less pronounced than those of an inguinal hernia; indeed a small femoral hernia may be unnoticed by the patient or disregarded for years, until perhaps the day it strangulates.
9. Spigelian hernial orifice lies in the aponeurosis of the oblique muscles of the anterior abdominal wall at the lateral edge of the rectus sheath, below the linea semilunaris. The defect is usually quite small and, if it lies in the upper part of this region, is easily distinguished from the external inguinal ring; the lower it lies however, the more likely is confusion to arise with an inguinal hernia.
10. An obturator hernia is most unlikely to be clinically distinguishable from a femoral hernia.
    Reason: The obturator hernial orifice itself is deep in the thigh, inaccessible to physical examination. As the obturator hernia progresses and comes to lie superficially in the clinically accessible subcutaneous tissue, occupies more or less the same position of a femoral hernia.
11. The majority of patients who are admitted with complicated groin hernias have not previously sought medical attention or been diagnosed with the condition in the outpatient department. This observation implies that most hernias that develop complications do so within a relatively short-time in the natural history of the disease.
12. Obstructed hernia usually culminates in strangulation. Although symptoms and signs of strangulated hernia have been described, often no clear distinction can be made between obstruction and strangulation particularly in early period of strangulation. Hence, the safe course is to assume that strangulation is imminent and treat urgently.
13. In a strangulated hernia, the commonly involved viscera is small intestine or/and omentum. For large intestine to become strangulated in an inguinal hernia is of the utmost rarity, even when the hernia is of sliding variety.
14. At the operation, after opening the inguinal canal, if an inguinal hernia is not found as per expectation of the clinical diagnosis, then flaps should be raised upwards/downwards to search for a spigelian / femoral hernia respectively.

HISTORY

- **Exact duration**?
- **Exact site**?
- **Is it reducible or irreducible**?
- **Details of reducibility**
  - Does it reduce spontaneously on lying down?
    OR
  - Does it require some manipulation? How difficult it is?
- **Details of irreducibility**
  - Duration? – Few hours OR weeks / months
    ↓
    = Constant, OR frequently recurrent?
  - Associated h/s/o intestinal obstruction / strangulation?
- **Mode of onset**?
  Did it appear all of a sudden following lifting of a heavy weight, or bout of cough, or straining at stools, or a spell of cry?
  OR Was it insidious?
- **Extent of the swelling at the time of its first appearance, and progress**?
  - Did the swelling reach the bottom of the scrotum straight at the time of its first appearance?
  OR – Was the swelling small to start with and gradually increase in size?

www.Ebook777.com

A preliminary judgment may be obtained at this juncture



- **Pain**?
  - Exact site?
  - Intensity: Mild / moderate / severe?
  - Nature: Dragging / vague?
  - Precipitating, aggravating, relieving factors?
  - Constant OR intermittent?
- **Relation to micturition**?
  - Does the size of the swelling appear larger just prior to micturition, and get diminished soon after micturition?
  - Does pressure on the swelling induce a desire for micturition especially when the swelling is in a distended state just prior to micturition?
- H/o **chronic bronchitis / chronic habitual constipation / chronic difficulty in micturition**?
- H/o **any surgery in the lower quadrant** of abdomen, such as appendicectomy for example?
- **Is this a recurrent swelling**, a surgery having been performed earlier for the same?
  If so, – Details of the previous surgery
    - When was it performed?
    - Technical method of repair (was a mesh used)?
  – Within how many days did you start strenuous activity after the surgery?
  – (If applicable -) could you control (reduce) your body weight?

INTERPRETATION OF SYMPTOMS, AND DISCUSSION

Free ebooks ==> www.Ebook777.com

Age

– Direct inguinal hernia is more common in old age
– Direct inguinal hernia is extremely rare in children
– Congenital hernia (as a rule, oblique inguinal) is seen in children, adolescents and sometimes in young adults
– A femoral hernia is rare before puberty.

Gender

– Direct inguinal hernia almost never occurs in women
– Inguinal hernia as such, is rare in women
– Femoral hernia is common in women.

Occupation

Occupations involving strenuous work predispose to development of hernia.

Details of reducibility

– Spontaneous reduction on lying down ⇒ - Direct inguinal hernia, unless descending in the scrotum which is unusual - Small 'early' oblique inguinal hernia - Saphena varix
– Reduction requiring some maneuver ⇒ - Oblique inguinal hernia—funicular/complete type - Occasionally, a direct inguinal hernia that descends in the scrotum - Femoral hernia
– In a child, spontaneous but slow reduction overnight ⇒ congenital hydrocoele.

Mode of onset

– Sudden mode of onset following lifting of a heavy weight, or bout of cough, or straining at stools, or a spell of cry, etc. ⇒ descent of abdominal contents in a preformed sac, in turn ⇒ essentially congenital (oblique) inguinal hernia
– Insidious mode of onset ⇒ direct inguinal hernia / acquired oblique inguinal hernia / femoral hernia / saphena varix / encysted hydrocele of cord / congenital hydrocele.

Extent of the swelling at the time of its first appearance, and progress.

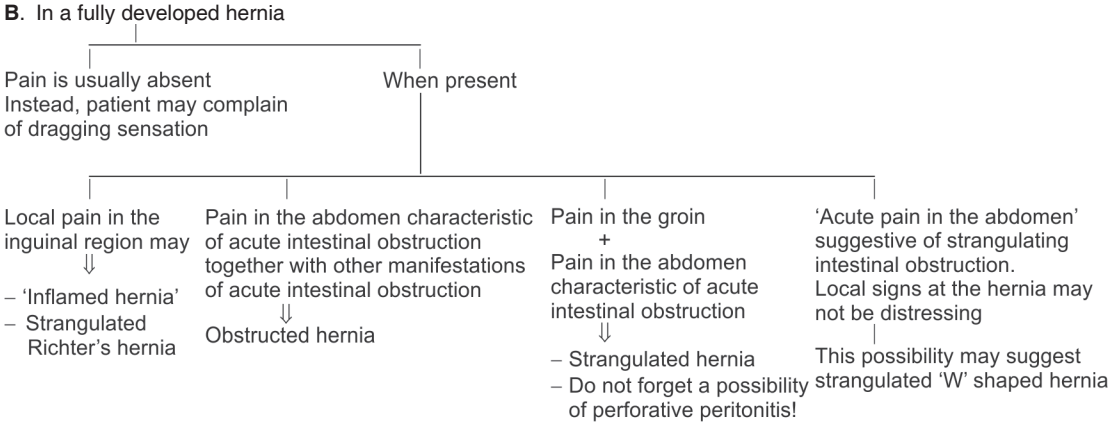
– A swelling reaching the bottom of the scrotum straight at the time of its first appearance ⇒ descent of abdominal contents into a preformed sac (patent processus vaginalis) ⇒ congenital hernia
– A swelling that was small to start which gradually increased in size ⇒ acquired hernia.

Pain

A. Pain may be present in a small developing hernia. It is caused by stretching of tissues by the protruding viscera.
  – Such a pain is often vague and usually intermittent
  – Drag on the mesentery or omentum may → epigastric pain/discomfort.

  Pain may be the only symptom in small nonpalpable hernias.

B. In a fully developed hernia



Relation to micturition

Size of the swelling appearing larger just prior to micturition and getting diminished soon after micturition; and if pressure on the swelling induces a desire for micturition especially when the swelling is in a distended state suggests cystocele in a direct inguinal hernia.

www.Ebook777.com

Chronic bronchitis / chronic habitual constipation / chronic difficulty in micturition predispose to the development of a hernia.

Any surgery in the lower quadrant of abdomen may predispose to the development of an inguinal hernia in particular of direct type, due to possible damage to ilioinguinal nerve.

LOCO-REGIONAL EXAMINATION

EXPOSURE: From umbilicus downwards, including both groins.

POSITION of the patient: Standing with legs apart. Also, ask the patient to cough a couple of times to make the (hernial) swelling achieve its fullest dimensions.

If a swelling in the inguinal region is not easily visible being small in size, inspect the region from the side.

POSITION of the clinician: Sitting on a stool with eyes at the level of the groin.

INSPECTION

Inspect the **apparently normal side first**.

- Any abnormal bulge? If not,
  ↓
- Ask the patient to cough again to confirm normalcy

Inspection of swelling on the **abnormal side**
↓

- **Number**?
- **Site and extent**?
  - Inguinal ⇒ - An 'early' oblique inguinal hernia
    - (The usual type of) direct inguinal hernia
  - Inguinoscrotal ⇒ - An oblique hernia
    - Rarely, a direct hernia: Very long-standing, OR Busoga type

    ↓

    Is the testicle separately visible?
  - Inguinolabial ⇒ hernia (oblique by default)
  - Femoral ⇒ - Femoral hernia
    - Saphena varix
    - Rarely, obturator hernia
  - In case of a child: An apparently scrotal* swelling is often due to congenital hydrocele / congenital hernia.
- **Margin**?
  - Well-defined margin all around, of a spherical or ovoid swelling (usually present at the root of the scrotum) may suggest encysted hydrocele of the cord
  - Margin of an inguinal hernia is ill-defined as the swelling lies deep to aponeurosis
  - In c/o a femoral swelling:
    - Well-defined margin may suggest saphena varix
    - Ill-defined margin suggests femoral hernia
- **Shape**?
  - *In c/o an inguinal swelling*
    - Ovoid shape with long axis parallel to the inguinal ligament ⇒ oblique hernia
    - Hemispherical shape ⇒ direct hernia

* From the point of view of pathologic anatomy, congenital hydrocele and congenital hernia are inguinoscrotal swellings. However, on inspection they appear to be scrotal for various reasons—1. In infants, the canal is anteroposterior. 2. The canal is quite short; consequently, the inguinal portion, which is only a small portion of the entire swelling, may not be easily visible enough to be differentiated from a pad of fat present in the region almost invariably in children.

Free ebooks ==> www.Ebook777.com

- Shape of *an oblique inguinoscrotal hernia* is pyramidal
- *A fully visible spherical / ovoid swelling at the root of the scrotum* ⇒ encysted hydrocele of the cord
- *In c/o a femoral swelling*
  - Retort shape ⇒ femoral hernia
  - Saccular shape ⇒ saphena varix
- *In a child*
  - Globular shape may suggest congenital hydrocele
  - Ovoid shape may suggest congenital hernia

- **Size**?
  - An exceptionally huge swelling suggests hernia, more so of oblique type—called 'a giant hernia' or 'scrotal abdomen'
  - No other reducible swelling can attain a very big size
  - Chances of developing strangulation in a huge hernia are less as the internal inguinal ring has become greatly enlarged
- **Pulsations**?
- **(Visible) Impulse-on-coughing**?

  Purpose
  - Confirmation of the diagnosis of hernia / saphena varix. Rules out possibility of encysted hydrocele of the cord
  - An impulse-on-coughing is said to be present, when on coughing / sneezing / crying, the swelling expands momentarily **OR**, the swelling increases in size **OR**, a swelling appears where there was none
  - The swelling **must expand**. **Slight movement-on-coughing should not be considered as a +ve test.**

  Procedure
  - Ask the patient to turn the face to one side to avoid droplet infection, and then ask him to cough, often repeatedly
  - *Observe* at the **external inguinal ring** for momentary expansion of the swelling / increase in size of the swelling / appearance of a swelling where there was none.

  Inference
  - +ve test ⇒ uncomplicated hernia
    However, the test is +ve *even in an irreducible hernia* (except for femoral)
  - –ve test ⇒ - Encysted hydrocele of the cord
    - An inguinal hernia complicated by obstruction, strangulation.

  Fallacies
  1. Slight movement-on-coughing should not be considered positive.
  2. Often –ve in a femoral hernia, even uncomplicated.
- **Peristalsis** (visible across the skin)?
  - ⇒ hernia and nothing else but hernia
  - Observe very carefully. *Movement of the scrotal skin due to contraction of dartos* should not be mistaken for peristalsis
- **Surface**?
- **Skin overlying the swelling**?
  - Red, tense and glossy ⇒ inflamed or strangulated hernia
  - Dermatitis?—the presence of dermatitis is a contraindication for an elective surgery
  - If the patient has been using a truss for a long time, the skin will show discoloration, wrinkling, and thinning due to atrophy of subcutaneous fat.
- **Scar** and its details?

  Scar overlying the swelling suggests recurrence of the present condition, usually a recurrent hernia.
- **Edema**?
  - Edema suggests inflamed or strangulated hernia
- **Minor bilateral bulging in the region of the inguinal canal** is normal in thin-built individuals.

www.Ebook777.com

PALPATION

Palpate the **apparently normal side first**.

- Any abnormal bulge? If not

↓

- Ask the patient to cough while palpating, to confirm normalcy

Palpation of the swelling on the **abnormal side**

↓

- **Number**?
- **Site and extent**?

  Extent on palpation may differ from that of the inspection finding.

  Is the testicle palpable separately?

  In a complete inguinal hernia, the testicle is not palpable as it is engulfed by the hernia.

  – A 'bubonocele' = an inguinal hernia confined to the inguinal canal. The misnomer 'bubonocele' is worth omitting, but may stay for want of an alternative term; may I suggest: 'purely inguinal' hernia?

  – A 'funicular' hernia = an inguinal hernia, which stops short of the superior pole of the testis
- **Relation to the pubic tubercle**: Is the swelling superomedial OR inferolateral to pubic tubercle?
  – Superomedial to pubic tubercle: Inguinal hernia, congenital hydrocele
  – Inferolateral to pubic tubercle: Femoral hernia, saphena varix
  – Inferolateral to pubic tubercle, but situated in an unusually medial position—may suggest a Laugier's femoral hernia.

  If a femoral hernia enlarges considerably in size, its various parts may have quite different relations to the pubic tubercle. However, **the neck** of the hernial sac always lies inferolateral to pubic tubercle.
- **Relation to femoral pulse**: Is the swelling medial OR lateral to the femoral pulse?
  – Psoas abscess lies lateral to the femoral pulse
  – Rest of the (reducible/irreducible) swellings lie medial to the femoral pulse
- (Local) **Temperature**?

  Increased local temperature ⇒ inflamed or early strangulated hernia.
- **Tenderness**?

  ⇒ complicated hernia—inflamed / irreducible / obstructed / strangulated.
- **Thrill**?
- **Pulsations**?
  – Look for pulsations as a rule although pulsations are not expected in the case of a reducible swelling in the groin.
- '**Getting above'—**applicable to only inguinoscrotal swelling, and a 'scrotal' swelling.

  Purpose: To differentiate a purely scrotal swelling from a hernia with inguinoscrotal extent.

  Principle: Upper limit of the swelling + normal spermatic cord and nothing else but normal spermatic cord should be palpable.

  Procedure

  Hold the **root** of the scrotum between the thumb in front and the fingers behind, and make an attempt to reach the upper limit of the swelling i.e. 'to get above the swelling', and also to palpate the normal spermatic cord.

  Inference

  – It is not possible to 'get above' in the case of a hernia and a congenital hydrocele
  – It may be possible to 'get above' in an encysted hydrocele of the cord

↓

In that case, perform the 'traction test'.

'Traction test'

Purpose: To ascertain the diagnosis of encysted hydrocele of the cord.

Principle: The swelling as well as the testis are attached to the same cord viz. the processus vaginalis. Traction on the testis leads to pulling and consequently restricting the mobility of the cord ('Cord' means the fibrous cord resulting from obliteration of processus vaginalis. 'Cord' in this context does not mean spermatic cord.).

Precaution: Do not grasp the testis itself as that causes a hurting sickening painful sensation.

Free ebooks ==> www.Ebook777.com

Procedure and observation

– Grasp the spermatic cord just above the upper pole of the testis, and apply a gentle downward traction
– Observe for any movement of the swelling as the traction is being applied and any restriction in the mobility of the swelling.

Findings in a c/o encysted hydrocele of the cord

1. The swelling moves down i.e. caudally.
2. Mobility (both craniocaudal as well as transverse) is restricted.

- **(Palpable) Impulse-on-coughing**?

  Procedure and observation

  Ask the patient to turn the face on one side to avoid droplet infection.

  – In c/o an inguinal swelling: Try to grasp and compress the entire swelling, or as much as is possible. If the swelling is too small to grasp between fingers and thumb, then place the fingers upon the swelling and compress firmly. Ask the patient to cough (often repeatedly)
  – In c/o an inguinoscrotal swelling: **Compress the swelling firmly**, preferably at the root of the scrotum, between the fingers and the thumb, and ask the patient to cough (often repeatedly)
  – In c/o a saccular swelling in the femoral region: If the swelling is not big enough to be grasped between the thumb and the fingers, then just place the fingers upon the swelling, and ask the patient to cough (often repeatedly)
  – If the femoral swelling is big enough such that it can be grasped, then grasp the 'root' (i.e. the part of the swelling as much flush with the deep fascia as possible) of the swelling between the thumb and fingers, and ask the patient to cough. Specific reason for going down to the 'root' of the swelling: If the swelling is a femoral hernia, then the chance of getting expansile impulse-on-coughing increases. If the fundus of the swelling is grasped, then impulse-on-coughing is less likely to be felt as fundus of the femoral hernia is often covered with a pad of fat. Temptation to grasp the fundus comes naturally as that is the largest portion of the hernia that attracts attention; that temptation should be curbed.

  Increase in tension and expansion of the swelling ⇒ +ve result. Increase in tension is better felt when the swelling is grasped and compressed firmly rather than when the fingers are simply placed upon the swelling.

  – In a small swelling, expansion may not be appreciated. ↑ in tension must be felt at the least to qualify for +ve result. ↑ in tension will be appreciated only by the clinician performing the test.

  Inference of +ve test

  1. Expansile impulse-on-coughing ⇒ uncomplicated hernia.
     However, the test is +ve *even in an irreducible inguinal hernia*.
  2. Vague / indefinite impulse is felt in congenital hydrocele, hydrocele of the canal of Nuck, and cold abscess along the cord (rare).
  3. Thrill-like impulse upon a femoral saccular swelling ⇒ saphena varix.

  Inference of –ve test

  1. Encysted hydrocele of the cord.
  2. Inguinal hernia complicated by obstruction, strangulation.

  Fallacies

  1. Slight movement felt on coughing should not be considered as a +ve test.
  2. The test is often –ve in a femoral hernia, even uncomplicated.

  A remark

  A swelling exhibiting impulse-on-coughing *need not necessarily be reducible*.

- **Margin**?

  – Well-defined margin all around, of a spherical/ovoid swelling (at the root of the scrotum) suggests encysted hydrocele of the cord

www.Ebook777.com

- In c/o an inguinoscrotal hernia, margin of the inguinal portion of the swelling is ill-defined; and margin of the scrotal portion of the swelling is well-defined
- In c/o a femoral swelling:
  - Margin of a femoral hernia is often ill-defined
  - Margin of a saphena varix cannot be really judged on palpation, as consistency of the swelling is highly soft; the swelling yielding to pressure very easily

- **Shape**?
- **Size**?
- **Surface**?

  Granular surface ⇒ omentocele.
- **Consistency**?
  - lastic ⇒ enterocele
  - Doughy ⇒ omentocele
  - Soft ⇒ saphena varix
  - Consistency of a congenital hydrocele is variable; it will depend on the tension of fluid within the sac.
- **Fluctuation**?
  - +ve in congenital hydrocele
  - An enterocele is also fluctuant, but in an enterocele, fluctuation can be practically tested in only one axis.
- **Transillumination**?

  +ve in:
  - Encysted hydrocele of the cord
  - Congenital hydrocele
  - Hernias in infants and neonates only
- **Moulding**?
  - Can the swelling be moulded i.e. indented?
  - If the swelling can be moulded, in a patient who is constipated, then it could be an 'incarcerated' hernia
- Insert **the little finger very gently** through the skin of the upper part of the scrotum and **through the external inguinal ring**
  - If this is possible, then it means that there is no swelling related to the spermatic cord, which in turn means that the hernial swelling under consideration must be a femoral hernia.

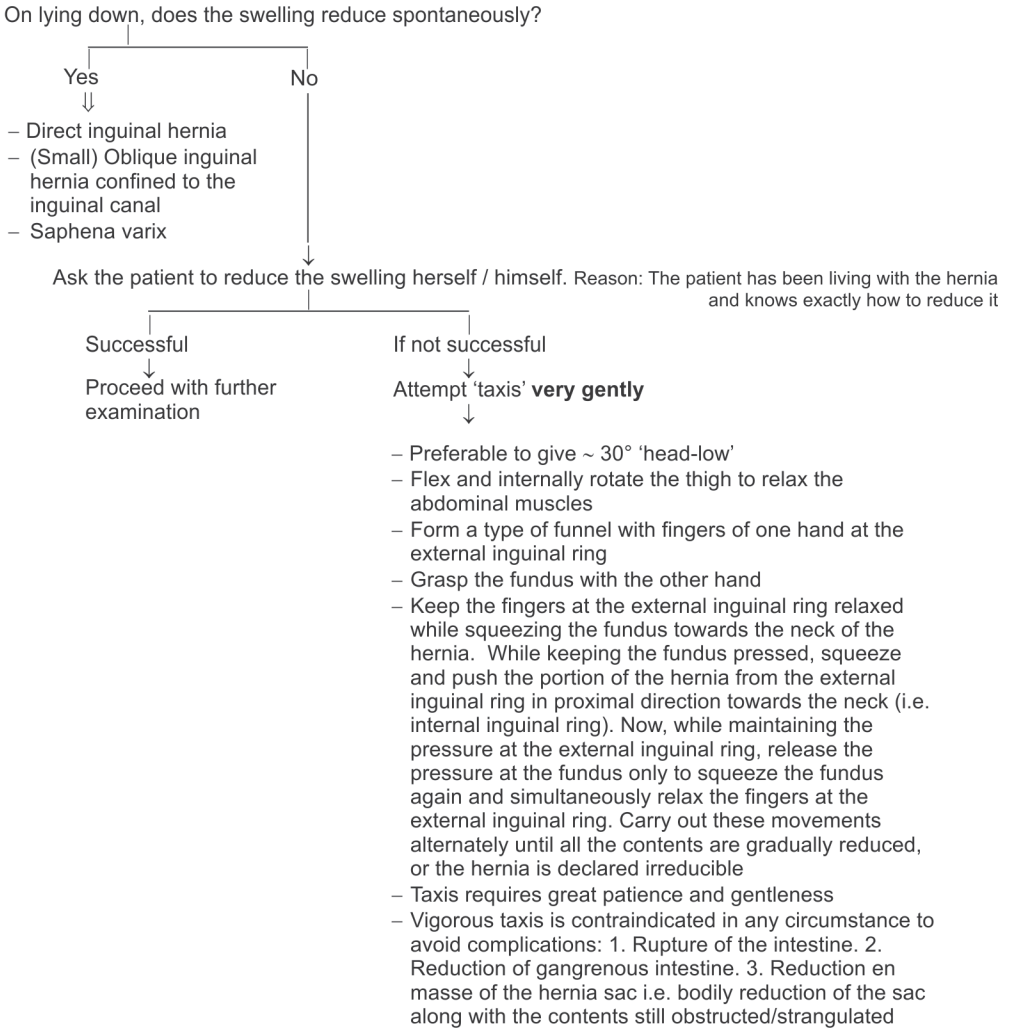
Now change the POSITION of the patient to lying down.

Further examination is carried out with the patient in lying down position.

- **Reduction**?

  A swelling exhibiting impulse-on-coughing *need not necessarily be reducible*.

Free ebooks ==> www.Ebook777.com



Points to be noted during reduction

1. Does the swelling initially gradually decrease in size and then disappear, or does it disappear in one stroke due to bodily movement of the entire swelling? The first way of disappearance is seen in hernia whereas the latter way of disappearance suggests encysted hydrocele of the cord or partially descended (enlarged) testis.
2. If there is any difficulty in reduction, is it during initial portion, or later portion?
   - Difficulty during initial portion ⇒ enterocele
   - Difficulty during later portion ⇒ omentocele.
3. Is reduction accompanied by gurgling? Gurgling suggests enterocele.
   - In case of an oblique inguinal hernia, when it is said that the hernia has been reduced, it means that the contents have been returned to the abdominal cavity; the sac stays
   - *Congenital hydrocele is not reducible instantaneously.* It reduces slowly when lying down position is maintained for a sufficiently long-time—practically at night when the child is asleep.

www.Ebook777.com

- **External inguinal ring test**

  The procedure can be painful or unpleasant for the patient and therefore should be performed if at all, with great gentleness.

  1. Try to invaginate the little finger very gently, through the skin of the upper/middle part of the scrotum and through the external inguinal ring. Normally, external ring admits only the tip of a little finger. Enlarged ring is an indirect e/o hernia, in the absence of a demonstrable swelling.
  2. Ask the patient to cough.

     An impulse-on-coughing palpable to the finger in the external inguinal ring suggests inguinal hernia. This may be the earliest sign of inguinal hernia, when even visible impulse is absent.
  3. With the finger-tip maintained through the external ring, ask the patient to raise both the lower limbs straight (without bending at the knees) at least through the angle of ~ 45° and judge (a) competency of the shutter mechanism, and (b) the site and size of the defect (weakness). Weakness lying just behind (i.e. posterior to) the external ring suggests direct hernia.

  A note on controversial point

  Some surgical books mention that the external inguinal ring test helps to differentiate direct from oblique inguinal hernia.–If the impulse-on-coughing hits the tip of the little finger within the canal, then that suggests oblique inguinal hernia. Impulse hitting the pulp of the little finger suggests direct inguinal hernia.

  Practically, that is difficult for the following reasons:

  1. Although the tactile discriminating power of a bear finger is excellent, the same degree of discriminating ability will not be present when a finger is passed through a few layers of coverings in a small area.
  2. While pushing the finger through the superficial ring, it invariably gets slightly flexed at the DIP joint, so that the tip may actually lie on the conjoint tendon rather than facing the anterorsuperior iliac spine.

  Remark

  Some authorities consider that this test does not yield extraordinarily useful information with respect to diagnosis and management; hence it may be omitted particularly because the test is unpleasant for the patient. This argument seems practically correct.

- **Internal inguinal ring occlusion test**

  – Applicable to a reducible inguinal/inguinolabial/inguinoscrotal swelling.

  Purpose:

  – To differentiate between direct and oblique types of inguinal hernia
  – In women, practically to confirm an oblique hernia; direct hernia being rare.

  Prerequisite

  – Hernia must be reduced
  – There is no indication to do this test in a hernia that is not reducible
  – Since a congenital hydrocele is not instantaneously reducible, the test cannot be applied to a congenital hydrocele.

  Principle

  – The internal inguinal ring is situated 1–1.5 cm superior to the midinguinal point (midway between the anterorsuperior iliac spine and the symphysis pubis). On occluding the ring and making the patient cough, oblique hernia is controlled but a direct hernia is not.

  Procedure

  – Occlude the internal ring with a thumb. In case of a large hernia, 2 fingers may be used instead of only thumb, to ascertain occlusion of the ring
  – If the hernia is big, the patient may be asked to cough while still in the lying down position
  – If the hernia is small, then ask the patient to stand while maintaining the occlusion, and then ask the patient to cough.

Free ebooks ==> www.Ebook777.com

Observation and Inference

1. Hernia 'controlled' i.e. no hernial swelling visible on coughing ⇒ oblique hernia.
   - Release the occlusion, and ask the patient to cough again. Appearance of the swelling confirms oblique inguinal hernia. If the hernia is funicular or complete, then the swelling actually exhibits an oblique path of its travel from the groin to the scrotum
   - One more clinical method may be applied to reconfirm oblique hernia (This clinical method has not been described anywhere else, but I have been practicing it, and it is worth trying): Reduce the hernia. Keep the tips/pulps of 4 fingers close together on the inguinal canal in a slightly oblique orientation matching to that of the canal, and also such that the little finger lies laterally and the index finger lies medially. Ask the patient to cough. A wave produced by the hernia bulging in the groin may be appreciated by the little finger first, successively followed by the ring finger, middle finger, and the index finger at the end, as the hernia travels from the groin towards the scrotum.
2. Hernia 'not controlled' ⇒
   - Direct inguinal hernia
   - Femoral hernia
   - Very rarely, obturator hernia—practically, clinically can not be distinguished from a femoral hernia.

   A direct hernia classically 'pops out' in the region of the external inguinal ring.

Fallacies

1. In c/o a 'pantaloon' (*syns.* 'dual', 'saddle-bag') hernia, the direct component will not be controlled. The oblique component will be controlled and consequently missed, leading to a true recurrence after surgery. Although pathologic anatomy describes a saddle-bag hernia as having 2 limbs/sacs saddled on the inferior epigastric artery, clinically, 2 different sacs cannot be distinguished separately; they produce single hernial swelling. Hence, during the surgery of a direct hernia, possibility of the presence of an indirect sac must be ruled out. There are 2 ways to do this. (a) The spermatic cord is examined after splitting the cremasteric and internal spermatic fasciae. (b) The direct hernial sac may be opened, and a finger passed inside and advanced laterally to explore the internal ring from within.
2. Medial displacement of the internal inguinal ring may occur in a long-standing huge hernia (in ~ 2–3 years time), due to hooking by visceral contents of the hernia over the medial margin of the ring. In this situation, pressure over the 'normal anatomical surface marking' will fail to occlude the internal ring; consequently, a hernia will not be controlled even if it is of oblique type.
3. Too big an internal inguinal ring: This possibility again can occur in a long-standing huge hernia. In this situation, the thumb will not be able to occlude the internal inguinal ring completely, thereby failing to control the hernia even if it is of oblique type.

Conclusion

1. Hernia controlled ⇒ oblique hernia.
2. Hernia not controlled ⇒
   - Direct hernia
   - Pantaloon (dual = saddle-bag) hernia
   - Long-standing huge oblique hernia.

Controversy

- Correct surface marking of the internal inguinal ring: 'Mid-inguinal point' OR 'inguinal mid-point'?
- Mid-inguinal point lies slightly medial to the inguinal midpoint.

As a long-standing huge hernia may displace the internal ring medially, the chance of getting a fallacious result will reduce if mid-inguinal point is considered as the correct surface marking of the internal ring. This is a practical philosophy to circumvent the controversial issue.

- **Saphenous opening occlusion test**
  - Applicable to a reducible femoral swelling.

  Purpose: To confirm a femoral hernia.

www.Ebook777.com

Prerequisite

Hernia must be reduced. There is no indication to do this test in a hernia that is not reducible.

Principle

The saphenous opening (*syn*. fossa ovalis femoris) is an opening in the deep fascia of the thigh, through which a femoral hernia emerges in the thigh to become clinically detectable. The center of this opening is situated 3 cm inferolateral to the pubic tubercle. On occluding the opening and making the patient cough, the femoral hernia is controlled.

Procedure

- Occlude the saphenous opening with a thumb. In case of a large hernia, 2 fingers may be used instead of only thumb, to ascertain occlusion of the opening
- If the hernia is big, the patient may be asked to cough while still in the lying down position
- If the hernia is small, then ask the patient to stand while maintaining the occlusion, and then ask the patient to cough.

Observation and inference

1. Hernia 'controlled' i.e. no hernial swelling visible on coughing ⇒
   - Femoral hernia
   - Very rarely, obturator hernia—practically, clinically cannot be distinguished from a femoral hernia.

   Release the occlusion, and ask the patient to cough again. Appearance of the swelling confirms the femoral hernia.
2. Hernia 'not controlled' ⇒ inguinal hernia.

- Examine the **testicles**
- Examine the **spermatic cord**

  Clinical method
  - Preferably palpate the spermatic cords of both sides simultaneously, for comparison
  - Grasp the spermatic cord between the fingers behind and the thumb in front
  - Judge the size (Diameter)
  - Roll the cord such that various longitudinal structures are actively left out of the grip one by one while trying to distinguish each constituent essentially by its consistency. For example, lymphatics have the softest consistency, and the ductus deference feels like a cord. The ductus deference can normally be felt clearly; number of other structures cannot be defined
  - Can a sensation similar to rustling together of two surfaces of silk be appreciated?

  Observation:
  - Is the cord normal OR thickened?
  - Can various constituents of the spermatic cord be distinguished?
  - 'Rolled silk sign' +ve or –ve?

  Inference: Thickening of the spermatic cord and inability to distinguish various constituents of the cord, and +ve 'rolled silk sign' are indirect evidences of an inguinal hernia [in the absence of a demonstrable swelling even on repeated straining at the time of clinical examination of a patient presenting with a h/s/o hernia (this situation is not rare)].
- Examine the **penis and perineum** for obstructive pathology in the lower urinary tract: Phimosis, pinhole external urethral meatus, stricture urethra.

PERCUSSION

- In my opinion, percussion has no practical significance in reducible / irreducible groin swellings
- An ability of percussion to differentiate between a strangulated hernia and acute funiculitis, as described in many books, is an oversimplified application of normal anatomy to a pathological condition
- A strangulated hernia is associated with exudation making the sac tense. As fluid gives a dull note, how will it help to differentiate a strangulated hernia from other conditions? Moreover, percussion in strangulated hernia as well as any other acute painful condition is very tender, hurting the patient, and hence best avoided.

Free ebooks ==> www.Ebook777.com

AUSCULTATION

– Auscultation of peristaltic sounds confirms the diagnosis of enterocele. The absence of peristaltic sounds however, does not rule out the presence of a hernia
– Sometimes a venous hum may be heard over a saphena varix.

EXAMINATION OF THE ABDOMEN

Particularly look for the following:

- **A scar** of any surgery in the lower quadrant?
- **Another concurrent hernia**? e.g. Umbilical / paraumbilical / epigastric, etc.
- **Tone of the anterolateral muscles of the abdominal wall**?
  – Ask the patient to simultaneously raise both the lower limbs straight (without bending at the knees) at least through ~ 45°. And palpate the abdominal wall to judge (1) tone of the muscles, and (2) defect (weakness) in the posterior wall
  – Head-raising instead of straight leg raising makes the test invalid, as raising the head leads to contraction of only the rectii and not of the oblique muscles.
- Examine the **back** to rule out dermatitis and disorder of the spine, both of which may contraindicate spinal anesthesia for the surgery if indicated.

DRE whenever indicated

– Rule out obstructive anorectal disorder, and enlarged prostate, both of which can predispose the development of a hernia
– In children, bimanual palpation may help to detect a bladder stone.

In GENERAL EXAMINATION

– *Particularly look for* varicosity of the long saphenous vein if the swelling in the groin seems to be a saphena varix.

In SYSTEMIC EXAMINATION

– *Particularly look for* chronic bronchitis.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. From the point of view of pathologic anatomy, inguinoscrotal hernia (i.e. a funicular and complete type of inguinal hernia), varicocele and lymph varix—all 3 are virtually inguinoscrotal swellings. However, presenting chief complaint of an inguinoscrotal hernia is different from that of varicocele and lymph varix.
   – A patient with an inguinoscrotal hernia presents as ‘a swelling in the groin’, whereas a patient with varicocele or lymph varix presents as ‘a swelling in the scrotum’. Varicocele and lymph varix basically being varicosities, blood or lymph respectively collects in the scrotal portion of the vessels due to gravity (in upright posture) to produce a scrotal swelling
   – The inguinal portion of these swellings is neither visible nor palpable as pressure of the fluid (blood/lymph) within these varicosed vessels is low, and moreover the inguinal portion of these vessels is covered by tough aponeurosis
   – The same argument is applicable to a congenital hydrocele as well. Parents of a child with a congenital hydrocele seek medical consultation for ‘a swelling in his scrotum’ even though from pathologic anatomy point of view it is an inguinoscrotal swelling.

www.Ebook777.com

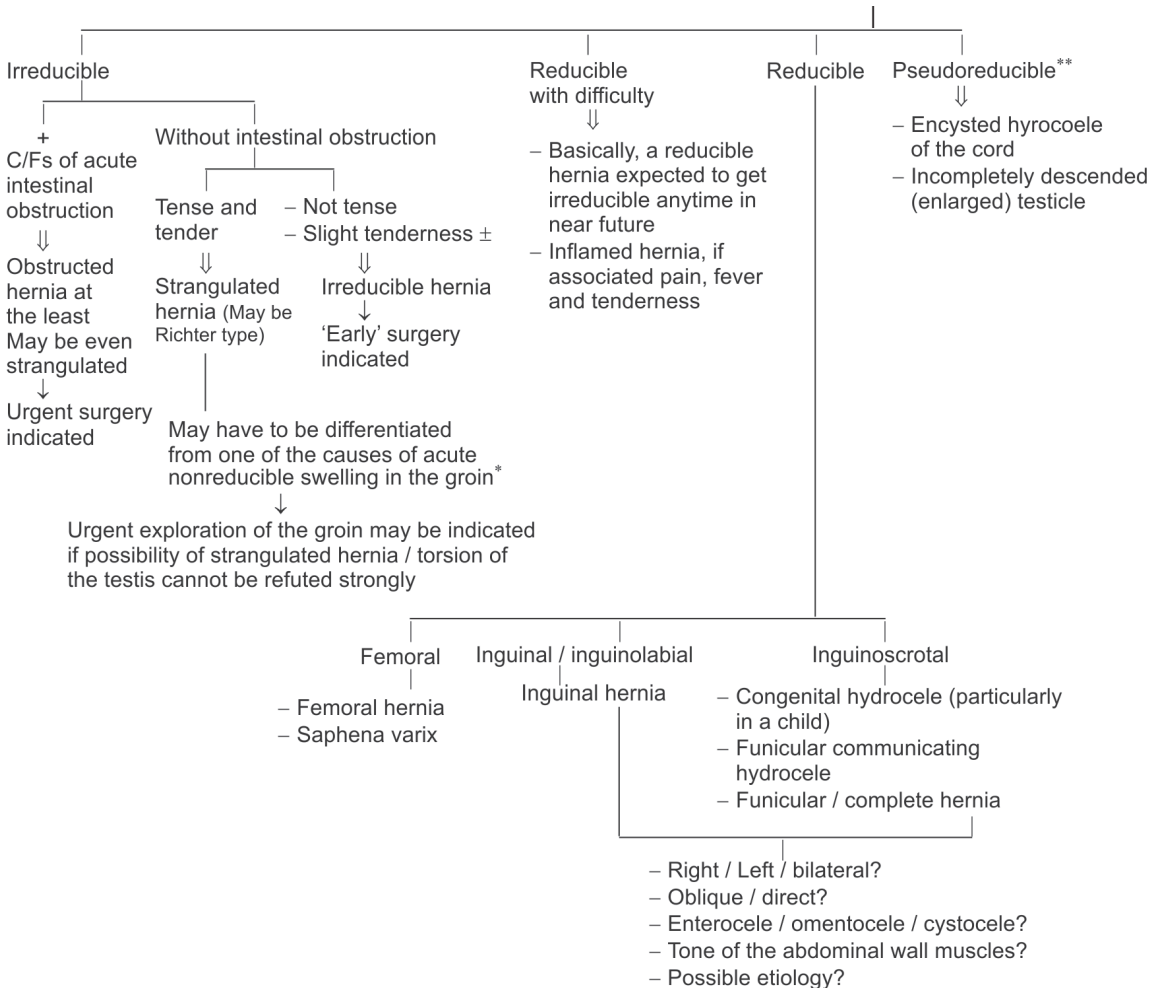
2. If a swelling lies at one of the hernial 'sites', then diagnosis of hernia is assumed unless there is specific clinical evidence in favor of some other disorder.
3. Hernia may be bilateral. Direct hernias are often bilateral, though one may be bigger than the other.
4. About 5% of inguinal hernias are associated with tunica vaginalis hydrocele of the same side.
5. There are no clinical findings that are pathognomonic of a sliding hernia, but it should be suspected in every large globular inguinoscrotal hernia.
6. Painful hernia may be caused by peritonitis! Irritant fluid in the peritoneal cavity may trickle in the hernial sac → painful hernia. If the 'case' is not taken in proper details, then the primary disease may be completely missed and the hernia operated on a mistaken diagnosis of 'strangulated hernia'.
7. A swelling exhibiting impulse-on-coughing *need not necessarily be reducible*.
8. 'Pseudoreducibility' = On applying (gentle) pressure, the swelling *seems to* disappear mimicking reducibility. In fact, there is bodily displacement of the whole swelling under the external oblique aponeurosis. The swelling will not first decrease in size before finally disappearing, but instead, move bodily and disappear in one stroke.
9. Indirect evidences of a congenital hernia in a child who presents with a h/s/o a reducible inguinal hernia but on physical examination there is no visible swelling:
   a. Thickening of the spermatic cord and inability to distinguish various constituents of the cord.
   b. +ve 'rolled silk sign'.
   c. Enlarged external inguinal ring.
10. The term 'incarcerated hernia' is not synonymous with 'obstructed hernia'. Incarcerated hernia means lumen of the portion of the colon occupying a hernial sac is blocked with feces. Scybalous contents of the bowel should be capable of being indented with finger.
11. A large femoral hernia may be mistaken as an inguinal hernia because as a femoral hernia progresses, it finally comes to overlie the inguinal canal. Hence, the importance of examining the relation of the swelling to the pubic tubercle at the outset. Moreover, such a femoral hernia is classically retort-shaped. With careful history-taking and physical examination, it should be possible to avoid a misdiagnosis.
12. Clinically, it may not be possible to differentiate enlarged femoral lymph node from a femoral hernia. The clinical test of mobility may be helpful.
13. A femoral hernia is more likely to get complicated because of narrow neck, rigidity of the femoral ring, and a tortuous path that it has to pursue as it enlarges.
14. A clinically nonreducible soft lobular swelling in the region of saphenous opening (3 cm inferolateral to pubic tubercle) may easily tempt one to give a diagnosis of lipoma. But, such a swelling means almost certainly a femoral hernia!

    A femoral hernia frequently develops a large pad of extraperitoneal fat when it reaches the thigh. The sac in such a femoral hernia is probably only a small fraction of the whole mass, at the root of the swelling. It is the large pad of fat that prevents the impulse on coughing from reaching the clinician's hand, and also prevents reduction of the hernia. Neither irreducibility nor the absence of a cough impulse in themselves suggests strangulation of a femoral hernia.
15. Endometriosis in a hernial sac may be mistaken for an incarcerated hernia, as may malignant tumors in the groin.
16. The Spigelian defect is usually quite small and, if it lies in the upper part of the region, is easily distinguished from the external inguinal ring; the lower it lies however, the more likely is confusion to arise.
17. Sometimes it may not be possible to distinguish between direct and oblique inguinal hernia clinically, in spite of careful examination, for the results of certain clinical tests may not be convincing. In such a situation, I have observed that if the hernia is not readily reduced on lying down and readily apparent on coughing, it will most likely turn out to be an oblique inguinal hernia at the time of operation.

Free ebooks ==> www.Ebook777.com

STEPWISE ANALYSIS OF DATA

Is the swelling **irreducible / reducible / pseudoreducible**?



* *DD more specifically applicable to a strangulated femoral hernia*
- Acute femoral lymphadenitis
- Torsion of an *ectopic* testis
- May be psoas abscess, in the absence of a bilocular nature of the swelling, and the supporting points.

DD more specifically applicable to a strangulated inguinal hernia
- Torsion of an *incompletely descended* testis
- Acute funiculitis
- Hematoma of the spermatic cord
- Acute lymphadenitis
- May be psoas abscess, in the absence of a bilocular nature of the swelling, and the supporting points.

** From the patient's point of view, it is a reducible swelling, as it may be disappearing off and on. However, truly it is not a reducible swelling, as it disappears due to bodily movement, under the cover of external oblique aponeurosis. There is not any transfer of content from one cavity to another. Hence, it is called 'pseudoreducible' or 'displaceable' swelling.

www.Ebook777.com

Femoral hernia v/s saphena varix

Femoral hernia	**Saphena varix**
Reduction not spontaneous. Rather, often not reducible	Reduces spontaneously on lying down
-	It never gets irreducible
-	Almost always associated with varicose veins
On inspection	
Margin—ill-defined	Margin—well-defined
'Retort' shaped	Saccular
On palpation	
Nature of impulse-on-coughing—expansile However, often absent even in an uncomplicated hernia	Thrill-like impulse-on-coughing
Consistency—soft / elastic / doughy	Consistency—highly soft

Congenital hydrocele v/s congenital hernia

Differentiating feature	**Congenital hydrocele in older children (above 2 years)**	**Congenital hernia**
Characteristic history	Gradual reduction of the swelling overnight. Starts filling-up during the day after the child gets up and becomes active (walking, running), and the swelling attains its biggest dimensions by evening	On lying down, reduces instantaneously and completely (completely, when uncomplicated) either spontaneously or on a little manipulation
Shape	Globular	Ovoid
Peristalsis overlying the swelling	Absent	May be visible (from across the skin) if enterocele
Impulse-on-coughing / crying	Vague	Expansile
Consistency	Soft or firm, depending on tension of fluid within the sac	– 'Elastic' if enterocele – 'Doughy' if omentocele
Transillumination	Brilliantly translucent	An enterocele may be translucent only in neonates/infants; but not as brilliantly as a hydrocele
Reducibility and reappearance	Does not reduce instantaneously (due to inverted ink-bottle effect*). Also, does not reappear instantaneously	On lying down, reduces instantaneously and completely either spontaneously or on a little manipulation. An enterocele may make gurgling noise during reduction. Re-appears instantaneously after some active maneuver like standing, crying or coughing

* 'Inverted ink-bottle effect': Imagine a flexible bottle with a narrow mouth and a long narrow neck. After inverting and then pressing on the fundus of such a bottle a tubular collar will develop around the neck which will accommodate some fluid and compress the neck of the bottle itself from outside. More the pressure applied more the compression.

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

Differentiating feature	**Congenital hydrocele in older children (above 2 years)**	**Congenital hernia**
Internal inguinal ring occlusion test	Not applicable	Result: The hernia is 'controlled'
Peristaltic sounds on auscultation of the swelling	Absent	– Presence definitely suggests enterocele – Absence usually suggests omentocele, though enterocele cannot be ruled out
Risk of complication	No; in fact not applicable	Present, such as obstruction/ strangulation

Congenital hydrocele in infants

– Unilateral or bilateral
– Often large, usually lax, and nearly always asymptomatic.

They have **a strong tendency to close down spontaneously**. Virtually all will have disappeared by the age of 1 year and surgery is only required if the hydrocele persists beyond 2 years of age.

Differentiating points of practical importance between oblique and (common type of) direct hernia

Differentiating feature	**Oblique inguinal hernia**	**Direct inguinal hernia (the usual type)**
Age	Any	More common in old age
Site and extent	Inguinal or inguinolabial or inguinoscrotal	Usually confined to the groin
Impulse-on-coughing	Travels obliquely	The hernia pops out anteriorly
Shape	– Of inguinal hernia: Ovoid – Of inguinoscrotal hernia: Pyriform	Usually hemispherical
Reducibility and reappearance	May not reduce and reappear as easily as a direct hernia	Reduces and reappears easily
On internal ring occlusion test	Hernia is controlled, provided the ring is not widely stretched and / or medially shifted	Not controlled
Risk of complication	+	Almost none

Enterocele v/s omentocele v/s cystocele

Differentiating feature	**Enterocele**	**Omentocele**
Surface	Smooth	Rather granular
Consistency / feel	Firm, elastic	Soft, 'doughy'
On percussion	Resonant note	Dull note
While reduction	The first portion is difficult to reduce Reduces with gurgle	The last portion is difficult to reduce NO gurgle

A cystocele

– A cystocele is essentially a direct inguinal hernia
– Urinary symptoms
– Size of the swelling appears larger just prior to micturition and gets diminished soon after micturition
– Pressure on the swelling induces a desire for micturition especially when the swelling is in a distended state
– Micturition is greatly helped if the hernia is pressed upon during the act.

www.Ebook777.com

Etiology of the hernia?

A. Congenital
B. Familial
C. Acquired

Predisposing factors

Poor muscle tone due to
(1) Nerve injury:
- Accidental
- Iatrogenic as during appendicectomy, kidney opearation, etc.

(2) Poliomyelitis rarely

Exciting factors

Raised intra-abdominal pressure either continuously or intermittently due to
(1) Occupation involving strenuous work
(2) 'Chronic' constipation
- Habitual: due predominantly to nonvegetarian diet / use of European type of commode for defecation / sedentary life-style, etc.
- Pathological e.g. due to anorectal stricture, etc.

(3) 'Chronic' straining-type of difficulty in micturition.—due to enlarged prostate (old age) / phimosis of 'long-duration' / pinhole meatus / stricture urethra / bladder stone
(4) A (big) lump in the abdomen
(5) Ascites
(6) Chronic bronchitis

Associated disorder?

DIFFERENTIAL DIAGNOSIS

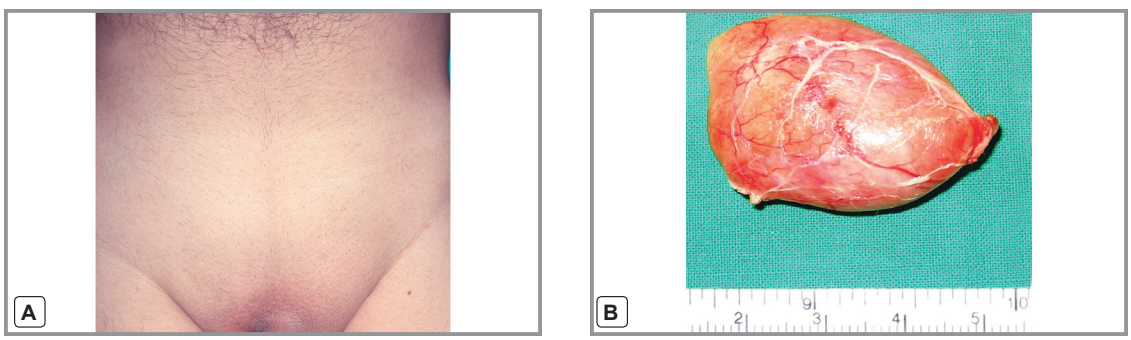
Encysted hydrocele of the cord

- Well-defined, globular (spherical / ovoid)
- No impulse-on-coughing
- Possible to 'get above'
- Traction test +ve
- (Brilliantly) Translucent.

Pathologic anatomy

A segment/portion of the processus vaginalis remains patent, being shut-off from the tunica vaginalis as well as on the proximal side at a variable point (and will never communicate with the peritoneal cavity). Distension of the patent segment with fluid → a hydrocele, called 'encysted hydrocele of the cord. ' Cord' means the fibrous cord resulting from obliteration of the processus vaginalis. 'Cord' in this context does not mean spermatic cord.

→ an inguinal, inguinoscrotal, or a scrotal swelling, depending on which segment of the processus remains patent and the length of the patent segment.



Figs 23-1A and B (A) The swelling was palpable, but not visible at the time of taking this photograph as it had slipped under the cover of the external oblique aponeurosis, **(B)** Excised specimen of the encysted hydrocele

Free ebooks ==> www.Ebook777.com

Obstructed inguinal hernia (Obstructed inguinal enterocele)

Basically

- An inguinal swelling, or an inguinoscrotal swelling superomedial to the pubic tubercle
- Irreducible
- Previous h/o one or more recurrent episodes of irreducibility ±.

Differentiating points

- Clinical features of (acute) intestinal obstruction
- Impulse-on-coughing absent
- The hernial swelling may be tender, but not very tense.

Strangulated inguinal enterocele

In addition to the clinical features of obstructed inguinal hernia

- Pain in the abdomen becomes constant, diffuse and severe
- Severe pain develops in the hernial swelling
- Prostration, thirst, pallor
- Tachycardia
- Impulse-on-coughing absent
- Tense and tender (due to exudation).

Acute funiculitis may have to be differentiated from a strangulated hernia. Refer to page 306.

Obstructed femoral hernia (Obstructed femoral enterocele)

Basically, an irreducible femoral swelling (– inferolateral to pubic tubercle)

Differentiating points

- Clinical features of (acute) intestinal obstruction
- Shape of the swelling essentially depends on its size. A small hernia may be hemispherical whereas a larger hernia will be ovoid or characteristically retort shaped
- Impulse-on-coughing absent
- The swelling may be tender, but not tense.

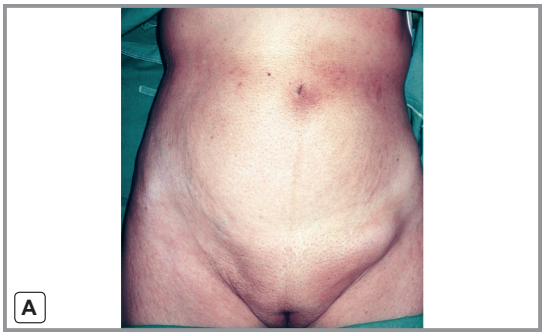
Strangulated femoral enterocele

In addition to the clinical features of obstructed femoral hernia

- Pain in the abdomen becomes constant, diffuse and severe
- Severe pain develops in the hernial swelling
- Prostration, thirst, pallor
- Tachycardia
- Shape of the swelling essentially depends on its size. A small hernia may be hemispherical whereas a larger hernia will be ovoid or characteristically retort shaped
- Impulse-on-coughing absent
- Tense and tender (due to exudation).

Strangulated omentocele

Basically, an irreducible inguinal, inguinoscrotal or a femoral swelling.







Figs 23-2A to C (A) The swelling can be easily confused with an inguinal swelling, if relation of the swelling to the pubic tubercle is not satisfactorily established. This was a femoral swelling. **(B)** Feculent NG tube aspirate, **(C)** The strangulated loop of intestine as seen during surgery

www.Ebook777.com

Differentiating points
- No e/o intestinal obstruction
- Severe pain develops in the hernial swelling
- In case of an inguinal hernia, infection of the strangulated omentum usually terminates as a scrotal abscess
- Patient looks distressed
- Tachycardia ±
- Impulse-on-coughing absent
- The swelling becomes tender and tense, but not as tense as a strangulated enterocele
- Doughy consistency may be appreciated.

Notes
- Unlike intestine, omentum can exist on a meager blood supply. The onset of necrosis is correspondingly delayed
- Infection is limited to the sac for days, and sometimes for weeks.

(Strangulated) Richter's femoral hernia
- = Only a part (a knuckle) of the wall of the intestine is herniated which gets strangulated
- Symptoms of intestinal obstruction or collapse are slight. On the contrary, patient may initially have loose motions due to irritation of the intestine. Absolute constipation is delayed until paralytic ileus supervenes
- Common in a femoral hernia and laparoscopic port site hernia
- Severe pain in the region of the swelling (groin in c/o a femoral hernia)
- No impulse-on-coughing
- Tender and tense
- Irreducible.

Irreducible inguinal hernia
Basically, an inguinal swelling, or an inguinolabial/inguinoscrotal swelling superomedial to pubic tubercle.

Differentiating points
- Impulse-on-coughing—*present*, in c/o enterocele; absent, in c/o omentocele
- Irreducible
- Not tense.

Additional clinical features which may be present
- Mild to moderate pain
- Constipation
- Slight tenderness.



Fig 23-3 This hernia was present for 49 years

Free ebooks ==> www.Ebook777.com

Causes of irreducibility of a hernia

Irreducibility of 'short' duration (few hours)

Due to increase in volume of the contents of the sac

Irreducibility of 'long' duration (weeks) / frequently recurrent episodes of irreducibility (over months)

Bedside reduction may be attempted by ~ 30° head-low, sedation and gentle taxis, provided obstruction / strangulation has been ruled out

(a) By virtue of mere size
Example—very large scrotal hernia. As a large portion of the viscera is used to remain 'outside the abdomen, in the hernial sac', the abdomen fails to accommodate the herniated contents back if attempts are made to reduce the hernia
(b) By virtue of the type: Sliding hernia
(c) Due to fibrosis
- Adhesions of contents to the sac
- Adhesions of contents to each other
- Narrowing of the sac at the external ring or at the neck of the sac
- Fibrosis at the site of pressure of truss (if the patient has been using one)

(d) Incarceration = impaction of feces in the lumen of herniated segment of large intestine

Inflamed hernia
- Pain and fever
- Overlying skin—red, edematous
- The swelling is tender, but may not be tense
- Impulse-on-coughing *present*
- Reduction may be difficult.

Causes: Appendicitis, salpingitis, Meckel's diverticulitis, a sore caused by an ill-fitting truss.

Essentially uncomplicated femoral hernia
- Practically, an irreducible femoral hernia may be considered 'otherwise' uncomplicated, because a femoral hernia is often irreducible. So mush so, that impulse-on-coughing may be absent even if the hernia is not complicated by obstruction or strangulation
- *Basically*, a femoral swelling (– inferolateral to pubic tubercle.).

Differentiating points
- Often ill-defined
- Shape of the swelling essentially depends on its size. A small hernia may be hemispherical whereas a larger hernia will be ovoid or characteristically retort shaped
- Expansile impulse-on-coughing ±, however, often absent
- Consistency—soft / elastic / doughy
- Reducible or irreducible; however, often irreducible.

Supporting point: Elderly woman.
A note: A small femoral hernia may be difficult to diagnose especially in obese women. The swelling may not be easily palpable and the cough impulse difficult to elicit.

Saphena varix
Basically, a reducible swelling (~ 3 cm) inferolateral to the pubic tubercle.

Differentiating points
- Very soft
- Disappears on lying down, without any manipulation
- Thrill-like impulse-on-coughing.

www.Ebook777.com



Fig 23-4 Saphena varix

Supporting points

– Blue color visible through the skin
– Associated presence of varicosity of the long saphenous vein the connection of which with the saphena varix can be demonstrated by Schwartz's test
– Sometimes, a venous hum may be heard on auscultation.

Mechanism of impulse-on-coughing in saphena varix: Coughing → increase in intrathoracic pressure → hampering of venous return through major veins → back pressure wave in the whole venous system—detected as an impulse.

Congenital hydrocele and congenital hernia

Basically, the presentation is with a reducible scrotal swelling in a young boy; sometimes may be first noticed even soon after birth.

– On physical examination, they are found not to be 'purely scrotal' but instead, inguinoscrotal
– Funicular variety: The fundus of the sac stops (ends) short of the upper pole of the testis. Testis can be palpated separately
– Complete variety: The sac communicates with the tunica vaginalis so that the fluid in case of hydrocele or the contents in case of a hernia practically engulf the testis (strictly excepting on the posterior aspect) so that the testis cannot be separately palpated.

For *differentiating points*, refer to the table above.

The basic pathologic anatomy of both the congenital hydrocele and hernia is essentially the same except for the following:

– When the neck of the patent processus vaginalis (i.e. the sac) is too narrow to admit viscera, but can only allow trickling of intraperitoneal fluid, a congenital hydrocele results. A congenital hernia results when the neck of the patent processus vaginalis is wide enough to allow descent of viscera
– A congenital hernia need not necessarily present only during childhood, but may present during adolescence/early young adulthood. Explanation: Presumably, increased abdominal pressure can stretch the fascial margins of the internal inguinal ring and open up a preformed peritoneal sac
– In bilateral congenital hydroceles, ascites or ascitic tuberculous peritonitis should be suspected.

Indirect evidences of congenital hernia in a child who presents with a h/s/o a reducible inguinal hernia but during consultation the swelling somehow refuses to make its appearance:

a. Thickening of the spermatic cord and inability to distinguish various constituents of the cord.
b. +ve 'rolled silk sign'.
c. Enlarged external inguinal ring.

Funicular communicating hydrocele

– The pathologic anatomy is same in both the congenital funicular hernia, and the funicular communicating hydrocele except for the size of the mouth (i.e. the opening at the internal inguinal ring)
– In case of a hernia the size of the mouth is big enough to admit an intra-abdominal viscus
– In case of a hydrocele the mouth is narrow and allows only tricking of fluid as in a congenital hydrocele.

Free ebooks ==> www.Ebook777.com



Fig 23-5 Oblique funicular inguinal hernia



Fig 23-6 Inguinolabial hernia

Uncomplicated oblique inguinal hernia

Basically, an inguinal swelling, or an inguinolabial / inguinoscrotal swelling superomedial to pubic tubercle.

Differentiating points

– The presence of expansile impulse-on-coughing
– Reducibility.

Supporting point: Widened external inguinal ring. Refer to the table on page 294 for distinguishing points between enterocele and omentocele.

Notes

1. *Indirect evidences of hernia, in the absence of an obvious swelling:*
   – In women, pain may be the only symptom in small non-palpable hernia
   – Pain at the hernial site on coughing
   – 'Rolled silk' sign.
2. The single most important pathognomonic feature of a hernia—reduction with gurgling. (This fact relates only to enterocele; not to omentocele).
3. *Risk factors for the development of complications in groin hernia:*
   – In adults:
     - Old age
     - Hernia of relatively 'short duration' (< 1 year)
     - Femoral > oblique inguinal > direct inguinal
     - Coexisting COAD
   – In children:
     - Very young. Infants
     - Male gender
     - Right-sided hernia
   – In general, any hernia with small neck and / or irreducibility predisposes to obstruction.
4. Sliding hernia and interstitial hernia are rare types of inguinal hernia.

Sliding hernia

– Part of the sac wall is formed by prolapsed viscus and for this reason these hernias are always large
– The condition can be suspected but not confirmed preoperatively on clinical examination. Possibility of sliding variety should be suspected in every large globular inguinoscrotal hernia.

Supporting points

– Age—nearly always > 40. Incidence rises with increasing age
– Occurs almost exclusively in males
– Left-side is more commonly affected.

Bilateral sliding hernias are exceedingly rare.

www.Ebook777.com

Interstitial hernia

- → inguinal rather than inguinoscrotal swelling
- Often associated with ectopic testis
- *Pathologic* anatomy: The sac lies in between different layers of the abdominal wall.

Types of interstitial hernia

a. Properitoneal: The sac lies between the transversalis fascia and the peritoneum.
b. Interparietal: The sac lies between the internal oblique and the external oblique muscles.
c. Extraparietal: The sac lies superficial to the external oblique muscle.



Fig 23-7 Bilateral direct inguinal herniae

(Common type of) Direct inguinal hernia

Basically, a hemispherical reducible inguinal swelling with expansile impulse-on-coughing.

Differentiating points

- Characteristically pops out anteriorly on standing / coughing
- Reduces and reappears easily
- Not controlled on internal inguinal ring occlusion test.

Notes

- Common after middle age
- Frequently bilateral.

Uncomplicated Busoga type of direct inguinal hernia

- Rare
- Busoga is an area in Uganda
- Pathologic anatomy: A narrow defect in the conjoint tendon or transversalis fascia

Basically, an inguinal or inguinoscrotal reducible swelling with expansile impulse-on-coughing.
Differentiating point: The swelling is not controlled on internal inguinal ring occlusion test.

Free ebooks ==> www.Ebook777.com

24 A NON-REDUCIBLE SWELLING IN THE GROIN

Definitions

1. *Groin* = an oblique strip of area 3 cm above + 3 cm below and parallel to the inguinal ligament.
2. '*Non-reducible swelling*' = a swelling which never disappeared (i.e. never got reduced).

GENERAL REMARKS

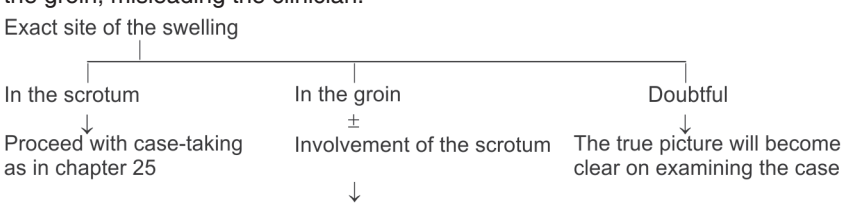
1. Before declaring a swelling as 'non-reducible', make sure you are not dealing with an 'irreducible swelling'. Occasionally, patients do present with h/o an acute/subacute non-reducible swelling when they are actually having an irreducible hernia which they had never noticed earlier in its reducible state.

2. Pubic tubercle is such an important landmark that the clinician must satisfy herself/himself by finding it rapidly and accurately, even in a fat or uncooperative patient. For clinical methods, refer to page 277.

HISTORY

- **Residence**?
  - Is filariasis common in the area of your residence?
  - Funiculitis and lymph varix of the cord are more common in the areas where filariasis is endemic.
- **Duration**?
- **Constancy**
  - Is the swelling continuously present?
    OR
  - Does it periodically recur, particularly in association with fever with chills? ⇒ filarial funiculitis.
- **Mode of onset**?
  - Examples of swellings which appear suddenly: Torsion of the testis, acute funiculitis, and acute hematoma of the cord.
- **Exact site**?

  Verification of the exact site is important as a patient with a scrotal swelling, out of shyness, may point to the groin, misleading the clinician.



  - **The site of commencement and the direction of subsequent enlargement?**
    - Did the swelling commence in the scrotum and subsequently enlarged towards the groin?
      OR
    - Did it commence in the groin and subsequently enlarged towards the scrotum?

  Examples of swellings which commence in the scrotum and subsequently enlarge towards the groin: Infantile hydrocele, malignant tumor of the testis.

www.Ebook777.com

- Is it **pulsatile**?
- **Progress**?
- **Pain**?
  - Exact site?
  - Intensity?
  - Nature: Throbbing / shooting / dragging, etc.?
  - Precipitating, Aggravating, Relieving factors?
    - Pain on exertion and/or at night, + stiffness, both leading to a limp ⇒ arthritis of the hip/SI joint (TB or nonspecific) leading to psoas bursitis
    - Disability (resulting from losing movements, muscle spasm, fibrosis of the joint capsule and bony changes in the joint) ⇒ OA with psoas bursitis
    - Backache **even if mild** may be the earliest and the only symptom of tuberculous osteomyelitis of the spine
- **Fever with chills**?
  ⇒ acute funiculitis (mostly filarial) / acute pyogenic abscess
  - Periodic attacks of fever ⇒ filarisasis
- Low grade fever especially in the evening + loss of appetite + (significant) loss of weight? ⇒ TB
  Examples:
  - Inguinal/femoral tuberculous chronic lymphadenitis
  - Cold abscess resulting from tuberculous arthritis of hip / SI joint
  - Psoas abscess
  - TB spine.
- Significant loss of weight? ⇒ TB, malignancy, AIDS
- **Any other swelling of similar nature in the same region or elsewhere**?
  For example, a lump in the iliac fossa may suggest psoas abscess.
- Symptoms s/o **arterial deficit** in the lower limb?
  - ⇒ compression or occlusion of the femoral artery. Occlusion can be a complication of aneurysm.
  - Paresis / paralysis
  - Coldness and deadness of the limb
  - Acute spreading ulcer with cellulitis
  - Blackening of one / more digits or more extensive area (s/o gangrene)

  } ⇒ Acute arterial insufficiency

  - Raynaud's phenomenon
  - Chronic indolent ulcer
  - Blackening of one / more digits or more extensive area (s/o gangrene)

  } ⇒ Chronic arterial insufficiency
- **Edema** of lower limb?
  Edema may suggest compression of the femoral vein.
- H/o **trauma**?
  Trauma may → – Hematoma of the spermatic cord
  – False aneurysm of femoral artery
  – AV fistula.
- H/o **septic wound / chronic ulcer on the foot / leg**?
  ⇒ Lymphadenitis.

LOCO-REGIONAL EXAMINATION

EXPOSURE of the patient: From the umbilicus downwards, including both groins and private parts.

POSITION of the patient
Standing with legs apart. Ask the patient to cough a couple of times to rule out a possibility of reducible swelling. Then ask the patient to lie down.

Inspection
- Number, site and extent?
- Margin, shape, size?

Free ebooks ==> www.Ebook777.com

- Pulsations, impulse-on-coughing?
- Surface, skin, scar?
- Dilated veins, color of the swelling?
- Surrounding area?
- External genitals?
- Pressure effects?

Palpation

- Number, site and extent?
- Relation to the pubic tubercle?
- Relation to femoral pulse?
- Temperature, tenderness?
- Margin, shape, size?
- Thrill, pulsations?
- Getting above: Practically it will not be possible to 'get above' inguinoscrotal swellings
- Traction test (if indicated e.g. if encysted hydrocele of the cord is suspected)
- Mobility? Test the mobility of the **root** of the clinically palpable swelling. This test may be useful to differentiate a hernia from other swellings. 'Root of the hernia' (maximally proximal palpable portion of a hernial swelling) is immobile
- Anatomical plane and fixity?
- Surface, consistency, fluctuation, transillumination?
- Moulding, compressibility, reducibility?
- Testicle, spermatic cord, penis and perineum?

Percussion

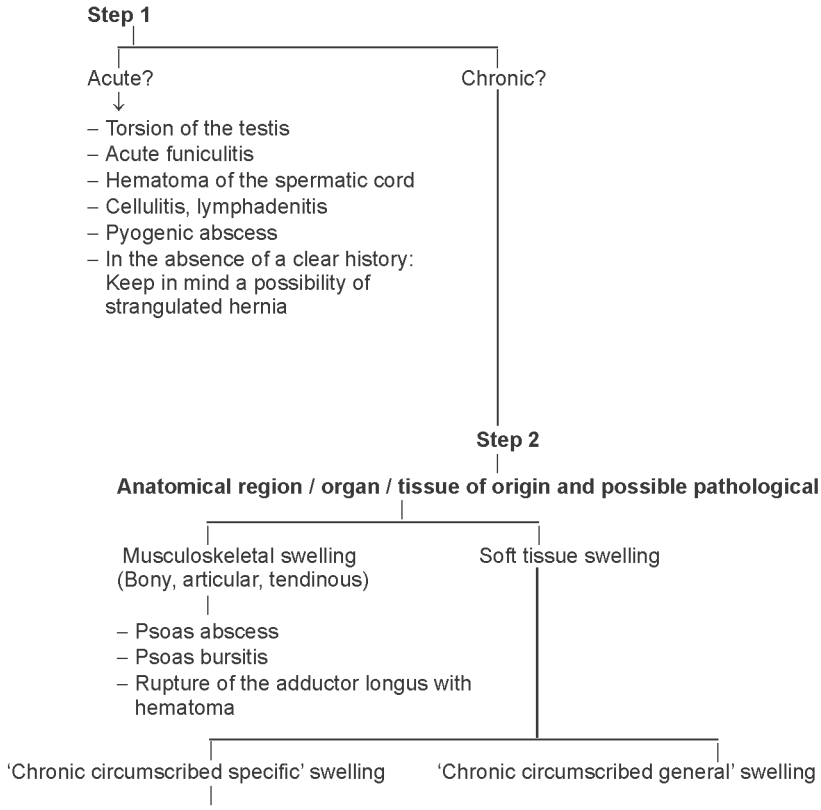
Auscultation

Examine the drainage area of the groin lymph nodes

- Routinely, umbilicus to toes
- Rarely, breasts.

www.Ebook777.com

Stepwise analysis of data



– Vascular swelling related to the femoral vessels: Aneurysm, AVF
– Hydrocele:
  - Encysted hydrocele of the cord
  - Hydrocele of the canal of nuck
  - Infantile hydrocele
  - Bilocular hydrocele
  - Hydrocele of the hernial sac
– Benign tumor e.g. lipoma of the cord
– Malignant tumor:
  - Malignant lymphadenopathy
  - Testicular tumor extending up the inguinal canal
– Chronic inflammation:
  - Inflammatory thickening of the cord
  - Chronic lymphadenitis
  - Cold abscess resulting from tuberculous arthritis of hip / SI joint
  - Psoas abscess
  - Psoas bursitis
– Miscellaneous:
  - Ectopic testis
  - Incompletely descended enlarged testis in the inguinal canal
  - Lymphadenovarix

A. ***Specific*** *swellings superomedial to the pubic tubercle i.e. inguinoscrotal swellings*

– Hydrocele: Encysted hydrocele of the cord, hydrocele of the canal of Nuck, infantile hydrocele, bilocular hydrocele
– Benign tumor e.g. lipoma of the cord
– Testicular tumor extending up the inguinal canal
– Inflammatory thickening of the cord
– Incompletely descended enlarged testis in the inguinal canal.

Free ebooks ==> www.Ebook777.com

B. ***Specific*** *swellings inferolateral to the pubic tubercle i.e. in the upper part of the femoral triangle*
- Aneurysm of the femoral artery
- Psoas abscess
- Psoas bursitis
- Femoral lymphadenopathy
- Femoral hernia.

Step 3: Complication?
Step 4: Etiology?
Step 5: Associated disorder?

DIFFERENTIAL DIAGNOSIS

Torsion of an incompletely descended testis
- Extremely painful, tender and tense inguinal swelling
- Slight fever ±.

Notes
- There may be vague pain in the lower quadrant of abdomen—the 'referred pain'
- H/o injury to the scrotum / groin ±, but this is characteristically minor / vague. It represents an attempt to relate the onset of symptoms, when in reality the minor injury merely draws attention to the already abnormal groin
- *It must be remembered that a hernia associated with an incompletely descended inguinal testis may be strangulated when the clinical diagnosis can become very much confusing*.

Acute funiculitis
- (Periodic attacks of) Fever with chills / rigors associated with painful and tender unilateral / bilateral inguinoscrotal swelling
- Elongated swelling with long longitudinal axis
- The overlying skin may become edematous
- Often associated with epididymo-orchitis
- Suppuration may take place due to secondary infection, and result in an abscess.
- Funiculitis is sometimes preceded by retroperitoneal lymphangitis, and represents its downward extension into the cord.

Funiculitis may need to be differentiated from a strangulated inguinal hernia.
Two signs may be helpful.
1. *K. Das's sign*: Palpate just above the internal inguinal ring.
   - Feeling of abdominal contents as they enter the ring suggests hernia
   - No such content can be felt in funiculitis.
2. S. Datta's sign: Try to fold the spermatic cord upon itself by elevating the scrotum upwards.
   - This is possible in funiculitis
   - Not possible in a strangulated hernia on account of high tension in the obstructed loop.

Pathology
- Acute funiculitis = acute inflammation of the spermatic cord, primarily its lymphatic component i.e. lymphangitis. However, thrombophlebitis is also associated
- The cord becomes congested, edematous and thickened with serous/seropurulent exudate.

Causes:
- Bacterial infection with pyogenic organisms
- Filariasis.

Hematoma (hematocele) of the spermatic cord
- Elongated swelling with long longitudinal axis
- Develops soon following trauma

www.Ebook777.com

– Ill-defined
– Nontranslucent.

Acute iliac lymphadenitis (*Syns.* **Acute iliac adenitis**, **iliac pyogenic abscess**): Refer to page 475.

Acute pyomyositis of iliopsoas (*Syns.* **Iliopsoas pyogenic abscess**, **psoas pyogenic abscess**): Refer to page 475.

Encysted hydrocele of the cord

– Well-defined. Spherical / ovoid
– Getting above may be possible if the swelling can be pulled down sufficiently, and the dimension of the swelling small
– Traction test +ve
– (Brilliantly) Translucent.

Pathologic anatomy

A segment/portion of the processus vaginalis remains patent, being shut off from the tunica vaginalis as well on the proximal side at a variable point (and will never communicate with the peritoneal cavity). Distension of the patent segment with fluid → a hydrocele, called 'encysted hydrocele of the cord. ('Cord' means the fibrous cord resulting from obliteration of the processus vaginalis. 'Cord' in this context does not mean spermatic cord.) → an inguinal, inguinoscrotal, or a scrotal swelling, depending on which segment of the processus remains patent and the length of the patent segment.

Hydrocele of the canal of Nuck

– Occurs in females
– Vague / indefinite impulse-on-coughing
– Brilliantly translucent.

Pathologic anatomy: The processus vaginalis in females also traverses the inguinal canal, and is called the canal of Nuck. It normally obliterates but sometimes may fail to do so and may lead to a hernia, or a hydrocele if its mouth is narrow.

Infantile hydrocele

– *From its name it may appear that it must be occurring only in infants, but that is not true*
– It may extend right up to the internal inguinal ring, and hence basically an inguinoscrotal swelling

  Pathologic anatomy: Processus vaginalis is closed at the internal inguinal ring only, but communicates with the tunica vaginalis. As the fluid in the sac surrounds the testis, it cannot be felt separately.
– May be associated with an inguinal hernia.

Bilocular hydrocele

– Cross fluctuation between the scrotal swelling and the abdominal extension of the swelling.

Pathologic anatomy: A pocket of hydrocele above the root of the scrotum communicates with the tunica vaginalis hydrocele by a patent track of variable size. The superior pocket of hydrocele lies within the abdominal cavity or in the inguinal region (superficial to or partly within the inguinal canal and it may insinuate itself in between the muscle layers).

Hydrocele of the inguinal hernial sac

– Previous h/s/o a hernia ±
– Testis can be palpated separately from the swelling—the fact that distinguishes it from infantile hydrocele
– Translucent.

Pathologic anatomy: Collection of fluid in the hernial sac, the opening at the internal inguinal ring being closed by a tag of omentum.

Hydrocele of the femoral hernial sac

– Previous h/s/o a hernia ±
– Translucent.

Pathologic anatomy: Collection of fluid in the hernial sac, the mouth / neck of the sac being closed by a tag of omentum or adhesions

Possible complications of the above-mentioned non-communicating hydroceles

1. Rupture—usually traumatic, but may possibly be spontaneous. On rare occasions a cure results after absorption of the fluid.
2. Transformation into a hematocele can occur both spontaneously or as a result of trauma.

Free ebooks ==> www.Ebook777.com

3. Hernia of the hydrocele sac may occur in long-standing cases. Tension of fluid → herniation through the dartos muscle.
4. Calcification of the sac may occur in long-standing cases.

Lipoma of the cord

Basically, an inguinoscrotal swelling.

– Lobular
– Soft consistency.

A note

The characteristic differentiating features of a lipoma viz. lobular surface and soft consistency will not be appreciated in case of a lipoma of the cord confined to the inguinal canal, as the swelling lies under the tough external oblique aponeurosis.

Testicular tumor extending up the inguinal canal

The presence of obvious malignant tumor of the testis is the clue.

Inflammatory thickening of the cord

→ general (diffuse) fullness of the cord.

Additional findings will vary according to the primary cause.

1. *If it occurs as an aftermath of attacks of filarial acute funiculitis:*
   – The cord feels nodular, thickened and indurated (around the dead adult worms in the lymphatics).
2. *If it occurs as an aftermath of tuberculous epididymitis:*
   – The ductus deference feels beaded
   – Associated tuberculous epididymitis / epididymo-orchitis (enlarged, nodular and hard ± a tuberculous sinus).

Cold abscess along the spermatic cord resulting from tuberculous arthritis of hip/SI joint.

– Vague impulse-on-coughing
– Consistency—(very) soft
– E/o primary tuberculous disease in the spine.

Tuberculous psoas abscess

Differentiating points

– Subacute course
– H/s/o osteomyelitis of the spine (X-ray changes will be evident)
– Nonspecific symptoms of TB
– Fixed tender lump in the inguinal area (just above the inguinal ligament)
– The mass will extend up to iliac crest, spine, inguinal ligament or in the femoral region
– Cross fluctuation between the two lumps across the inguinal ligament
– Expansile impulse-on-coughing.

Supporting point: Painful fixed flexion deformity at the hip joint.

Psoas bursitis

– A tense swelling beneath the inguinal ligament
– Disappears when the thigh is flexed
– OA of the hip joint.

Ectopic testicle: Refer to page 337.

Incompletely descended enlarged testis in the inguinal canal

As such a testicle is covered by tough external oblique aponeurosis, and also because an incompletely descended testis is usually underdeveloped, an incompletely descended testis in the inguinal canal will present as a swelling only if enlarged i.e. pathological.

– Typical 'testicular sensation' on pressing the swelling **very gently**. However, testicular sensation will be decreased or abolished if the testicular swelling has resulted from a cancer
– Scrotum on the affected side will be empty.

www.Ebook777.com

Notes

– Appreciation of shape, size and consistency may be slightly underestimated as such a testicle is covered by tough external oblique aponeurosis
– An incompletely descended testicle is associated with an oblique inguinal hernia.

Lymphadenovarix

Bilateral swelling in the groin. On lying down the size of the swelling diminishes slowly.

– Mode of onset—insidious
– Painless, except during acute attacks of lymphadenitis
– Patulous
– A vague impulse on coughing
– Surface—lobular
– Consistency—soft, doughy. Nodules of firmer consistency are also felt at places
– Compressible.

Notes

– May be mistaken for a hernia
– Lymphadenovarix is the result of obstruction to the retroperitoneal lymphatics and represents structural adaptation to the proximal obstruction and acts as safety valve and buffer for any tendency to sudden rise of lymphatic pressure
– Removal of these safety valve mechanisms may initiate lymphedema of the limb; hence surgery is best avoided.

***Acute funiculitis v/s inguinal strangulated hernia**, particularly in the absence of a clear history*

– Apart from supporting symptoms and signs, 2 signs may help to differentiate
– K. Das's sign and S. Datta's sign: Explained earlier

An enlarged femoral lymph node v/s femoral hernia

– The difficulty never arises if multiple lymph nodes are enlarged
– The need to distinguish arises only when single femoral lymph node (of Cloquet) is enlarged
– Diagnosis may be impossible unless there is a clue, such as a primary focus of infection in the drainage area (limb / perineum)
  - 'Root' of the femoral hernia is immobile
  - Enlarged lymph node may be wholly mobile.

Free ebooks ==> www.Ebook777.com

25 SCROTAL SWELLING OR SCROTAL PAIN

SCROTAL SWELLING

HISTORY

- **Duration**?
- **Mode of onset**?
- **Progress**, **regress**, **fluctuation** in size of the swelling?
- **Painful**?

At this juncture: Decide whether the swelling is acute or chronic.

Details to be asked in a case of acute painful scrotal swelling

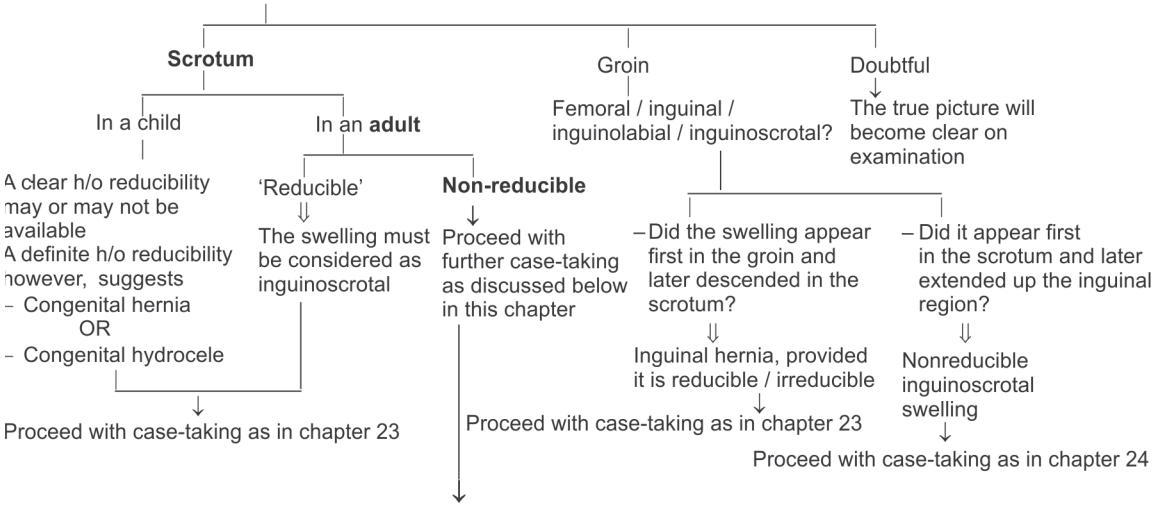
- Was it reducible (earlier / to begin with)?
  A previous h/o reducibility raises a strong possibility of strangulated hernia.
- H/o trauma? Any scratch / bruise?
  Did the swelling appear as well as gain its full size immediately following trauma; and if so, has the swelling been stationary in size ever since?
  Trauma may cause hematocele, or trauma especially mild, may merely draw attention to the pre-existing scrotal disorder.
- Any procedure upon the urinary tract e.g. dilatation of stricture of urethra / I & D of a periurethral abscess, etc?
- Have you been suffering from a bleeding disorder?
- Have you been on any anticoagulant therapy?
- Recent episode of UTI?:
  - Increased frequency of micturition?
  - Pain while passing urine?
  - Passage of red / brown-colored or smoky urine?
- H/s/o gonococcal urethritis in the last 2–3 weeks period?:
  - Itching of the external urethral meatus?
  - Scalding pain throughout the act of micturition?
  - Profuse, thick, yellow-grey colored purulent urethral discharge?
- Did you recently undergo a urethral instrumentation e.g. catheterization / dilatation of a stricture, etc?
- In the recent past, did you anytime pass urine by violent straining while the bladder was full?
- H/s/o poor flow? Poor flow suggests bladder outflow obstruction, one of the causes of which is stricture of urethra
  ↓
  - Intermittency?
  - How is the caliber of stream? Has it reduced than before?
  - Does the stream fall close to the feet rather than away?
  - Terminal dribbling?
  - Do you have to strain to pass urine (by contracting abdominal muscles)?
  - Does straining improve the stream or weaken the stream?
  - Do you take longer than normal time to complete the act of micturition?

www.Ebook777.com

- Did you recently suffer from mumps parotitis / influenza?
- Of late, did you suffer from a few attacks of self-resolving pain in the testicle / groin / lower abdomen?
- Was the swelling preceded immediately by straining at stools / lifting heavy weight / sexual activity, etc?
- Fever?

Details to be asked in a case of chronic scrotal swelling

- **Exact site** of the swelling?



- Have you been getting **recurrent attacks of fever with chills** simultaneously associated with **painful swelling in the inguinoscrotal region**?
- Did you suffer from **pulmonary TB**?
- – Urethral discharge?
  – Mild ache in the perineum?
  – Painful, sometimes blood-stained ejaculations?

  } These symptoms suggest **tuberculous prostatitis**
- – Progressively increasing frequency both by D & N for months?
  – Painless passage of red-brown colored or smoky urine?
  – Vague discomfort / pain in the loin?
  – A lump in the loin?

  } These symptoms suggest **renal tuberculosis**
- Loss of appetite, loss of weight and low-grade evening fever with night sweats?
- Symptoms of 'poor urinary flow'?

} – **The nonspecific constitutional symptoms of TB**.

LOCO-REGIONAL EXAMINATION OF AN ACUTE SCROTAL SWELLING

EXPOSURE: From umbilicus down to both mid-thighs, including both groins and private parts.

INSPECTION

Inspect the **apparently normal side first**.
Inspection of the **abnormal side.**

- Number?
- Site and extent?
- Margin?
- Shape?

Free ebooks ==> www.Ebook777.com

- Size?
- Surface?
- Skin overlying the swelling?
- Lie of the testicle: Does it appear vertical / oblique?

PALPATION

Palpate the **apparently normal side first**.
Palpation of the **abnormal side**.
↓

- **Number**?
- **Site and extent**?
- **Is the testicle palpable separately**?
- (Local) **Temperature**?
- **Tenderness**?
- **Edema**?
- Is it possible 'to **get above**' the swelling?
  Purpose: To differentiate a purely scrotal swelling from inguinoscrotal swelling.
  Principle: Upper limit of the swelling + normal spermatic cord and nothing else but normal spermatic cord should be palpable.
  Procedure: Hold the **root** of the scrotum between the thumb in front and the fingers behind, and make an attempt to reach the upper limit of the swelling i.e. 'to get above the swelling', and also to palpate the normal spermatic cord.
  Inference
  – Ability to get above the swelling ⇒ the swelling is purely scrotal
  – It is not possible to 'get above' an inguinoscrotal swelling e.g. an inguinoscrotal hernia, acute funiculitis, acute hematoma of the spermatic cord.
- **Margin**?
- **Shape**—globular OR Does it conform to that of epididymis and cord?
- **Size**?
- **Surface**?
- **Consistency**?
- **Fluctuation**?

Can the testis and epididymis be distinguished?

- Examine the **testis**:
  – Can it be felt separately?
  – Site and lie? Does it reach the bottom of the scrotum?
  – Shape, size, surface, consistency?
  – Is the characteristic 'testicular sensation' preserved or not?
- Examine the **epididymis**: – Can it be felt separately?
  – Tenderness?
  – Shape, size, surface, consistency?
- Examine the **spermatic cord.**
  It is often tender and thickened in acute epididymitis.
- **Elevate the scrotum of the affected side** to see if pain is relieved or increased.

LOCO-REGIONAL EXAMINATION OF A CHRONIC SCROTAL SWELLING

EXPOSURE: From umbilicus down to both mid-thighs, including both groins and private parts.

www.Ebook777.com

POSITION of the patient: Standing with legs apart.
Also, ask the patient to cough a couple of times to look for impulse-on-coughing so that differentiation between a scrotal and an inguinoscrotal swelling can begin from the outset.
Additional reason for standing position: Clinical method and observation of 'getting above the swelling' is found to be easiest in this position.

POSITION of the clinician: Sitting on a stool with eyes at the level of the groin.

INSPECTION

- **COMPARE the 2 sides** for—the **levels** of the bottom of the scrotum, the **size and shape**, and **rugosity**
  - Normally the left half of the scrotum hangs lower than the right half due to longer spermatic cord of the left side This may be reversed in case of dextrorotation of viscera
  - Small size and flat shape ⇒ underdeveloped scrotum ⇒ maldescent of the testicle
  - 'Massive' enlargement is usually seen in primary hydrocele, and filariasis of the scrotum
  - Rugosity is diminished in the following situations—warm atmosphere, old age, debilitation, and tuberculous epididymitis.

  Inspection of the enlarged hemiscrotum
  ↓
- **Number** of swelling/s?
- **Site and extent**?
  In case of a child: An apparently scrotal swelling is often due to congenital hydrocele or congenital hernia.
  From the point of view of pathologic anatomy, congenital hydrocele and congenital hernia are inguinoscrotal swellings. However, on inspection they appear to be scrotal for various reasons – 1. In infants, the canal is anteroposterior. 2. The canal is quite short; consequently, the inguinal portion, which is only a small portion of the entire swelling, may not be easily visible enough to be differentiated from a pad of fat present in the region almost invariably in children.
- **Margin**?
- **Shape**?
  Is there **any constriction around the middle of the swelling**?
  - *In a child*:
    - Globular shape suggests congenital hydrocele
    - Ovoid shape suggests congenital hernia
  - A peculiar constriction around the middle of the swelling may be observed characteristically in a (primary) tunica vaginalis hydrocele.
- **Size**?
- **Pulsations**?
- **(Visible) Impulse-on-coughing**?
  *Purpose*s
  1. Confirmation of the diagnosis of hernia.
  2. A hernia may be associated with a scrotal swelling. An unwary clinician may miss the diagnosis of a hernia if he concentrates all his attention on the scrotal swelling and forget to carry out this simple clinical test.

  Inference
  - +ve test ⇒ - Uncomplicated hernia
    - The test is +ve *even in an irreducible hernia* (except for femoral)
  - –ve test ⇒ - Encysted hydrocele of the cord
    - An inguinal hernia complicated by obstruction, strangulation
- **Peristalsis** visible across the skin ⇒ hernia and nothing else but hernia.
  - Observe very carefully. *Movement of the scrotal skin due to contraction of dartos* should not be mistaken for peristalsis
- **Surface**?

Free ebooks ==> www.Ebook777.com

- **Skin overlying the swelling**?
  For inspection of the skin of the posterior aspect of the scrotum, lift the scrotum up.
- **Scar**—if present, its details?
- **Ulcer**, **sinus**?—if present, their details?
  - Gummatous ulcers (tertiary syphilitic ulcer of the testis) and malignant ulcer resulting from cancer of the testis are situated on the anterior aspect of the scrotum whereas a tuberculous ulcer/sinus is situated on the posterior aspect
  - These findings can alter in case of rotational anomalies of the testicle.

PALPATION

Palpate the **apparently normal side first**, f/b the abnormal side.

- **Skin**
  Feel for temperature, texture, moisture, thickness, and pitting edema.
- **Number** of swelling/s?
- **Site and extent**?
- (Local) **Temperature**?
- **Tenderness**?
- **Thrill, pulsations**?
  Look for these features as a rule although they are not commonly expected in the case of a scrotal swelling.
- Is it possible 'to **get above**' the swelling?
  Purpose: To differentiate a purely scrotal swelling from an inguinoscrotal swelling.
  Principle: Upper limit of the swelling + normal spermatic cord and nothing else but normal spermatic cord should be palpable.
  Procedure: Hold the **root** of the scrotum between the thumb in front and the fingers behind, and make an attempt to reach the upper limit of the swelling i.e. 'to get above the swelling', and also to palpate the normal spermatic cord.
  Inference
  - Inability to 'get above' ⇒ inguinoscrotal swelling
  - Ability to 'get above' ⇒ a (purely) scrotal swelling
  - It may be possible to 'get above' in an encysted hydrocele of the cord

    ↓

    Perform the 'traction test'.

  'Traction test'
  Purpose: To ascertain the diagnosis of encysted hydrocele of the cord. For details, refer to chapter 23.
- Examine the **spermatic cord.**
  Clinical method: Refer to page 289.
  Inferences
  - Thickening of the spermatic cord and inability to distinguish various constituents of the cord, and +ve 'rolled silk sign' are indirect evidences of an inguinal hernia [In the absence of a demonstrable swelling even on repeated straining at the time of clinical examination of a patient presenting with a h/s/o hernia (this situation is not rare)]
  - Voluminous cord that feels like a 'bag of worms' ⇒ varicocele
  - Mildly tender, thickened, nodular cord with 'beading' of ductus deference ⇒ tuberculous funiculitis (usually associated with tuberculous epididymitis)
- **(Palpable) Impulse-on-coughing**?
  Inference of +ve test
  1. Expansile impulse ⇒ uncomplicated inguinal hernia. The test is +ve *even in an irreducible inguinal hernia*.
  2. Vague/indefinite impulse is felt in congenital hydrocele, hydrocele of the canal of Nuck, and cold abscess along the cord (rare).
  3. Thrill-like impulse ⇒ varicocele / lymph varix.

www.Ebook777.com

Inference of –ve test

1. Encysted hydrocele of the cord.
2. Inguinal hernia complicated by obstruction, strangulation.

- **Does the swelling feel heavier**?

  Clinical method: Lift both testicles on fingers. Compare their weight and take a judgment if one feels 'heavier than the normal'.

  Clinical significance of heaviness

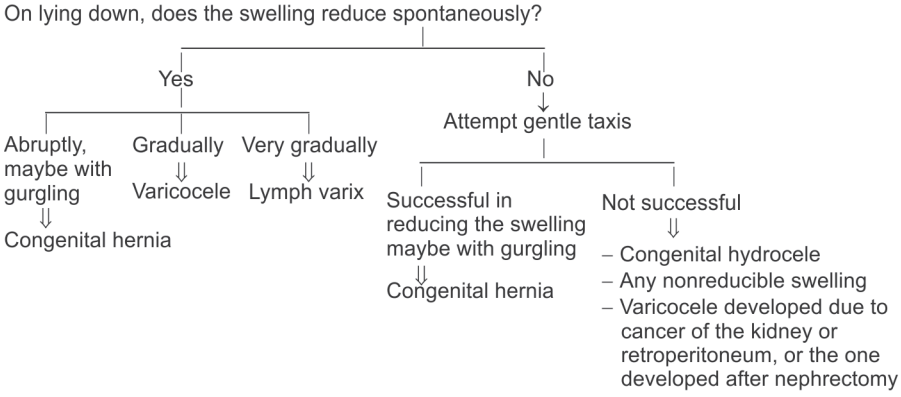
  1. Testicular tumors, hematoceles, gumma, and very huge scrotal swellings feel heavy. Related to the size, the testicle does not feel so heavy in gumma as compared to a tumor or a hematocele.
  2. A heavy swelling may lead to hypertrophy of the cremaster muscle, which in turn may cause slight thickening of the cord, which may give a false sense of the swelling being inguinoscrotal. Even in such a case however, easily palpable cord structures and ductus deference rules out the possibility of the swelling being inguinoscrotal.

Now change the POSITION of the patient to lying down.

Further examination is carried out with the patient in lying down position.

- **Does the scrotum appear to 'stand out'** (forward projecting)?
- **Reduction**

  A swelling exhibiting impulse-on-coughing *need not necessarily be reducible*



  – At this juncture, it will be clear whether one is dealing with an inguinoscrotal swelling or a purely scrotal swelling
  – For an inguinoscrotal swelling, refer to chapter 23/24.

In case of a purely scrotal swelling, proceed as follows:

- **Margin**?
- **Shape**
  – Is there **any constriction in the middle** of the swelling?
- **Size**?
- **Surface**?
- **Consistency**?
- **Fluctuation**?

  For clinical method, refer to chapter 15.

  Evaluation of fluctuation to draw conclusion.

  – +ve test ⇒ cystic swelling containing fluid ↓ moderate tension e.g. hydrocele, chylocele
  – A fallacy: Fluctuation will be absent if the swelling is very soft (lax) or tense (- false –ve)
  – If the swelling is not fluctuant, look at the consistency of the swelling
  – Firm/hard consistency suggests a solid testicular mass, or a cystic (tunica vaginalis) swelling that is thick-walled (may be due to calcification)

    Thus, the presence of fluctuation draws a definite conclusion, but the absence of fluctuation carries no definite significance.

Free ebooks ==> www.Ebook777.com

- **Transillumination**?
  For clinical method, refer to chapter 15.
  A mistake which must be avoided: Do not place the torch posteriorly as the testis stands in the way of light.
  Inference
  - Brilliant translucence ⇒ thin walled cyst containing clear fluid e.g. hydrocele (of all kinds), cyst of the epididymis
  - Translucent, but not 'brilliantly': Spermatocele
  - Negative in cystic swellings not containing clear fluid e.g. hematocele, pyocele, chylocele, sebaceous cyst, etc; and swellings of 'non-cystic' nature i.e. inflammatory, neoplastic.

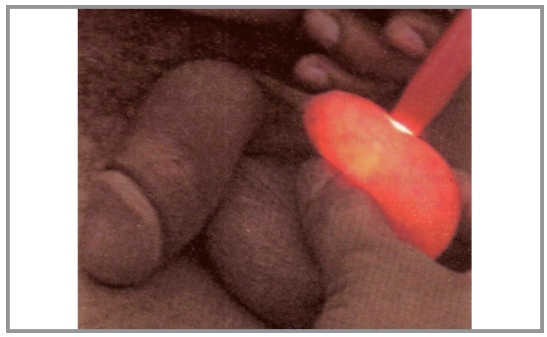


Fig 25-1 Transillumination

  Fallacies
  - False +ve in hernias in neonates and infants if the gut (which is thin-walled in these age groups) is distended with gas
  - The test may be false –ve, if wall of the sac / skin / subcutaneous tissue / all have become thick; for example, due to chronicity leading to calcification of the wall, recurrent attacks of inflammation leading to fibrosis of the wall.

  Therapeutic application: Position of the testicle can be located before tapping a hydrocele (if indicated).
- **Moulding**?
- **Can the testicle be palpated separately from the swelling**?
- **Testis**
  - Location and lie? (Does it reach the bottom of the scrotum?)
  - Shape, size, surface, consistency?
  - Is the characteristic 'testicular sensation' preserved or not? The greatest gentleness must be exercised in eliciting this sign for the fear of disseminating malignancy.

  Characteristics of a normal adult testis: Vertically ovoid, lies slightly obliquely (upper pole tilted anterolaterally and the lower pole tilted posteromedially), the lower pole reaches the bottom of the scrotum, $4 \times 2.5 \times 3$ cm, smooth, soft, and has a characteristic sickening 'testicular sensation'.
  - Small size ⇒ atrophy
  - Decrease in / absence of testicular sensation occurs in malignant tumor and in gumma.
- **Epididymis**
  - It is felt as a distinct ridge of tissue on the posterior and superior aspects of testis
  - **When testis is enlarged, the outline of normal epididymis cannot be made out**
  - **When epididymis alone is enlarged, the separate outline of the enlarged epididymis and the attached testis can be made out**
  - Details of the epididymis to be palpated:
    - Tenderness?
    - Shape, size, surface, consistency?
  - Slightly tender, enlarged, nodular and hard ⇒ tuberculous epididymitis
  - 'Woody hard' consistency is a feature of filarial epididymitis
- **Ductus deference sign**
  - This sign helps to differentiate between a (old clotted) hematocele and a testicular tumor
  - Identify the ductus in the spermatic cord above the swelling, trace it down, and note where it stops
  - If it stops above the swelling, then the swelling in question is a testicular swelling (tumor)
  - If it can be traced going to the lower pole behind the swelling, then it is a hematocele.
- Examine the **penis and perineum** for obstructive pathology in the lower urinary tract: Phimosis, pinhole external urethral meatus, stricture urethra.

www.Ebook777.com

SPECIFIC TEST when varicocele is suspected

– Ask the patient to lie down, elevate and hold the scrotum up—this makes the swelling disappear
– Apply pressure at the external inguinal ring; maintaining the pressure, ask the patient to stand up.
Observation and inference: Varicocele will reappear despite pressure at the external inguinal ring being maintained.

EXAMINE THE REGIONAL LYMPH NODES

– For primary lesion of the skin of the scrotum, examine the horizontal chain of the inguinal group of lymph nodes
– For primary lesion of the testis/epididymis, examine the para-aortic lymph nodes at the level of L1/2 i.e. above the umbilicus, and the left supraclavicular lymph nodes.

DRE

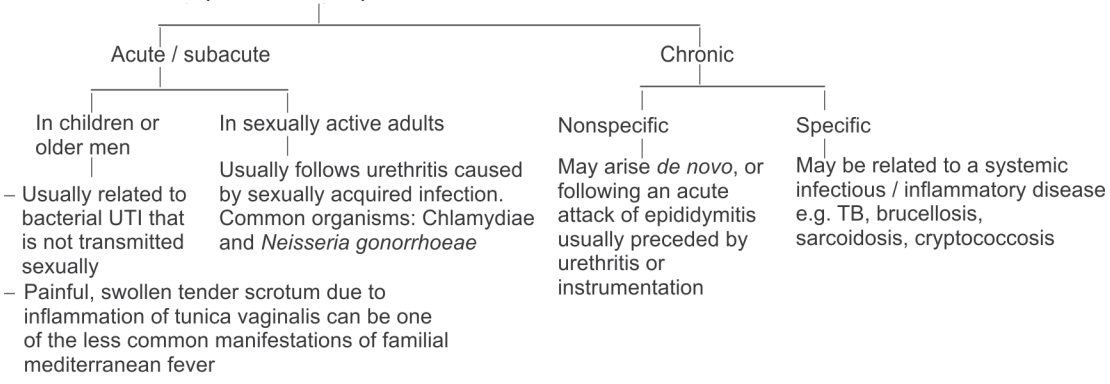
– This is essential in suspected case of tuberculous epididymitis
– One or more well-defined nodules consistency of which is not stony hard, or a large solitary mass occupying a more central position in the prostate ⇒ tuberculous prostatitis.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. A heavy scrotal swelling may lead to hypertrophy of the cremaster muscle, which in turn may cause slight thickening of the cord, which in turn may give a false sense of the swelling being inguinoscrotal. Even in such a case however, easily palpable cord structures and ductus deference rules out the possibility of the swelling being inguinoscrotal.
2. *Epididymitis / epididymo-orchitis*.
   – In many instances inflammation remains confined to the epididymis alone in which case it is referred to as epididymitis
   – When infection in the epididymis spreads to the adjoining testis → the clinical picture of epididymo-orchitis.

   Classification from practical standpoint



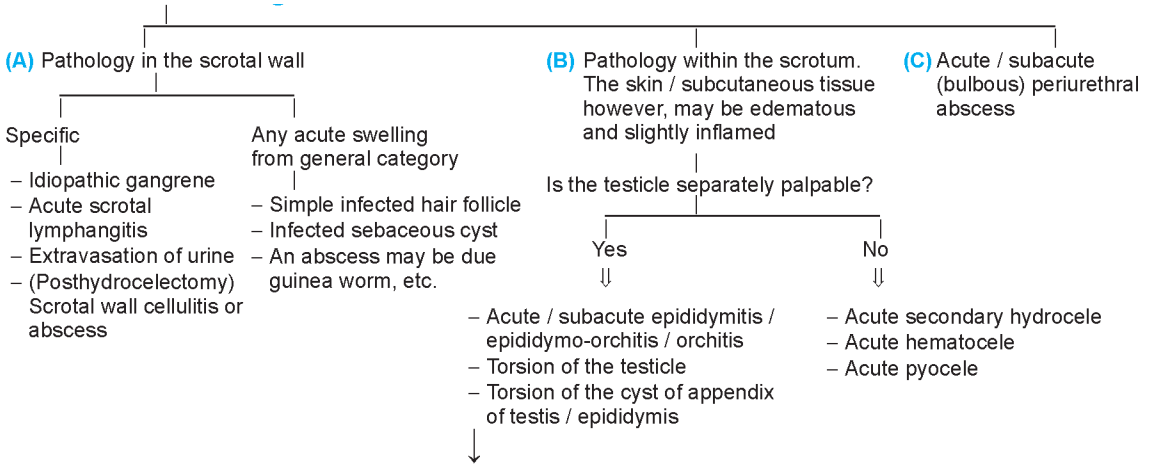
3. *Orchitis*
   Routes of spread of infection to the testis: (a) By direct extension from the ductus and epididymis, (b) Via the lymphatics, (c) Blood-borne. A focus of pyogenic orchitis may also occur after an episode of septicemia.
   Orchitis alone is relatively rare
   – The common causes of orchitis are those associated with epididymitis
   – Non-bacterial causes—viral, parasitic, chemical, traumatic
   – Syphilitic and filarial orchitis can occur in endemic areas.

Free ebooks ==> www.Ebook777.com

4. Not uncommonly, some patients are completely unaware that they have been suffering from a hernia! Even in such patients, a tender, irreducible, often tense swelling without impulse-on-coughing should be taken seriously, as it may be a strangulated hernia; it may be mistaken for acute epididymo-orchitis.
5. Often a large hydrocele obscures a small inguinal hernia, even if the latter has been especially looked for.
6. From the point of view of pathologic anatomy, inguinoscrotal hernia (i.e. a funicular and complete type of inguinal hernia), varicocele and lymph varix – all 3 are virtually inguinoscrotal swellings. However, presentation of an inguinoscrotal hernia is different from that of varicocele and lymph varix.
   - A patient with an inguinoscrotal hernia presents as 'a swelling in the groin', whereas a patient with varicocele or lymph varix presents as 'a swelling in the scrotum'. Varicocele and lymph varix basically being varicosities, blood or lymph respectively collects in the scrotal portion of the vessels due to gravity (in upright posture) to produce a scrotal swelling
   - The inguinal portion of these swellings is neither visible nor palpable as pressure of the fluid (blood/lymph) within these varicosed vessels is low, and moreover the inguinal portion of these vessels is covered by tough aponeurosis
   - A patient does not know the technical term—'inguinoscrotal'. A patient with varicocele or lymph varix therefore presents with a h/o 'a swelling in the scrotum'
   - The same argument is applicable to a congenital hydrocele as well. Parents of a child with a congenital hydrocele seek medical consultation for 'a swelling in his scrotum' even though from pathologic anatomy point of view it is an inguinoscrotal swelling.
7. A swelling on the anterior or inferior aspect of the testis may be caused be an epididymal or vestigial structure pathology if an inversion of the testicle exists in the given case. Inversion is a congenital anomaly; its possibility should be kept in mind as otherwise it can cause confusion in diagnosis.
   - In anterior inversion, the epididymis lies anteriorly, and the body of the testis and the tunica vaginalis posteriorly
   - In complete polar inversion, the globus major and vestigial structures lie inferiorly.
8. A painless nodule in the testis or epididymis requires exploration at the earliest.

STEPWISE ANALYSIS OF DATA

ACUTE SCROTAL SWELLING



The distinction between testis and epididymis is often blurred,and the whole mass of testis and epididymis together may be quite tender.

Specific symptoms, signs and pitfalls to differentiate the swellings in category (B):

– **H/o trauma** may suggest acute hematocele
– **Shape** of the swelling
  - Globular ⇒ hematocele / pyocele

www.Ebook777.com

- In epididymo-orchitis, the testis often being fairly normal, the swelling tends to take the shape of the epididymis and cord

– In torsion of the cyst, **the testis** will be **felt separate** from the tender mass
– **The testicle lying at a higher level** than the opposite (normal) side, and also may be lying obliquely ⇒ torsion of the testicle. Testis lies high in the scrotum due to shortening of the cord.
A pitfall: Edema of the skin of the lower part of the scrotum can occur in epididymo-orchitis or pyocele, and that may make the testicle appear at a higher level, but in these cases, the testicle touches the bottom of the scrotum, while in torsion of the testicle, there is a gap between the lower pole of the testis and the bottom of the scrotum
– (With patient in lying-down position) **Elevate the scrotum of the affected side** to see if pain is relieved or increased
  - Relief of pain occurs in epididymo-orchitis, pyocele
  - Increase in pain suggests torsion of the testicle / strangulated hernia
– **Every effort must be made to distinguish between 2 important conditions**, since their management is quite different—**acute nonspecific epididymo-orchitis, and torsion of the testis**
– The common mistake is to treat a painful testis as infection without making a definite diagnosis
– A definite discrimination cannot always be achieved. In many patients in whom the diagnosis is not clear-cut, a decision has to be made whether to explore as a case of torsion, or whether to treat conservatively as a case of UTI (epididymitis / epididymo-orchitis).
Clinical clues:
1. The younger the patient, and more acute the mode of onset, more likely it could be torsion of the testis.
2. UTI in the male is much commoner in older men than in childhood and pubescence.
3. If urine has no pus cells or bacteria, infection is unlikely.

CHRONIC SCROTAL SWELLING

Decide—is the swelling **scrotal or inguinoscrotal**?

– Nonreducibility
– Ability to 'get above' the swelling
– Absence of impulse-on-coughing

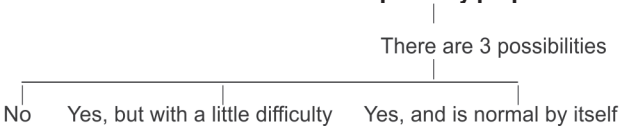
⇒ Scrotal swelling

– H/o reducibility
– Inability to 'get above' the swelling
– +ve impulse-on-coughing
– Swelling actually getting reduced at the time of physical examination

⇒ Inguinoscrotal swelling

Pitfalls
– Lymph varix reduces very gradually
– A congenital hydrocele almost never reduces at the time of physical examination.

Anatomical space / tissue of origin of the swelling?
– Scrotal swelling will be in connection with one of the following structures: Skin / subcutaneous tissue/ spermatic cord / vestigial structures / testis / epididymis / tunica vaginalis.
– *The key question to differentiate one from the other*: '**Is the testis separately palpable from the swelling**?'



A. *If the testis is not separately palpable from the swelling* ⇒ the swelling is in connection with tunica vaginalis / testis itself / both

1. *Key features helpful for differentiation*
  a. Duration. Long duration in many months / years ⇒ usually a tunica vaginalis swelling.

Free ebooks ==> www.Ebook777.com

b. Size of the swelling *vis-à-vis* presence or absence of metastatic disease.
   - 'Large' size in the absence of metastatic disease ⇒ tunica vaginalis swelling
   - The presence of metastatic disease (irrespective of size of the scrotal swelling) ⇒ testicular malignant tumor.

c. Consistency. Solid mass ⇒ testicular (often malignant) swelling.

d. Cystic nature i.e. fluctuant and/or translucent ⇒ tunica vaginalis swelling (abnormal amount or / and abnormal nature of fluid collection in the tunica).

2. *Clinical evaluation of a tunica vaginalis swelling*

a. What is the nature of the fluid?: The key clinical test is transillumination.
   - +ve transillumination ⇒ hydrocele
   - Causes of –ve transillumination:
     i. Truly –ve ⇒ chylocele / pyocele / hematocele.
     ii. False –ve, if wall of the sac / skin / subcutaneous tissue / all have become thick; for example, due to chronicity leading to calcification of the wall, recurrent attacks of inflammation leading to fibrosis of the wall.

b. What is the condition of the testis within?

Clinical clue
- In most of the cases, the testis is normal—primary hydrocele
- If it might be a secondary hydrocele, then look at the duration of the swelling
  - In weeks / months ⇒ inflammatory / neoplastic testicular pathology
  - In years ⇒ inflammatory pathology

Or / and

Imaging investigation
- Normal testis ⇒ primary hydrocele.
- Abnormal testis ⇒ secondary hydrocele.

Aspiration and re-examination
- Amber-colored (urine-like) aspirate ⇒ (essentially) primary hydrocele
- Milky aspirate ⇒ chylocele
- Blood-stained aspirate ⇒ hematocele / testicular tumor

A primary hydrocele may not reaccumulate after the 1st tapping. Should it do so, a definitive treatment will be indicated.

3. *If the testis is not obscured by fluid/blood in the tunica vaginalis*, the alternative possibility is that the palpable mass is testis itself which has been altered in shape and size by a disease process so as not to be recognizable as testis; normal testicular sensation may have also been destroyed.

B. *If the testis can be felt although with a little difficulty* ⇒ the testis is surrounded by 'a relatively small' amount of fluid under minimal tension in the tunica vaginalis (i.e. usually a lax secondary hydrocele).
- The testis is more likely to be diseased (e.g. from an inflammatory or a neoplastic pathology) rather than being normal.

C. *If the testis is separately felt from the swelling* (the testis itself being normal) ⇒ the swelling must be in connection with one of the following: Skin / spermatic cord / epididymis / vestigial structures (at the upper pole of the testis)/subcutaneous tissue.
- Swellings in connection with the skin: Clinical features will be similar to dermal swelling occurring elsewhere in the body; hence they may be easily diagnosed clinically
- Elongated swelling with long longitudinal axis
  - The swelling moving down when the testicle is pulled down
  - And when the testicle is kept pulled down, it moves only transversely

  } ⇒ Swelling of the spermatic cord
- Epididymal swelling is situated on the posterior and superior aspect of the testis.
  Exception:
  - In c/o anterior inversion, it will lie on the anterior aspect of the testis
  - In c/o polar inversion, it may lie on the inferior aspect
- Chronic inflammatory epididymal swelling (which is the common epididymal pathology) usually shows associated involvement of the spermatic cord as well
- Swellings of the vestigial structures are situated above, and fixed to the upper pole of the testis
  Exception: When there is complete polar inversion

www.Ebook777.com

– The swelling which is not in connection with the skin / spermatic cord / epididymis / vestigial structures must be in the subcutaneous tissue. Swellings arising from the subcutaneous tissue (fat, dartos muscle, etc.) are rare anyway.

Inguinoscrotal swellings which present with the chief complaint of 'scrotal swelling':
– Congenital hernia / hydrocele
– Varicocele, lymph varix.

Pathological nature of the swelling?

Common tunica vaginalis swellings:
– Tunica vaginalis hydrocele (primary / secondary)
– Chylocele, (old clotted) hematocele, pyocele

Testicular swellings:
– Usually malignant tumors
– Rarely—tuberculous orchitis, syphilitic orchitis, leprous orchitis, subacute mumps orchitis, (foreign body) granuloma
– Very rarely—benign tumor
– *A solid mass in the testis* should always be considered to be neoplastic until proved otherwise
– A large number of diseases can be entertained as a differential diagnosis, but despite full investigation one usually has to end up exploring the mass on a working diagnosis of tumor
– Unless the clinical evidence in favor of tuberculous / syphilitic orchitis is very strong, and there is an almost immediate response to appropriate chemotherapy, exploration is carried out (through an inguinal incision).

Dermal / Subdermal swellings

Specific (i.e. commonly occurring in the scrotum) | Any swelling from the 'general' category

– Sebaceous cyst/s—often small and multiple
– Cancer of the scrotum: Could be due to occupational exposure to carcinogens e.g. in chimney sweeps, cotton spinners. As it extends, it may involve the underlying testis
– Nonspecific abscess due to guinea-worm
– Small pustules due to infection of hair follicles—can attain more significant form (boil / abscess) in patients with reduced immunity / DM
– Lymph scrotum
– Lymphedema, elephantiasis
– Nonfilarial elephantiasis
– Scrotal edema due to hypoproteinaemia / hepatic failure / renal failure / cardiac failure
– Idiopathic scrotal edema
– Fat necrosis.

Spermatic cord swellings:
– Encysted hydrocele of the cord
– Benign tumor e.g. lipoma, rarely myxoma
– Chronic inflammatory i.e. funiculitis. Usually the cord and epididymis are affected together
– Malignant—usually an extension from testicular malignant tumor
– Varicocele
– Lymph varix.

→ An inguinoscrotal swelling

Epididymal swellings:
– Cysts—of the epididymis / of the appendix of epididymis
– Tumors are very rare
– Inflammatory i.e. epididymitis—nonspecific, tuberculous, filarial, gonococcal, viral (mumps).

Swellings in connection with the **vestigial structures**: Example—cyst of the appendix of testis.

Free ebooks ==> www.Ebook777.com

Spermatocele

Some of the cysts (such as cyst of the appendix of testis / epididymal cyst, etc.) contain cloudy material; microscopic examination of this fluid demonstrates sperms. Those cysts are referred to as spermatocele. Spermatoceles are not diagnosable as such clinically. It is a retrospective diagnosis on aspiration or operative excision. Spermatoceles have no special clinical significance.

Solid masses in the spermatic cord / epididymis other than tuberculous epididymitis cannot be diagnosed with confidence clinically; cannot usually be diagnosed even when exposed at operation. However, should the tumor look yellow it is likely to be an interstitial cell tumor, usually benign.

Miscellaneous conditions: An example—hernia testis.

DIFFERENTIAL DIAGNOSIS

– **Idiopathic gangrene of the scrotum** (*Syn*. Fournier's gangrene) refer to figure 13-4.
– *Basically*, acute necrotizing nonclostridial infective inflammation leading to subcutaneous ganagrene of the scrotum.

Cardinal features

1. Sudden mode of onset and very rapid progress of scrotal wall inflammation in the midst of apparently good health.
2. Rapid onset of gangrene.
3. Idiopathic in over half the cases.

It may follow however, minor injuries or procedures in the perineum such as scratch, bruise, dilatation of stricture, opening up of a periurethral abscess, etc.

Untreated, the cellulitis spreads along those planes so well-known in superficial extravasation of urine. Sometimes the entire scrotal coverings slough off, leaving the testes hanging exposed, bared to their tunica, though, remarkably free from gangrene.

Notes

– It is a vascular disaster of infective origin
– Cutaneous gangrene is secondary to thrombosis (obliterative arteritis) of the perforating vessels coursing through the infected necrotic area.

Acute scrotal lymphangitis (filarial)

– Acute painful scrotal enlargement occurs due to widespread cellulitis of the scrotal (± penile) skin, associated with constitutional symptoms
– The infection is generally self-limiting and when it subsides, it leaves behind characteristic thickening of the skin
– The condition may be mistaken for extravasation of urine.

In fulminating infection, the constitutional symptoms and local necrotizing inflammation are severe → suppuration and sloughing off of the scrotal (± penile) skin. At times, massive idiopathic type of gangrene develops. Line of demarcation forms in about 10–15 days (Operative procedure of making multiple incisions to release edema and purulent exudate will usually have been carried out before this).

Superficial extravasation of urine

Basically, in superficial variety of extravasation of urine, the pathology/injury occurs superficial to the transverse perineal membrane whereas in deep variety, it occurs deep to the transverse perineal membrane.

Causes of superficial extravasation of urine

1. Neglect of complete rupture of the bulbous urethra i.e. when operation is not undertaken within 12–24 hours.
2. Rupture of a bulbar periurethral abscess.

The extravasated urine passes (a) into the scrotum, (b) beneath the superficial layer of the penis, and (c) up the abdominal wall beneath the deep layer of the superficial fascia. The swelling is of the nature of edema. In fair-skinned patients, the water-logged condition can be easily ascertained by pinching the skin of the scrotum and penis.

Infection predisposed to by the extravasated urine → constitutional manifestations of sepsis.

www.Ebook777.com

Posthydrocelectomy scrotal wall cellulitis / abscess
Particularly after those operative procedures for tunica vaginalis hydrocele e.g. excision of the sac or eversion of the sac, if infection develops, then one must remember that a pyocele cannot result as there is no more potential space left in which pus can form and accumulate. The infection essentially involves the scrotal wall layers and hence termed as a scrotal wall abscess.

Acute / subacute **secondary hydrocele**
The testis may not be palpable, or may be palpable with a little difficulty depending upon the amount of fluid in the tunica vaginalis. In the case of acute epididymo-orchitis usually a secondary hydrocele subsides *pari passu* with the primary inflammatory lesion.

Acute / subacute **hematocele**
- *Basically*, an acute painful and tender scrotal swelling, in which the testicle is not separately palpable
- Fluctuation ±.

Supporting points
- H/o trauma. In that case, the swelling appears as well as gains its full size immediately following trauma, and subsequently the size remains stationary
- Bleeding disorder/patient on anticoagulant therapy
- Skin—red or blue depending on the interval between the time of injury and consultation
- Nontranslucent.

Causes
A. In case of a pre-existing hydrocele:
   1. As a complication of tapping.
   2. Transformation into a hematocele either spontaneously or as the result of accidental trauma.
B. In the absence of any pre-existing hydrocele, i.e. acute bleeding occurring into a previously normal tunica vaginalis:
   1. A blow on the testis.
   2. A testicular tumor may sometimes present in this way.

Pyocele
Basically, an acute painful and inflammatory scrotal swelling; the testicle is not separately palpable.
- Fluctuation—usually present
- Transillumination—absent
- Pyoceles usually develop as complication of another primary pathology
- Sometimes the pyocele may burst within the scrotal sac and produce a subcutaneous collection and a collar-stud abscess.

Acute / subacute **epididymitis / epididymo-orchitis**
Basically, an acute painful and inflammatory swelling of the epididymis alone or epididymis + testis together as a single mass.

Supporting points
- Constitutional symptoms
- Associated funiculitis.

Clinical classification of severity
- Epididymis thickened and tender
- Testis separately palpable and non-tender

} = Mild

- Entire hemiscrotum inflamed
- Loss of intrascrotal landmarks

} = Moderate

- Inflammatory mass fixed to scrotal wall
- Cord tender and indurated.

} = Severe

Clinical course
- The epididymis may become adherent to the scrotal skin and may soften, and later, discharge
- Resolution is heralded by scaling of the scrotal skin
- Complete resolution of the swelling in the epididymis may take 6–8 weeks.

Free ebooks ==> www.Ebook777.com

Causes: Nonspecific, gonococcal, tuberculous, filarial, viral (mumps), postoperative (e.g. post-prostatectomy), 'reflux', brucellosis, lymphogranuloma venereum, epidemic myalgia.

Complications: abscess formation, testicular infarction, chronicity, subfertility (due to inflammatory obstruction of sperm transport). Abscess may produce extensive tissue destruction of epididymis ± testis.

Acute nonspecific epididymitis due to *E. coli*, streptococcus, staphylococcus or proteus.
Suppuration may occur if correct antibiotic therapy is not commenced early.

Secondary to UTI	Acquired by blood-borne infection
– E/o recent episode of UTI +	– As a rule there is no e/o UTI
– Obvious e/o factors predisposing to UTI may be present e.g. chronic retention of urine, urethral instrumentation, etc.	– In the absence of any local cause, the infection is assumed to be blood-borne

Acute gonococcal epididymitis

- In 90% of cases the infection is unilateral
- Epididymitis commences during the 2nd or 3rd week of gonococcal posterior urethritis
- By about the 3rd day after commencement of epididymitis, the skin of the scrotum often becomes adherent to the globus minor, but in cases where the gonococcus is the sole infecting organism suppuration does not occur
- After the 5th day the symptoms commence to abate, and by the 8th to 10th day the condition subsides
- Induration of the globus minor may persist for months
- In cases of persistent infection of the corresponding seminal vesicle, subacute attacks of recurrent epididymitis are not infrequent.

Acute tuberculous epididymitis

- This condition is not rare.
- In cases where the ductus deference is thickened and there is little response to the usual treatment (for a provisionally diagnosed case of nontuberculous epididymitis), the possibility of the infection being due to tuberculosis must receive due consideration.

Acute / subacute filarial epididymo-orchitis

Differentiating points

- Filarial infestation is more recurrent rather than truly chronic and continuous
- The presence of eosinophilia and microfilariae in blood during the acute attacks.

Supporting points

- Patient residing in the endemic area
- Fever with rigors.

Natural history

- Inflammation subsides, leaving behind residual hydrocele, fibrous thickening of the spermatic cord, and nodules in the epididymis
- In endemic areas, epididymal nodules are often seen during the operation for tunica vaginalis hydrocele. A big nodule may be mistaken for a tumor.

'Reflux' epididymitis

- Epididymitis can develop during an unusual excretion or violent strain while the bladder is full, and it is caused by the urine being forced along the ductus deference. The degree of inflammation that results depends upon whether the urine is infected, or if there is an active or latent infection in the prostate or seminal vesicles
- Reflux epididymitis can also occur due to the presence of a stricture in the urethra.

Acute orchitis

Testicular swelling is prominent and there is often a reactive hydrocele.

Causes:

1. Associated with mumps. Mumps orchitis is rare in prepubertal children. Mumps virus has little affinity for the testis until after sexual maturation. It occurs about 5 days after the parotitis.

www.Ebook777.com

2. Influenza and coxsackie viruses.
3. Typhoid and other eruptive fevers.

Acute mumps epididymo-orchitis

– Usually develops as the parotitis is waning
– Especially in infants, mumps epididymo-orchitis can occur without parotitis.

Natural history

– Resolution or fibrosis
– In about half the cases atrophy of the testis follows. It often takes many months for signs of testicular atrophy to become apparent. A small incision into the tunica vaginalis and drainage if undertaken early in the attack, prevents testicular atrophy and relieves the intense pain
– Suppuration does not occur.

Torsion of the testis

Basically, acute painful and exquisitely tender scrotal swelling.

Differentiating points

– Sudden, often dramatic mode of onset
– Agonizing pain
– The testicle lies at a higher level than the opposite (normal) side, and also may be lying obliquely.

Supporting points

– Younger age—between 5 and 20 years, especially between 12 and 16
– H/o preceding self-resolving attacks of pain probably representing episodes of torsion
– The attack of pain immediately preceded by straining e.g. at stools / lifting heavy weight / sexual activity, etc.

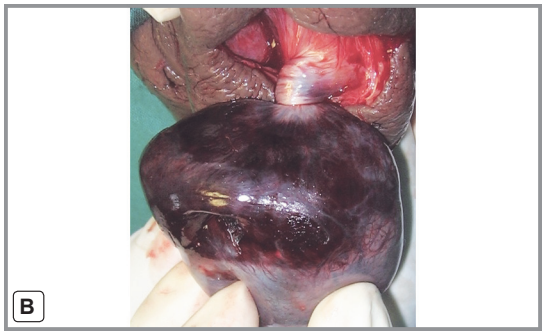
Additional clinical manifestations which might be present

– Pain in the groin or even in the lower abdomen
– Nausea and vomiting.

Exciting causes

– Straining e.g. at stools/lifting heavy weight/sexual activity, etc. Mechanism: Any of these activities → violent contraction of the abdominal wall musculature → contraction of the cremaster → intravaginal torsion of those testes which are not anchored in the scrotum properly due to anomaly e.g. 'a bell-clapper' deformity or long mesorchium
– Often no exciting cause is apparent. Occasionally, torsion develops during sleep.







Figs 25-2A to C **(A)** The left testis raised and lying obliquely, **(B)** The testis had undergone necrosis as the patient presented very late (3 days after the commencement of pain), **(C)** The right side explored at the same time; shows a long mesorchium

Notes

– A h/o mild trauma just before the onset ±, however that does not play a significant role in arriving at the diagnosis
– The actual pathological event is torsion of the spermatic cord. Ischemia of the testis → pain
– The testicular cells suffer ischemic damage within 6 hours
– Torsion of a testicle is a surgical emergency; if the blood supply is not restored within 12 hours, the testis infarcts and must then be excised
– After the neonatal period, torsion becomes relatively less common until the approach of puberty when rapid enlargement of testis predisposes to twisting (*intravaginal torsion*) of those testes which are not anchored in the scrotum properly due to some anomaly e.g. 'a bell-clapper' deformity or long mesorchium.

Free ebooks ==> www.Ebook777.com

Torsion of testicular or epididymal appendages

– → scrotal pain and swelling out of all proportion to their actual size
– Localized tenderness
– The twisted cyst may be seen as a blue-black spot through the scrotal skin (in fair-skinned patients).

If the diagnosis is certain, surgical exploration is not essential, but may be needed if testicular torsion cannot be ruled out.

Tunica vaginalis hydrocele

– *Basically*, a scrotal swelling where the testicle is not palpable separately
– *Differentiating points that are usually present*: Fluctuation and tansillumination
– One or both the differentiating points may be absent as false –ve observations
– *Differentiation between primary and secondary tunica vaginalis hydrocele* is as follows:

Clinical feature	***Primary** tunica vaginalis hydrocele*	***Secondary** tunica vaginalis hydrocele*
Age	Young adults	Middle / old
Duration	Months / years	Weeks to months
Pain	Absent	±
Fever	Absent	±
Side/s	May be bilateral	Usually unilateral
Spermatic cord	Normal	May show some thickening
Size	Variable. Could be massive	Usually 'small'
Consistency / tension	Firm / usually tense	Soft / usually lax
Testis	Not palpable	Can be felt inside the swelling through the fluid
After tapping (if tapped)	The testis feels normal	Testis and / or epididymis may show primary disease
Fluid	Clear, straw-colored	Clear, straw-colored, or may be blood-stained

Additional differentiating points which may be evident in primary tunica vaginalis hydrocele:

– If the swelling is tense, the scrotum 'stands out' (forward projecting) even when the patient lies down
– A peculiar constriction around the middle of the swelling.

Notes on tunica vaginalis hydrocele

– Although most commonly occurs in adults, it is not uncommon in early childhood
– The inconvenience of its size usually leads the patient to seek advice
– The fluid collects in between the visceral and the parietal layers of tunica vaginalis testis. Rest of the processus vaginalis testis is obliterated
– The cause of most hydroceles is unknown (idiopathic)—the primary hydrocele
– The secondary hydrocele develops as a result of trauma or some disease in the testis and / or epididymis e.g. epididymo-orchitis, tumor of the testis, etc; or after an inguinal hernia repair
– Occasionally the question as to whether a hydrocele is primary or secondary can be settled only after aspiration of contents.

Possible complications of essentially primary hydrocele

1. Rupture—usually traumatic, but may possibly be spontaneous. On rare occasions a cure results after absorption of the fluid
2. Transformation into a hematocele can occur both spontaneously or as the result of trauma
3. Hernia of the hydrocele sac may occur in long-standing cases. Tension of fluid → herniation of a portion of the hydrocele sac through the dartos muscle
4. Calcification of sac may occur in long-standing cases.

Post-hernia repair hydrocele

– = a secondary hydrocele that may appear after an operation for inguinal hernia in which there was no evidence of a hydrocele preoperatively
– Possible mechanism: Damage of lymphatic vessels of the tunica vaginalis.

www.Ebook777.com

Chylocele

– Fluctuation is often present, but transillumination is –ve
– Evidence of filarial affection of the scrotal and/or penile skin ±
– Filarial limb edema ±
– Patient may be residing in endemic area.

Pathology of chylocele

The fluid in the tunica vaginalis is milky and is laden with high proteins and fine fat globules called 'chylomicrons'. Chylocele results from obstruction to the intestinal lymphatics near the para-aortic lymph nodes with regurgitation of chyle into testicular lymphatics.

Old clotted hematocele

Causes

1. Idiopathic: Curiously, slow bleeding into the tunica vaginalis occurs spontaneously, and apparently painlessly. Most of the patients give neither a h/o trauma nor of pain. An old clotted hematocele simulates a testicular tumor so closely that preoperative differential diagnosis is sometimes impossible.
2. As a consequence of an acute hematocele that was left without prompt appropriate surgical treatment.

Differentiating clinical feature	***(Old clotted) Hematocele***	***(Malignant) Tumor of the testis***
Age	Any	(Young) Adults
Duration	Months / years	Weeks to months
Progress	Stationary	Persistently progressive
Loss of weight	No	May be marked
Anemia	No	May be marked
Shape	Pyriform with broad diameter above A constriction in the middle ±	Pyriform with broad diameter below No constriction
Surface	Smooth	May be nodular
Fluctuation	±, although usually absent	Absent
Spermatic cord	Normal / slightly thickened	May show extension of the tumor
Ductus deference sign	The ductus can be traced going to the lower pole of the testis behind the swelling	The ductus stops above the swelling
Para-aortic lymphadenopathy	Absent	±
Distant metastases	Absent	±

Testicular tumor

– Every testicular swelling must be assessed with the possibility of tumor in mind
– Early diagnosis and accurate staging of malignant tumor is essential. Prognosis is good for early stage and low bulk disease
– Clinically, it may be difficult to pinpoint the type of malignancy.

Typical clinical features of malignant testicular tumor:

Symptoms

– A painless enlargement of the testis × 4–6 months or more
– A sensation of heaviness (when the testis has reached 2–3 × its normal size)
– Slight injury if at all, merely calls attention to the testicular enlargement.

Signs

– The spermatic cord remains normal for a considerable time. It then becomes thickened due to cremasteric hypertrophy and enlargement of testicular vessels
– The testicle feels heavier than normal
– Size—variable. A teratoma for example, varies in size from that of a peanut to that of a coconut
– Surface—initially smooth; later becomes irregular / nodular
– Consistency may be variable due to areas of necrosis, however usually firm / hard

Free ebooks ==> www.Ebook777.com

- A uniformly firm consistency may suggest seminoma. A variegated consistency may suggest teratoma
- The loss of characteristic testicular sensation on gentle compression. In no other disease is testicular sensation lost so early and so completely
- (Secondary) Hydrocele.

The common errors in diagnosis result from:

1. The presence of pain (which may result from bleeding into the tumor), and the case may then be misdiagnosed as epididymo-orchitis.
2. Hydrocele (secondary hydrocele is not an uncommon accompaniment of tumor).
3. A h/o 'trauma' which may merely bring the swelling to attention.

Atypical presentations of malignant testicular tumors:

- Hurricane type: The tumor is small and yet spreads and kills the patient fast within a matter of weeks.
- Slow-growing type: Slowly growing over a period of about 3 *years*
- Epididymo-orchitis like
- Gynecomastia
- Malignant cachexia
- Presentation with metastatic disease: Pain/lump in the abdomen, lumbar pain due to retroperitoneal metastatic disease, left supraclavicular lymphadenopathy, pulmonary metastatic disease.

Common types of testicular tumor:

1. Seminoma:
   - Arise from the seminiferous tubular epithelium
   - Peak incidence in the age group of 35–40
   - The tumor marker β-HCG levels occasionally ↑
   - Relatively slow growing.
2. Teratomas = Nonseminomatous germcell tumors
   - Peak incidence in the age group of 25–30; however, may also occur in childhood
   - Tumor markers—α-FP and β-HCG—one / both ↑
   - Types depending on the degree of differentiation: Well-differentiated, intermediate differentiated, teratomas containing yolk sac / trophoblastic elements, and undifferentiated. Well-differentiated malignant teratoma has the least malignant potential whereas the undifferentiated variety is the worst
   - A differentiated teratoma in childhood can be considered as a benign tumor, but this is not so in adults.
3. Mixed: Teratomatous element may not be histologically detected within a seminoma, however, increased levels of α-FP suggests the presence of a teratomatous element. Prognosis relates to the teratomatous element; hence it is managed along the lines of a teratoma.
4. Malignant lymphoma occurs in patients above 60 years of age.
5. Carcinoma *in situ*:
   - Usually asymptomatic, however, may produce a slight tenderness
   - Poor semen quality
   - NO ↑ in the levels of tumor markers
   - Semen analysis may reveal malignant germ cells.

Leukemia often involves the testes. After initially successful treatment, leukemia may recur especially in the CNS (meninges) and the testis.

Rare tumors: Leydig cell tumors, Sertoli cell tumors, etc.

A Leydig cell tumor masculinises; a Sertoli cell tumor feminizes. For clinical reasons, these tumors are classified as those occurring before or after puberty.

Prepubertal Leydig cell tumor

This is characterized by an excessive output of androgens, which is responsible for sexual precocity and extreme muscular development. → an infant Hercules. This is in all respects similar to the condition produced by an adrenal cortical tumor, except that on palpation a swelling of the testis can be elicited.

Exceptionally it is bilateral, but it must be noted that in this condition the contralateral normal testis frequently becomes hypertrophied.

www.Ebook777.com

Postpubertal tumor
In a high percentage of cases the tumor arises in the cells of Sertoli, and consequently the output of feminizing hormone becomes excessive.
- → gynecomastia, loss of libido, aspermia
- Aschheim-Zondek test is +ve.

Gumma of the testis (Tertiary syphilitic orchitis)
Basically, chronic testicular swelling (unilateral or bilateral).
Differentiating points
- Slight to moderate enlargement; i.e. 2–3 × the size of normal testis
- Smooth surface
- Firm-to-hard consistency
- The loss of characteristic testicular sensation on gentle compression.

Supporting points
- Adherent to the skin anteriorly. Softening then occurs and a gummatous ulcer forms
- The presence of other syphilitic stigmata.

Interstitial fibrosis—the least common type of tertiary syphilitic orchitis.
- Bilateral asymptomatic gradual destruction of the seminiferous tubules
- The testes are *not* enlarged. They are firmer than normal, and there is a gradual loss of testicular sensation.

Granulomatous orchitis
- The testis is enlarged and tender
- Nonspecific inflammation may occur in the testis, epididymis and spermatic cord following infection, exposure to chemicals, trauma, and occasionally after vasectomy. The etiopathogenesis is often not clear; probably produced by extravasation of spermatic material into the interstitial tissues of the testis in consequence of minor trauma.

Encysted hydrocele of the cord
- Well-defined, globular (spherical / ovoid)
- No impulse-on-coughing
- Possible to 'get above'
- Traction test +ve
- (Brilliantly) Translucent.

Pathologic anatomy of encysted hydrocele: Refer to page 295.

Funiculitis: Refer to chapter 24.

Varicocele
Basically, the patient may present with a reducible swelling in the scrotum or / and dragging ache / pain in the scrotum after prolonged walking / standing.
Differentiating points
- Thrill-like impulse-on-coughing (with the patient in standing position)
- A 'bag of worms' feel (with the patient in standing position)
- The swelling reduces after the patient lies down and the scrotum held up. After reduction, there is no trace of palpable swelling
- No thickening of the spermatic cord at all
- When patient stands up from lying-down position, the swelling is seen to fill from below upwards.

Notes
- Usually left-sided due to various anatomical / physiological reasons.



Fig 25-3 Varicocele

Free ebooks ==> www.Ebook777.com

- Right-sided varicocele must arouse suspicion of its being secondary to some disease and must be investigated thoroughly
- In c/o a rapidly progressing varicocele in a middle-aged man, look for a renal cell carcinoma or retroperitoneal cancer on the same side
- Testis of the affected side may feel softer
- 16% of normal adult men have abnormal dilatation of the pampiniform plexus of veins
- Most varicoceles cause no symptoms
- The relationship of varicocele to subfertility is controversial.

Lymph varix (*Syns.* **Lymphangiectasis, lymphangiocele, diffuse hydrocele of the cord.**)
Basically, the patient presents with dragging ache and soft swelling in the scrotum which becomes less prominent on lying down.

Clinical features

- Past h/o periodic attacks of fever with chills with simultaneous development of painful and tender swelling in the inguinoscrotal region (funiculitis) ±
- The swelling appears to extend from the external inguinal ring to the top of the testis
- Thrill-like impulse-on-coughing
- Diffuse
- Soft, doughy and compressible. + a few firm nodules are felt. In later stages however, the soft and doughy feel is less marked because of fibrofatty infiltration
- The swelling becomes less prominent on lying down.

DD

1. In the early stage it may be mistaken for a varicocele. The lymph varix however, is not as soft and compressible and as completely reducible as a varicocele.
2. In the later stages it may be mistaken for an inguinal hernia as the soft-to-firm consistency of fibrofatty cord closely resembles the consistency of an omentocele.

Pathology of lymph varix

Lymph varix is produced as a result of obstruction to the lymphatics of the cord, epididymis and testis. Because of the surrounding loose cellular tissue, the lymphatics dilate and become varicosed. Recurrent attacks of inflammation → considerable fibroblastic reaction → firm nodules.

The condition generally remains stationary and is self-limiting.

Cysts of the epididymis

Basically, epididymal swelling/s, in a middle aged adult.

- Multiple small tense (brilliantly) translucent cysts → nodular surface of the mass of cysts as a whole—feel like a bunch of tiny grapes
- The brilliant translucency is finely tessellated because of numerous septa, giving an appearance like that of a lantern
- A solitary epididymal cyst may even resemble a testis → fables of 3 testes!
- Often bilateral.

(Chronic) Tuberculous epididymitis / epididymo-orchitis

Basically, a slightly painful chronic epididymal swelling.

Differentiating points

- Surface—nodular
- Consistency—hard
- Skin of the posterior wall of the scrotum is adherent to the swelling, with or without sinus formation
- Ductus deference—thickened and beaded (due to submucous tubercles).

Supporting points

- Young adult
- Nearly 90% of cases of chronic epididymo-orchitis are tuberculous
- The loss of normal rugosity of the scrotal skin
- Associated renal TB (Refer to page 805)
- Associated tuberculous prostatitis (Refer to page 803)

www.Ebook777.com

Clinical course

Early feature: A discrete slightly tender indurated nodule in the globus minor; rarely in the globus major. As the disease progresses other nodules appear, and eventually the entire epididymis becomes involved. A cold abscess forms in the posteroinferior aspect of the scrotum. Bursting of the abscess → a chronic discharging sinus.

- A lax secondary hydrocele may develop
- Untreated, after a varying time (from a few months to a year or so), the contralateral epididymis becomes similarly diseased
- The testis remains uninvolved for a long-time, often years.

Notes

- In 2/3rds of cases there is either active e/o TB in the renal tract or e/o previous disease. In the remainder the patient is apparently healthy
- It is reasonable to accept the clinical diagnosis of tuberculous epididymitis as proven to consider prescription of ATT
- Routes of spread of infection to the epididymis: (a) Commencement of the disease in globus minor suggests retrograde spread along the ductus deference from an infected seminal vesicle. (b) Commencement of the disease in globus major suggests a blood-borne infection.

Chronic nonspecific epididymitis

- It may arise *de novo*, or following an acute attack of epididymitis usually preceded by urethritis or instrumentation
- The condition is difficult to distinguish clinically from tuberculous epididymitis, but as a rule the epididymis in nonspecific epididymitis is larger and smoother
- Rule out a stricture in the urethra.

(Residual) Nodule/s in the epididymis most probably due to filariasis

- This diagnosis may be entertained if there has been a definite previous h/s/o recurrent attacks of acute filarial epididymo-orchitis in the patient residing in endemic area
- Associated features which are likely to be present: Hydrocele, and fibrous thickening of the spermatic cord
- In endemic areas, epididymal nodules are often seen during the operation for tunica vaginalis hydrocele. A big nodule may be mistaken for a tumor.

Nodularity in the epididymis due to TB *v/s* due to filariasis

Apart from other differentiating features between the two, as far as the feature of the abnormal epididymis is concerned, if any softening is detected within the firm epididymis it is very strong evidence in favour of tuberculosis.

Spermatocele: Refer to page 322.

Cyst of the appendix of testis

Situated above, and fixed to the upper pole of the testis; however, it may not be so if there is polar inversion of the testis.

Sebaceous cysts

Often multiple whitish or yellowish round nodules of varying size, and may be scattered over the entire scrotum.

Lymph scrotum

- The scrotum is 'moderately' enlarged, and it hangs down as a loose bag with folds
- Surface—irregular due to vesicles (innumerable varicose lymphatic villi bulging out under the epidermis of the skin)
- Feels soft and velvety (because of innumerable varicose lymphatic villi bulging out under the epidermis of the skin).

During an attack (of lymphangitis) or due to friction, the dilated dermal lymphatics (the vesicles) burst, oozing clear / turbid lymph from the surface. If the abdominal lymphatics are occluded, then chyle may ooze out.

Natural history: Elephantoid changes may take place subsequently.

Free ebooks ==> www.Ebook777.com

Filarial elephantiasis of the scrotum

Clinical presentation and features

- Chronic painless diffuse thickening of skin and subcutaneous tissue of the scrotum occurring in those who dwell, or have dwelt, in tropical and subtropical countries
- Previous h/o recurrent acute attacks of epididymo-orchitis/funiculitis, lymph scrotum and scrotal lymphangitis will be available
- **If the skin of the elephantoid scrotum becomes thickened and inelastic in early stages, scrotal enlargement is considerably restricted**. If on the other hand these changes in the skin do not occur early, giant enlargement reaching even below the knee level is produced



Fig 25-4 (Filarial) Elephantiasis

- In long-standing cases the scrotum becomes immense, thick, and the penis is buried within it causing a mechanical hindrance to walking and passing urine
- Urine trickles over the anterior surface of the scrotum → dermatitis and ulceration
- Intercourse is practically impossible
- Hygiene of the prepucial sac cannot be maintained → balanoposthitis
- The pathological process of elephantiasis stops short above of the abdominal wall, and below of the thighs
- There is no distortion of the anal orifice.

DD: Elephantiasis of the scrotum may be mistaken for giant bilateral tunica vaginalis hydrocele and giant inguinoscrotal hernia.

Pathology in brief

Elephantiasis of the scrotum and penis may develop independently of each other which show that the lymph drainage in the inguinal region is separate for the scrotum and the penis.

Elephantiasis is most massive at the fundus of the scrotum and tapers off towards its root.

The elephantiatic changes involve only the skin and the subcutaneous tissue; they do not extend beyond the spermatic fascia. The spermatic cord and its contents never undergo elephantiatic changes. The inner layer of the prepucial skin remarkably maintains its normal elasticity and it never becomes thickened and elephantiatic.

Nonfilarial elephantiasis of the scrotum

- Results from fibrosis of lymphatic vessels due to lymphogranuloma venereum
- Never reaches the extravagant proportions of tropical (filarial) elephantiasis.

Idiopathic scrotal edema

- This is a benign self-limiting condition of unknown cause, occurring in young children
- The erythema and edema are painless and may affect both sides and extend onto the groin and penis
- Spontaneous resolution occurs in a few days.

Fat necrosis of the scrotum

This is a rare condition occurring in stout boys.

- Usually bilateral, small, comma-shaped, tender swellings within the scrotal wall
- Usually a h/o swimming in very cold water is obtained.

Congenital hydrocele and congenital hernia

Basically, the presentation is with a reducible scrotal swelling in a young boy; sometimes may be first noticed even soon after birth.

- On physical examination, they are found not to be 'purely scrotal' but instead, inguinoscrotal
- For differential diagnosis, refer to page 293 in chapter 23.

Infantile hydrocele

From its name it may appear that it must be occurring only in infants, but that is not true.

- It may extend right up to the internal inguinal ring, and hence basically an inguinoscrotal swelling.

www.Ebook777.com

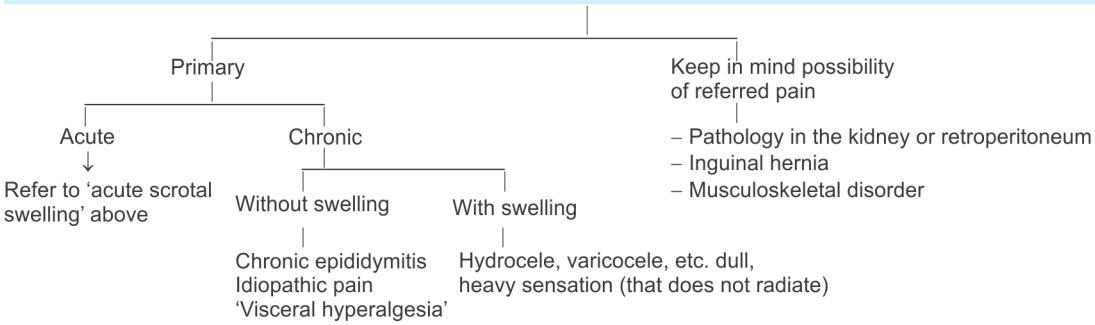
Pathologic anatomy: Processus vaginalis is closed at the internal inguinal ring only, but communicates with the tunica vaginalis. As the fluid in the sac surrounds the testis, it cannot be felt separately

– May be associated with an inguinal hernia.

Hernia testis

– = protrusion of a granulating mass of testis through the tunica albuginea, scrotal wall layers and finally the skin of the scrotum, resulting from septic penetrating injury or suppurative infection of the testis
– The condition may also develop in a burst gumma.

SCROTAL PAIN



'Visceral hyperalgesia' is a difficult problem to manage

– Refer the patient to a pain clinic
– Surgery virtually never helps and commonly results in transfer of the pain to the incision or opposite testis.

Free ebooks ==> www.Ebook777.com

26 IMPALPABLE TESTICLE EMPTY SCROTUM

This chief complaint (impalpable testicle / empty scrotum) suggests maldescent of the testicle.
(Testis and epididymis together is referred to as 'testicle').

GENERAL REMARKS

1. Undescended testicle/incompletely descended testicle/ectopic testicle qualify for maldescent.

Undescended / incompletely descended testicle	*Ectopic testicle*
– The testicle either does not descend at all (rare), or the descent is arrested somewhere along its normal path of descent	– The testis has often developed well, if not fully, but after passing through the superficial inguinal ring its further descent is impeded
– The testis is smaller than normal, and the cord is short	– The testis is normal in size, and its cord is normal too (or slightly bulkier)
– Orchidopexy is difficult	– Orchidopexy is achieved without difficulty

2. A superficial inguinal pouch is a sort of pouch created by an ectopic testicle (covered by external spermatic fascia) in the anatomical plane that is superficial to the external oblique aponeurosis (i.e. in the subcutaneous tissue).
3. Retractile testicle is a variant of normal; the testicle otherwise being normal, there is no indication for treatment as such.
4. By definition, congenital incompletely descended/undescended testicle is one that has failed to reach the bottom of the scrotum by 3 months of age.
5. An incompletely descended or ectopic testicle is always associated with a patent processus vaginalis (which must be taken care of at the time of operation).

During interview and examination of the patient, stress or tension should be avoided. The examination room should be preferably warm. Reason: Anxiety and cold stimulate cremasteric reflex pulling the testicles up.

HISTORY

- Was / were the testicle/s visible low in the scrotum (the normal position) at the time of birth?
- Is / are the testicle/s seen to be residing spontaneously in the scrotum at least some of the time during a day?
  [The best time to observe this is while taking bath (with warm water)].

LOCO-REGIONAL EXAMINATION

Initially examine the patient in standing position
Reason:
– This is the best position to reveal whether the scrotum is of normal size and contains the testicle, or whether it is small and flat (i.e. underdeveloped) and does not seem to contain a testicle

www.Ebook777.com

– And subsequent examination is carried out in lying down position
– Initially examine the apparently normal side, and then the apparently abnormal one.

A point to be specifically noted on the apparently normal side: Is the testis normal or **hypertrophied**?

INSPECTION of the scrotum and the inguinal region

– Scrotum: Normal OR small and flat (i.e. underdeveloped)?

Does it seem to contain a testicle? If not, the testicle may be lying at the level of the root of the scrotum where it is concealed due to thick layer of subcutaneous fat.

In a 'small' boy	In an 'older' boy
↓	↓
Get the child to squat on the examination table	Ask the boy to sit on the table, with the knees pulled up against the chest

This position causes relaxation of cremaster, allowing the testicle to be seen in the scrotum

– Any swelling in the inguinoscrotal region?

PALPATION of the scrotum and the inguinal region

Is the testicle palpable in the scrotum?

If not palpable

↓

Without touching the scrotum itself, **retract the pubic skin cranially.** This maneuver tends to elevate the scrotum to reveal the testicle. **Get above the testicle and gently pull it caudally**

If palpable

↓

Is it **normal?**

Criteria of normalcy in children	Criteria of normalcy in adults
↓	↓
< 2.5 cm, vertically ovoid smooth, soft, and has characteristic sickening 'testicular sensation'	4 x 2.5 x 3 cm, vertically ovoid, lies slightly obliquely (upper pole tilted anterolaterally), the lower pole reaches the bottom of the scrotum, smooth, soft, and has a characteristic sickening 'testicular sensation'

If this maneuver fails to demonstrate the testicle	If this is possible without undue tension on the cord, and on leaving the cord, the testicle continues to occupy the normal position for sometime (a few minutes at least)
⇓	⇓
Maldescended testicle	Retractile testicle

Place flat of the fingers overlying the inguinal region in the area of superficial inguinal pouch. By gently rolling the fingertips round and round, the testicle may be felt as a spherical mobile structure. Once the testicle is located in this way, **make an attempt to deliver it down into the scrotum**. Grasp the testicle firmly by compressing the spermatic cord just above the top of the upper pole of the testicle (*the testis must never be held by itself*), and gently pull it down into the normal scrotal position.

Observations to be noted during this maneuver:

1. Can the testicle be manipulated to the bottom of the scrotum without undue tension on the cord?
2. On leaving the cord, does the testicle continue to occupy the normal position for sometime (a few minutes at least) or, does it spring back to the higher (original) position?
3. Is the testis normal or hypoplastic (smaller and softer than normal)?

Inference that can be drawn at this juncture:

– Normal distance between the superior pubic crest and midpoint of the normal testis = 4–6 cm (minimum acceptable is 4 cm). If a testicle cannot be manipulated beyond this distance, then it is declared as incompletely descended

Free ebooks ==> www.Ebook777.com

– If a testicle can be manipulated down, but only with a tension on the spermatic cord, such that on leaving the cord, the testicle springs back to the higher (original) position (the testicle cannot remain in the bottom of the scrotum even for a while) ⇒ incomplete descent
– A hypoplastic testis ⇒ incomplete descent.

If the testicle cannot be palpated by this juncture, then carry out further maneuver to look for an ectopic testicle: Roll a finger over the spermatic cord as it emerges from the external inguinal ring. Follow the cord in an attempt to trace the (ectopic) testicle. The maneuver may be successful in locating the ectopic testicle. If testicle is not found in the superficial inguinal pouch (the commonest position of an ectopic testicle), then also make a search in the femoral triangle and perineum.

CLINICAL DIAGNOSIS

Principles and Pitfalls

1. Normally, both testicles are in the scrotum by the age of 6 months.
2. In neonates, descended (normal) testicles are easy to see through the flaccid scrotum and very thin skin.
3. In infancy, 80% of non-apparent testicles are retractile.
4. Size of the normal testis hardly changes between 6 months and 10 years of age. It starts growing rapidly with the onset of puberty. On this background, make an assessment for a hypertrophied testis.
5. In later childhood and puberty the incomplete descent of the testicle frequently remains unrecognized. In a few cases the presence of a hernia, pain in the inguino/scrotal region, or acute torsion direct attention to the abnormality.
6. In small children, there is a tendency to underestimate how far superior to the scrotum is the superficial inguinal pouch; that may lead to (a) failure to locate an ectopic testicle, and (b) misdiagnosis of an enlarged lymph node for a testicle.

To be able to categorize the problem and consequently decide on the correct management, the following questions need to be answered satisfactorily:

1. Is the problem unilateral/bilateral?
2. Is the testicle palpable / intermittently palpable / impalpable?
3. If palpable,
   – What is the exact site?
   – How much of the greatest distance can be achieved by manipulation (in c/o a maldescent) between the pubic crest and the midpoint of the testis?
4. If impalpable: Is the testis present at all? And if present, where is it?: An imaging investigation is usually necessary to answer this question.
5. Is the side of the scrotum under consideration—normal or underdeveloped?
6. Contralateral testis if present in the normal location (i.e. scrotum)—normal / hypertrophied?

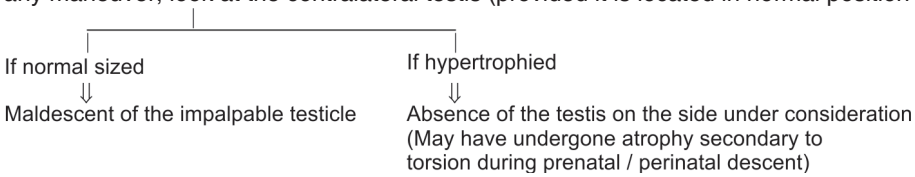
Following conclusions can be drawn to arrive at the clinical diagnosis, by combining inferences of various points in history and physical examination:

1. A testicle that is intermittently palpable in a well-developed scrotum (this can be also put as 'intermittently impalpable') ⇒ a retractile testicle.
2. A testicle that may be intermittently palpable in the area of the superficial inguinal ring, with the scrotum of the concerned side being relatively less-developed ⇒ an incompletely descended testicle. Such a testicle may be intermittently palpable due to its movement into and out of the inguinal canal. At times, this may be difficult to differentiate from an ectopic testicle in the superficial inguinal pouch.
3. A testicle (normal sized or hypoplastic) in the inguinal canal is not palpable as there is no resisting floor underneath and also, it is shielded by external oblique aponeurosis.
4. A palpable testicle in the inguinal canal with underdeveloped ipsilateral hemiscrotum ⇒ an incompletely descended testicle and the testis may be assumed to have been (pathologically) enlarged.

www.Ebook777.com

5. An impalpable testicle ⇒ a testicle that is in the inguinal canal / intra-abdominal / atrophic / absent.

If a testicle is impalpable at all times, and during physical examination if it cannot be brought into view by any maneuver, look at the contralateral testis (provided it is located in normal position i.e. in the scrotum).



In practice, most impalpable testicles are in the inguinal canal or close to the internal inguinal ring.
An impalpable testis is associated with higher incidence of sequelae/complications, than a palpable maldescended one. An incompletely descended, or an ectopic testicle is always associated with a patent processus vaginalis.

Sequelae / complications of incomplete descent

1. Pain—particularly in inguinal testis, due to oft-repeated trauma.
2. An associated oblique inguinal or interstitial hernia. In adolescent and adult patients it is frequently the hernia that causes the symptoms.
3. Epididymo-orchitis. Right-sided epididymo-orchitis in an incompletely descended testis can be difficult to differentiate from acute appendicitis.
4. Torsion.
5. Sterility in bilateral cases.
6. Increased liability to cancer.

Hazard of ectopic testicle: Liability to injury.

DIFFERENTIAL DIAGNOSIS

Retractile testicle

- The scrotum is normal
- A h/o the testicle being visible low in the scrotum (i.e. normal position) at birth may (should) be available
- The testicle is seen to be residing spontaneously in the scrotum at least some of the time during a day, usually while taking bath (with warm water; not cold water)
- The testicle can be brought down to the bottom of the scrotum without undue tension on the cord, and on leaving the cord, it continues to occupy the normal position for sometime (for a few minutes at least)
- The testicle is normal in size
- The condition is usually bilateral.

(Unilateral) Incompletely descended testicle

- The contralateral hemiscrotum and the testis are normal
- Scrotum on the affected side is underdeveloped and empty; the testicle is impalpable
- Occasionally however, the testicle may be intermittently palpable in the area of superficial inguinal ring. If at all such a testicle can be manipulated down into the scrotum, it is possible only with a tension on the spermatic cord such that on leaving the cord the testicle springs back to the higher (original) position (i.e. the testicle cannot remain in the bottom of the scrotum even for a while)
- In practice, most incompletely descended testicles lie in the inguinal canal or close to the internal inguinal ring.

Ectopic testicle

- Scrotum on the affected side is underdeveloped and empty
- The ectopic positions include: Superficial inguinal pouch (most common), femoral, perineal, pubopenile
- The testicle itself is normal otherwise (well-developed)
- Typical 'testicular sensation' on pressing the swelling **very gently** is also preserved
- The spermatic cord is normal, or bulkier and longer.

Free ebooks ==> www.Ebook777.com



Fig 26-1 Most probably, the absence of (unilateral) testicle



Fig 26-2 Bilateral incompletely descended testicles (Cryptorchidism)

Notes

- Appreciation of shape, size and consistency will depend on the plane in which the testicle lies—superficial or deep
- Associated with an interstitial hernia.

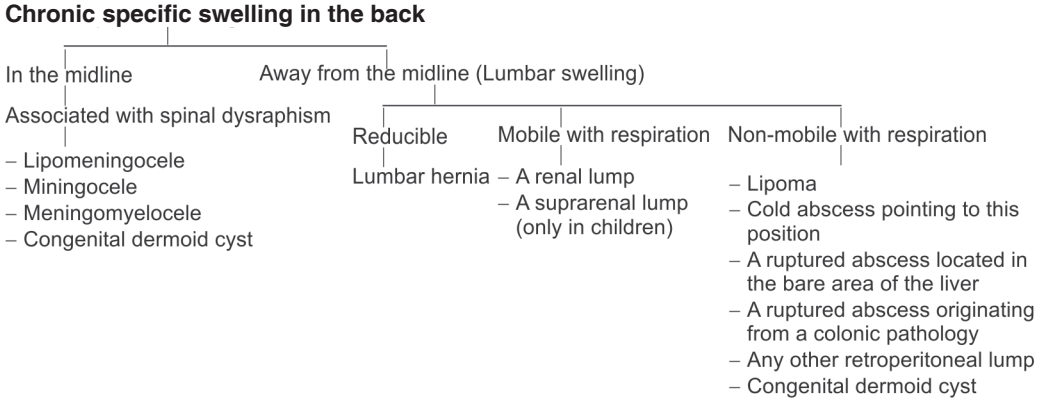
Most probably, the absence of (unilateral) testicle

- The contralateral hemiscrotum is normal and the testis is hypertrophied
- Scrotum on the affected side is underdeveloped and empty
- The testicle is impalpable at all times.

www.Ebook777.com

27 CHRONIC SWELLING IN THE BACK

Chronic specific swelling in the back



NOTES ON SELECTED SWELLINGS

Lipomeningocele

= lipoma occurring in association with spinal dysraphism.

Such lipomas range from purely intraspinal swellings to very large masses extending along with neural tissues through the bony defect. All are adherent to the conus and closely related to the lumbosacral roots, preventing complete removal and increasing operative hazards.

Meningocele

- 80% occur in lumbosacral region
- Size: A small bulge to an enormous protrusion
- The skin overlying the swelling may be normal or occasionally ulcerated
- Consistency: Usually soft, and fluctuation present
- Occasionally, it may be tense, or becomes tense when the baby cries
- Cross-fluctuation between the swelling and the anterior fontanels ±
- Impulse-on-crying / coughing ±. The absence of expansile impulse does not rule out the possibility of meningocele, as the neck may be extremely narrow, and the communication practically closed
- Mild hydrocephalus ±.

Known predisposing factors: Family history, teratogens e.g. sodium valproate.

Meningomyelocele

- The spinal cord and roots protrude through the bony defect and lie within a cystic cavity, lined with meninges and / or skin
- In most patients, the meningeal covering rupture and the spinal cord and roots lie exposed to the air
- The swelling is always accompanied by neurological deficit
- Mortality is high.

Free ebooks ==> www.Ebook777.com



Fig 27-1 Sacrococcygeal teratoma



Fig 27-2 Thoracic meningocele

Lumbar hernia

Gains exit through the lumbar triangle.

- A h/o reducible / irreducible swelling usually with an insidious mode of onset
- H/o exciting factor viz. raised intra-abdominal pressure either continuously or intermittently ±
- Expansile impulse-on-coughing
- Peristalsis may be visible across the skin in case of an enterocele.
- Hernia may be controlled on occlusion of the defect;—the test applicable to only a reducible hernia.

Lumbar cold abscess

- Diffuse hemispherical swelling
- Impulse-on-coughing +, but rather vague
- Partially reducible
- Features of chronic osteomyelitis of spine: Deformity, rigidity, tenderness
- X-ray of the spine may show features of tuberculous osteomyelitis.

Differentiating features	*Lumbar hernia*	*Lumbar cold abscess*
– Impulse-on-coughing	Expansile	Rather vague
– Reducibility	May be completely reducible, unless complicated	Only partially reducible

Renal lumps – Refer to chapter 35.

www.Ebook777.com

28 CHRONIC SWELLING IN THE PERINEUM AND GLUTEAL REGION

CHRONIC SPECIFIC SWELLING IN THE PERINEUM

1. **Perineal hernia** (*Syn.* **Hernia through the pelvic floor**)
   a. A hernial swelling gaining exit through the **scar after resection of rectum**.
      = *Postoperative hernia through the perineal scar*.
   b. A **perianal** hernial swelling
      = *Posterolateral perineal hernia*: Hernia passes through levator ani to enter the ischiorectal fossa.
   c. A hernial swelling of the **labium majus**
      = *Anterolateral perineal hernia.*
2. **Cyst of bulbourethral gland**
   → a unilateral swelling in the anterior part of the perineum.
3. **Chronic periurethral abscess** (In the male)
   – Almost invariably the result of a long-standing stricture of the bulbous urethra
   – *Possible complication*: Urethral fistula either spontaneously, or as a result of incision of the abscess
   – An indolent periurethral abscess that after incision not only fails to heal, but if the periurethral induration increases, think of the possibility of **carcinoma of the urethra**.
4. **Urethral diverticulum**—in children
   Manifestations: Difficulty in micturition, hematuria, a palpable swelling in the perineum or in the penoscrotal region.
   In a few cases, the symptoms may appear 'out of the blue' or after minor trauma.
5. **Condylomata**
   – Situated at mucocutaneous junctions e.g. vulva, penis, scrotum, anus, mouth
   – Fungating sessile growth / round or oval papules covered by a gray exudate with moist sodden surface and typical (offensive) smell
   – Usually due to syphilis. Contagious.
6. **Condyloma acuminatum = venereal warts**
   – Situated around the genitalia
   – A single or multiple cauliflower-like dry masses of variable size
   – Usually due to gonorrhea.

CHRONIC SPECIFIC SWELLING IN THE GLUTEAL REGION

1. **Gluteal aneurysm**
2. **Hernia:**
   – **A gluteal** hernia passes through the *greater* sciatic foramen, above / below the piriformis
   – **A sciatic** hernia passes through the *lesser* sciatic foramen.
3. **Tumor**
   – Benign e.g. lipoma
   – Malignant e.g. fibrosarcoma.
4. **A cold abscess**: Tuberculous / nonspecific.

Free ebooks ==> www.Ebook777.com

29 SWELLINGS / BULGES RELATED TO VULVA / VAGINA / URETHRA

GENERAL REMARK

The presence of a painless mass in the vagina is always of concern because of the possibility of cancer.

DIFFERENTIAL DIAGNOSIS

Vulval varicosities

Symptoms:

– Pain or heaviness
– Occasionally, bleeding during or after intercourse.

Pathologic anatomy: The varicosities arise from within the pelvis from branches of the internal iliac vein.

Carcinoma of vulva

– Ulcerated lesion with typical features s/o a carcinoma
  OR
– Raised red vulvar lesion in an elderly patient should raise a strong suspicion of carcinoma.

Vaginal wall (cystic) swelling

– History is generally nonspecific
– In most cases, discovered incidentally during routine vaginal examination
– (When not complicated by infection) Non-tender, well-defined, mobile
– Cancer should always be ruled out.

Vaginal wall (cystic) swellings have differing causes. Clinical diagnosis may be guessed from location of the swelling.

Location	*Possible diagnosis*	*Remarks*
– Anterior and lateral wall	Gartner cyst	However, may be present at any place
– Lateral introitus, just medial to the labia minora. It may encompass the entire lateral vaginal wall	Bartholin cyst	May be small or large If infected, and if abscess seems to have been formed, look for pus coming out of the duct or erythema around the duct opening
– (Mainly) Posterior	Sebaceous cyst	Hemispherical, small, firm nodules in the labia Punctum ± Sometimes yellowish in color
– Upper 3rd of vagina. Single / multiple	Adenosis	–
– Any place. Cyclic changes	Endometriosis	–

Cystocele

– A bulge of the anterior vaginal wall, together with the bladder above it; caused by weakening of the supporting tissues
– The upper 2/3rd of the vaginal wall is involved.

www.Ebook777.com

Cystourethrocele
The entire anterior vaginal wall, together with the bladder and the urethra is involved in the bulge. A groove sometimes defines the border between urethrocele and cystocele, but is not always present.

Rectocele
Herniation of the rectum into the posterior wall of the vagina producing a bulge. Results from a weakness or defect in the endopelvic fascia.

Urethral caruncle
– Common in middle-aged and elderly women
– Symptoms: Pain on micturition, intercourse. Bloody spotting even with mild trauma
– A raspberry-like pedunculated, granulomatous, soft mass about the size of a pea
– Attached to the posterior urethral wall near the external meatus
– Usually asymptomatic
– Symptoms when present: Increased frequency, pain during and after micturition, blood-stained discharge independent of micturition.

DD
1. Carcinoma of the urethra: Felt as thickened or nodular indurated mass.
2. Prolapse of the urethral mucosa: Insert a probe very gently. The mass is exquisitely tender, and bleeds readily.

Prolapse of urethral mucosa
– Congenital / acquired
– Age-groups at which acquired prolapse is more common: Before menarche / past menopause
– Asymptomatic, unless associated with senile urethritis, or if more than half of the circumference prolapses. Local discomfort, especially on walking
– Appears as a swollen red ring around the urethral meatus
– Position of the external urethral meatus?:
  - Central ⇒ complete prolapse
  - Eccentric ⇒ partial prolapse
– In a young girl, keep in mind another possibility of prolapse of a (ectopic) ureterocele.

Cyst of a minor vestibular gland (Skene)
– Patient may be otherwise asymptomatic, or develops symptoms due to complication
– Voiding dysfunction
– Dyspareunia
– Urethral discharge
– Recurrent UTI not responding to therapy
– Infection of the cyst and consequently formation of an abscess

The abscess may drain into urethra. Re-entry into urethra thus forms a diverticulum
The abscess may drain into vagina forming a small fistulous tract.

Urethral diverticulum
– Severe urethral pain
– Urethral discharge
– Voiding dysfunction
– Distal urethra may appear inflamed. Pus can be drained by pressing the periurethral area
– In some cases a small / large tender mass may be found.

Condylomata: Refer to chapter 28.

Condyloma acuminatum = venereal wart: Refer to chapter 28.

Free ebooks ==> www.Ebook777.com

30 DISORDERS OF THE FEMALE BREAST

GENERAL REMARKS

1. The female breast lies against the anterior chest wall. It extends from the clavicle and 2nd rib down to the 6th rib, and from the sternum across to the midaxillary line.
   Its surface area is **generally** (vertically) **rectangular rather than round**.
2. The normal adult breast may be soft, but it often feels granular/nodular/lumpy. This uneven texture is normal and may be termed 'physiological nodularity'. It is often bilateral. It may be evident throughout the breast or only in parts of it. The nodularity may increase premenstrually—a time when breasts often enlarge and become painful/tender.
3. *Changes in breasts with aging*: Breasts tend to diminish in size as glandular tissue atrophies. They often become flaccid and more pendulous. The ducts surrounding the nipple may become more easily palpable as firm, stringy strands.
4. The best time for examination of breasts is 5–7 days after the onset of menstruation.
5. To describe/document clinical findings, the breast may be divided in 5 regions viz. 4 quadrants and the central areolar region. The 4 quadrants are: superomedial, inferomedial, inferolateral, and superolateral including axillary tail. The superolateral quadrant contains a large amount of glandular tissue; this is the most common quadrant where 'cyclical nodularity' and cancers develop.
6. A practical dictum worth remembering: "Any swelling in the breast should be considered malignant, unless proved otherwise".
7. Fibrous tissue develops around carcinomatous cells. Collagen fibers in the fibrous tissue contract. These pathological aspects lead to the following signs in a case of CA-breast:
   a. Change in the direction of nipple. The nipple deflects in the direction of the tumor.
   b. Retraction of the nipple—due to pull on the lactiferous ducts.
   c. Dimpling of the skin—due to pull on Cooper's ligaments.
   d. 'Orange peel' appearance of the skin—due to obstruction of subcutaneous lymphatics leading to peculiar cutaneous edema.
   e. Hard consistency—due to fibrous tissue.
8. Although a long duration with slow progress suggests benign tumor (apart from cystic swelling), an atrophic scirrhous carcinoma may be present for years.
9. An inflammatory carcinoma, which is usually rapidly growing, may be mistaken for an abscess, and inadvertently incised!
10. Sometimes, granulomatous type of TB leads to a swelling, which is clinically indistinguishable from carcinoma.
11. Paget's disease represents a tumor of lower-grade malignancy and its prognosis is generally good.
12. Quite apart from the period of lactation, the adult female breast secretes a small quantity of fluid at all times. This fluid passes along the lactiferous ducts to the nipple, and there it emerges on to the surface epithelium to be removed by evaporation and by contact with clothes, or to be washed off in the bath. In normal circumstances a woman is unaware of this secretion. Occasionally, however, the amount of secretion may increase. In such cases (which are presumably physiological), or if the cause of discharge is pathological, then the woman will notice a variable amount of moisture appearing at one or both nipples and she will present with "discharge from the nipple/s".

www.Ebook777.com

13. The most important pointer to the diagnosis of breast disorders is the age of the patient.
14. All palpable lesions should be measured with callipers and the dimensions and site (using a clock system) recorded in the document.
15. Clinical assessment of axillary lymph nodes is not always accurate.

Symptoms of breast disorder

- Swelling
- Discomfort
- Chronic pain (Chronic mastalgia)
- Withdrawal of the nipple
- Discharge from the nipple
- Eczema of the nipple-areola
- Cracked nipple
- Pain and hypersensitivity in the nipple
- Small size
- Big size
- Asymmetry
- Amazia = congenital absence of breast
- Polymazia = accessory breast anywhere along the milk-line viz. axilla, groin, buttock, thigh; the most frequent site is axilla.

SWELLING IN (THE REGION OF) THE BREAST

HISTORY

- **Age**?

  Age incidence of various swellings
  - Teens and early twenties: Fibroadenoma
  - 20s and 30s: Benign nodularity
  - 40s and early 50s: Cyst/s.

  Although cancer occurs most frequently between 45 and 55, it could occur at any age after puberty. In practice, one should not rule out possibility of a cancer on the basis of age alone.
- **Swelling**?
  - Exact site?
  - Duration?
  - Mode of onset?
  - Progress?

    A slow progress does not rule out possibility of cancer, e.g. an atrophic scirrhous CA may be present for years.
  - Did it regress in size anytime during the course?
  - Does it fluctuate in size? An example of a swelling that fluctuates in size—benign nodularity.
- **Pain**?
  - Exact site? Does it radiate?
  - Duration?
  - 'When did it begin? Did it begin before the appearance of the swelling, OR Did it begin after the appearance of the swelling?'
  - Is it related to periods? How many days does the pain last?
  - Do the episodes of 'pain related to periods' 'wax and wane'? If so, how long do the episodes last?
  - Intensity?
  - Nature?
  - Precipitating, aggravating, relieving factors?

Free ebooks ==> www.Ebook777.com

For cancer of the breast

- Fever?
- Significant loss of weight?
- 1. Previous history of breast cancer?
  2. Family history of breast cancer, especially in 1st degree relatives?
     Sister, mother and daughter constitute the 1st degree relatives.
     Grandmother, granddaughter, aunt and niece constitute the 2nd degree relatives.
  3. Consumption of high estrogen-containing OC pills?
     Did you use OC pills before the age of 20?
     The risk is elevated during, and for 10 years after the use of pills, and with the use of pills before the age of 20.
  4. What was your age at the time of menarche?
     Age at menopause?
     Younger age at menarche, and older age at menopause increase the risk.
  5. At what age did you have your 1st pregnancy?
     How many pregnancies?
     Did you breastfeed the babies?
     Later age at 1st pregnancy (age over 30), lesser number of pregnancies, and the absence of breastfeeding increase the risk.
  6. Did you take HRT (Hormone replacement therapy)?

  – High-risk factors for development of CA breast

- 1. Axillary or/and supraclavicular swelling?—lymphadenopathy—with clinical features typically s/o mets viz. duration in weeks, rapid progress, often painful, hard consistency. Manifestations of brachial plexus infiltration ±.
  2. Anorexia, nausea, lethargy? ⇒ hepatic mets.
  3. Bone pains? ⇒ mets to bones, OR malignant hypercalcemia.
  4. (Pathological) Fracture? ⇒ mets to bones.
  5. Abdominal symptoms including pains, nonspecific deterioration of health, confusion, dehydration? ⇒ malignant hypercalcemia.
  6. Anemia, (repeated) infections (due to leukopenia)? ⇒ (red) bone marrow involvement.
  7. Breathlessness? ⇒ pleural effusion due to pleural mets/pulmonary (parenchymal) mets/ascites pushing up the diaphragm.
  8. Dry cough? ⇒ pulmonary (parenchymal) mets.
  9. Distension of abdomen (ascites)? ⇒ peritoneal mets.
  10. Manifestations of intracranial SOL? ⇒ mets in the brain.

  – For regional / distant metastatic disease

LOCOREGIONAL EXAMINATION

Girls and women may feel apprehensive during examination of breasts. Be reassuring and adopt a courteous and gentle approach.

EXPOSURE: Both breasts must be completely exposed before examination is commenced. However, later in the course of examination, it may be appropriate to cover one breast while the other one is being palpated.

POSITION of the patient: To begin with, sitting with arms hanging by the side. Reason: Slight elevation of the breast (as inferred from a change in the level of nipples, on comparison) in an early cancer is better appreciated in sitting position.

www.Ebook777.com

INSPECTION

Methods

1. From **above the head** of the patient—compare the size and contour of the breasts.
2. From **front**—compare the size and contour of the breasts, and also the level of the nipples.
   Cancer can → both increase, **as well as** a decrease in size of the breast
   Compare the contours by starting inspection from the anterior axillary folds of both sides and ending in the midline. A bulge or an indentation may suggest a pathology underneath
   Raised level of one nipple suggests subareolar fibrosis due to any cause, frequently a carcinoma
   Conclusion: Asymmetry in size of the breasts, when pathological, suggests definite presence of abnormality in either of the 2 breasts.
3. **Hands raised above the head** to look for asymmetry in the level of nipples, and also to look for dimpling in the skin which might not have been evident otherwise, with the hands by the side. Raising the hands puts lactiferous ducts and suspensory ligaments on a stretch.
4. **Bending forwards** from the waist so that the breasts fall away from the body. Failure of one nipple to fall away from the chest suggests abnormal fibrosis behind the nipple, the pathological process extending down (i.e. deep) to the plane of pectoralis major at least.



Fig 30-1 Note the findings on comparison

Preliminary interpretation of asymmetry in the size of 2 breasts

- Developmental
- Pathological*
  - Large size
    - 'Moderate' ⇓
      1. A swelling in the breast
      2. Any of the causes of 'massive' enlargement during their evolution
    - 'Massive' ⇓
      (A) Massive swelling in the breast
      1. Giant fibroadenoma
      2. Phyllodes tumor
      3. Malignant tumor, particularly sarcoma, medullary carcinoma
      4. Other e.g. cystic hygroma, rarely, galactocele, etc.

      (B) Massive enlargement of the breast itself
      1. Diffuse hypertrophy
      2. Filarial elephantiasis
    - 'Apparent'
      1. Due to scoliosis
      2. Due to retromammary swelling
  - Small size
    The side of small size could be the seat of a disease!
    An example: Scirrhous CA

*' Pathological asymmetry' occurs at a later time; i.e. anytime after the development of the breasts to a definitive size and shape at the end of puberty having been achieved

– Although many of the pathological conditions lead to an increase in size, some of the conditions **may lead to a decrease in size** of the breast, e.g. scirrhous carcinoma.

SEX MATURITY RATING IN GIRLS ON THE BASIS OF DEVELOPMENT OF BREASTS

Stage 1: Preadolescent—elevation of nipple only.
Stage 2: Elevation of breasts + nipple as a small mound; enlargement of areolar diameter.
Stage 3: Further enlargement of elevation of breast and areola, with no separation of their contours.
Stage 4: Projection of areola and nipple to form a secondary mound above the level of breast.
Stage 5: Mature stage—projection of nipple only. Areola levels with general contour of the breast, although in some normal individuals the areola continues to form a secondary mound.

Free ebooks ==> www.Ebook777.com



Fig 30-2 Retracted nipple

Inspect the apparently normal breast first, followed by the abnormal side.

1. **Nipple**
   - **Change in the direction**?
     Deflection occurs in the direction of malignant tumor.
   - **(Unusual) Prominence**?
     It may be due to an underlying swelling.
   - **Withdrawal**?
     Withdrawal can occur due to 'inversion' or 'retraction'.

Inversion	v/s	*Retraction*
= Instead of protruding normally, the nipple lies in a pit in the areola		= Withdrawal of the previously upstanding nipple.
Often only partial and slit-like		– Slit-shaped OR circumferential?
		⇓ ⇓
– Otherwise normal, i.e. not broadened, distorted, thickened or fixed.		Periductal mastitis CA. – Distorted/thickened? ⇒ CA.

Cause of 'inversion' is unknown. For unknown reason, the nipple does not develop with the breast; nipple lies in a pit in the areola. There is no underlying ominous pathology

Causes of 'retraction': Periductal mastitis associated with mammary duct ectasia, CA

Basic mechanism of retraction—pull on the lactiferous ducts.

- **Fissure (*syn.* Crack)**?
  Cause: Neglect of hygiene especially during lactation.
- **Rash**?—may suggest Paget's disease.
- **Ulcer**?
  Complete destruction with replacement by a red flat ulcer = Paget's disease.
- **Crusting**?
  Crusting could simply be the dried products of secretion—occurs in a patient having 'discharge from nipple'.
- **Eczema**?
  Rare. Bilateral
  Important differential diagnosis: Paget's disease. Paget's disease is an unusual form of ductal CA.

2. **Areola**
   - **Size**?
     – Enlarged, with the development of secondary areola: This can be due to pregnancy
     – Decrease in size around a retracted nipple ⇒ CA

www.Ebook777.com

- **Color**?
  Darkening, with development of 'Montgomery's tubercles': In pregnancy
  Bright red may suggest early stage of Paget's disease
- **Dimpling (= Puckering)**?
  Refer to dimpling of skin. The same discussion applies to dimpling of the areola.
- **Orange peel appearance**?
  Areola is the commonest site where orange peel appearance occurs first.
- **Rash**?
- **Ulcer** ?
- **Eczema**?

} Refer to '1. Nipple' above. The same discussion applies to these features affecting the areola.

3. Skin

- **Color**?
  Red and shiny (Red, tense and glossy)? - Causes: - Acute inflammations
  - Rapidly growing tumor like inflammatory CA.
- **Dimpling (= Puckering)**?
  Mechanism: Fibrosis involving Cooper's ligaments due to any cause, e.g. CA, TB, fat necrosis → contraction and shortening of the ligaments → abnormal traction on the skin → dimpling (= puckering)
  Dimpling ⇒ infiltration of the ligaments of Cooper; *not* of the skin directly
  Clinical significance: In small lesions, especially in patients with large or pendulous breasts, dimpling may be the earliest sign.
  Ask the patient to raise her arms above the head
  - Dimpling, otherwise not evident with hands by the side, may become evident
  - Dimpling evident with hands by the side may become accentuated

  Mechanism: Raised arm puts more traction as compared to when the arm lies by the side.
- **Orange peel appearance**?
  Orange peel appearance becomes more obvious *by gently squeezing the skin*
  Mechanism: Cutaneous edema leading to swelling of skin except at the hair follicles, which appear as deep pits. Orange peel appearance usually suggests infiltration of the skin by CA
  It can also result form axillary tuberculous lymphadenitis.



Fig 30-3 Orange peel appearance

- **Fungation**?
  ⇒ infiltration of skin by CA
  Conditions, which may lead to an appearance of fungation (false fungation): massive swellings like giant fibroadenoma, phyllodes tumor, rapidly growing swelling like rapidly growing sarcoma, chronic abscess.
  Pathogenesis: Pressure necrosis of the overlying skin leading to ulceration, fungating appearance

  Clinical test to differentiate true fungation from false fungation: Make an attempt to pass a probe under the edge
  - If possible ⇓ False fungation
  - If not possible ⇓ True fungation—due to fungating CA

Free ebooks ==> www.Ebook777.com

- **Nodule/s**?
  ⇒ infiltration of the skin by CA.
- **'Cancer-en-Cuirasse' appearance**
  Chest wall studded with carcinomatous nodules leading to the appearance of a coat of armor. ± Brawny edema of arm
  Usually, though not necessarily, seen in cases with local recurrence after mastectomy.
- **Engorged veins**
  More commonly encountered with sarcoma than carcinoma
  Physiological cause: Venous pattern becomes increasingly visible (rather than gross engorgement) as pregnancy progresses.

4. **A swelling in the breast may not always be (big enough to be) easily visible.** However if visible, then note the details as usual.
5. **Arm**
   Edema of the arm can occur due to blocking of the lymphatics by carcinomatous fibrosis.

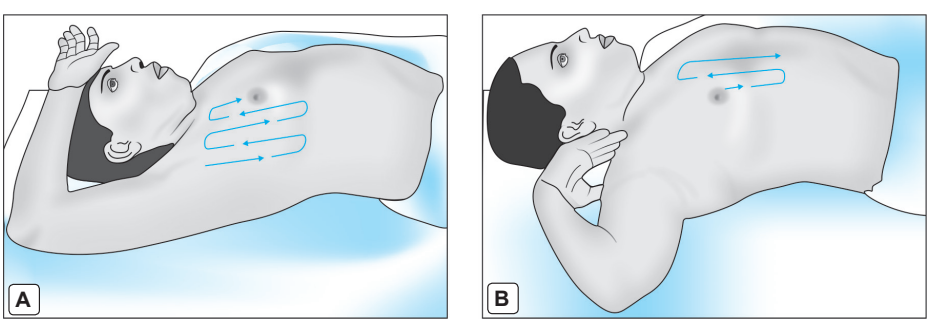
PALPATION

- **POSITION** of the patient**: Lying down**
- Reason: Breast spreads and flattens on the chest. Even a small swelling can then be detected without getting missed
- Palpation in sitting position is insensitive and inaccurate.
  – The **arm should be abducted to ~ 90°**. Reason: The breast tends to fall laterally, making the medial quadrants even more flat.
  – **General method of palpation of the breast** (excluding nipple)
    - Palpate with flat of 4 fingers close together
    - Make gentle rotating movements of the fingers in an attempt to roll and detect even a small swelling between the fingers and the chest wall
    - Palpate in a fixed sequence systematically.

  1. 4 quadrants f/b the central region.
     OR
  2. The vertical strip pattern—a better sequence than the quadrant
     ↓ sequence

  - Begin palpation in the axilla, moving in a straight line down to the bra line (/ inframammary fold)
  - Move medially and then in a vertical strip up the chest to the clavicle
  - Continue in this up-and-down vertical strip pattern between the clavicle and the bra line (/ inframammary fold) until the midsternal strip is reached, keeping in mind to release the breast to allow it to fall naturally laterally, after palpation of the lateral half of the breast is over

– **Nipple** should be palpated between the thumb and the index finger
– Specific methods of palpation of a swelling, and of a withdrawn nipple are discussed in respective sections.



Figs 30-4A and B Vertical strip-pattern of palpation

www.Ebook777.com

Steps of palpation of the breasts

A. Palpate the apparently normal breast first.

- Benign nodularity (fibroadenosis) is frequently bilateral
- Some of the other diseases may be often bilateral, e.g. fibroadenoma, cysts, etc.
- Cancer may be rarely bilateral.

B. Palpation of the abnormal breast

1. The distorted area

- Temperature?
- Tenderness?
- Extent and size?
- Surface?
- Consistency?

2. The swelling (which may be the chief complaint) or the region suspected to have a pathology as judged on history and inspection, is grasped between the thumb and fingers.

Palpate the same swelling again with flat of the fingers

Palpate the same swelling again with flat of the fingers.

If the swelling is palpable to the same extent with roughly the same features as when palpated between thumb and fingers

⇓

True swelling

↓

Palpate in details in the usual manner
Use tips/pulps of fingers as necessary to obtain finer details

Not palpable
Instead, diffuse shotty or granular surface is felt, and suppleness of normal breast is replaced by elastic consistency (in the affected region)

⇓

(a) Nodularity
(b) Normal breast containing milk

Two points specifically applicable to a c/o 'swelling in the region of the breast' are discussed below.

Impulse-on-coughing?

Impulse-on-coughing may suggest that the swelling may be an intercostal cold abscess or empyema necessitans, rather than a swelling arising from the breast.

Anatomical plane and fixity?

a. Fixity *v/s* tethering to the skin.
   - Fixity to skin, as determined by pinching, gliding and lifting up tests ⇒ infiltration of the skin, OR a swelling arising from the skin
   - Normally, a swelling of the breast can be moved independently of skin for some distance—the distance being < the radius of (shortened) ligaments of Cooper. Forced movement of the swelling beyond this distance leading to dimpling is called 'tethering'. Tethering ⇒ involvement of Cooper's ligaments by a chronic inflammatory process or cancer.

b. Fixity to the breast tissue.
   Fix the breast with one hand and move the swelling with the other
   Method of fixing the breast: Place the thumb and fingers on the breast much beyond the margins of the swelling, and press the breast against the chest wall
   Restricted or absence of mobility of the swelling with dimpling in the skin may suggest CA.

c. Fixity to muscle: After putting the appropriate muscle in 'isometric' contraction,

↓

   - Look at the size of the swelling—Does the swelling become more prominent/less prominent/remain unaltered?
   - Does that produce any dimpling in the skin? Appearance of dimple suggests that the swelling infiltrates both the pectoralis major and the skin.
   - Is the mobility of the swelling restricted?

Free ebooks ==> www.Ebook777.com

- Slight restriction of mobility ⇒ tethering of the swelling to pectoral fascia
- Gross restriction of mobility ⇒ direct infiltration of pectoralis major muscle.

(The inferolateral quadrant is related to serratus anterior muscle. Rest of the whole breast is related to pectoralis major.)

d. Fixity to the chest wall.

If the swelling is fixed irrespective of contraction of muscle, then it means that it is fixed to the chest wall

Chest wall includes ribs, intercostal muscles and serratus anterior muscle, but not the pectoral muscle.

3. Further palpation of **the rest of the breast** is carried out with flat of the fingers.

a. Engorgement in a lactating lady?

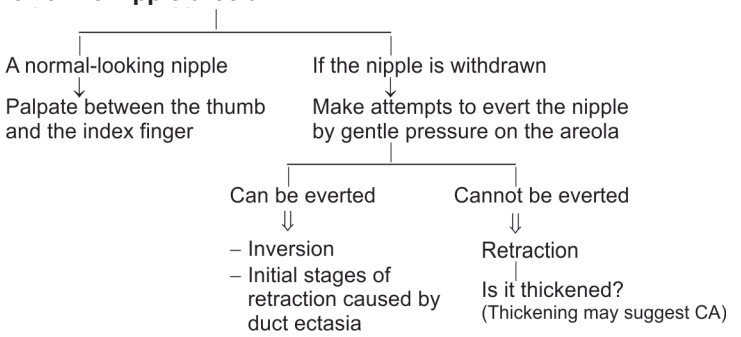
It may be caused by: - Inadequate drainage with residual milk
- Blockage of lactiferous ducts by epithelial debris.

Usually occurs about the weaning time.

b. Pitting edema of skin?—may suggest acute bacterial mastitis, which has most probably progressed to form an acute pyogenic abscess, provided other clinical manifestations support the possibility.

c. Tender cords? ⇒ mammary duct ectasia.—a benign but sometimes painful condition of dilated ducts.

4. Palpation of the **nipple-areola**



5. **Regional examination**

Examine the axillary, supraclavicular **and inguinal** groups of lymph nodes

Lymph from the skin of the breast may be drained to the opposite breast and the inguinal lymph nodes via the lymphatics passing along the abdominal wall.

6. **Examination of the arm** for edema

Edema can occur due to blocking of the lymphatics by carcinomatous fibrosis.

IF THERE IS AN ULCER, examine it in the usual way.

IF THE BREAST IS FOUND TO BE NORMAL, examine the spine for scoliosis.

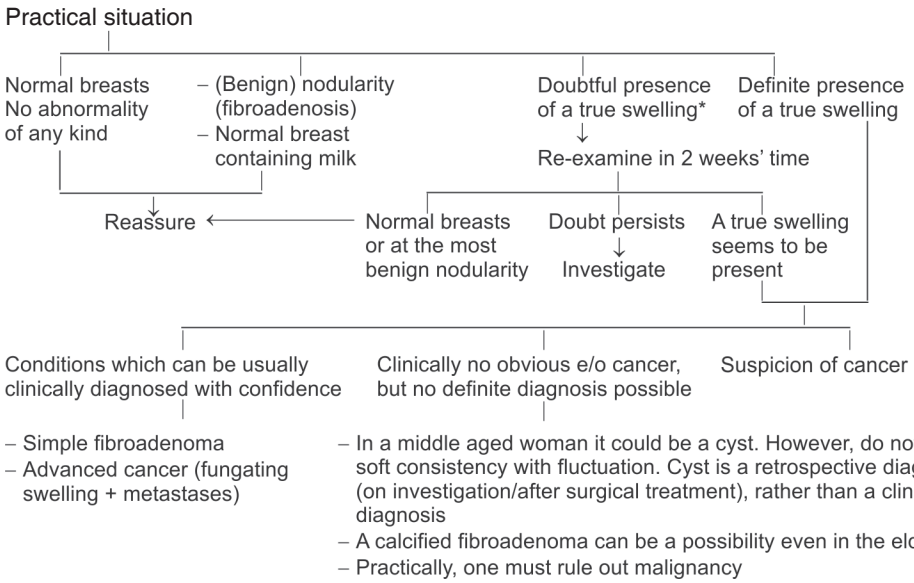
PITFALLS in interpretation of physical findings:

1. When pressing deeply on the breast, a normal rib may be mistaken for a hard swelling in the breast.
2. Nodules in the tail of the breast may be mistaken for enlarged axillary lymph nodes and vice versa.
3. There may be a firm transverse ridge of compressed tissue along the lower margin of the breast, especially in large breasts. This is a normal inframammary ridge, NOT a tumor.
4. Carcinoma usually leads to single swelling.

PRELIMINARY IMPRESSION

It should be possible to arrive at a 'preliminary clinical impression' at this juncture.

www.Ebook777.com



* This is a relatively frequent circumstance. There is a wide spectrum in the degree of 'lumpiness' of normal breast tissue and areas where the irregularity of texture is extreme can be, on first examination, impossible to differentiate clearly from a true swelling. Since the lumpiness seems to depend on the phase of the menstrual cycle, usually being worst during the premenses, it is reasonable to ask the patient to return for re-examination in 2 weeks' time i.e. at the opposite phase of her menstrual cycle.
During pregnancy, breasts become tender and nodular; a pathological mass may be difficult to isolate.

RE-EXAMINE (especially palpation) AFTER EMPTYING THE BREAST OF MILK, where applicable.

ARRIVING AT THE DIAGNOSIS of a swelling in the region of the breast

PRINCIPLES AND PITFALLS

1. Well-defined margin/regular shape/smooth surface/soft consistency does not necessarily rule out cancer.
2. Distortion of the breast rather than a swelling may suggest an invasive lobular carcinoma.
   Diffuse infiltrating nature of invasive lobular carcinoma → distortion of the breast rather than a well-circumscribed swelling.

Free ebooks ==> www.Ebook777.com

STEPWISE ANALYSIS OF DATA

Step 1

- Swelling in the region of the breast, excluding the nipple
- Benign swelling of the nipple
  - Cyst: e.g. sebaceous cyst
  - Adenoma
  - Papilloma (Fig 30-5)
  - Fibroepithelial polyp



Fig 30-5 Duct papilloma

Step 2

- Acute
  - Acute mastitis
  - Acute retromammary abscess, etc.
- Chronic

Step 3

1. Arising from skin/fat of the superficial layer of the superficial fascia,* or from the chest wall
2. Arising from, and confined to the breast
3. Arising from retromammary space
4. Arising
   (a) from the breast and infiltrating the underlying muscles,
   OR (b) basically from the pec major/serratus anterior,
   OR (c) from axillary tail of the breast,
   OR (d) from any of the axillary soft tissue other than axillary tail of the breast, e.g. lipoma / fibroma / enlarged lymph node etc.

Discussion

Basically, when a patient presents with a history of 'a swelling in the breast', one should not take it for granted that the swelling is arising from the breast. It may be arising from any structure in any plane from skin to chest wall bounded by the perimeter of the breast.

Differentiating features under the 1st heading (in step 3 above) are straightforward. *: A swelling arising from the skin or the superficial layer of superficial fascia moves with the skin i.e. it does not have a significant mobility independent of skin. And a swelling arising from the chest wall will be fixed irrespective of contraction of muscle.

Clinically, it is difficult to differentiate between a swelling arising from the breast and a swelling arising from the retromammary space.

Regarding the 4th heading: Clinically, it is not possible to differentiate (a) from (b), and (c) from (d).

However, as per the dictum—'a swelling arising in a specific anatomical area should be considered to be arising from the organ/tissue specifically located in that anatomical area unless proved otherwise', a swelling arising in the region of the breast should be considered to be arising from the breast unless proved otherwise. Also, a swelling arising from the breast is much more common than a swelling arising from any other tissue in this location; particularly for example, rhabdomyoma/rhabdomyosarcoma is very rare.

Step 4

Nature of the swelling arising from the breast?

- Normal breast containing milk
  Breasts start becoming nodular from the 3rd month of gestation onwards
- (Benign) nodularity

- True swelling
  - Vascular: Rare.
  - Benign: Aberration, cyst, tumor
  - Malignant: usually a (primary) carcinoma
  - Chronic inflammatory swelling
  - Miscellaneous

www.Ebook777.com

Aberrations of normal development/involution:
- Fibroadenoma group of swellings: Arise from breast lobule. There is both glandular and CT proliferation.
  - Simple fibroadenoma
  - Giant fibroadenoma
  - Phyllodes 'tumor'
- Simple cysts—represent cystic dilatation of the ducts
- Polycystoma: Cyst/s of benign nodularity (fibroadenosis).

Cysts proper:
- Galactocele
- Any other cystic swelling which can occur elsewhere in the body, e.g. cystic hygroma, blood cyst, dermoid cyst, hydatid cyst, etc.

Benign tumors:
- Cystadenoma
- Any other swelling from 'general category', i.e. which can occur elsewhere in the body, e.g. neurofibroma, etc.

Malignant tumors:
- Primary: Carcinoma, sarcoma, lymphoma
- Secondary.

Chronic inflammatory swellings:
- Chronically inflamed swelling—antibioma.
- Chronic inflammatory swelling *de novo*:
  - Nonspecific
  - Specific: TB, actinomycosis, syphilis.

Miscellaneous:
- Traumatic fat necrosis
- (Resolving) Hematoma
- Thrombophlebitis of the superficial veins of the breast and chest wall
- Filarial nodules.

Step 5: Etiology?

Step 6: Sequelae/complications?

Step 7: Associated disease—closely related/otherwise?

DIFFERENTIAL DIAGNOSIS of a chronic swelling in the breast

Distortion of the breast, rather than a well-circumscribed swelling
- That is the way an invasive lobular carcinoma presents
- Clinical diagnosis may be difficult due to diffuse infiltrating nature
- Can be bilateral.

(Benign) Nodularity
- Age: Anytime after puberty (—during child-bearing period). More prone in spinsters, childless married women and multiparous women who have not suckled their children
- Frequently bilateral
- What patient calls swelling is actually fine nodularity; each fine nodule corresponding to the size of a rice grain.

  Not a 'true swelling' meaning thereby it may appear as a (big) swelling when grasped between fingers and thumb; and the same so called swelling disappears when palpated with flat of the fingers; instead, diffuse shotty or granular surface is felt, and suppleness of normal breast is replaced (in the affected area) by elastic consistency. The finding is somewhat similar to the feel when a gunny-bag filled with rice-grain is felt from outside
- May be associated with cyclical pronounced mastalgia
- Tenderness appears, or becomes pronounced in the week just preceding menstruation
- Usually diffuse, (but may be focal).

Free ebooks ==> www.Ebook777.com

- Size of the swelling also varies with menstrual cycle. However, if severe and prolonged, then it may loose its cyclical pattern
- Axillary lymph nodes may be slightly enlarged and tender.

Notes

- Nodularity is nothing more than a variation of the normal theme of cyclical proliferation and regression. Some women are worried due to pain and swelling, that is how making it a 'disease entity'. Many women are affected without being even aware of it
- Nodularity may be diffuse or focal
- The normal breast may be lumpy (due to diffuse nodularity). Diffuse nodularity is normal; it is not associated with any underlying pathological abnormality. Diffuse nodulairty *should not be* labelled with the diagnosis 'fibroadenosis'
- Focal nodularity may be labeled with fibroadenosis (*syns.* fibrocystic disease, chronic cystic mastitis). Some selected cases of focal nodularity may have to be investigated to rule out cancer
- Nodularity is usually a self-limiting condition. Pregnancy and lactation may bring about permanent relief. Permanent relief may also be brought about after completion of menopause.

Simple fibroadenoma

- Usual age-group: 15 ↔ 25. Mean age: ~ 30. This age-span is the period of greatest growth and activity of the breast (If remains undiagnosed/untreated until menopause, then it may be detected on mammogram as a calcified structure.)
- Painless slow-growing swelling. May rapidly increase in size during pregnancy
- Usually solitary. However, can be multiple. Occasionally, the whole breast may be replaced by fibroadenomas
- Multiple recurrences possible
- Well-defined. Non-tender. Ovoid/spherical
- Size—< 5 cm. Surface—smooth or lobular. Consistency—firm
- **Freely mobile. However, mobility may be decreased in the following 2 situations:**
  1. If situated adjacent to nipple-areola.
  2. With increasing age (due to involution of fibrotic tissue)
- **In elderly, it can still present as a rather hardened, freely mobile swelling!**

Natural history:

1. Occasionally may disappear spontaneously.
2. May become a hard calcified mass.
3. Extremely rarely, malignant transformation.

Causes of recurrence (after excision):

1. A true metachronous tumor.
2. Incomplete excision/a tumor completely missed.
3. It might be actually a phyllodes tumor.

Giant fibroadenoma

- Age: Bimodal presentation. - At the extremes of reproductive life. 15 ↔ 20, and 45 ↔ 50
- Nipple: Displaced
- Size of the swelling: > 5 cm
- Skin overlying the swelling: Characteristically shiny. Dilated veins ±
- Pressure-necrotic ulcer ±.

A girl may present with the chief complaint of 'enlarged breast', not appreciating that there is a swelling inside-within the breast. Differential diagnosis in that case: 'Diffuse hypertrophy'. Differentiating features in favor of hypertrophy: There are no cutaneous or venous changes.

Phyllodes tumor

- Large size involving most of the breast
- Skin: Reddened. Later on, pressure-necrotic ulcer

www.Ebook777.com

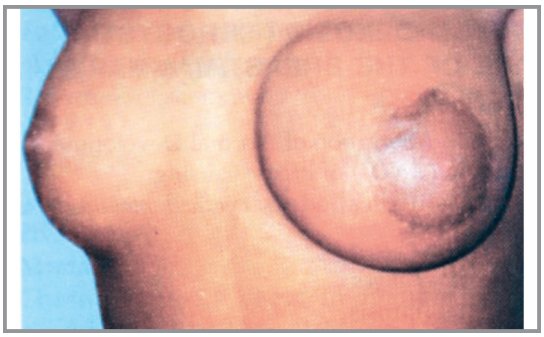


Fig 30-6 A giant fibroadenoma in a 14 year old girl



Fig 30-7 Phyllodes tumor

– Mobile, even if large
– Unevenly bosselated surface.
– *Can present as a small swelling,* clinically indistinguishable from a simple fibroadenoma.

Simple cyst/s

– Age: Classically perimenopausal—45 ↔ 55. However, can be seen outside this range, especially in women receiving HRT
– Mode of onset: Characteristically sudden. Explanation: Cyst usually exists beforehand in a flaccid state subclinically. Additional accumulation (of even small amount) of fluid → disproportionate increase in the intracystic pressure (La Place's law)
– Painless/uncomfortable/frankly painful
– Number: Usually single, but can be multiple. In extreme cases, the whole breast contains plenty of cysts
– Size: 2 mm (obviously clinically impalpable) to several centimeters
– Surface—smooth
– **Mobile, but not as freely as a fibroadenoma**.

A note on 'cyst'

Do not expect soft consistency with fluctuation. Cyst is a retrospective diagnosis (on investigation/after surgical treatment) rather than a clinical diagnosis; clinically, simple cyst can be only suspected, not diagnosed.

Clinically worrisome cysts

1. Fresh blood/blood-stained aspirate.
2. Residual mass after aspiration. (Residual mass should be aspirated for cytological examination.)
3. Cysts which recur more than 2 times after aspiration.

Duct ectasia-periductal mastitis

– About 40 years of age
– Basically presents as an acute case, followed by a painless mass adherent to skin
– Often bilateral multifocal thick opalescent discharge of varying colors
– Transverse slit-shaped retracted nipple.

Notes

Duct ectasia-periductal mastitis complex is a spectrum of inflammatory process where the subareolar ectatic enlarged ducts are surrounded by a mild inflammatory infiltrate.

Galactocele

= a retention cyst in the main duct occurring during lactation.
– Usually starts about the second week of lactation
– Single, subareolar, globular, fluctuating swelling
– Can become enormous
– Squeezing of the swelling → discharge of milk from the nipple.

Free ebooks ==> www.Ebook777.com

Hydatid cyst
- Usually single
- A relatively superficially located cyst may be brilliantly translucent.

Cystadenoma
- 35–45 years of age
- Rapid enlargement → large size
- May fungate
- Freely mobile
- Lobulated
- Partly solid and partly cystic
- No regional lymphadenopathy.

Schirrous/atrophic schirrous CA
= a clinical type of invasive ductal carcinoma
- Atrophic scirrhous CA occurs in very old patients, and enlarges very slowly over years
- Painless
- The affected breast—smaller in size than the normal one. Atrophic scirrhous CA is particularly smaller
- Irregular nodular swelling of hard consistency
- Nipple—deflected/retracted
- Regional lymphadenopathy after about 3 months of origin of the primary.

Medullary CA
= a clinical type of invasive ductal carcinoma
- Slightly painful
- Affected breast enlarged
- Nipple normal
- Consistency—firm; sometimes soft
- Skin does not show dimpling.

'Lipomatous' CA
- True lipoma of the breast is extremely rare
- A scirrhous carcinoma may sometimes contract a covering of soft breast and subcutaneous tissue around itself to mimic a lipoma
- It is, therefore, extremely dangerous to diagnose lipoma of the breast without a histological proof.

Inflammatory carcinoma
= carcinoma occurring during pregnancy/lactation. The tumor grows so rapidly that it excites an acute inflammatory reaction
- Diffuse, brawny induration of the skin with an erysipeloid edge, usually with no underlying mass
- If a mass is palpable, then the clinical features of the mass are similar to medullary carcinoma, because inflammatory carcinoma is not a special pathological type of cancer; but a cancer (invasive ductal) occurring during pregnancy/lactation.

Acute mastitis v/s inflammatory carcinoma
Features in favor of inflammatory CA:
- Intensity of pain and tenderness—lesser
- Fever without rigors
- Consistency—firm with no area of softening
- Nodularity of the surface ±
- No leukocytosis.

Sarcoma
Arises either as a malignant change in a soft fibroadenoma, or *de novo*.
- H/o a swelling of long duration with a recent rapid increase in size ⇒ malignant transformation of a pre-existing fibroadenoma

www.Ebook777.com

- Short duration ⇒ a sarcoma arising *de novo*
- Usually attains a large size
- Consistency—variegated i.e. hard in one part while soft and fluctuating in another part
- Involvement of skin occurs in late stages
- No regional lymphadenopathy until very late.

TB
- Chronic abscesses with clinical features typical of TB—Refer to chapter 15
- Sinus(es)
- Regional tuberculous lymphadenitis
- Usually associated with pulmonary TB.

Traumatic fat necrosis
- 35–50 years of age
- Stationary after initial growth
- H/o trauma (might have been forgotten). The nature of trauma can be direct (a blow, seatbelt injury), or indirect (violent contraction of pectoralis major)
- Large sized breasts
- Often tender
- Irregular
- Size—usually not > 2 cm in diameter
- Usually attached to skin, muscle.

Note: On bisection of an excised specimen, characteristic chalky white area of fat necrosis becomes evident.

(Resolving) hematoma
- H/o trauma ±
- In the uncertainty of h/o trauma and the absence of external mark of injury, diagnosis will be difficult.

D/D of CA breast:
- Focal nodularity (fibroadenosis)
- Very long-standing fibroadenoma
- Antibioma
- (Granulomatous) TB
- Traumatic fat necrosis.

DIFFERENTIAL DIAGNOSIS of chronic swelling in the skin/subcutaneous tissue overlying the breast

Spontaneous thrombophlebitis of the superficial veins of the breast and anterior chest wall
- Indurated subcutaneous thrombophlebitic cord of ~ 3 mm in diameter and of varying length
- Consistency—likened to that of ductus deference
- When the skin over the breast is stretched by raising the arm, a narrow shallow subcutaneous groove alongside the cord becomes apparent
- No antecedent trauma or infection. The cause is obscure
- Self-limiting. Subsides within a few months.

Filarial nodule/s
- Swelling/s in the subcutaneous tissue plane
- Firm consistency
- Clear-cut h/s/o filariasis may not be available
- Clinical diagnosis is not always possible, unless patient gives a clear-cut h/s/o filariasis. Diagnosis is confirmed on excision-biopsy
- These swellings are formed by granulomatous reaction around an adult worm lodged in the peripheral lymphatics in subcutaneous tissues of the breast.

Free ebooks ==> www.Ebook777.com

Filarial elephantiasis of the breast

– Previous h/s/o recurrent acute attacks of lymphangitis of the breast and axillary lymphadenitis will be available
– Thickening and edema → orange-peel appearance.

Common causes of elephantiasis of the breast in Africa: Axillary tuberculous lymphadenitis, chronic pyogenic lymphadenitis.

BENIGN SWELLINGS OF THE NIPPLE

1. **Cyst**: Sebaceous cyst, retention cyst arising from Montgomery tubercles.
2. **Adenoma**
   – Uncomfortable swelling beneath the nipple
   – Can get ulcerated, and then be confused with Paget's disease.
3. **Papilloma**
   – Features of a cutaneous papilloma. Pedunculated
   – Sometimes may grow to the size of a cherry.
4. **Fibroepithelial polyp.**

EXAMINATION OF A MASTECTOMIZED PATIENT

EXAMIINATION OF THE OPERATED SIDE

- **Scar**
  Palpate gently. Scar may be sensitive.
  – Change in color
  – A localized are of cellulitis
  – Nodularity
  – Mass

  } May suggest a local recurrence of cancer
- **Rest of the chest of the operated side**
- **Axilla**—examine for lymphadenopathy.
- **Arm**—lymphedema.

Examine the CONTRALATERAL BREAST in usual manner.

EXAMINE THE NECK, CHEST, AND ABDOMEN to rule out metastatic disease.

DISCOMFORT IN THE BREAST/S

Causes:

– Pregnancy
– OC pills.

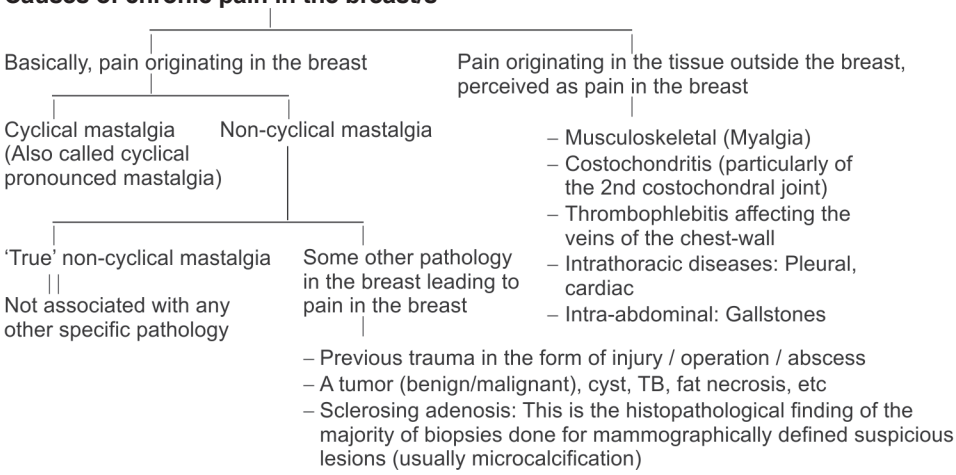
CHRONIC PAIN IN THE BREAST (CHRONIC MASTALGIA)

Definition: Pain in the breast of sufficient severity to seek medical advice

– Cyclical pronounced mastalgia is the commonest cause
– Cancer can occasionally present with pain, without a clearly palpable swelling.

www.Ebook777.com

Causes of chronic pain in the breast/s



HISTORY

- **Age**?
  - 30s: Cyclical pronounced mastalgia
  - 40s: Non-cyclical mastalgia
  - 45↔55: Cyst/s in the breast.
- **Pain**
  - Exact site? Does it radiate?
  - Duration?
  - Is it related to periods? How many days does the pain last?
  - Do the episodes of 'pain related to periods' 'wax and wane'? If so, how long do the episodes last?
  - Intensity: Mild/moderate/severe?
  - Nature: Throbbing/shooting/'heaviness'/burning/dragging, etc.?
  - Precipitating-aggravating-relieving factors?
  - Past h/o any breast disorder/injury to the breast/any operation upon the breast?
- **Any swelling**?
  - Did the pain start before the appearance of the swelling, OR after the appearance of the swelling?'
  - Exact site?
  - Duration?
  - Mode of onset?
  - Details of progress?
  - Did it regress in size?
  - Does it fluctuate in size? An example—benign nodularity.

LOCOREGIONAL EXAMINATION

In addition to the examination of breasts in the usual manner, test for 'shifting tenderness' (Turn the patient on her side so that the breast moves away from the chest wall, and then examine the chest wall for any abnormality and tenderness. This test will be able to point out the site of origin of pain—whether in the breast or in the chest wall).

Free ebooks ==> www.Ebook777.com

DIFFERENTIAL DIAGNOSIS of chronic mastalgia

Cyclical pronounced mastalgia

'Cyclical', because pain has obvious cycle-related pattern associated with menstrual cycle; and 'pronounced' because the pain is pronounced by virtue of intensity/duration/both. Intensity can be such that marital relations may be affected, or mothers may not be able to allow their children near them.

– Age: Generally the condition of premenopausal women. Usually in 30s. Ceases with menopause
– Site: Outer half of the breasts. Often bilateral.

Spectrum of the disorder with respect to severity of pain and duration

1. Mild discomfort only, few days prior to menstruation; patient not unduly concerned.
2. Pain worst in the days premenses.
3. Severe symptom lasting throughout the cycle; some relief only during menstruation.

– Descriptor: Often 'heaviness'-like
– Characteristically waxing and waning. Episodes of discomfort/pain lasting for some months, followed by relief may be for years, followed by recurrence again
– Often associated with palpable nodularity in the breast, especially in the week just before menstruation is due.

Non-cyclical mastalgia

– Age: Occurs in both pre- and postmenopausal women. Usually 40s
– Site: Medial quadrant or central. Usually unilateral
– Descriptor: Burning/dragging, rather than heaviness
– Continuous/random pattern
– Not associated with nodularity to the same degree as in cyclical mastalgia.

'Trigger-point' pain—a specific type of non-cyclical mastalgia.

– Perfectly localized to a specific spot. Unilateral
– Nature: Burning/dragging
– Abscess-like acute exacerbation ±
– Spontaneous, OR may be precipitated by touch
– Aggravated by cold
– Current or past association of 'duct ectasia/periductal mastitis' complex ±.

WITHDRAWAL OF THE NIPPLE

HISTORY

- Which **side**?
- **Duration**?
  – If 'long', i.e. in years, then—is it present since puberty or birth?
  – Was the nipple previously upstanding?

  Withdrawal since puberty/birth = 'Inversion'

  ↓
- Have you had a child; and if so, was/has there been any **difficulty in breastfeeding**?

  Withdrawal of previously upstanding nipple = 'Retraction'

  ↓
- **Discharge from nipple**?—If yes, then its details – refer to the section 'Discharge from the nipple/s'.
- Any **past h/o surgery** upon the breast?
- **Any swelling** in the breast?
- Also ask history for CA breast: Refer to page 346.

www.Ebook777.com

LOCOREGIONAL EXAMINATION

INSPECTION

- Frankly completely inverted, OR (only) flattened?
- Is it linear transverse slit-like?
- Does it appear distorted?
- Any ugly periareolar scar?

PALPATION

- Soft, OR thickened?
- Can it be digitally everted?
- Palpate the breast in usual manner, especially to look for any swelling/s.
  - Swelling/s could be suggestive of non-lactational chronic abscesses associated with periductal mastitis, or cancer.

ARRIVING AT THE DIAGNOSIS—in a c/o withdrawal of the nipple

PRINCIPLES AND PITFALLS

1. As 'inversion' is present since puberty/birth, it is natural to have been present for a 'long duration' (i.e. often in years).
   - Even 'retraction' can be present for 'long-duration', if it is caused by duct ectasia or injudicious previous surgery
   - 'Retraction' of 'short-duration' (= just of few weeks or months duration) could be suggestive of cancer
   - Hence, no rigid conclusion should be drawn regarding the possible cause only on the basis of 'duration' of withdrawal of the nipple.
2. Duct ectasia, cancer can occur in a breast with inverted nipple!

ANALYSIS OF DATA

A. Withdrawal since puberty/birth = **'Inversion'**.

The degree of inversion?

A degree of flattening of nipple is common Does not really interfere with breastfeeding	Frank complete inversion leading to difficulty in breastfeeding is not so common

Complications of inversion of nipple

1. Difficulty in breastfeeding.
2. Acute infective mastitis (even abscess formation). Pathogenesis of infection: Inversion, especially of long-standing, leads to local excoriation by retained sebaceous secretions. Excoriation predisposes to infection.

B. Withdrawal of the previously upstanding nipple = **'Retraction'**.

- Associated with discharge from the nipple
- Often bilateral
- Linear, transverse slit-like

} ⇒ Duct ectasia.

- Unilateral
- Past h/o surgery upon the breast
- Ugly periareolar scar/defect
- Cannot be digitally everted

} ⇒ Postsurgical retraction

- Unilateral
- Distorted and thickened. Cannot be digitally everted
- Often associated a swelling characteristic of cancer

} ⇒ CA breast

Free ebooks ==> www.Ebook777.com

DISCHARGE FROM NIPPLE/S

When a woman is giving a h/o having noticed a discharge from the nipple/s, she very frequently and quite unconsciously gives her breast a squeeze if she has been in the habit of doing this to demonstrate the discharge. Warn her against doing this while the case-taking is in progress, so that the discharge can be reproduced exactly when required during the examination, and which can also be collected on a slide for cytological examination.

HISTORY

- **Duration**?
- **Amount**?
- **Nature**—milky/blood-stained/clear watery/thick opalescent of which color?
- Which **side**?
- Does it come from a single specific point or multiple points round the nipple (i.e. **unifocal or multifocal**)?
- **LMP**?
- Are you **pregnant/lactating**?
- **When did you stop lactating**? (–if applicable)
- Have you been taking **any medicine** for some time (e.g. haloperidol/chlorpromazine/metoclopromide/methyldopa, etc.)? If so, how long?
- Have you taken **any treatment** for the same?–What (–**details**)?
- **Any swelling** in the breast?
- Also ask history for CA breast: Refer to page 346.

LOCOREGIONAL EXAMINATION

INSPECTION

Of nipple, areola, skin and a visible swelling, if any—in the usual manner (Refer to page 348.)
(Wipe away any crusting from nipple, if present.)

PALPATION OF THE NIPPLE AND AREOLA

To reproduce the discharge and also to determine whether it arises from one or more ducts

1. Apply pressure with a finger on the areolar margin at 12 O'clock position to begin with, and move around step-wise in a clock-wise direction.
   Points to be noted
   - Could the discharge be reproduced?
   - Unifocal/multifocal?
   - Nature of the discharge?:
     - Colostrum (thick yellowish) during mid and late pregnancy/milk (milky)/clear watery/blood-stained (bright fresh blood or darker (due to the presence of products of degradation))/cream/green/almost black colored thick opalescent?
   - Any subareolar dilated duct/nodule/swelling palpable?

   If this maneuver to reproduce discharge fails, then

   ↓

2. Pick up the tissue immediately beneath the nipple-areola and gently compress to reproduce discharge.
3. Palpate the breast segment-wise in radial direction from periphery to center to reproduce discharge. This maneuver will determine the possible segment that is diseased, as in cyclical nodularity for example.

Collect the discharge on a glass slide, smear, air-dry for half a minute and send for cytological examination with/without fixation as per the policy of cytologist.

Sometimes discharge cannot be reproduced at the time of clinical examination. Patient may give h/o discharge only once/twice. In such a situation, if investigations rule out malignancy, then keep the patient under follow-up for at least six months, especially in case of blood-stained discharge.

Palpate the (rest of the) breast in the usual manner.

www.Ebook777.com

ARRIVING AT THE DIAGNOSIS—in a c/o discharge from the nipple/s.

PRINCIPLES AND PITFALLS

1. There is no absolutely unequivocal descriptor of either physiological or pathological discharge, except for milk in the neonate, and in a lactating woman.
2. Cancer although the most important, is not the only cause of discharge from nipple. There are many benign causes too.
3. Since blood/serous type of discharge can occur in cancer, investigation to exclude this possibility needs to be much more thorough than for other types of discharge.
4. A possibility of inflammatory CA must be kept in mind during pregnancy and lactation. Reason: Discharge of blood can sometime occur from engorged breast of pregnancy, normally.
5. **Physiological discharge can be unilateral.**
6. The woman who complains of discharge from the nipple may be wrong in her description. The complaint may be referable to some other condition which might reasonably be confused with a discharge from the nipple. The common conditions are discharge from the surface of the nipple, and discharge from some focus on or near the areola.

Basically,

Discharge at the orifice of the lactiferous duct/s
⇓
Discharge coming from within the duct/s
||
A 'true' c/o 'discharge from the nipple'
↓
Various factors to be taken into consideration for arriving at the diagnosis:
- Is the patient pregnant / lactating / otherwise?
- Is there a swelling in the breast?
- Type of discharge?
- Unilateral / bilateral?
- Unifocal / multifocal?
- Refer to the 'causes of discharge from the nipple/s'

Discharge not seen at the orifice of the lactiferous duct/s; instead, it originates from a lesion on the surface of the nipple or a lesion on the areola
⇓
- Eczema of the nipple-areola
- Paget's disease
- Mamillary fistula
- Furuncle/other minor septic lesion on the areola.

CAUSES OF DISCHARGE FROM THE NIPPLE/S

A. *Based on the nature (type)* of discharge

1. **Thick yellowish from mid to late pregnancy**—called colostrum.
2. **Milk**
   - Neonates: Physiological—due to transplacental passage of maternal hormones
   - During pregnancy and lactation.—obviously physiological
   - 'Galactorrhea'. Could be physiological or pathological.
3. **Clear, colorless or slightly yellow, homogeneous and only slightly sticky**
   Possibly physiological; however, a discharge that is described in this way is no proof that it is physiological.
4. **Clear watery**: Pathological. Could be due to intraductal CA.
5. **Thick opalescent** of varying colors: Pathological.
6. **Blood-stained**
   - During pregnancy—most likely to be physiological
   - Otherwise, pathological. - Duct papilloma, intraductal CA, duct ectasia (in perimenopausal and older age-group patients).

Free ebooks ==> www.Ebook777.com

B. *Based on the etiology*

1. **Physiological**
   - In a neonate: Milk
   - From mid to late pregnancy: Colostrum
   - During late pregnancy: Milk, or blood-stained discharge
   - During lactation: Milk
   - Apart from the periods of pregnancy/lactation, in a premenopausal woman: Milk - 'galactorrhea'.
2. **Galactorrhea**: = secretion of milk during period other than pregnancy and lactation.

   Causes

   a. Physiological:
      - For some period (even years) after cessation of breastfeeding
      - During menarche/menopause
      - Mechanical stimulation of breast
      - Stress.

   b. Pathological:
      - Drug-induced (side-effect of drugs) (enhancing dopamine activity), e.g. haloperidol, chlorpromazine, metochlopromide, methyldopa, etc.
      - Hyperprolactinemia—due to prolactin-secreting pituitary tumor, bronchogenic CA.
3. **Duct ectasia**-periductal mastitis
   - Characteristically bilateral and multifocal
   - Thick opalescent discharge of varying colors
   - Retracted slit-shaped nipple.

   In perimenopausal and older age-groups, discharge can be unifocal and frankly blood-stained.
4. **Cysts**

   Exact meaning of 'cysts' - ? - Probably simply dilated ducts. Discharge is produced simply by compressing an area of cystic change.
5. **Duct papilloma**
   - Discharge—unifocal, often blood-stained; *however, can be watery*
   - More centrally located papillomas tend to cause 'discharge from nipple'
   - More peripherally located papillomas tend to cause a swelling, rather than discharge from nipple
   - Papillomas carrying increased risk of malignant transformation: Multiple/affecting > 1 duct/peripherally located.
6. **Intraductal carcinoma** → unifocal discharge—blood-stained/watery/any other type.
7. **Idiopathic**: = cause cannot be found even with biopsy.

ARRIVING AT THE DIAGNOSIS BY COMBINING THE ABOVE-MENTIONED VARIABLES

A. DISCHARGE ASSOCIATED WITH A SWELLING OR ANY OTHER ABNORMALITY IN THE BREAST

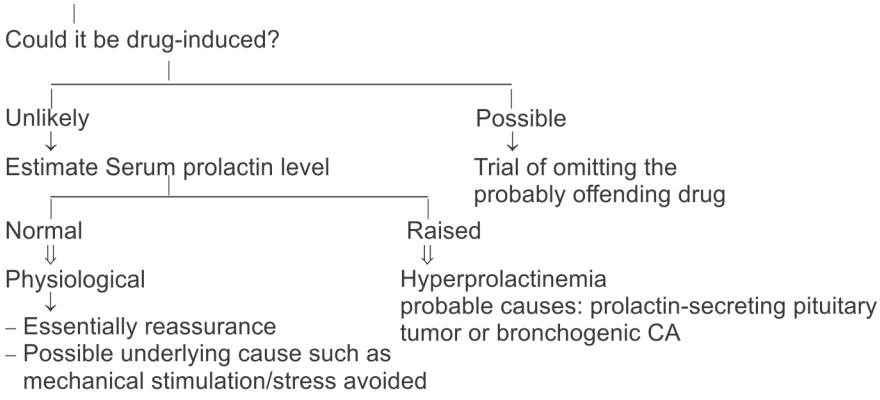
Discharge from the nipple *per se* is probably of secondary importance. Appropriate management of the swelling is the dominant consideration, as discharge most probably could be secondary to the swelling.

B. DISCHARGE NOT ASSOCIATED WITH ANY SWELLING:

- Galactorrhea
- Blood-stained discharge during pregnancy
- Unifocal discharge (irrespective of the type)
- Multifocal discharge (irrespective of the type).

www.Ebook777.com

1. **Galactorrhea**



2. **Blood-stained discharge during pregnancy**
   - Most commonly physiological
   - However, if cancer is suspected due to any reason,—Investigate.
   - If cancer is ruled out, review the patient after completion of pregnancy to rule out a pathological cause of blood-stained discharge.
3. **Unifocal discharge** irrespective of the type
   Only 2 common causes:
   - Duct carcinoma
   - Duct papilloma.

   Clinically, it is not possible to distinguish one from the other. However, as a rule duct carcinoma is not seen below the age of 40.
4. **Multifocal discharge** irrespective of the type
   Causes:
   - Physiological
   - Duct ectasia-periductal mastitis.
5. **Idiopathic**, when cause cannot be found even with biopsy.

MAMMARY DUCT FISTULA

= a fistula between the epithelium of a lactiferous duct and the skin.
– A preceding h/s/o recurrent attacks of subacute abscess or a chronic subareolar abscess
– Opening of the fistula is often situated on the areolar margin
– Nipple inverted or retracted.

Causes

1. Inversion of nipple
2. Mammary duct ectasia/periductal mastitis

} → Attacks of subacute abscess or a chronic subareolar abscess

ECZEMA of the nipple, usually extending onto the areola

Causes

1. Mammary duct ectasia leading to chronic nipple discharge. The chronic discharge in turn leads to eczema.
2. Any cause that may lead to eczema of skin anywhere else.
3. Paget's disease.

Paget's disease

= a clinical type of invasive ductal carcinoma.
– Eczema-like condition which persists in spite of local treatment
– A tumor of lower-grade malignancy; and its prognosis is generally good.

Free ebooks ==> www.Ebook777.com







Figs 30-8A to C Examples of Paget's disease

– It develops in the main lactiferous ducts; extends to the skin to infiltrate nipple and areola leading to eczematoid changes. The nipple-areola is eroded slowly. Eventually, they may be completely destroyed and replaced by a red flat ulcer
– If eczema does not resolve after a few weeks of appropriate medication, biopsy of the involved skin is essential.

DD of Paget's disease: Ulcerated adenoma of the nipple.

CRACKED NIPPLE

Cause: Neglect of hygiene during lactation.

Complication: Acute infective mastitis.

PAIN AND HYPERSENSITIVITY IN THE NIPPLE

This condition is difficult to explain.

SMALL SIZE of one or both breasts

This symptom might be highly subjective, especially if the woman has some psychological problem.

Cause of small size: Underdevelopment, which may have an endocrinal basis. There may not be any abnormality as such. A patient may present only for a cosmetic concern.

Thus, a patient complaining of 'small size' may fit in one of the 2 groups from point of view of planning the treatment.

1. 'Apparent underdevelopment'—especially if the patient is known to have a psychological problem.
2. 'Underdevelopment of cosmetic concern'.

www.Ebook777.com

BIG SIZE

Uilateral

Possible causes: Diffuse hypertrophy, filarial elephantiasis, wrong interpretation of actually a big tumor within the breast (such as a giant fibroadenoma) or in the retromammary space

Bilateral

More likely cause: Diffuse hypertrophy.
Much less possible: Remaining causes under the heading of 'unilateral', occurring on both sides simultaneously

Diffuse hypertrophy

- Bilateral, but can be unilateral
- Occurs sporadically in otherwise healthy girls, or during the first pregnancy
- The breasts may attain enormous dimensions, and may even reach below the knees when the patient is sitting.

ASYMMETRY

Developmental

= During development of breasts at the time of puberty, both the breasts may not develop to the same extent, and some degree of asymmetry may persist for ever. There is neither physiological, nor pathological abnormality

Pathological

= Occurring at a later time; i.e. anytime after the development to a definitive size and shape at the end of puberty having been achieved.
Although, many of the pathological conditions lead to an ↑ in size of the breast, some of the conditions **may lead to a ↓ in size** leading to asymmetry e.g. scirrhous CA

POLYMAZIA

The supernumerary breast and nipple remain unnoticed until puberty.

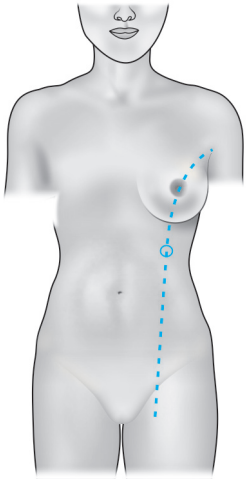


Fig 30-9 The mammary line



Fig 30-10 An example of polymazia

Free ebooks ==> www.Ebook777.com

31 DISORDERS OF THE MALE BREAST

Disorders of the breast, which occur in males:
- Most common, in the boys at puberty: Mastitis of puberty
- Gynecomastia
- Benign nodularity
- Mastitis from local irritation
- Rare: Tumors, both benign and malignant, including Paget's disease.

SWELLING IN (THE REGION OF) THE BREAST

HISTORY

- **Age**?
  Age incidence of various swellings
  - Infants: Physiological gynecomastia
  - Puberty: Mastitis of puberty = Painful and tender self-limiting gynecomastia
  - Young adults: Primary (idiopathic) gynecomastia
  - Elderly: Senescent gynecomastia.
- **A circumscribed swelling**
  This is a more typical presenting symptom of a localized swelling in the (region of the) breast.
  - Exact site?
  - Duration?
  - Mode of onset?
  - Details of progress?
  - Did it regress in size anytime during the course?
- **Prominence of the breast/s**
  This is a more typical presenting symptom of gynecomastia.
- **Pain**
  - Exact site? Does it radiate?
  - Duration?
  - 'When did it start? Did it start before the appearance of the swelling, OR Did it start after the appearance of the swelling?'
  - Intensity: Mild/moderate/severe?
  - Nature?
  - Precipitating, Aggravating, Relieving factors?

For gynecomastia
- Do you have a swelling in the scrotum?
- Have you been consuming alcohol since a few years?
- Are you suffering from a chronic liver disease?
- Did you suffer from CA prostate? If so, which treatment have you received? – Stilbestrol, orchidectomy?

www.Ebook777.com

- Did you suffer from leprosy?
- Have you been on medicines like—digitalis, antihypertensive, spironolactone, tricyclic antidepressant, diazepam, isoniazide, ketoconazole, etc.

For CA breast

- H/o **significant loss of weight**?
- **Previous history** of breast cancer?
- **Family history** of breast cancer?

} – High-risk factors for development of CA breast.

- 1. Axillary or/and supraclavicular swelling? - lymphadenopathy - with clinical features typically s/o mets viz. duration in weeks, rapid progress, often painful, hard consistency. Manifestations of brachial plexus infiltration ±.
  2. Anorexia, nausea, lethargy? ⇒ hepatic mets.
  3. Bone pains? ⇒ mets to bones, OR malignant hypercalcemia.
  4. (Pathological) Fracture? ⇒ mets to bones.
  5. Abdominal symptoms including pains, nonspecific deterioration of health, confusion, dehydration? ⇒ malignant hypercalcemia.
  6. Anemia, (repeated) infections (due to leukopcenia)? ⇒ (red) bone marrow involvement.
  7. Breathlessness? ⇒ pleural effusion due to pleural mets/pulmonary (parenchymal) mets / ascites pushing up the diaphragm.
  8. Dry cough? ⇒ pulmonary (parenchymal) mets.
  9. Distension of abdomen (ascites)? ⇒ peritoneal mets.
  10. Manifestations of intracranial SOL? ⇒ mets in the brain.

} - for regional/distant metastatic disease.

For mastitis from local irritation

- Have you been wearing ill-fitting brace?
- Do you routinely carry heavy equipment across your shoulder?

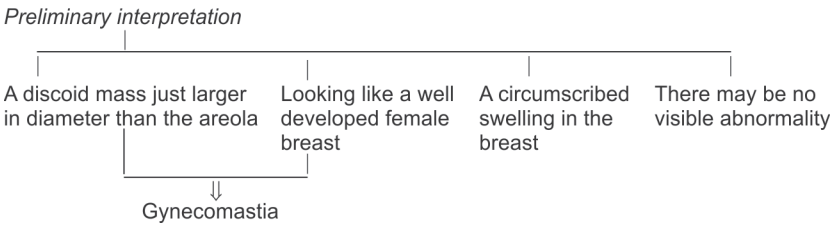
LOCOREGIONAL EXAMINATION

EXPOSURE: Both breasts must be completely exposed before examination is commenced.

INSPECTION

Methods

1. **From front**—compare the size and level of the nipples.
   Raised level of one nipple suggests subareolar fibrosis due to any cause, frequently a carcinoma.
2. **Hands raised above the head** to look for asymmetry in the level of nipples, which was otherwise not evident with hands by the side, and also to look for dimpling in the skin.



Inspect the apparently normal breast first, followed by the abnormal side.

1. **Nipple**
   - **Change in the direction**? Deflection occurs in the direction of malignant tumor
   - **(Unusual) Prominence**? – It may be due to an underlying swelling
   - **Retraction**? – Distorted/thickened?
     – A retracted nipple that is distorted/thickened ⇒ CA
   - **Ulcer**? Complete destruction with replacement by a red flat ulcer = Paget's disease

Free ebooks ==> www.Ebook777.com



Fig 31-1 Slightly retracted nipple, evident only on careful observation

2. **Areola**
   - **Size**? Decrease in size of the areola around a retracted nipple ⇒ CA.
   - **Dimpling (= Puckering)**?
   - **Orange peel appearance**? Areola is the commonest site where orange peel appearance occurs first.
   - **Ulcer**?
   - **Eczema**?
3. **Skin**
   - **Dimpling (= Puckering)**?

     Mechanism: Fibrosis involving Cooper's ligaments due to any cause e.g. CA, TB etc. → contraction and shortening of the ligaments → abnormal traction on the skin → dimpling (= puckering).

     Dimpling suggests infiltration of the ligaments of Cooper; not of the skin directly.

     Ask the patient to raise his arms above the head

     Dimpling, otherwise not evident with hands by the side, may become evident

     Dimpling evident with hands by the side may become accentuated

     Mechanism: Raised arm puts more traction on the ligaments as compared to when the arm lies by the side.
   - **Orange peel appearance**?

     Orange peel appearance becomes more obvious *by gently squeezing the skin*

     Mechanism: Cutaneous edema leading to swelling of skin except at the hair follicles, which appear as deep pits

     Orange peel appearance usually suggests infiltration of the skin by CA

     A cause other than cancer: Axillary tuberculous lymphadenitis.
   - **Fungation**?

     ⇒ Infiltration of the skin by CA.

     Conditions, which may lead to an appearance of fungation: Rapidly growing swelling like rapidly growing sarcoma, chronic abscess

     Pathogenesis: Pressure necrosis of the overlying skin leading to ulceration, → fungating appearance.
   - **Nodules**?

     ⇒ Infiltration of the skin by CA.
   - **'Cancer-en-cuirasse' appearance**?

     Chest wall studded with carcinomatous nodules leads to the appearance of a coat of armor

     This may be associated with brawny edema of the arm

     Usually, though not necessarily, seen in cases with local recurrence after mastectomy.
   - **Engorged veins**?

     More commonly encountered with sarcoma than carcinoma.

www.Ebook777.com

4. **If there is a discoid swelling beneath the areola OR if the whole breast seems to have been well-developed like a female breast**
   ↓
   - Definition and dimensions?

     A well-defined swelling with regular shape suggests gynecomastia.
5. **A swelling in the breast may not always be (big enough to be) easily visible** (e.g. benign nodularity). However if visible, then note the details as usual.
6. **Arm**

   Edema of the arm can occur due to blocking of the lymphatics by carcinomatous fibrosis.

PALPATION

POSITION of the patient**: Lying down**.

The **arm should be abducted to ~ 90**° so that the medial quadrants of the breasts become flat.

– **General method of palpation of the breast** (excluding nipple)
  - Palpate with flat of 4 fingers close together
  - Make gentle rotating movements of the fingers in an attempt to roll and detect even a small swelling between the fingers and the chest wall
  - Palpate in a fixed sequence systematically:
    1. 4 quadrants f/b the central region.

       OR
    2. The vertical strip pattern—a better sequence than the quadrant sequence.

– **Nipple** is routinely palpated between the thumb and the index finger.

Steps of palpation of the breasts

A. Palpate the apparently normal breast first.

B. Palpation of the abnormal breast

1. **Discoid swelling beneath the areola or well-developed breast like a female breast**?
   - Temperature?
   - Tenderness?
   - Margins?
   - Dimensions?
   - Surface?
   - Consistency?

   Well-defined shape, smooth surface and firm consistency ⇒ gynecomastia.
2. **The swelling**

   Grasp the swelling between the tips of the thumb and fingers.

   Palpate the same swelling again with flat of the fingers

   If the swelling is palpable to the same extent with roughly the same features as when palpated between thumb and fingers
   ⇓
   True swelling
   ↓
   Palpate in details in the usual manner
   Use tips/pulps of the fingers necessary to obtain finer details

   Not palpable. Instead, diffuse shotty or granular surface is felt, and suppleness of normal breast is replaced by elastic consistency (in the affected region)
   ⇓
   Benign nodularity (fibroadenosis)—can occur in males

   Two points specifically applicable to a c/o 'swelling in the region of the breast' are discussed below.

Free ebooks ==> www.Ebook777.com

Impulse-on-coughing?

Impulse-on-coughing may suggest that the swelling may be an intercostal cold abscess or empyema necessitans, rather than a swelling arising from the breast.

Anatomical plane and fixity?

a. Fixity *v/s* tethering to the skin.
   - Fixity to skin, as determined by pinching, gliding and lifting up tests ⇒ infiltration of the skin, OR a swelling arising from the skin
   - Normally, a swelling of the breast can be moved independently of skin for some distance—the distance being < the radius of (shortened) ligaments of Cooper. Forced movement of the swelling beyond this distance leading to dimpling is called 'tethering'. Tethering ⇒ involvement of Cooper's ligaments by a chronic inflammatory process or cancer.

b. Fixity to the breast tissue.
   – Fix the breast with one hand and move the swelling with the other
   – Method of fixing the breast: Place the thumb and fingers on the breast much beyond the margins of the swelling, and press the breast against the chest wall
   – Restricted or absence of mobility of the swelling with dimpling in the skin may suggest CA.

c. Fixity to muscle: After putting the appropriate muscle in 'isometric' contraction,
   ↓
   - Look at the size of the swelling—Does the swelling become more prominent/less prominent/remain unaltered?
   - Does that produce any dimpling in the skin? Appearance of dimple suggests that the swelling infiltrates both the pectoralis major and the skin
   - Is the mobility of the swelling restricted?
     - Slight restriction of mobility ⇒ tethering of the swelling to pectoral fascia
     - Gross restriction of mobility ⇒ direct infiltration of pectoralis major muscle.

   (The superolateral, superomedial and inferomedial quadrants, and the central zone of the breast are related to pectoralis major. The iinferolateral quadrant is related to serratus anterior muscle.)

d. Fixity to the chest wall.
   – If the swelling is fixed irrespective of contraction of muscle, then it means that it is fixed to the chest wall.
   – Chest wall includes ribs, intercostal muscles and serratus anterior muscle, but not the pectoral muscle.

3. **Palpation of the nipple-areola**

   Nipple is routinely palpated between the thumb and index finger.

4. **Regional examination**
   - Examine the axillary, supraclavicular **and inguinal** groups of lymph nodes.
   - Lymph from the skin of the breast may be drained to the opposite breast and the inguinal lymph nodes via the lymphatics passing along the abdominal wall.

5. **Examination of the arm** for edema

   Edema can occur due to blocking of the lymphatics by carcinomatous fibrosis.

IF THERE IS AN ULCER: Examine it in the usual way.

IF THE BREAST IS FOUND TO BE NORMAL: Examine the spine for scoliosis.

In **SYSTEMIC EXAMINATION,** specifically look for liver disorder and testicular disorder.

www.Ebook777.com

Step 1

- **Benign swelling of the nipple**
  - Cyst: e.g. sebaceous cyst
  - Adenoma
  - Papilloma
  - Fibroepithelial polyp
- **Swelling in the region of the breast, excluding nipple** → **Step 2***
  - Arising from the skin/fat of the superficial layer of the superficial fascia,* or the chest wall
  - Arising from, and confined to the breast
  - Arising from retromammary space
  - Arising from the breast and infiltrating the pectoralis major/serratus anterior, OR arising from the pectoralis major/serratus anterior, OR arising from the axillary soft tissue other than axillary tail of the breast

*For discussion, refer to chapter 30.

Step 3

Nature of the swelling arising from the breast?

- **Benign nodularity can occur in males**
- **True swelling**
  - **A discoid mass just larger in diameter than the areola**
  - **Looking like a well-developed female breast**

  ⇓ **Gynecomastia**
  - **A circumscribed swelling in the breast** ↓
    - Vascular
    - Cyst
    - Benign tumor
    - Chronic inflammatory swelling
    - Malignant tumor

Gynecomastia

- **Physiological**
  - In the neonate
  - Mastitis of puberty
  - May be seen in otherwise normal midddle-aged men (~ 1/3rd of population)
  - Senescent gynecomastia in the elderly
- **Pathological**
  - Mastitis from local irritation
  - Idiopathic gynecomastia
  - Gynecomastia secondary to
    - Hypogonadism from any cause (bilateral testicular trauma/tumor/infection; failure of proper descent, bilateral orchidectomy, failure of the pituitary—testicular axis, chromosomal aberrations, etc.)
    - Antiestrogen therapy often for CA prostate
    - Pituitary disease / adrenal disease / bronchial carcinoma (these conditions produce ectopic hormone)
    - Liver failure
    - Leprosy
    - Drugs, e.g. digitalis, antihypertensive, spironolactone, tricyclic antidepressant, diazepam, isoniazide, ketoconazole, etc.
- **Pseudogynecomastia**
  - Due to local fat deposition no breast tissue development

Free ebooks ==> www.Ebook777.com

DIFFERENTIAL DIAGNOSIS of chronic swelling in the male breast

Physiological gynecomastia in the neonate

= transient breast development in the neonate due to maternal estrogens.

Mastitis of puberty

= physiological gynecomastia occurring in boys at puberty.

– A button-like painful and tender swelling under the areola
– Tenderness subsides within 2 weeks
– Induration takes many months to subside.

Physiological mastitis in the middle-aged man

One-third of otherwise normal middle-aged men have some development of their breasts.

Senescent gynecomastia

= physiological gynecomastia occurring in elderly.

Mastitis from local irritation

Causes:

– Wearing of ill-fitting braces
– Carrying heavy equipment across the shoulders as by soldiers for example.

Idiopathic gynecomastia

– Usually occurs in young adults
– H/o prominence of the breast
– Unilateral/bilateral
– A discoid mass just larger in diameter than the areola and concentric with the nipple and areola
– ~ 5 cm in diameter.



Fig 31-2 Idiopathic gynecomastia

DD

1. Lipoma, although lipoma in the breast is very rare.
   Differentiating point: If it is a lipoma, then the swelling and the nipple-areola will move separately. In gynecomastia, there will be no relative movement between the swelling and the nipple-areola.
2. Pseudogynecomastia in the obese.

Notes

– Gynecomastia is defined as excessive development of the male mammary glands, even to the functional state
– Gynecomastia presents such a typical clinical picture that a confident clinical diagnosis can be made and accepted as a basis for management.

Gynecomastia secondary to ……

– Physiological cause ruled out
– Underlying cause evident.

Pseudogynecomastia

– The tissue lacks the well-defined appearance and firm consistency of true gynecomastia
– Occurs in obese individuals.

Fibroadenoma, rarely lipoma

– All have the same clinical features as in the female
– Beware of a lipoma; in fact it may be a 'lipomatous carcinoma'

Carcinoma

Basically, a malignant tumor.

– Starts as a firm nodule
– Clinical features are similar to a carcinoma in the female breast

www.Ebook777.com

– Greater tendency to fungate quite early
– Predisposing causes: Gynecomastia, and diseases associated with excess estrogen.

Sarcoma

Basically, a malignant tumour.

– Age 30–40
– Features—similar to a sarcoma in the female breast
– Clinically, it may be impossible to distinguish it from a medullary carcinoma.

Other cancers which can occur in male breast: Lymphoma, secondaries.

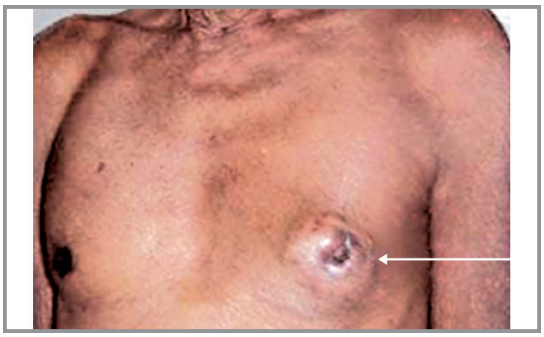


Fig 31-3 Malignant tumor

Free ebooks ==> www.Ebook777.com

32 DISORDERS OF THE CHEST WALL

1. Congenital anomalies.
2. Lung hernia.
3. Tumors:
   - Of bone/cartilage
   - Of soft tissue.
4. Costochondritis.
5. Infections:
   - Of bones
   - Of soft tissue.

CONGENITAL ANOMALIES

- Congenital anomalies of the bony thorax are often incidental finding on routine chest radiography
- With anomaly in one component, search for anomaly in another component; for example with anomaly in the ribs or sternum, look for anomaly in the vertebral column
- The majority of **rib abnormalities** are of little significance and only if extensive do they present a clinical problem, e.g. cervical rib with neuro/vascular symptoms
- **Pectus excavatum** is a deformity of the anterior chest wall characterized by depression of the sternum.
  - It may be localized to the lower sternum but most often begins at the manubriosternal junction.
  - Accommodations occur in other structures within the chest wall and the area of the rib-ends at the costal cartilages becomes prominent.

Pigeon chest deformity

- 2 main types
  1. At the manubrial level: The manubrium and the body of the sternum are almost at right angles to one another
  2. Near the xiphisternum
- Symmetrical or asymmetrical?
  - In case of asymmetrical deformity the sternum lies obliquely with depression of the costal cartilages on one side and elevation on the other

- *Symptoms* directly due to such physical deformities are rare
- Patients may be very conscious of the unusual shape of their chest wall and be unwilling to participate in activities/sports which involve exposure of the trunk such as swimming, etc.
- Limitation of peak cardiac output may occur in severe pectus excavatum as maximal venous return is impaired
- Most patients with moderate to severe degrees of depression, even with displacement of their heart to the left, have no clinical disability
- *An ejection systolic murmur may be audible* at the left sternal edge, which is from the pulmonary artery flow in a distorted vessel

www.Ebook777.com

- *The degree of abnormality may be assessed radiologically* by measuring the distance between the body of the sternum and the vertebral column in the lateral chest X-ray
- *Routine PFTs* usually show that lung volumes and dynamic function are within normal limits.

LUNG HERNIA

= protrusion of pulmonary tissue outside the normal pleural boundaries.

Causes: following trauma or surgery, but may also occur spontaneously.

Types

External

1. Intercostal: Usually occurs either alongside the sternum or the vertebral column where the intercostal muscles are relatively incomplete
2. Into the (root of the) neck through a (congenital) deficiency in the Sibson's fascia (the aponeurosis overlying the apex of the lung). This usually develops in adults, particularly those with bronchitis and emphysema, and is occasionally seen in people whose occupation involves maintaining high expiratory pressures, such as trumpeters, glass blowers. These hernias are usually asymptomatic

Internal

Through the diaphragm

TUMORS

Of the bone/cartilage

Secondary (Metastatic) ↓

These form the majority of tumors in ribs and thoracic vertebrae

Majority → bone destruction → pathological fracture

Exception: Osteoblastic mets from the prostate

Primary

The most common

Chondromata and chondrosarcomata ↓

Visible and palpable swelling in the costochondral junction area

Other

Any of the bony tumors

Solitary plasmacytoma is the commonest tumor of the sternum

Primarily pleural or pulmonary tumor invading the chest wall and causing bone destruction

Soft tissue tumor

Anyone from the general category

COSTOCHONDRITIS

– Painful and tender swelling in the area of the 2nd or 3rd costochondral junction
– No inflammatory changes in the overlying skin
– Usually a self-limiting condition
– Radiography of the area is not helpful in confirming the diagnosis.

INFECTIONS OF THE BONE

1. A destructive osteomyelitis of the sternum can occur as a complication of median sternotomy; the most common organism responsible—staphylococci.
2. Tuberculosis or fungal infections may involve the chest wall by direct extension from underlying infection of the lungs, pleura or lymph nodes.
3. Osteomyelitis of the ribs/sternum may occur like anywhere else from hematogenous spread.

Free ebooks ==> www.Ebook777.com

INFECTIONS OF SOFT TISSUES

Serious deeper chest wall infections

1. Subpectoral and subscapular abscesses, usually due to streptococci or staphylococci, originate in the ribs or the scapula respectively
2. Empyema necessitans. Refer to page 171
3. Cold abscess—a collar-stud abscess which primarily originates in the caries of a vertebra and subsequently tracks along the perforating neurovascular bundles finally into the subcutaneous tissues of the chest wall
4. Deep cervical infection will spread onto Sibson's fascia and may present below the clavicle as a fluctuant swelling

The skin and subcutaneous tissues of the chest wall are also subject to all the common infections which occur anywhere else in the body

www.Ebook777.com

33 CHRONIC PAIN IN ABDOMEN (CHRONIC ABDOMEN)

Any one or more of the following chief complaints qualify for chronic pain in abdomen.

– Actual pain.
– 'Pressure' / 'Fullness' / 'Heaviness' in the abdomen or lower chest.
– Heartburn, i.e. feeling of warmth behind lower part of the sternum.
– Acid eructation, i.e. sour-tasting fluid in the throat after belching.
– Feeling afraid to eat, or feeling sorry after eating.

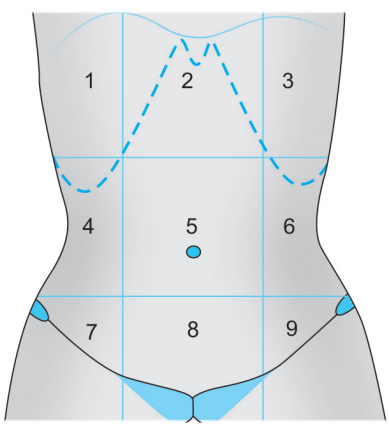
Definitions of commonly used clinical anatomical terms.

- *Flank:* the side of the body between the ribs and ilium.
- *Loin:* the part of the back between the thorax and pelvis.
- *Lumbar:* pertaining to the loins.
- *Groin:* an oblique strip of area 3 cm above and 3 cm below and parallel to the inguinal ligament.

Regions of the abdomen: There are two ways to regionalize the abdomen for localization of symptoms and signs.

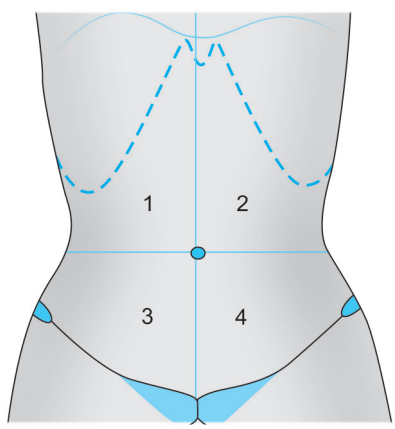
1. Nine regions created by 2 longitudinal and 2 transverse lines. The longitudinal lines pass through midinguinal points. The upper transverse line—the transpyloric line and the lower one—the transtubercular line.
2. Four quadrants created by 1 longitudinal middle line and 1 transverse line, both passing through the umbilicus.

In practice, it may be often convenient to localize the site of pain and/or lump by using the 4-quadrant system.



1 and 3: Hypochondrial regions
4 and 6: Lumbar regions
7 and 9: Iliac regions
2: Epigastrium
5: Umbilical region
8: Hypogastrium / Suprapubic region

Fig 33-1 Nine regions



1: Right upper quadrant (RUQ)
2: Left upper quadrant (LUQ)
3: Right lower quadrant (RLQ)
4: Left lower quadrant (LLQ)

Fig 33-2 Four regions

Free ebooks ==> www.Ebook777.com

GENERAL REMARKS

1. More than one disease may be present in the abdomen.
2. A carefully taken history is very important, as physical signs may be too meager.
3. Organs which are not amenable to direct physical examination: Esophagus, duodenum, ureters and adrenal glands.
4. **Concept of 'referred pain'**
   Referred pain means pain perceived at a site different from the site of its origin.
   Mechanism
   Both the sites, i.e. the actual site of pathology and the site of reference share a final common pathway to the brain.
   This is a characteristic phenomenon of pain originating in deep-seated organs.
   Examples
   a. Pain due to pathology in the testis may be perceived as pain in the abdomen.
   b. Pain due to pathology in the extra-abdominal site may be perceived as pain in the abdomen.
   c. Pain due to pathology in the abdomen may be perceived at a site away from the abdomen, e.g. pain due to pathology in the sub-diaphragmatic region being perceived in the shoulder.
5. **'Colicky' pain**
   Definition: Paroxysmal spasmodic pain due to violent peristaltic contractions of a hollow viscus or a duct such as stomach and intestines (including appendix), extrahepatic biliary tract, pancreatic duct, renal pelvis and ureters, uterus and uterine tubes.
   Causes (Mechanisms): – to overcome mechanical obstruction of the organ concerned.
   – result of irritation, e.g. irritation caused by inflammation, e.g. gastritis, enteritis, etc.
   Characteristics
   – Appears and disappears suddenly.
   – Makes the patient restless and roll about in agony, unable to find a comfortable position.
   – Often associated with vomiting and sweating.
   – Pain-free interval between paroxysms, during which patient feels and looks comfortable.
   – Pressure over the abdomen may relieve/reduce the intensity of pain.
   – Antispasmodic medicine may relieve colicky pain, especially that caused by irritation.

HISTORY

- **Gender**?
- **Residence**?
  Urinary stone disease, filariasis, etc. are common in endemic areas.
- **Occupation**?
  Lead poisoning in painters, arsenic poisoning in industry → chronic intermittent pain.

PAIN

- **Observe** as the history is unfolding: Does the pain seem to be definitely present, or does it appear to be 'vague'?
  'Definite' presence of pain suggests organic, intractable (undiagnosed), or functional pain; whereas 'vague' pain could be psychogenic.
- **Intermittent or Constant (Continuous)**?
  |
  If intermittent,
  - How long does each episode last?
  - How much is the pain-free interval?
  - During the so-called pain-free interval, are you completely asymptomatic, or do you continue to have some discomfort/dyspepsia?

www.Ebook777.com

- Are the durations of pain-episodes and pain-free intervals more-or-less constant, or are they variable?
- Does the site of pain remain constant during each episode, or does it vary?

- **Duration**?
- **Exact site**? Observe as the patient points out the site. Does it appear to be segmental?

Is it accurately located by pinpointing with a fingertip? — or — Is it vaguely located over a more diffuse area by putting the flat of a hand, or by moving around spread-out fingers?

  - **In c/o pain in RUQ/epigastrium**: • Does it radiate to the right lower back/the right shoulder?
  - **In c/o pain in lumbar region**: • Do you feel more comfortable moving around, holding the flank?
    • Does the intensity of pain alter due to changing position?
    • Nature of pain?

Colicky, or Continuous with fluctuating intensity? — or — Continuous with steady intensity?

⇓ Obstructive pathology of the upper urinary tract

Further details to be asked

↓

- Does it radiate to groin/vulva/testicle/penis/inner thigh?
- Is it precipitated by running/jolting?
- Hematuria? If yes, then

↓

- Did pain come first, followed by hematuria?

or

- Did hematuria come first, followed by pain?

- Similar past history?

Examples of obstructive disorders
- Ureteric colic (*synonyms* renal colic)
- Renal stone which may be obstructing the neck of a calyx

⇓ Inflammatory pathology of the upper urinary tract

Further details to be asked

↓

- Intensity of pain - Very severe OR Not so severe?

– Severe pain ⇒ inflammation involving the parenchyma
Examples: Pyelonephritis, pyonephrosis, carbuncle of kidney, infarct of kidney, perinephric abscess, mismatched blood transfusion, toxicity because of drugs like sulfa

– Less severe pain OR, Discomfort rather than pain ⇒ inflammation of mucosa
Examples: pyelitis, pyelonephritis, ureteritis

  - **In case of pain in suprapubic region**: • Is the pain associated with (other) urological symptoms?
    • Does the pain radiate to the tip of the penis?
  - **In case of pain in the lower quadrant**: Does the pain come particularly on coughing/straining?
- Is pain in the abdomen associated with **pain in the back**?
  - Exact site in the back? And other details?

↓

a. Between the scapulae or either lumbar regions?
b. If in the mid-back: Rhythmic or persistent? Does it decrease after food?
c. If in the loin: Any urinary symptoms?
d. Mid-sacral?

- Is pain in the abdomen associated with **pain in the lower limb**?
- **Nature**: Colicky / constricting / burning / dragging / throbbing / twisting / shooting / vague?
- Does the pain **initiate or get aggravated by**
  - Eating? If so, how much time after eating - immediately / ~ half-one hour later / ~ 2–3 hours later?
  - Consuming particular type of food such as hot (chilly)/spicy/oily/fatty?
  - Stooping forwards?
  - Lying down flat?
  - Jolting / running / traveling on a bumpy uneven road?

Free ebooks ==> www.Ebook777.com

- Have you noticed any particular **relieving factor**? - such as
  - Eating
  - Vomiting (often 'induced')
  - Taking antacid
  - Assuming sitting position
  - Passing flatus / stools / taking enema
  - Applying heat
  - In cose of pain in lumbar region: Do you feel more comfortable moving around and holding the flank?

DISCUSSION ON PAIN

Intermittent pain: Multiple episodes of pain, separated by pain-free intervals.

- Completely symptom-free interval ⇒ possibility of peptic ulcer disease.
- The presence of a mild discomfort even during the so-called pain-free interval ⇒ possibility of 'appendicular dyspepsia' or gallbladder disease.
- Progressively increasing duration of pain-episodes with decreasing duration of pain-free intervals ⇒ advancing disease, whichever it may be.
- Variability in the site of pain during each episode does not necessarily mean pain of 'psychogenic' origin. If the 'case' appears to be genuine and the presence of pain seems to be definite, then it may be due to modification/advancement in the disease process; for examples, obstructing element (usually a stone, occasionally a foreign body) may be moving distally; an inflammatory or neoplastic pathology getting worsened, etc. On the other hand, if the 'story' of the illness does not appear to be genuine and the details of pain vague, then pain could be functional or psychogenic in origin.

Continuous pain: A pain that is present for hours or even days without any period of complete relief. The constancy implied is one of the *presence* of the pain in time, not necessarily one of its *intensity*; the intensity may vary.

The presence of continuous pain ⇒ – Disease of the anterior/posterior abdominal wall, or disease of a solid organ.
– Inflammation / perforation / torsion / bleeding.
– Possibility of disease of gallbladder/CBD must be kept in mind.

In general terms, continuous pain requires more urgent attention and intervention as compared to intermittent pain.

The pain accurately located by pinpointing with a fingertip; usually of 'short' duration and of sharp character ⇒ **somatic pain.**

Somatic pain: The pain arising from structures of the **anterior abdominal wall / posterior abdominal wall / dome of the diaphragm.**

Vaguely located pain over a more diffuse area by putting flat of one/both hands, or by moving around spread-out fingers ⇒ **visceral pain.**

Segmental pain means pain experienced **along a 'dermatome'** ⇒ irritation of a sensory nerve root/trunk – because of disorder of vertebral column / spinal cord / herpes zoster.

In case of pain in lumbar region: – Feeling more comfortable moving around and holding the flank ⇒ more likelihood of renal pathology.
– Alteration of intensity of pain due to changing position ⇒ radicular pain.

Suprapubic pain associated with urological symptoms suggests urological disorders like chronic cystitis, chronic retention of urine, etc.

Suprapubic pain not associated with urological symptoms, is unlikely to be caused by urological disease.

The site of pain helps to denote the organ involved.

Site of pain	*Probable site of disease*
– Shoulder	– Subdiaphragmatic. Examples: free air under the diaphragm, pathology under the diaphragm like cholecystitis, subdiaphragmatic abscess, liver abscess, splenic abscess/hematoma, etc. – 'Referred' from a disease in the chest, e.g. pleurisy, angina, etc.
– High epigastrium or xiphoid region	Esophagogastric junction.
– Epigastrium (especially lower), and umbilical	– Stomach, small intestine, appendix, biliary tract, pancreas, small intestinal mesentery (mesenteric ischemia). – Colon (e.g. 'irritable colon').

Contd...

www.Ebook777.com

Contd...

Site of pain	*Probable site of disease*
– RUQ/Epigastrium	Biliary tract, liver, pancreas, stomach, duodenum, kidney (dull aching in nature – called 'fixed renal pain; caused by stretching of renal capsule due to hydronephrosis, tumor), subphrenic space, appendix.
– LUQ/Epigastrium	Stomach, spleen, colon, kidney (dull aching in nature – called 'fixed renal pain; caused by stretching of renal capsule due to hydronephrosis, tumor), subphrenic space.
– Lumbar	Kidney/Perinephric tissue, esophagogastric junction, costal nerves.
– Suprapubic region	Pelvic organs, e.g. urinary bladder, uterus and adnexa.
– LLQ	Colon.
– Along a dermatome	Sensory nerve root/trunk.
– Pain migrating (i.e. shifting) from the epigastrium → RUQ ⇒ disease of the gallbladder. – Pain migrating (i.e. shifting) from the epigastrium → RLQ ⇒ appendicitis.	

Radiation of pain: When definite, it is very useful in determining the diseased organ.

Radiation from	*The most probable diseased organ*
– Epigastrium/RUQ → right side of the back (infra-scapular) / right shoulder	Gallbladder/bile ducts.
– Loin → lower quadrant of abdomen/groin/scrotum/penis/labium/inner thigh	Ureter.
– Suprapubic region → tip of the penis	Trigone of the urinary bladder.
– Back → round the flank → anterior abdominal wall, specifically following a dermatomal pattern	Spine (intercostal pain). – Subcostal pain (T8) on the right side may mimic gallbladder pain, and on the left side gastric ulcer pain. – RLQ pain (T11) can mimic appendicitis.

Associated pain in other areas with some other details	*Probable pathology*
– Mid-back, rhythmic, decreasing after food	Penetrating peptic ulcer.
– Mid-back, persistent	Pancreatitis.
– Loin, urinary symptoms	Renal disease.
– Mid-sacrum	Disease of prostate, seminal vesicles, uterus and adnexa.
– Relentless, severe gnawing which seems to bore through from the back.	Spinal bone pain.
– Pain similar to bone pain, but which is worse on movement, and when severe, can make the patient terrified to move or even cough.	Spinal instability pain.

Causes of **colicky pain**

1. 'Colics' (commonly in children).
2. Obstruction of tubular/hollow viscera, e.g. intestine, ureter, appendix, etc. Exception—so-called gallbladder/CBD colic. Pain caused by obstruction of gallbladder or CBD is only rarely truly colicky.
3. Paralysis of a segment of intestine due to vascular catastrophe or focus of infection in the vicinity.
4. Intestinal spasm due to exogenous/endogenous intoxication.
5. Attacks of colicky pain in the LUQ may be caused by splenomegaly.

Constricting pain ⇒ ischemia.

Burning pain ⇒ mucosal injury / inflammation. Examples: Reflux esophagitis, peptic ulcer, urethritis, cystitis, pyelonephritis, etc.

'Ulcer-like' pain, apart from peptic ulcer, may be caused by recurrent pancreatitis.

Shooting type of pain ⇒ neurological pain.

Vague pain ⇒ organic deep-seated pathology / functional pain / psychogenic pain.

Bending forward, hands supporting the abdomen while walking slowly (typical 'appendix shuffle') ⇒ acute appendicitis.

Free ebooks ==> www.Ebook777.com

Precipitating (Initiating) / Aggravating (Enhancing) factor	*Probable disease*
– Eating	– Gastritis, gastric ulcer. – Mesenteric ischemia (abdominal angina). – Visceroptosis. – Colitis, large intestinal partial obstruction. Mechanism: Gastrocolic reflex.
– Jolting / running / traveling on a bumpy uneven road	Ureteric colic, (recurrent) appendicitis.

Relieving factors	*Probable disease*
– Assuming sitting position	Hiatal hernia, pancreatic disease.
– Passing flatus/stools/taking enema	Colonic disease, reflex spasm of colon due to gastric disease.
– Applying heat	Smooth muscle spasm. Heat → relaxation of smooth-muscle spasm and relieves all colics.

Causes of **terminal** pain (end of micturition)

1. Pathology in the bladder neck (including prostate): Stone, trigonitis, tuberculosis, malignancy, acute prostatitis, acute prostatic abscess.
2. Stone impacted in the lower end of ureter.
3. Adhesion of bladder to surrounding structure → dragging pain.

Causes of pain **throughout the act of micturition**

1. Pathology in urinary bladder: Cystitis, advanced malignancy.
2. Urethritis.
3. Meatal ulcer.
4. Balanitis / balanoposthitis.

Pain while passing urine and **relieved after micturition** ⇒ urethritis / cystitis.

Mid-menstrual pain ⇒ rupture of Graafian follicle.

Pain before and during menstruation ⇒ dysmenorrhea / uterine and adnexal disease.

Pelvic pain at monthly interval may suggest endometriosis.

Symptoms which may give clue about pathological nature of the disease

- **Fever**?

  Causes: Dehydration, chronic infection, occult intra-abdominal abscess, autoimmune disorder, hematologic malignancy like lymphoma.

- **Loss of appetite**?
  - Nonspecific (i.e. for all foods), or qualitative (i.e. for particular type of food such as meat/fatty, etc.)?
  - Mild or severe?

  Inference: – Nonspecific anorexia suggests TB, malignancy in general. However, if severe, then it suggests malignancy.
  – Anorexia particularly for meat may suggest CA stomach.
  – Anorexia particularly for fatty food may suggest gallbladder disorder.

- **('Significant') Loss of weight**?

  Causes (relevant to 'chronic abdomen'): Effect of persistent vomiting and diarrhea; disorders of esophagus/stomach/small intestine/liver/gallbladder/pancreas (esophageal obstructive disease, gastric ulcer, 'abdominal angina', hepatitis, cirrhosis, chronic pancreatitis); worm infestation; TB; malignancy; malabsorption syndrome.

  However, some patients with cancer of stomach/large intestine may maintain their weight surprisingly, until advanced stage.

Nonspecific symptoms which may give clue about the tissue/organ/system affected

- '**Indigestion**'? ⇒ GIT disorder.
- (**Excessive**) **Belching and eructations**?

  Causes (relevant to 'chronic abdomen'): Disorders of the esophagogastric junction/stomach/gallbladder; psychogenic.

– **Nausea**?

  Causes (relevant to 'chronic abdomen'): Disorders of stomach/gallbladder; psychogenic.
  Nausea should not be taken lightly. It could be an early symptom of CA-stomach.

www.Ebook777.com

- **Vomiting**?
  Frequency, amount, color, contents, relation to eating?
  Causes (relevant to 'chronic abdomen'): Disorders of stomach-duodenum/gallbladder; (subacute/chronic) intestinal obstruction; ('chronic') appendicitis, renal lithiasis, pregnancy and pregnancy-related disorders, psychogenic.
- Episodes of **vomiting of blood**? (**Hematemesis**)
  Causes (relevant to 'chronic abdomen'): Chronic peptic ulcer; esophagogastric varices (due to cirrhosis/chronic pancreatitis/pancreatic tumor); GERD complicated by Barrett's ulcer penetrating into esophageal branch of aorta; ingested (especially sharp) foreign body (either leading to esophageal mucosal injury or penetrating into aorta); aortic aneurysm forming fistulous communication with esophagus.
- Black or brown colored stools of tarry consistency? (**Melena**)
  Cause: GI bleeding from anywhere proximal to sigmoid colon (inclusive of sigmoid colon).
- **(Persistent) wind-ball movement** and **'borborygmi'**? ⇒ subacute/chronic intestinal obstruction.
  (Occasional wind-ball movement may be experienced in an otherwise normal individual with 'indigestion'.)
- **Diarrhea**?
  Frequency, amount, consistency (watery or semisolid); is it associated with gripping pain in central abdomen; does it contain mucus/pus/fat; does it float in the pan; is it foul-smelling?
  Causes (relevant to 'chronic abdomen'): Disorders of small intestine/large intestine/pancreas; malabsorption syndromes.
- **Constipation**?
  Causes (relevant to 'chronic abdomen'): Greatly reduced food intake (including effect of persistent vomiting); (subacute/chronic) intestinal obstruction; uremia; psychogenic.
- **Abnormal bowel habits / bloating**: may suggest irritable bowel syndrome, intermittent intestinal obstruction.
- **Dark colored urine, pale stools and itching**: Features suggestive of obstructive jaundice.
- Present or recent past history of **jaundice**?
  Causes (relevant to 'chronic abdomen'): Disorders of liver/biliary tract.

Associated Specific symptoms

Specific symptoms of **Esophageal** disorder **(including esophagogastric junction and occasionally gastric)**

- Do you get difficulty in swallowing? Do you feel that the bolus of food/liquid gets stuck on its way to the stomach after you have swallowed it?—**Dysphagia**.
- **Excessive belching**?
- Do you find it **difficult/unable to belch**?
- Do you get pain while swallowing?—**Odynophagia**.
- Did you (accidentally (or otherwise)) **swallow any foreign body recently or in the past**?
- Do you experience warmth/burning sensation starting in the epigastrium and radiating up the chest behind lower part of the sternum for variable distance, brought on or aggravated by stooping or lying flat?—**Postural heartburn**.
- Did your heartburn if there was any, disappear spontaneously?—**Vanishing heartburn**.
- Do you experience passive return of swallowed material to the throat?—**Regurgitation**.
- Do you get sour-tasting fluid in the throat after belching?—**Acid eructation**.
- Do you experience a sudden filling of the mouth with clear, frothy salty fluid?—**Waterbrash**.
- Pain in the chest? ('**Esophageal angina':** pain or distress in the chest, often clinically indistinguishable from angina pectoris; caused in some (10–20%) of the patients with gastroesophageal reflux.)
- Hoarseness of voice?
  Cough, stridor, recurrent attacks of pneumonia? } **Aspiration and respiratory symptoms**.
- Sensation of a substernal lump?—**Globus**.

Specific symptoms of **Gastroduodenal** disorder

Classical symptoms of chronic peptic ulcer (duodenal ulcer/gastric ulcer)

- Have you been getting periodic pains in upper abdomen since a few months / years?
- Have you noticed that the periods of exacerbations are particularly associated with periods of stress / smoking / dietary or alcoholic indiscretions?

Free ebooks ==> www.Ebook777.com

- What time / when do you get the pain, i.e. on empty stomach / early morning / induced by food?
- Is the pain relieved – by taking food, or by vomiting (which may be induced)?
- How about appetite? Is it so that your appetite is good, but you are afraid of eating due to fear of pain?
- How about your weight – Have you gained or lost weight?
- Do you feel full soon after eating much smaller quantity of food as compared to before?—Early satiety.
- Hunger and appetite having been preserved, do you still feel afraid to eat?
- Do you feel sorry after eating?
- Do you feel fullness in the upper abdomen (epigastrium) after eating?
- Regurgitation?

} **Indigestion,** i.e. Dyspepsia

RECURRENT DYSPEPSIA AFTER HAVING UNDERGONE SURGERY FOR PEPTIC ULCER

- Duration?
- How much is the symptom-free interval between the operation and commencement of the present symptoms?
- Pain? – Details?
- Hematemesis / Black tarry stools?

Possible causes of recurrent dyspepsia

1. Recurrent ulcer. It is not always easy to diagnose recurrent ulceration.
2. Complications of ulcer surgery, e.g. enterogastric reflux, gastric stasis.
3. Unrelated pathology, e.g. GER, gallstones, pancreatitis.

Pertaining recurrent ulceration

– Presentations: - Pain: Nature of pain may be similar to that experienced before surgery, or different in site, type or both.
- May present with bleeding or perforation with no accompanying dyspepsia.
– Rapid return of symptoms/bleeding/perforation, particularly in a younger patient should suggest the possibility of ZES.

Symptoms due to **SEQUELAE OF GASTRODUODENAL SURGERY**

Type of surgery and consequently more likely sequelae *(or complications)*

– Vagotomy → Early satiety.
– Truncal vagotomy and Drainage → Iron-deficiency anemia, severe intractable explosive diarrhea.
– Gastroenterostomy (in general) → Development of CA stomach.
– Antecolic gastroenterostomy → Afferent loop obstruction especially with long afferent loop (> 20 cm)
– (Extensive) gastrectomy → Loss of weight; Iron-deficiency anemia, megaloblastic anemia (vitamin B_{12} deficiency), 'small stomach syndrome', development of CA in the remnant.
– Gastric resection with duodenal exclusion (Polya) → Bone disease (several years postoperative).

Various symptoms (and their possible causes)**.**

1. Same symptoms as before the operation ⇒ Recurrence of the disease.
2. Transient dysphagia after any type of vagotomy may be caused by edema of the abdominal esophagus.
3. Hematemesis: Acute jejunogastric intussusception, erosive gastritis due to enterogastric reflux.
4. Acute pain in abdomen
   a. Characteristically associated with nonbilious vomiting: may indicate acute afferent loop obstruction.
   b. Acute jejunogastric intussusception. Intra-abdominal mass may be palpable.
5. Chronic/intermittent epigastric pain. Enterogastric reflux.
6. Vomiting (usually but not necessarily bilious): Does it occur within minutes of eating, and associated with other gastrointestinal symptoms of dumping? Cause: Dumping.
   – Other causes of vomiting: Enterogastric reflux/reflux (erosive) gastritis, (usually intermittent) obstruction of the afferent or efferent loop of gastroenterostomy, gastroesophageal reflux/esophagitis, and recurrent ulceration.
   a. *Enterogastric reflux / reflux (erosive) gastritis*
      – Epigastric pain of burning nature, aggravated by food, not relieved by antacids.
      – Vomiting of bile-stained fluid 1–2 hours after meal culminating the attack.
      – Less commonly: Nocturnal burning pain followed by vomiting in early morning.

www.Ebook777.com

b. *Chronic/intermittent afferent loop obstruction*
   - Nausea, fullness and cramp-like pain within 1 hour of eating.
   - Vomiting of copious amounts of bile-stained fluid → relief of symptoms.

   (Various possible causes of extrinsic loop obstruction: internal herniation, kinking of the anastomosis, adhesions, volvulus, stenosis, jejunogastric intussusception, and development of carcinoma of the gastric remnant.)

c. *Gastroesophageal reflux / esophagitis*

   Possible mechanism: damage to esophageal attachments, particularly phrenoesophageal membrane, during mobilization of the esophagus may → cardioesophageal incompetence → reflux.

7. Weakness, tiredness, dizziness, headache, fainting/wanting to lie down, warmth, palpitations, dyspnea, sweating within minutes of eating; attacks being typically precipitated by carbohydrate meals—Systemic (vasomotor) symptoms of 'dumping'.
8. Fullness, epigastric discomfort/heaviness, nausea, vomiting, distension, excessive borborygmi, diarrhea: Gastrointestinal symptoms of 'dumping'.

   Cause of dumping: Rapid (precipitous) gastric emptying.
9. Hypoglycemic symptoms occurring 2–3 hours after a meal—weakness, sweating, tremors, difficulty in concentration, rarely fainting—'Reactive hypoglycemia'.
10. Early satiety: Causes: Vagotomy (because of the loss of receptive relaxation of stomach during eating), (extensive) gastrectomy (because of small remnant of stomach).
11. 'Inability to eat' and many other symptoms after extensive gastrectomy leading to inadequate oral intake leading to gross malnutrition refractory to conservative treatment—'Small stomach syndrome'.
12. Loss of, or failure to gain weight: Nutritional, severe postcibal symptoms.
13. Weakness: Anemia, myopathy associated with bone disease (hypercalcemia).
14. Anemia: Iron-deficiency (hypochromic microcytic), vitamain B_{12} deficiency (megaloblastic).
15. Diarrhea.
    a. Frequent loose motions.
    b. Intermittent episodes of short-lived diarrhea.
    c. Severe intractable explosive diarrhea. Characteristics: Extreme urgency often causing incontinence during an acute attack.

       Precipitated by food. Often accompanied by dumping symptoms. A sequel of truncal vagotomy.
16. Bone pains (several years postoperative): Bone disease.
17. Other complications: Phytobezoars, development of CA stomach in the remnant (particularly after a gastrectomy or gastroenterostomy).

Specific symptoms of **COLORECTAL** disease

- Colicky pain ± fullness (particularly in the flanks) relieved by passing flatus/stools?
- Passage of excessive mucus and/or blood per rectum?
- Passage per rectum, of altered blood mixed with mucus/pus (especially apart from defecation), and tenesmus?
- Recent alteration of bowel habit?

Specific symptoms of **HEPATIC** disease

- Fatigue, malaise, myalgia, arthralgia, headache: Although more or less 'nonspecific', these symptoms suggest hepatitis.
- Jaundice? The presence of jaundice ⇒ active liver disease.
- Forgetfulness, confusion, poor concentration, personality changes ⇒ advanced hepatic cirrhosis.

History to be asked in a suspected case of **CHRONIC RELAPSING PANCREATITIS**

Possible causes of pain: 1. 'Pancreatic intractable' pain.
2. Low-grade cholangitis.
3. Development of CA.

- Site of pain?
- Intensity?: If severe, how much is narcotic need?

Free ebooks ==> www.Ebook777.com

- Nature: Constant / colicky / dull and vague?
- Precipitating / aggravating / relieving factors?
- Alcoholism controlled or not?
- Frequency of getting attacks of pain: Leads to how much of disruption of life?
- Associated symptoms: - Fever with chills?
  - Jaundice, pale-colored stools, itching?
  - Hematemesis / melena?
  - Anorexia, nausea, vomiting, dyspepsia, weight-loss?

INTERPRETATION of pain apparently caused by pancreatic disorder

Site of pain: Inconstancy in the site of pain may suggest CA.

Intensity of pain

– 'Pancreatic intractable' pain is severe.
– If severe, and is because of CA, then it suggests invasion of perineural lymphatics or posterior parities.
– Pain because of CA is of variable intensity.

Nature of pain

– Usually constant, however, that does not necessarily differentiate one condition from the other.
– Dull and vague in CA.
– Colicky pain may suggest compression of D3/4 by CA.

Pancreatic intractable pain may be ***precipitated by*** alcohol.

Associated symptoms

– Fever with chills ⇒ ascending cholangitis ⇒ long-standing biliary obstruction, which in turn is suggestive of CA.
– Obstructive jaundice ⇒ CA - head / neck of the pancreas.
– Hematemesis / melena. Possible causes: Gastric/duodenal mucosal invasion, or (sectorial) portal HT caused by CA.
– The nonspecific symptoms (Anorexia, nausea, vomiting, dyspepsia, weight-loss) ⇒ CA.

Specific **UROLOGICAL** symptoms

- Pain while passing urine? If so, is the pain experienced almost throughout the stream, or is it experienced particularly at the end of micturition?
- Pain in the perineum?
- Sensation of fullness/weight in the perineum?
- Pain during defecation?
- Increased frequency of micturition, nocturia, urgency, urgency incontinence?
- Hesitancy, poor flow, feeling of incomplete emptying, after dribbling?
- Urethral discharge?
- Any noticeable change in the urine passed - Color: Red/Brown/Stale tea-like/Smoky/'Cloudy'/Turbid / White?
  - Presence of gravel/stone, fragments of a growth, etc.?
- Not passed urine (at all/satisfactorily) since a few hours?
- Incontinence?

Specific symptoms to be asked in case of **CHRONIC PELVIC PAIN IN A FEMALE PATIENT**

- **Pain**
  – Duration?
  – Cyclical or Constant? Pelvic pain at monthly interval may ⇒ endometriosis.
    |
    Mid-cycle or otherwise?
  – Is it related to MC? - Mid-menstrual, or just before and during menstruation?
    - Mid-menstrual pain ⇒ rupture of Graafian follicle.
    - Pain before and during menstruation ⇒ dysmenorrhea / uterine and adnexal diseases.
  – Exact site?
  – Deep dyspareunia?
  – Pain on defecation?
  – Is intensity of pain worse at the end of the day?
  – Nature: Colicky / constricting / dragging / burning / throbbing / twisting / shooting / vague?

www.Ebook777.com

- **Dyspareunia**?
- **Any lump in the pelvis**?
- **SCOPV**?
- **Obstetric** history?
- **Menstrual** history?

NOTES ON CHRONIC PELVIC PAIN IN A FEMALE PATIENT

There are no universally accepted criteria, but it may be defined as cyclic/noncyclic pain in the lower abdomen or pelvis or lower back or buttocks for at least 6 months duration that affects the quality of lite (QOL) and motivates the patient to seek medical help. Up to 90% of patients with chronic pelvic pain will undergo one/more unsuccessful and often unnecessary gynecologic procedures.

The differential diagnosis of chronic pelvic pain is extensive, and the cause is often multisystem and multifactorial.

Acute pelvic pain may be defined with the same criteria, but which lasts for < 1 month.

Past history

- Past history of any surgery?

  Previous surgery may be responsible for—postoperative adhesive intestinal obstruction (beyond 7 days after surgery) / pelvic adhesions leading to chronic pelvic pain in female patient.

Symptoms suggestive of 'referred pain'

– Swelling in the scrotum?
– Any chest-symptoms?

ABDOMINAL EXAMINATION

Examination-bed

It is preferable to have a hard bed with a thin mattress as it helps to maintain the normal lumbar lordosis, thereby opening up the maximum area of the anterior aspect of the abdomen.

If the mattress is thick and soft, the back will sag and arch, diminishing the abdominal area available for examination.

POSITION of the patient: **Lying down.**

- With or without flexion of the lower limbs?

Flexion of thighs → relaxation of mainly the iliopsoas; relaxation of the anterior abdominal wall being only minimal. Moreover, there is a disadvantage of flexed thighs—they come in the way of palpation of lower abdomen. Thus, the disadvantage nullifies the advantage gained if at all by flexing the lower limbs. Hence, it may be preferable to keep the lower limbs straight.

Avoid keeping a pillow under the head. Reason: With a pillow under the head, the head is automatically raised to some extent allowing the patient to see (although with a little difficulty) what is being done to the abdomen. Out of curiosity, there is a tendency to raise the head a bit further up off the pillow leading to contraction of the anterior abdominal-wall muscles, which is not desirable.

EXPOSURE of the patient

Initially only from nipples ↔ groins.

At the end of palpation, exposure is extended to include the private parts and down to mid-thighs.

Exposure of the private parts tends to make the patient apprehensive and shift the position in an effort to cover up the private parts. This prevents relaxation of both mind and body, which precludes proper examination. Exposing the private parts at the end would help to keep the patient relaxed through the initial 'main' course of palpation.

INSPECTION

Methods

1. From top.
2. From side, i.e. tangential—This often requires some bending even if the examiner has acquired sitting position by the bed-side.

   Tangential inspection helps to detect smaller bulges and pulsations.

Free ebooks ==> www.Ebook777.com

Movements—of the anterior abdominal wall associated with (normal) respiratory excursions

– Normal / diminished / absent?
– If diminished or absent – confined to a region, or all over the abdomen?

Contour

- In the anteroposterior diameter: Concave (i.e. scaphoid) / flat / bulging?
- In the flanks: Concave / flat / bulging?
- Apparently generalized distension?
- Any localized fullness?

Clinical method to define the contour:
Imagine a plane passing through a straight line between the xiphoid and symphisis pubis.

– Anterior abdominal wall parallel to (i.e. coinciding with) this plane—'flat' abdomen.
– Anterior abdominal wall lying below this plane—boat-shaped—'scaphoid' abdomen.
– Anterior abdominal wall lying above this plane—'bulging' abdomen—distension.

Interpretation

1. In the anteroposterior diameter: Flat contour ⇒ nothing particular. However, an abnormality may not be ruled out.
2. Causes of scaphoid contour in the anteroposterior diameter: a. Normal in a thin-built individual.
   b. Undernourishment/cachexia.
3. Causes of bulging contour in the anteroposterior diameter
   a. Constitutional (Normal for the individual).
   b. Due to intra-abdominal pathology: – (Distal) intestinal obstruction / Gastric outlet obstruction.
   – A large intra-abdominal lump.
   – Tympanitis.
   – Paralytic ileus.

   Fullness in the **epigastrium and upper umbilical** region more on the left side ⇒ distended stomach.
   Central abdominal fullness, i.e. around the umbilicus ⇒ – distal small intestinal obstruction.
   – later stages of large intestinal obstruction, if back pressure is transmitted across the incompetent IC valve.

   Fullness in the **right lumbar region** ⇒ distension of cecum and ascending colon.
   Fullness in the **left lumbar region** ⇒ distension of the left colon.
   Fullness all along the periphery of the abdomen ⇒ distension of the entire large intestine ⇒ obstruction in the pelvic colon/rectum.
   Severe distension is encountered in distal small intestinal obstruction, left colonic obstruction, volvulus of pelvic (sigmoid) colon, Hirschprung's disease, acute colonic pseudo-obstruction.
4. Bulging mainly in the flanks; **bulging in the anteroposterior diameter being only slight** (i.e. bulging in the flanks much more than bulging in the anteroposterior diameter) suggests free fluid in the peritoneal cavity.
5. **Suprapubic** fullness ⇒ distension of urinary bladder or a big pelvic lump.
   At a volume of about 500 ml distended bladder becomes visible in thin-built patients.

Asymmetry?

Slight asymmetry can only be made out if patient lies well relaxed and without twisting. Asymmetry helps in localization of the disease. Asymmetry may result from a lump/distension that is too small to be detected as an obvious objective finding.

Any visible lump?

Note the features of the lump as usual.

Skin

– Discoloration: • Hyperpigmentation in the midline occurs in pregnancy.
• Otherwise, skin pigmentation may indicate intestinal polyposis.

www.Ebook777.com

– Striae (furrows): bands/streaks/lines distinguished from the tissue in which they are found by color/depression/elevation/texture.
  Striae develop due to rupture of reticular dermis as a result of sustained stretching.
  Bands of thin wrinkled skin, initially red, becoming white with passage of time.
  Causes: Pregnancy, obesity, ascites, Cushing's syndrome, abdominal carcinomatosis.
  Other sites in the body where cutaneous striae are found—Thighs and buttocks.

Dilated veins

Around the umbilicus (in radial fashion), or on the sides of the abdomen?

– Dilated veins around the umbilicus in radial fashion usually ⇒ portal HT.
– Dilated veins on the sides of the abdomen ⇒ IVC or SVC obstruction.

Umbilicus

– Site: • Normal / Upward displacement / Downward displacement?
  • Normal: Midway between xiphoid process and symphysis pubis.
  • Upward displacement is caused by a large pelvic lump.
  • Downward displacement is seen in ascites.
– In case of a distended abdomen – Is it depressed (i.e. depth accentuated) OR Transversely stretched OR Flush/everted?
  • Depth is accentuated in obesity.
  • Transversely stretched umbilicus ⇒ free fluid in the peritoneal cavity.
– Is it bulging? · If bulging, does it lie on top of the bulge OR near the base of the bulge?
  Bulging umbilicus may suggest irreducible umbilical hernia OR a swelling in the anterior abdominal wall close to the umbilicus. Differentiation between the two may be difficult. However, • the umbilicus lying on top of the swelling suggests umbilical hernia.
  • the umbilicus lying near the base of the swelling suggests solid tumor of abdominal wall.
– Any discharge: s/o sinus/fistula?
– Stone (umbilicolith)?
– Endometrioma: Bleeding during menses.
– Hard nodule (malignant)?
– Granuloma/polyp?

Visible peristalsis?

Clinical method

Ask the patient to hold breath at the end of expiration, and inspect the abdomen.

Is there a clinical background already suggestive of (chronic) gastro/intestinal obstruction? If so, then the visible peristaltic waves are abnormal.

Site and direction
- Upper abdomen. Left to right ⇒ gastric outlet obstruction.
- Multidirectional* ⇒ small intestinal obstruction.
- From below upwards ⇒ peristalsis in ascending colon.
- From right to left ⇒ peristalsis in transverse colon.
- From above downward ⇒ peristalsis in descending colon.

Visible peristalsis ⇒ subacute/chronic gastric/intestinal obstruction, provided symptoms and other signs agree.

Only rarely are they seen in acute obstruction; and if they do, they will be seen only during the late stage of obstruction. Peristaltic waves may be visible even in individuals otherwise not suffering from a significant abdominal disease.

Examples: – Elderly multiparous women with divaricated recti and thin atrophic abdominal wall muscles.
– Large abdominal wall herniae with thin coverings.
– Scrotal hernia containing loops of intestine.

* **'Ladder pattern' appearance:** Central abdominal distension with horizontal grooves. The appearance results from distended loops of obstructed small intestine; seen in chronic obstruction, rather than in acute.

Free ebooks ==> www.Ebook777.com



Fig 33-3 'Ladder pattern'. The inset shows the scar of previous laparotomy

Pulsations

Clinical method

Ask the patient to hold breath at the end of expiration, and inspect the abdomen **tangentially**.

Pulsations could be normal or abnormal.

Fullness (bulging) of intercostals spaces

Causes: Large hepatic abscess, subphrenic abscess.

PALPATION

POSITION of the examiner: Sitting **comfortably** on the right side of the patient.

Method

First and foremost, ask the patient to palpate her/his own abdomen and tell the findings regarding tenderness and lump.

– Is there any tenderness? - If yes, what is the exact site?
– Is there any lump?

1. Patients have a natural tendency to conclude that tenderness may be felt at the same site as that of pain. Patient should be explained that she/he may not experience tenderness at the site of pain; on the contrary, she/he may even feel relief on pressing at the site of pain (as in case of colics because of spasm).
2. If patient localizes an area of tenderness (which may be either at the same site as that of pain, or different), the clinician can be most gentle while palpating that particular area.
3. This method also exhibits caring attitude of the clinician, which helps to win the patient's confidence. This time is actually well spent in making the patient relaxed, dispel fear and secure cooperation throughout further examination.

Sequence of palpation

– The region that is farthest from the site of tenderness if any, as perceived by the patient herself/himself, or farthest from the site of pain/lump.
– If the chief complaint has been other than actual pain, and apparently if there is no lump (as judged from history or on inspection), then begin palpation in the LLQ. Reason: LLQ is the least common site of pathology. Subsequently, proceed anticlockwise i.e. LLQ→LUQ→RUQ→RLQ→Umbilical region.
  To achieve a relaxed abdomen continuously during palpation, it is essential to distract patient's attention from abdominal examination. Keep talking to the patient frequently/as required. **The patient should not be allowed to talk** as talking necessitates contraction of the anterior abdominal-wall muscles.

A. First round of palpation: Gentle 'superficial' palpation with flat of the fingers

1. Any **hyperesthesia in well defined areas**?

Viz. Shoulder / Boas' area / Sherren's triangle / lumbar area.

Inference

– Hyperesthesia in shoulder region ⇒ irritation of undersurface of the diaphragm due to blood/pus (hemoperitoneum / subdiaphragmatic abscess respectively).

www.Ebook777.com

- Hyperesthesia in Boas' area (over 9th, 10th and 11th ribs posteriorly on the right side) ⇒ cholecystitis.
- Hyperesthesia in Sherren's triangle (formed by pubic symphysis, umbilicus and highest point of iliac crest) ⇒ appendicits.
- Hyperesthesia in lumbar area may favor a disorder of nerves (radiculopathy, neuritis, etc.) thus differentiating it from a renal disorder.

2. Abdomen—**Soft, OR Contracted**? **If contracted**, proceed as in chapter 34, page 439.
3. **Tenderness** present or absent? If present - in which area/quadrant?
4. **Lump**?
5. (Gross) **Organomegaly**?

B. Next round of palpation: 'Deep' palpation to obtain details

1. Localization of tenderness

Purpose

Tenderness is a **very useful sign** and **perhaps the only sign available in most of the cases** of chronic pain in abdomen. Hence, it is essential to elicit localization of tenderness properly. **Site of localized tenderness has got diagnostic significance**; the site of disease corresponds to the site of tenderness. As against this, the site of pain may not denote the site of disease.

Pre-requisite

Relaxed muscles of the anterior abdominal-wall. **Voluntary contraction/guarding may mask tenderness**.

Difficulty

Children and apprehensive adult patients become instinctively afraid of palpating hand which results in voluntary contraction of the anterior abdominal wall muscles, precluding satisfactory palpation. Application of the chest-piece of stethoscope, instead of hand, to the anterior abdominal wall to localize the tenderness may circumvent this difficulty.

Practical mistakes to be avoided

i. Avoid jerky palpation.
ii. Do not apply pressure over a very broad area.
iii. Do not touch/press two different areas with two (different) hands simultaneously.

Principle

The presence of tenderness should be concluded only if the patient **winces the face and flinches the body**. If any one of these two minimum criteria is absent, then think twice before concluding the presence of tenderness *even if a patient otherwise overreacts*!

Clinical method

(a) **If a broad area of tenderness was detected** on earlier 'superficial palpation', then within the perimeter of that broad area, apply **gentle persistent pressure with a finger-tip**; and attempt to localize a **tender *spot*, around which the patient does not feel any tenderness**.

In addition, (b) search for **localized tenderness at the sites of surface-markings of certain organs, which are commonly prone to disease.**

The organ	*Point of tenderness*	*Surface marking*
– **Pyloric** part of the **stomach**	Gastric point	On trans-pyloric plane (midway between xiphoid and umbilicus), 2.5 cm to the left of the midline.
– **D1**	Duodenal point	On trans-pyloric plane, 2.5 cm to the right of the midline.
– **Gallbladder**	1. Murphy's point 2. Boas's point 3. Superior phrenic point	Tip of the (right) 9th costal cartilage (where the trans-pyloric plane cuts the right costal arch; OR where the left spinoumbilical line cuts the right costal arch). To the right of the 10th interspinous ligament. Between the 2 heads of sternocliedomastoid muscle.
– **Appendix**	McBurney's point	Junction of lateral 1/3 and medial 2/3 of the right spinoumbilical line.
– **Retrocecal appendix**	No specific name.	A little inferior to the renal angle.

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

The organ	*Point of tenderness*	*Surface marking*
– **Uterine tubes**	No specific name.	In suprapubic region, on both sides of the midline.
– **Kidney**	Renal angle	Angle between the sacrospinalis and the last rib.
– **Subphrenic space (abscess)**	1. Anterior phrenic point 2. Posterior phrenic point 3. Superior phrenic point	Tip of the 10th rib. In the angle between sacrospinalis and the 11th rib. Between the 2 heads of sternocliedomastoid muscle.

(c) Ask the patient to **raise both the lower limbs straight** (i.e. without flexing the legs at the knee joints), and press again at the **previously determined tender spot.** Compare the degree of tenderness.

- Tenderness soon on contracting the muscles, even before pressing suggests pathology in musculo-aponeurotic tissue of the anterolateral abdominal wall.
- Tenderness to almost the same degree or even more in comparison to the degree of tenderness with the abdominal wall muscles in relaxed state suggests pathology possibly in the abdominal wall.
- Marked decrease in the degree of tenderness or disappearance of tenderness suggests intra-abdominal pathology.

Conclusion

The site of localized tenderness has diagnostic significance; the site of disease corresponds to the site of localized tenderness.

2. Any **Lump**?

If palpable, examine the details as described in chapter 35.

3. Organomegaly?

None of the normal abdominal viscus is palpable.

Occasional exceptions: Liver, right kidney, cecum and sigmoid (pelvic) colon.

Liver

Clinical methods of palpation

a. Place the hand in RLQ with fingers parallel to the costal margin and pointing towards the left axilla. Ask the patient to breathe deeply.

   Liver edge moves down 3–4 cm during deep inspiration.

   If hepatomegaly is 'massive', then the edge of the liver will strike the radial border of index finger and also, the finger may be felt to ride over the edge of the liver.

   If not, then shift the hand up a bit towards the right costal margin. Continue shifting the hand cranially bit-by-bit synchronizing with deep breathing excursions of the patient, without lifting the palpating hand off the abdominal wall much, until either the liver-edge is felt or the right costal margin is reached.

b. Place fingers of one or both hands lateral to rectus abdominis, pointing towards the ribs. If resistance is encountered, keep moving the fingers caudally until resistance disappears. Maintain the position of the fingers at that level. Ask the patient to breathe deeply.

   At the zenith of inspiration press the fingers firmly, cranially and inwards.

   The fingertips will be felt to ride over the edge of the liver.

c. In obese/muscular individuals: Place the left hand in the loin and push the posterior abdominal wall forwards (anteriorly). And palpate the liver with the right hand from anterior aspect as usual (as in (a)).

d. Technique for a minimally enlarged liver: Place the palm of hand on the thoracic cage with fingers hooking around the costal margin. Ask the patient to breathe deeply. During zenith of inspiration the edge of the liver may be felt.

e. Dipping method: This method is useful in the presence of ascites.

   Principle—Dipping fingers displace the fluid away making the mass palpable.

   Place fingertips of one or both hands on the abdominal wall (taking care not to hurt with untrimmed fingernails). Depress the fingers by a quick push to feel the organ (or lump). The use of this method can be extended for palpation of other organs and lumps as well.

www.Ebook777.com

A pitfall

A fibrous intersection running transversely through the rectus muscle at about the level of the tip of the 9th costal cartilage may be mistaken for the edge of the liver.

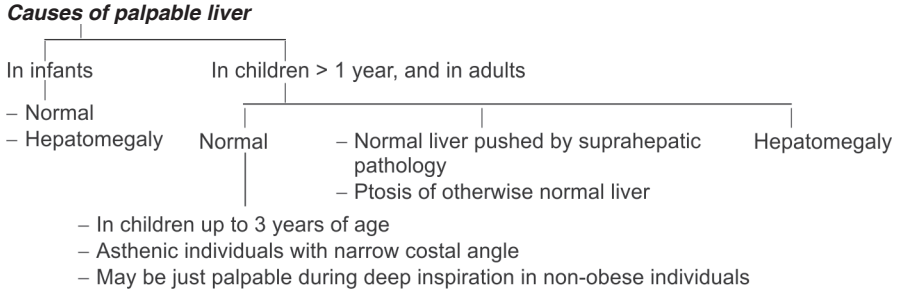
After palpation, determine the superior margin of liver by percussion.

Upper limit of normal liver dullness in	Lies at the level of
– Mid-sternal line	Xiphoid process
– Right mid-clavicular line	5th IC space
– Mid-axillary line	7th IC space
– The line of scapular angle	9th IC space

Details of hepatomegaly to be noted

- Tender or non-tender?
- Extent of enlargement inferior to the costal margin: in the 'unit' of centimeters or fingerbreadths.
- Surface: smooth / nodular / irregular?
- Consistency: soft / firm / hard?

Causes of palpable liver



Gallbladder

Normal gallbladder is not palpable.

Enlarged gallbladder can be palpated in the area of Murphy's point.

Gallbladder enlarges towards the umbilicus; the axis of enlargement being the left spinoumbilical line.

Clinical method of palpation

Place the hand in RLQ with fingers parallel to the costal margin and pointing towards the left axilla. Ask the patient to breathe deeply. Watch for the rounded border of enlarged gallbladder to strike the radial border of index finger.

If not, then shift the hand up a bit towards the right costal margin. Continue shifting the hand cranially bit-by-bit synchronizing with deep breathing excursions of the patient, without lifting the palpating hand off the abdominal wall much, until either the enlarged gallbladder is felt or the right costal margin is reached.

Spleen

Normal spleen is not palpable except very rarely in thin-built individuals.

Spleen has to enlarge at least twice in size before it becomes clinically palpable.

When enlarging, it extends obliquely from the left costal margin → RLQ. Enlargement in a straight downward axis towards the LLQ is apparently prevented by phrenico-colic ligament. Anterior border is sharp with one/two characteristic notches.

Clinical methods of palpation

a. Place the hand in the RLQ with the fingers pointing towards the left hip. Press 'slightly' backwards (i.e. posteriorly). Ask the patient to breathe deeply.

   If the spleen is enlarged 'massively', the edge will slip under the fingers during the zenith of inspiration.

   If not, then shift the hand up a bit towards the left costal margin. Continue shifting the hand cranially bit-by-bit synchronizing with deep breathing excursions of the patient, without lifting the palpating hand off the abdominal wall much, until either the splenic edge is felt or the left costal margin is reached. The spleen is identified by its typical notched border.

b. Bimanual palpation is useful to palpate minimally enlarged spleen that is not palpable by the method described above.

Free ebooks ==> www.Ebook777.com

Insinuate the right hand under the (left) costal margin. Place the left hand behind left side of the lower chest and lift it upwards. Slightly lateral position helps. Try to feel for the spleen with the right hand.

c. 'Hooking' method of palpation: This is yet another method to palpate minimally enlarged spleen that is not palpable by the method described under (a).
   Stand on the left side of the patient.
   Place the palm of hand on the thoracic cage with fingers hooking around the costal margin. Ask the patient to breathe deeply. During zenith of inspiration the edge of the spleen may be felt.
d. Dipping method: As described in palpation of liver (e).

After palpation, determine the superior margin of spleen by percussion in the (left) midaxillary line.
Upper limit of normal splenic dullness in the left midaxillary line lies at the level of the 9th IC space.
Normal extent of dullness in the midaxillary line spans over 9th to 11th ribs.
Splenic dullness cannot be consistently elicited in areas other than in the midaxillary line, because of the presence of gas-filled stomach and splenic flexure of colon.

Kidneys

In adult, kidneys are normally difficult to palpate because of anatomical position.

In children and thin-built women, it may be possible to palpate the lower pole of the right kidney with deep inspiration. In neonates, kidneys can be felt quite easily.

In men, it is usually not possible to palpate either kidney. Reasons: 1. Lesser degree of downward movement with respiration. 2. Thicker muscular layer of abdominal wall.

Left kidney is almost always impalpable unless it is abnormally enlarged.

Clinical methods of palpation

a. For palpation of the right kidney, stand/sit on the right side of the patient. Place the left hand in the loin. Place the right hand anteriorly on the flank. Press both hands towards each other with 'slight' pressure. Ask the patient to breathe deeply. During deep inspiration, the downward moving kidney may tend to separate both the hands. Try to anticipate this, and during expiration, try to grasp the kidney and feel the details.
   For palpation of the left kidney, stand on the left side of the patient and reverse the roles of the hands.
b. Single-handed bimanual palpation: This method is used to palpate both kidneys simultaneously. This method can be applied *only in children and thin individuals*.
   Place the fingers posteriorly in the loins. Place the thumbs anteriorly on the flanks.
   Lift the kidneys with fingers and try to feel them with thumbs.
c. Ballottement. Purpose—essentially to determine the mobility of the kidney, and consequently also to prove that the lump is in connection with the kidney (i.e. arising from the kidney).
   Stand on the respective side of the kidney that is being examined.
   Place one hand posteriorly in the loin (the displacing fingers) and the other hand anteriorly on the anterior abdominal wall overlying the lump (the watching hand).
   Impart short quick forward thrusts with the displacing fingers → Bouncing sensation (as if a ball is striking) felt by the watching hand ⇒ positive test. The peculiar sensation is due to the impact of the swelling on the anterior abdominal wall.
   Fallacy: If the hand is not kept properly in the renal angle but instead a little lateral to the renal angle, OR if the forward push is brisk rather than gentle, then the sign of ballottement may appear to be falsely positive even in lumps which are not related to kidney.

Percussion

Sacrospinalis and psoas muscles completely mask the dullness of a normal-sized kidney. Only when the kidney enlarges enough to reach the flanks does it produce dullness which is continuous with the paravertebral dullness.

Urinary bladder

It may be worth attempting palpation of bladder in children and thin-built adults.
Clinical method: Lift up the lumbar spine with one hand while pressing the other hand into the midline of the lower abdomen.
Note: In adult, normal bladder is neither palpable nor percussable.

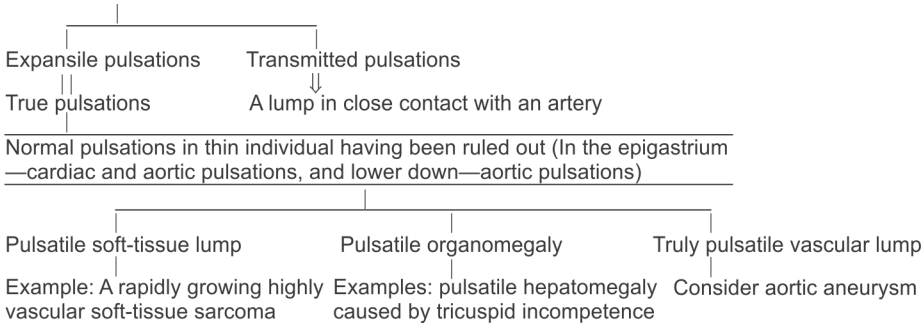
www.Ebook777.com

4. **Hernia of the linea alba**?

   A hernia of linea alba may be missed completely especially in fatty patients, unless specially searched for. It is felt as a nodule in the midline, usually somewhere close to the transpyloric plane.

   In the absence of a palpable swelling, tenderness may be suggestive, especially on contracting the recti.

5. **Pulsations of the aorta and iliac arteries**?



6. **Para-aortic enlarged lymph nodes**?

7. **'Succussion splash'**?

   Clinical method

   a. For gastric succussion splash: Grasp the lower rib cage with both hands and shake gently from side-to-side.

   b. For intestinal succussion splash: Grasp the pelvis with both hands and shake gently from side-to-side.

   Pre-requisite

   The patient should not have eaten or drunk for 3 hours because a normal stomach especially when full, can give a succusion splash.

   Observation and inference

   Splashing sound (heard without the aid of a stethoscope) suggests the presence of gas and fluid in a (hugely) dilated part of GIT or a cavity.

   Causes of succussion splash

   – Normal stomach especially when full, *up to 3 hours after a meal*.
   – (Abnormally) Dilated stomach due to atony/gastric outlet obstruction.
   – Huge dilatation of the colon/small intestine.
   – Hydropneumothorax.
   – Perforative peritonitis, abscess containing gas: In practice however, no patient with suspected perforation/abscess should be subjected to this test; it hurts the patient.

8. **Determine the direction of blood-flow in dilated veins,** if any

   Clinical method

   Two segments of vein, one each, above and below the umbilicus must be tested.

   Place two fingers on a segment of a dilated vein to be tested. Separate the two fingers apart maintaining gentle pressure to empty about 5 cm long segment of the vein.

   Lift only one of the two fingers, say caudal one, and observe if the segment of the vein fills up or not. Filling up of the vein suggests that the direction of blood flow is from caudal to cranial.

   If the vein does not fill up, then repeat the procedure, reversing the role of the fingers.

9. **Measurements**

   Compare the length of both spinoumbilical lines.

   In case of a large lower abdominal lump, the lump may be assumed to be arising from that side on which the spinoumbilical length is longer.

Free ebooks ==> www.Ebook777.com

PERCUSSION

Aims of percussion:
- to determine, in combination with palpation, the size of normal organs viz. liver, spleen and kidneys.
- to determine the structure (content) of a lump.
- to detect a distended urinary bladder.
- to detect abnormal collection of fluid/gas.

Clinical methods for fluid-thrill, horse-shoe-shaped dullness, and shifting dullness are described below.

'Fluid-thrill'

Purpose: To detect large amount of free or loculated fluid within the abdomen.

Procedure

- Ask the patient or assistant to keep the ulnar border of her/his hand firmly pressed on the anterior abdominal wall in the midline.
  Purpose of applying firm pressure: (a) to cut off the wave of thrill passing through the subcutaneous fat of the abdominal wall, and (b) to increase the intra-abdominal tension.
- Keep the flat of one hand and fingers gently pressed in one flank (the perceiving hand). Give sharp taps with fingers of the other hand, on the opposite flank.

Observation: Thrill felt by the perceiving hand ⇒ positive test.

Inference: positive test ⇒ ascites, large ovarian cyst, large hydronephrosis (large—so large that borders reach both the flanks).

Fallacy: False positive result may be obtained due to wrong technique in obese patients: If enough pressure is not applied in the midline, then a sensation of thrill may pass along the subcutaneous tissue (carried by fat of the subcutaneous tissue).

'Horse shoe shaped dullness'

Purpose: Demonstration of 'moderate' amount of *free* fluid in the peritoneal cavity occupying both the flanks and pelvis.

Procedure

Begin percussion from any fixed point in the midline in the epigastrium towards any of the two flanks. Mark the point if and where resonant note changes to dull note. Repeat percussion from the same fixed point in the epigastrium caudally in an axis that is medial to the previous one by a few degrees. Keep repeating until, after crossing the midline, finally the opposite flank is reached. Each time, keep marking the point at which resonant note changes to dull note. Join all the marked points.

Inference

a. The portion of the abdomen beyond the curved line contains abnormal fluid.
b. 'U'-shaped curved line with concavity facing cranially ⇒ free fluid in the peritoneal cavity occupying both the flanks and pelvis.
c. Curved line with convex border facing all sides ⇒ a cyst/solid mass arising from the pelvis.

'Shifting dullness'

Purpose: Demonstration of *free* fluid in both the flanks.

Principle

In supine position free fluid gravitates in the flanks and the air-containing loops of intestine float subjacent to the anterior abdominal wall. Therefore, percussion note is resonant in the central abdomen, whereas it is dull in the flanks. With change of position the free fluid shifts and so does the dullness.

Procedure

Begin percussion in the midline say at the level of the umbilicus and proceed towards any one of the flanks. Maintaining the position of the finger at which point resonant note changed to dull note, ask the patient to turn on the opposite side. Wait for a minute to allow the fluid to gravitate in the opposite (dependent) flank and intestinal loops to float in the upper (non-dependent) flank.

Percuss again at the same point where position of the finger was maintained.

If there was free fluid in the flank, then dull note will be replaced by resonant note.

Repeat the test in the other flank; only after which the test is complete and consequently the inference be considered valid.

www.Ebook777.com

Dullness may also shift in case of a mobile cyst (i.e. cyst with a long pedicle)**!**
Differentiation from free fluid: Apart from the fact that the dullness produced by a cyst will have convex borders on all sides, on change of posture, the site of dullness may shift *but the shape remains the same*.

Clinical method to differentiate massive ascites from very large cyst
Place the edge of a ruler over abdomen just above the anterior superior iliac spines and press it firmly backwards.
Transmission of aortic pulsations to the fingers holding the ruler ⇒ cyst.
Ascitic fluid does not transmit aortic pulsations.

AUSCULTATION

Aims of auscultation: to look for
- abnormal peristaltic sounds
- abnormal sounds overlying the liver and spleen
- bruit overlying aorta/its main branches
- to determine the size of (enlarged) stomach.

1. **Peristaltic sounds**: Normal / 'hyperactive' / hyperperistaltic / diminished / absent. For details, refer to page 442.
   'Borborygmi'—peristaltic sounds that are heard without the aid of a stethoscope. Applicable to only hyperactive and hyperperistaltic sounds.
2. **Abnormal sounds overlying the liver**
   Rubbing sound
   Causes: Perihepatitis (associated with abscess), hepatic neoplasm.
   Pathologic anatomy: Irregularity of visceral/parietal/both layers of peritoneum overlying the liver.
   Venous hum
   Basically, 'hum' means an indistinct low pitched prolonged sound. Venous hum means a continuous blowing or humming sound heard on auscultation overlying a vein.
   Causes of hepatic venous hum: Cavernous hemangioma of liver, cirrhosis, portal HT.
3. **Abnormal sounds overlying the spleen**
   Rubbing sound
   Causes: Perisplenitis (associated with abscess), splenic neoplasm.
   Pathologic anatomy: Irregularity of visceral/parietal/both layers of peritoneum overlying the spleen.
4. **Bruit** over the aorta/its main branches (such as renal arteries, etc.)
   Basically, arterial bruit means a noise produced by low frequency vibrations of the arterial wall caused by disturbances in blood flow.
   Causes: Coarctation of aorta, arterial stenosis (such as of the renal artery), aneurysm, A-V fistula, atherosclerosis, etc.

Auscultation with scraping
Purpose: to determine the inferior border, i.e. the greater curvature (and consequently the size) of the stomach.
Procedure
Place the bell of a stethoscope just inferolateral to xiphisternum. Make scraping movements with a finger along the lines radiating downwards from this point.
Observation and inference: Peculiar sound is heard as long as the finger is moving overlying the stomach. Nature of the sound changes suddenly after crossing the limit of the stomach.
Clinical application: Inferior border, i.e. the greater curvature of stomach can be mapped by joining several such points at which the peculiar sound changes suddenly.

PALPATION IN SITTING POSITION with hands locked and resting up on the head

'Hands up on the head' → lifting up of the rib cage → 'opening up' a wider area of the abdomen for examination. Abdomen can be palpated in this manner more satisfactorily in thin-built individuals rather than in overweight or obese.

Free ebooks ==> www.Ebook777.com

Purpose

Upper abdominal organs/lumps descend to some extent due to gravity, and become amenable to more detailed examination. Some physical findings may be detected if they were missed while examining the patient in lying-down position.

1. **Gallbladder tenderness**
   Gently hook the fingers under the right costal margin in the area of Murphy's point. Ask the patient to take deep breaths. Patient wincing with a 'catch' confirms tender gallbladder.
   The same procedure may be carried out on the left side for comparison.
2. Feel for **localized tenderness in the rest of the abdomen.**
3. **Any lump** palpable now, which was not palpable earlier when the patient was being examined in lying-down position?
4. **If a lump/organomegaly was noticed earlier, are there any changes in the physical findings** with change in the patient's position?
5. **Kidney**
   - Mark the renal angles on both sides. (Angle formed between the lowest rib and lateral border of sacrospinalis.)
   - After making sure that the patient's trunk is not twisted, inspect the renal angle for fullness, and palpate for a 'mass'. A visible fullness or/and a palpable mass ⇒ enlarged kidney.
   - Fullness or a mass lateral to the renal angle ⇒ possibility of a retroperitoneal mass (other than that connected to the kidney).

EXAMINATION OF GROINS AND EXTERNAL GENITALIA

1. **Pulsations of external iliac and femoral arteries**
   Absent / weak pulses on both sides ⇒ – Disease of the aorta – abdominal or higher up.
   – Rupture of a dissecting aneurysm.
   – Abdominal angina.
   (Diseases of abdominal aorta may occasionally cause pain in abdomen.)
2. **Inguinal lymph nodes**
3. **Hernial sites**: Epigastric, umbilical and paraumbilical, laparoscopoic port sites, inguinal, femoral, obturator, gluteal, sciatic.
4. **Scrotum.**

PELVIC EXAMINATION

- In a female patient: Vaginal and rectal examination.
- In a male patient: Rectal examination.

On PV/E: – Prolapse?
– Tender nodules in the pouch of Douglas?
– Uterus mobile or fixed?

Digital Rectal Examination (DRE)

Secondary deposits from gastric cancer may be felt in the rectovesical and rectovaginal pouch, and on the ovary.

DISCUSSION / INFERENCE on some selected clinical points

Age

(Commoner) Causes of chronic abdomen **in children**: Hiatal hernia, megacolon, chronic dysentery, UTI, urinary stone disease, TB of spine/pleura (→ referred pain), secondary to chronic coughing/vomiting, pica, trichobezoar, worm infestation, 'abdominal' TB, psychogenic to escape from going to school.

Gender

Conditions more common in women: Visceroptosis, gallbladder disease, functional pain.

Residence

Urinary stone disease, filariasis, etc. are common in endemic areas.

Occupation

Lead poisoning in painters, arsenic poisoning in industry → chronic intermittent pain.

www.Ebook777.com

PAIN

'Silent areas of GIT' are those areas where pain resulting from a disease is perceived very late. Examples - Middle portion of the body of stomach, ascending colon, and posterior peritoneum.

DYSPEPSIA / INDIGESTION

Dyspepsia is not a disease. Patients use the term 'indigestion' to denote 'dyspepsia'. Dyspepsia means episodic or persistent abdominal symptoms, often related to intake of food, which patient/physician believes to be due to disorder of the proximal GIT.

Symptoms which qualify for dyspepsia: – Pain/discomfort in upper abdomen.
– Early satiety.
– Epigastric fullness and regurgitation.
– Nausea and vomiting.

The *important practical issue in the management of dyspepsia* relates to which patients should be investigated by UGI endoscopy.

Indications for UGIE (usually considered in combinations rather than in isolation)

- Age > 45.
- Recent onset dyspepsia.
- Constant pain in upper abdomen.
- Loss of appetite.
- Loss of weight.
- Evidence of overt/occult bleeding (Hematemesis / chronic hypochromic microcytic anemia).

} These symptoms are called **'sinister' symptoms'**.

Dilated veins

Seen in emaciated patient, portal HT and vena caval obstruction.

- In portal HT, periumbilical subcutaneous veins of systemic venous system become varicosed → 'caput medusae'. Veins from the supraumbilical region drain into the axillary vein, whereas blood from the infraumbilical region drains into the saphenous vein.
- In vena caval obstruction, inguinoaxillary veins become varicosed.

In SVC obstruction, blood through these varicose veins flows in craniocaudal direction.

In IVC obstruction, blood through these varicose veins flows from below upwards, i.e. from caudal to cranial direction.

Voluntary abdominal wall contraction versus guarding versus rigidity

Refer to page 444.

Rigidity is a sign, which may be encountered in a case of 'acute pain in abdomen'; but not in a case of 'chronic pain in abdomen'.

In a case of 'chronic pain in abdomen', voluntary contraction, or occasionally guarding of the anterior abdominal-wall may be encountered.

ARRIVING AT THE DIAGNOSIS

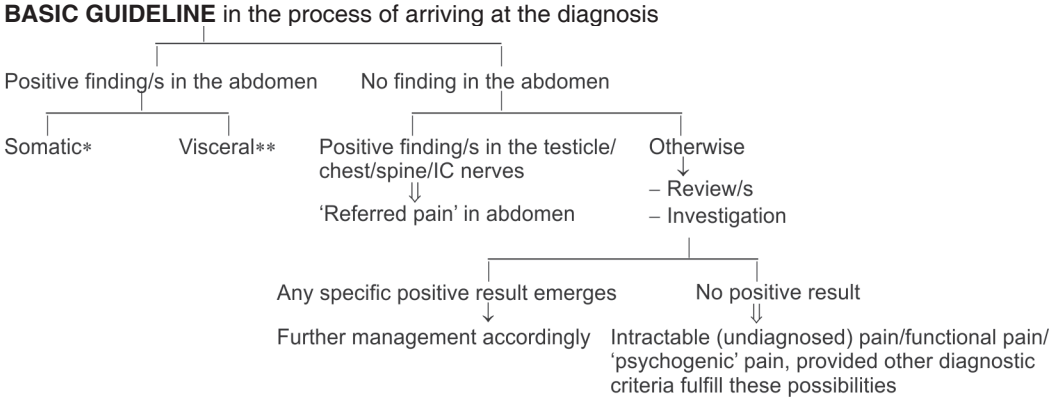
PRINCIPLES AND PITFALLS

1. More than one disease **may** co-exist in the abdomen.
2. A hernia of the linea alba may lead to pain that mimics the pain of a peptic ulcer.
3. **Interpretation of tenderness overlying a distended viscus**: Distended viscus can be tender, but that may not be necessarily the site of disease. The actual site of disease is most likely to be distal to it. For example, obstructing CA of hepatic flexure will produce distension of cecum and tenderness in the RLQ. Such a case may be misdiagnosed as appendicitis.
4. **Gaseous distension of certain viscera** like stomach and cecum **may be mistaken for a lump.**
5. **Functional pain may be clinically indistinguishable from organic pain.**
   – *Primary* functional pain may originate as a psychosomatic disorder but later on it may be enhanced and maintained by improper food habits/abuse of laxatives, antispasmodic or other medicines.
   – *Secondary* functional pain may completely mask the picture of the original organic disease.
6. Recurrent salpingitis is an inadequate diagnosis and should be used with caution. Long-term or recurrent antibiotics are not likely to be helpful if both partners have been treated. Investigation (laparoscopy) is indicated to exclude other causes of pain.

Free ebooks ==> www.Ebook777.com

7. Dull aching pain in the renal angle ⇒ renal pathology.
   The side of renal pain does not necessarily indicate the side of disease. Pain could be due to physiological compensatory hypertrophy caused by painless disease of the contralateral kidney. Hypertrophy → stretching of capsule → pain.

BASIC GUIDELINE in the process of arriving at the diagnosis



* Somatic: Pain arising from structures of the anterior abdominal wall, osseous/musculoaponeurotic components of the posterior abdominal wall, or dome of the diaphragm.

** Visceral: Esophagogastric junction, stomach, duodenum, small intestine, IC junction, appendix, colon including upper part of the rectum, liver, gallbladder, bile ducts, pancreas, spleen, mesentery and omentum, peritoneum including subdiaphragmatic spaces, lesser sac and pouch of Douglas, urogenital, uterus, ovary, uterine tube, broad ligament, retroperitoneal CT, retroperitoneal lymph nodes, abdominal aorta and IVC, nerves, visceroptosis.

Abdominal somatic disorders

– Granulomatous peritonitis.
– Peritoneal mesothelioma.

Abdominal visceral disorders

Esophagogastric junction

– Gastroesophageal reflux disease (GERD), hiatal hernia.
– Cardiospasm.
– Cancer.

Stomach

– Gastric outlet obstruction.
– Passive congestion, e.g. in cirrhosis of liver, CCF.
– Inflammation: Peptic ulcer (gastric, gastrojejunal), (chronic) gastritis, fungal gastritis, tuberculous gastritis.
– End result of inflammation: Fibrosis—'hour-glass' deformity complicating chronic peptic ulcer.
– Tumor: Carcinoma, lymphoma, pseudolymphoma, carcinoid and other endocrine tumors, adenomas and other benign tumors.
– Diverticulum.
– Chronic gastric volvulus.
– Gastroptosis.
– Hyperacidity.
– Functional: Pylorospasm, gastric atony.
– Reflex causes from other abdominal viscera.
– Sequel(ae) of gastroduodenal surgery.
– Presence of foreign body: Example – Trichobezoar.

Duodenum

– Inflammation: Peptic ulcer, duodenitis, TB.
– Obstruction: – Gastric outlet obstruction due to chronic duodenal ulceration/fibrosis.
  – Chronic ileus
  – Annular pancreas
  – TB.

www.Ebook777.com

- Diverticulum.
- Infection/Infestation: Ankylostomiasis.
- Tumor: Primary duodenal carcinoma.
- Presence of foreign body: Example—Trichobezoar.
- Functional: Pseudoulcer syndrome (irritability and spasm of duodenum).

Small intestine

- Obstruction.
- Malabsorption syndromes.
- Inflammatory conditions: Regional enteritis, eosinophilic enteritis, radiation enteropathy.
- Infective conditions, e.g. TB, actinomycosis, HIV enteropathy.
- Roundworm infestation.
- Abnormal position: Malrotation, herniation.
- Abnormal shape: Diverticula, Meckel's diverticulum.
- Ischemia: Vascular insufficiency.
- Presence of foreign body: Examples—Tricobezoar, phytobezoar.
- Tumor: – Benign, e.g. adenoma, lipoma, neurogenic tumor etc.
  – Malignant, e.g. adenocarcinoma, carcinoid tumor, lymphoma, mesenchymal tumor.
- Peutz-Jeghers syndrome.

Ileocecal junction

- Infective conditions, e.g. TB, actinomycosis, amebiasis.
- Inflammatory conditions: Regional enteritis, mesenteric lymphadenitis (nonspecific / tuberculous).
- Obstruction: Chronic intussusception.
- Tumor, e.g. carcinoma of cecum, carcinoid tumor, etc.

Appendix

- Appendicular dyspepsia.
- Mucocele.
- Tumor: Adenocarcinoma, carcinoid tumor, mucinous tumors, lymphosarcoma.

Colon (including upper part of the rectum)

- Colitis of various types.
- Inflammatory bowel disease: Ulcerative colitis, Crohn's colitis.
- Malignant tumor: Examples—Adenocarcinoma, squamous cell carcinoma.
- Vascular insufficiency: Ischemic colitis.
- Functional and structural disorders: Constipation, idiopathic megacolon and megarectum, pneumatosis coli, volvulus, colonic pseudo-obstruction, IBS (Irritable Bowel Syndrome), diverticular disease, spastic colon (reflex spasm).
- Ptosis.

Liver

- Amebic abscess.
- Cysts: 'Simple', dermoid, post-traumatic, ciliated hepatic foregut cyst, hepatic cystadenoma, Caroli's syndrome, hydatid cyst.
- Metastatic disease.
- Primary malignant tumor: HCC (Hepatocellular carcinoma), hepatic cholangiocarcinoma, angiosarcoma, epithelioid hemangioendothelioma, etc.
- Benign solid tumor: Hemangioma, hamartoma, liver cell adenoma, etc.
- Disorders of hepatic vasculature: Hepatic artery aneurysm, chronic form of Budd-Chiari syndrome.
- Fatty liver.
- Passive congestion.
- Actinomycosis.

Biliary tract

- Chronic cholecystitis.
- Acalculous chronic gallbladder disease: Adenomyomatosis of gallbladder (*synonyms*: adenomatous hyperplasia, diverticulosis of gallbladder), cholesterosis ('strawberry gallbladder).
- Ductal stones.
- Sclerosing cholangitis.

Free ebooks ==> www.Ebook777.com

- Cystic disease of the biliary tract: Diffuse choledochal cystic dilatation, localized dilatation of the supraduodenal bile duct, supraduodenal diverticulum, intraduodenal diverticulum (choledochocele), etc.
- Gallbladder dyskinesia (spasm of sphincter of Oddi).
- Tumors of the gallbladder: (a) Benign: Adenomas, papillomas. (b) Malignant: Carcinomas, carcinoid, melanoma, ACTH-secreting apudoma.
- Cholangiocarcinoma (malignant tumor of bile ducts).
- Parasitic infestation: (Adult) roundworms, (adult) Clonorchis sinensis, etc.
- Post-cholecystectomy syndrome.

Pancreas

- Pancreatitis.
- Fibrocystic disease.
- Cancer.
- Ectopic pancreas.

Mesentery and Omentum

Sclerosing mesenteritis.

Peritoneum / peritoneal cavity

- Granulomatous (chronic) peritonitis: TB, sarcoidosis, 'starch' peritonitis.
- Tumor: Secondary peritoneal carcinomatosis, pseudomyxoma peritonei, peritoneal mesothelioma.
- Chronic adhesion syndromes.
- Abscesses: Intraperitoneal, extraperitoneal.
- Pseudocyst of pancreas.

Uterus and adnexa

- Endometriosis.
- Adhesions.
- Pelvic congestion.
- Ovarian mass.
- Endometrioma.
- Fibroids.
- Adenomyosis.
- Prolapse.
- Recurrent salpingitis: debatable.

Urinary bladder

- Cystitis.
- Stones.
- Diverticulitis.
- Bilharziasis.
- Cancer.
- Various obstruction syndromes: Megacystitis syndrome.

Kidney and ureter

- Floating kidney.
- Polycystic disease.
- Pyelonephritis.
- Pyelitis.
- Perinephric abscess.
- Hydronephrosis.
- Pyonephrosis.
- Renal stone disease.
- Primary malignant tumor: Wilm's tumor, renal cell carcinoma, transitional cell carcinoma of renal pelvis/ureter.
- Renal infarct.
- TB.

Blood vessels

- Aneurysm.
- Abdominal angina.
- Chronic mesenteric ischemia.

www.Ebook777.com

Retroperitoneal disorders

- Aortitis.
- Sclerosing mesenteritis.
- Pain due to painful retroperitoneal swelling.
- Retroperitoneal fibrosis.

STEPWISE ANALYSIS OF DATA

Step 1

Ascertain whether pain is: – Chronic constant
OR – Chronic intermittent
OR – Acute-on-chronic, i.e. acute exacerbation of a chronic (usually intermittent) pain.

Step 2

The most probable anatomical site of disorder (applicable to both organic and functional)?

Pain	*Associated symptoms*	*Physical signs*	*Remarks*	*Anatomical site of the disorder/ disease*
Site/s: High epigastrium / xiphoid region ± between the scapulae/ either lumbar region. **Nature**: May be burning. **Precipitated/ aggravated**: immediately after eating (particularly large meal) OR on stooping/lying down flat. **Relieved**: by assuming sitting position	Excessive belching, ± Specific symptoms of esophageal disorder	Tenderness high in the epigastrium ±	-	**Esophagogastric junction**
Site: Epigastrium/umbilical region **Related to meals** in some way	– Excessive belching – Nausea – Vomiting – Early satiety – Epigastric fullness – Regurgitation – Specific symptoms suggestive of gastric ulcer ±	– Tenderness at the 'gastric point' – 'Succussion splash'	-	**Stomach**
Site: Epigastrium/umbilical region **Related to meals** in some way	– Vomiting – Early satiety – Epigastric fullness – Specific symptoms suggestive of duodenal ulcer ±	Tenderness at the 'duodenal point'	_	**Duodenum**
Site: Epigastrium (especially lower)/umbilical region/all over abdomen. **Colicky** / consciousness of painful peristaltic activity	– Vomiting – Diarrhea	– Central distension – Visible peristalsis – Vague tenderness in the umbilical region – Increased peristaltic sounds (in the early stages)	Repeated attacks of intestinal obstruction	**Small intestine**

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

Pain	*Associated symptoms*	*Physical signs*	*Remarks*	*Anatomical site of the disorder/ disease*
Site: Epigastrium (especially lower)/umbilical region/RLQ/all over abdomen. **Colicky** / consciousness of painful peristaltic activity	– Vomiting – Diarrhea	– Central distension – Visible peristalsis – Tenderness in the RLQ – Lump in the RLQ – Increased peristaltic sounds (in the early stages)	Repeated attacks of intestinal obstruction ±	**Ileocecal junction**
Site: Central/RLQ. **Intermittent/ Constant**	Vomiting ±	Tenderness in the RLQ – Greatest along the line of mesentery – 'Shifting tenderness'	-	**Ileocecal mesentery**
Site: Epigastrium (especially lower), and umbilical. Mild discomfort even during the so-called pain-free interval. Migrate → RLQ. **Nature**: Colicky/continuous. **Precipitated by** jolting/running / traveling on a bumpy uneven road	– Vomiting – Diarrhea	Tenderness: (1) in the RLQ (McBurney's point). (2) A little inferior to renal angle in case of retrocecal appendicitis	Repeated attacks ±	**Appendix**
Site: LLQ. May be **precipitated/ aggravated** immediately after eating. **Relieved**: by passing flatus/ stools OR by taking enema	– Fullness (particularly in the flanks) relieved by passing flatus/stools – Diarrhea – Excessive mucus and/ or blood per rectum – Altered blood mixed with mucus/pus (especially apart from defecation), and tenesmus – Melena – Recent alteration of bowel habit	Tenderness overlying (affected part of) the colon	-	**Colon including upper part of the rectum**
Site/s: Right hypochondrium / Shoulder. **Nature**: Dull aching	– Jaundice – Loss of weight – Fatigue, malaise, myalgia, arthralgia, headache – Forgetfulness, confusion, poor concentration, personality changes	Hepatomegaly	-	**Liver**

Contd...

www.Ebook777.com

Contd...

Pain	*Associated symptoms*	*Physical signs*	*Remarks*	*Anatomical site of the disorder/ disease*
Site/s: Epigastrium (especially lower), umbilical, shoulder. Mild discomfort even during the so-called pain-free interval. **Migrate** → RUQ. **Radiation** from epigastrium/RUQ → right side of the back (infrascapular) / right shoulder. **Precipitated/ aggravated** by fatty food	– Excessive belching – Nausea – Vomiting – Present / recent past h/o jaundice – Anorexia particularly for fatty food – Loss of weight	– Tenderness at Murphy's point, Boas's point, superior phrenic point – Palpable lump in RUQ	-	**Biliary tract**
Site: Epigastrium (especially lower), and umbilical. **Precipitated/ aggravated** by lying down flat or consumption of alcohol. **Relieved** by assuming sitting position	– Diarrhea – Loss of weight	-	Persistent pain in the mid-back (may be due to pancreatitis)	**Pancreas**
Site: LUQ. **Nature**: Chronic dragging, OR acute attacks of colicky pain. **May occur when lying on one side**	– Early satiety – Specific symptom/s suggesting the cause of splenomegaly	– Splenomegaly – Sign/s due to the underlying disease causing splenomegaly	Instead of pain, patient may experience only discomfort	**Spleen**
–	– Vomiting – Diarrhea	Ascites	-	**Peritoneal cavity**
Site/s: Suprapubic, mid-sacrum. May occur at monthly interval. **Related** to menstruation in some way	-	Tenderness in suprapubic region, on both sides of the midline	-	**Uterus and adnexa**
Site: Suprapubic. **Radiation** from suprapubic region → tip of the penis. **Related** to micturition in some way	(Other) urinary symptoms	Tenderness in suprapubic region	-	**Urinary bladder**
Site: Suprapubic and one or both lower quadrants. **Often worse after** ejaculation	– Disabling chronic pain in the perineum radiating along the urethra, to the sacral area and scrotum, and in a sciatic distribution – Lower urinary tract symptoms, particularly increased frequency – Scanty watery, or gleety urethral discharge	On DRE: Size of the gland smaller or larger. Irregular surface	May or may not follow acute prostatitis	**Prostate (Recurrent subacute or chronic prostatitis)**
Site: Lumbar region. **Radiation** from loin → groin / testis / vulva. **Nature**: Dull ache. May **feel comfortable** moving around and holding the flank	(Other) urinary symptoms	– Tenderness in renal angle – Renal lump	-	**Kidney (/ Perinephric mass)**

Notes: – There is no specific symptom to pinpoint small intestinal disorder.
– There is no specific symptom (other than pain), to pinpoint biliary tract/pancreatic disorder.
– Ureteral pathology that develops slowly and causes 'mild' (incomplete) obstruction **does not** cause pain.

Free ebooks ==> www.Ebook777.com

Step 3

With respect to ***Pathology****, pain may be classified as* diagnosable (intermittent or constant), intractable (undiagnosed), functional, or psychogenic.

A. **Diagnosable** (a) **chronic intermittent pain** (*synonyms* recurrent acute pain)

Mechanical	***Inflammatory***	***Neurologic and metabolic***	***Poisoning***	***Miscellaneous***
– Obstructive pathology Examples of intermittent intestinal obstruction -Hernia, intussusception, adhesions, volvulus. – Ampullary stenosis. – Gallstones	– Recurrent (/? 'chronic') appendicitis. – Inflammatory bowel disease. – Chronic relapsing pancreatitis. – Retroperitoneal lymphangitis (filarial). – Endometriosis/ endometritis. – Sclerosing mesenteritis. – Familial Mediterranean fever.	– Nerve-root compression /entrapment. – Diabetic radiculopathy. – Abdominal epilepsy. – Abdominal migraine. – Uremia. – Acute intermittent porphyria.	– Lead poisoning in painters. – Arsenic poisoning in industry.	– Functional abdominal pain (non-ulcer dyspepsia, IBS, sphincter of Oddi dysfunction, etc.). – Chronic mesenteric ischemia, median arcuate ligament syndrome – caused by isolated coeliac artery compression. – Mid-cycle pain.

A. **Diagnosable** (b) **chronic constant pain**
- Abscess
- Chronic relapsing pancreatitis.
- Cancer (primary / secondary).
- Psychiatric (depression, somatoform disorder).

B. Chronic **intractable** (**undiagnosed)** pain
- Pain ≥ at least six months without diagnosis despite appropriate evaluation.

Characteristics: – Described in vague, peculiar terms.
– Exacerbated by (psychological) stresses.
– Associated with multiple other somatic complaints.

Other features: Unresponsive to standard treatment often resulting in multiple unnecessary procedures.

C. Chronic **functional** abdominal pain
- Because of spasm of various sphincters and neuromuscular imbalance in the wall of intervening segments of the organ.

Definition: Disorders in which the symptom of pain cannot be explained by any detectable anatomical, biochemical or physiological abnormality.

This is different from psychogenic pain.

Causes of chronic functional abdominal pain:

a. Psychosomatic.
b. Secondary to (organic) disease in a viscus or structure in the adjoining or distant area. Examples— (i) Colonic spasm because of cholecystitis/duodenal ulcer. Clinical picture of colonic spasm may completely mask the picture of cholecystitis or duodenal ulcer. (ii) Pyloric spasm because of diseased gallbladder. Pyloric spasm in turn may lead to clinical picture of peptic ulcer that may mask the picture of gallbladder disease.
c. Secondary to addictions, medicines, food irritants, allergy and external pressure.

Examples of chronic functional abdominal pain:
- Noncardiac chest pain in the esophagus.
- Nonulcer dyspepsia.

www.Ebook777.com

- Sphincter of Oddi dysfunction.
- Nausea.
- Idiopathic vomiting.
- Painless diarrhea.
- Irritable bowel syndrome.
- Constipation.

D. Chronic **psychogenic** pain, e.g. Hysteria, malingering.
Clues – Shifting and depressive personality.
- Vagueness of symptoms.
- Long duration without remissions and without any change towards increase or decrease in intensity of symptoms.
- Unusual distribution of pain.
- Shifting of site of pain and tenderness.

Step 4

Possible etiology (in general):

1. ***As an effect of some other symptom/act*** *leading to strain on the abdominal wall musculature*
   a. Chronic (vigorous) coughing / sneezing / vomiting / persistent hiccough / dyspnea.
   b. Occupational strain. Examples – Musicians playing on blowing-instruments such as saxophone, trumpet, shehnai, etc.
   – Heavy-weight lifting.
   c. Heavy-duty outdoor games such as football, rugby, etc.
2. ***Diet*** *related*
   – Irritation caused by adulterated food, spicy food.
   – Hypersensitivity to particular food.
3. **Addiction**: Alcohol, tobacco, etc.
4. **Worm-infestation.**
5. **Tight abdominal clothing / corsets / belts / supports**.
6. **Iatrogenic**
   - Gastric irritation caused by various medicines like NSAIDs, steroids, etc.
   - Colonic irritation caused by certain alkaloids, magnesium, salicilates; chronic atony because of repeated purgation.
7. **Poisoning.**
8. **Pre-existing (medical) diseases** such as DM, uremia, etc.

Apart from the etiological factors mentioned above in general, search for specific cause that may be responsible for a specific disease in the given case.

Step 5

A. *Which **physiological life processes** seem to have been **affected**?*
B. *How much is the **degree of homeostatic imbalance**?*

Step 6

Sequelae / Complications:

- Disruption of life—Increased dependency.
- Disruption of work, family and social relationships.
- Altered self-image.

The above-mentioned problems often → psychological disturbances.

Step 7

Associated disease: Closely related and/or otherwise?

Free ebooks ==> www.Ebook777.com

DIFFERENTIAL DIAGNOSIS

Retroperitoneal lymphangitis (**filarial**)
Refer to page 480.

Familial Mediterranean fever (FMF)
Recurrent attacks of fever, and pain in abdomen, chest and joints (the latter symptom is because of serositis).
Characteristics of attacks: – Mode of onset – sudden, often without warning. Occasionally however, attacks occur after vigorous exercise/stress.
– Duration – 2 to 3 days.
– Peak intensity of symptoms occurs within 12 to 24 hours.

Symptom-free interval – a week ↔ many months. Patient feels normal during symptom-free intervals.

Typical manifestations:
– Fever 100° to 104° F.
– Pain in abdomen – Resembles acute appendicitis / acute cholecystitis / acute peritonitis / renal colic. Patients may even undergo laparotomy because abdominal episodes of FMF are not recognized as such.
– Pain in chest, pleural effusion.
– Pain and swelling in joints.
– Constipation followed by diarrhea.
– A red rash on legs.
– Splenomegaly.

Less common manifestations:
– Severe myalgia lasting 3 to 6 weeks.
– PID (Pelvic inflammatory disease).
– 'Acute scrotum' (painful, swollen tender scrotum)—because of inflammation of tunica vaginalis.
– Vasculitis – Henoch-Schölein purpura, polyarteritis nodosa.

Notes
– An inherited inflammatory (incurable) disorder usually occurring in people of Mediterranean origin. But it may affect any ethnic group.
– The disorder declares itself for the first time in childhood (usually < 20 years).
– Complications: amyloidosis, nephrotic syndrome, chronic arthritis, etc.

Nerve-root compression/entrapment
– In most (*but not all*) cases, pain clearly radiates from the spine round the flank to the anterior abdominal wall, and follows a dermatomal pattern.
– Pain may be increased by coughing or by movement.
– Sensory disturbance: paresthesias—almost as common as pain.
– May be associated with neurological deficit.
– No specific physical signs in the abdomen.
– Subcostal pain (T8) on the right side may mimic gallbladder pain, and on the left side gastric ulcer pain.
– RLQ pain (T11) can mimic appendicitis.

Abdominal epilepsy
Paroxysmal pain in upper mid-abdomen, bloating, nausea, diarrhea.
+ Headache, disorientation/confusion, syncope.
+ Tiredness or deep sleep after attacks. Patient gets normal after waking up.
Sometimes, pain in abdomen is the only symptom.
Pain: – Frequency of attacks – variable.
– M/o onset – abrupt.
– Duration of each episode – a few minutes. May recur several times a day × several days.
– Intensity – severe.
Abdominal symptoms may be similar to IBS.

www.Ebook777.com

The GI symptoms cannot be explained by other pathophysiological mechanisms, and are seen to improve upon anticonvulsant treatment.

Differentiating points

– Altered LOC during some of the attacks.
– Tiredness after attacks.
– Abnormal EEG (findings specific for a seizure disorder).

Abdominal migraine

Recurrent identical episodes of intense pain in abdomen often associated with migraine-like symptoms (headache, nausea, vomiting, diarrhea, leg pains, photophobia, pallor, perspiration, bradycardia).

Pain: – Frequency of attacks: variable—once a week to several times a year.
Duration of each episode—2 hours to several days.
– Symptom-free interval: several weeks to several months.
– Site: anywhere in the abdomen, although more common in the upper mid-abdomen.

Differentiating point: The typical presentation.

Supporting point: Family history.

Uremia

Azotemia and metabolic alterations and secondary cardiovascular, neuromuscular and gastrointestinal manifestations incident to renal damage.

Acute intermittent porphyria

Acute intermittent porphyria is the most common subtype of porphyria.

Porphyria is a (group of at least about 8) hereditary disorder resulting in low levels of the enzyme responsible for the production of hem.

– Most patients become symptomatic at the age of between 18 and 40.
– Female > Male.

Precipitating factors can strongly influence the occurrence and severity of the symptoms and signs—Exposure to sunlight, stress, dieting/fasting, alcohol, smoking, certain drugs (phenobarbital, sulfonamide), estrogen, deficiency of vitamin C, menstruation.

Basically, acute episodes of abdominal symptoms (pain is the most common) and psychiatric symptoms and peripheral neuropathy, mainly motor. In between the episodes, the patient is usually healthy except perhaps for HT.

– Pain in abdomen: - Often epigastric, severe, colicky.
- Duration of each episode of pain – several days to weeks.
Pain of < 1 day duration or chronic constant pains are unusual.
– Nausea and vomiting.
± Constipation – frequent; can be very severe.
± Retention of urine.
– Depression: may last long time.
– Disorientation/confusion, hysteria, seizure, cortical blindness, coma.
– Diffuse pain in the upper trunk.
– Paresthesia in digits.
– Paralysis: Death may result from respiratory paralysis.
– Color of palms and face may get darker.
– Skin may blister and ulcerate.
– Fever ±
– Tachycardia.
– HT: may persist between attacks.
– Motor neuropathy, more predominant in lower limbs.
– Areflexia.

Abdominal examination: negative.

Biochemical feature: ↑ed urinary excretion of δ-aminolevulinic acid and porphobilinogen.

Free ebooks ==> www.Ebook777.com

Granulomatous peritonitis

Causes: TB, sarcoidosis.

Clinical manifestations:

- Cramp-like pain in abdomen, and tenderness.
- Distension of abdomen (Causes—ascites, paralytic ileus).
- Anorexia, nausea, vomiting.
- Low-grade fever.
- Loss of weight.
- 'General breakdown of health'.
- On palpation of abdomen: 'Doughy' feel.

Peritoneal mesothelioma

Various modes of presentation (Specific manifestations)

1. Pain in abdomen and progressive abdominal distension due to intractable ascites.
2. Fever of unknown origin—Signifies aggressive tumor with a bad prognosis.
3. A mass.
4. Gastric outlet obstruction or intestinal obstruction.

Nonspecific symptoms: Anorexia, marked loss of weight, fatigue.

Uncommon modes of presentation: Dysphagia secondary to pseudoachalasia, chronic pancreatitis and regional lymphadenopathy.

GERD (**G**astro-**E**sophageal **R**eflux **D**isease): Refer to page 572.

Reflux esophagitis (a spectrum of inflammatory changes in the esophageal mucosa discernible by endoscopy) represents a complication of GERD.

The cardinal clinical feature of reflux esophagitis is postural heartburn.

Heartburn itself without postural link is common in many patients with peptic ulcer or gall stone disease, or indeed without any serious underlying disease.

Heartburn without postural link may also be caused by '**non-reflux esophagitis**' (Refer to p. 573).

Gastric outlet obstruction

Differentiating features:

- Effortless, projectile, non-bilious vomiting. Vomitus contains partially digested food consumed hours or even days previously.
- Distension in the epigastrium and upper umbilical region.
- Visible peristalsis passing across the upper abdomen from left to right.
- Palpable dilated stomach.
- Succussion splash.
- Hyperperistaltic sounds.

Supporting features:

- Borborygmi.
- Constipation, because of repeated vomiting and failure to eat.
- Underweight, dehydrated, anemic (iron-deficiency anemia).
- Disturbances of the level of consciousness and tetany because of metabolic disturbances (metabolic alkalosis and hypocalcemia respectively).

Diarrhea is an *unusual feature (It is unexplainable)*.

Causes of gastric outlet obstruction:

A. Common

1. Chronic duodenal ulceration/fibrosis:
   - Long-standing symptoms of duodenal ulcer ±.
   - As obstruction develops, characteristic pain s/o of DU may change to become more of a generalized upper abdominal discomfort.

www.Ebook777.com

2. CA-stomach (antral, 'leather-bottle stomach'): Short preceding history with little in the way of characteristic peptic ulcer pain.
3. CA-head of the pancreas.

B. Rare: Mucosal diaphragm, adult hypertrophic pyloric stenosis, duodenal hematoma, gastric/duodenal TB, Crohn's disease of the duodenum, benign tumor, lymphoma, annular pancreas, arteriomesenteric compression, megastomach-megaduodenum.

Note: Chronic duodenal ileus may mimic gastric outlet obstruction.

Gastric ulcer

Basically, chronic periodic epigastric pain; with periods of exacerbations particularly being associated with periods of stress, smoking, dietary or alcoholic indiscretions ⇒ chronic peptic ulcer.

Differentiating points in favour of chronic GU

- Pain induced by food.
- Vomiting (often self-induced) or consumption of an antacid relieves pain.
- Appetite is maintained but patient is afraid to eat.
- Loss of weight.

Supporting point: Tenderness at the gastric point.

Notes

- Nature of pain – burning or dull aches.
- The more indigestible the food the more severe the pain.
- As the disease progresses, attacks last longer with shorter pain-free intervals. And ultimately persistence of pain suggests development of a complication like adhesion to the pancreas, gastric outlet obstruction, etc.
- **All patients may not have classical clinical features. Instead, patients may have only vague epigastric discomfort.**

  Some patients may be totally asymptomatic with active ulcer, and may present straight with complication as the first evidence of peptic ulcer disease.

Gastrojejunal ulcer

Differentiating points:

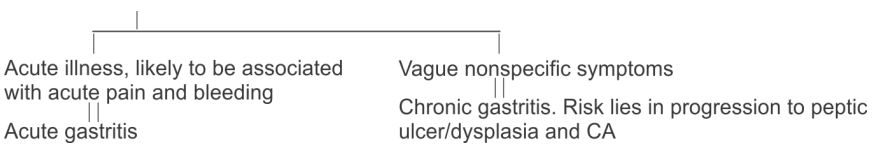
- The patient has had a gastrojejunostomy.
- Hematemesis precedes the pain.
- Pain especially on empty stomach/during early morning; relieved by food. The pain is relatively more severe. It characteristically radiates from the left of the umbilicus to the LLQ.
- Good appetite. Weight-gain ±.
- Vomiting: (more) frequent and gives temporary but complete relief of pain.

Notes:

- Usually seen within 2 years or so of the operation (gastrojejunostomy).
- Complications: Perforation leading to peritonitis, penetration into the transverse colon leading to Gastrocolic or gastrojejunocolic fistula leading to true fecal vomiting and diarrhea.

Gastritis

Clinical classification



Histological classification, i.e. types based on categorization of mucosa on histology

- Normal
- Non-atrophic *H. pylori* gastritis
- Atrophic *H. pylori* negative or positive.

Free ebooks ==> www.Ebook777.com

- Special forms: - Lymphocytic
  - Reactive/Erosive
  - Gastritis cystica polyposa
  - AIDS gastritis
  - Eosinophilic
  - Suppurative (Phlegmonous)
  - Granulomatous and Crohn's
  - Emphysematous.

Carcinoma of the stomach

Mode of onset of the symptom/s—insidious.

No positive physical findings in the initial period of evolution of the disease.

(Unfortunately), it may even respond to acid-inhibition treatment, consequently with improvement in symptoms.

Clues to suspect cancer (i.e. when to have high index of suspicion essential for early diagnosis)

- Onset of symptoms referable to the stomach (refer to the table – 'step 2' above) after 40 years of age.
- Symptoms are usually progressive.
- Significant loss of weight.
- Severe nonspecific anorexia, or qualitative anorexia for meat.
- Person residing in high-risk area.

Notes:

- This disease does not always produce pain.
- Anorexia and loss of weight, without the pain, constitute sufficient grounds for a presumptive diagnosis of CA stomach.
- Early symptoms of primary gastric lymphoma are indistinguishable from those of carcinoma, although nausea, vomiting and weight loss are less common. Some patients develop diarrhea, which is often persistent.

Volvulus of the stomach

Chronic gastric volvulus → chronic epigastric distress and vomiting.

Duodenal ulcer

Basically, chronic periodic epigastric pain; with periods of exacerbations particularly being associated with periods of stress/smoking/dietary or alcoholic indiscretions ⇒ chronic peptic ulcer.

Differentiating points:

- Pain especially on empty stomach/during early morning; relieved by food.
- Good appetite. Weight gain.
- The heavier the meal the longer is the interval (between two episodes of pain), but the worse the pain.

Supporting points:

- An 'executive' who does not observe proper schedule in routine daily life inclusive of feeding; gulps his meals hastily at improper times (late night/midnight) as well as at irregular intervals between cigarettes and telephone calls.
- H/o melena.
- Tenderness at the duodenal point.

All patients may not have classical clinical features. Instead, patients may have only vague epigastric discomfort. Some patients may be totally asymptomatic with active ulcer, and may present straight with complication as the first evidence of peptic ulcer disease.

Ankylostomiasis (Hookworm infestation)

- Severe dyspepsia.
- (Microcytic) anemia.
- Frank GI bleeding may occur especially in children.

www.Ebook777.com

Primary duodenal cancer

It is a distinct entity and should be differentiated from ampullary and periampullary tumors.

Presenting clinical features:

– Pain.
– Iron-deficiency anemia because of occult blood loss.
– Vomiting because of obstruction.
– Jaundice (50% cases).

Intraperitoneal adhesions → chronic adhesion syndromes or acute intestinal obstruction.

Chronic pain and episodic subacute obstruction constitute the chronic adhesion syndromes.

1. *Chronic pain.*
   – Site of pain – variable; and is not necessarily beneath the abdominal wall scar.
   – Nausea ±.
   – No positive physical signs in the abdomen, except perhaps minimal tenderness.
2. *Episodic subacute intestinal obstruction.*
   – Duration of episode is variable: Usually few days to a week or two.
   – Subsequent natural history: (a) the episodes may terminate in diarrhea leading to relief of both pain and distension. (b) may develop acute obstruction any time.

Causes of intraperitoneal adhesions: Abdominal surgery ('late postoperative adhesions'), peritonitis from any cause, sclerosing peritonitis (usually drug-induced, e.g. certain β blockers), inflammatory bowel disease, radiation enteritis.

Regional enteritis (Terminal ileitis)

Abdominal cramping pain, diarrea and weight loss.

A doughy mass of inflamed ileum ±.

Tumor

Clinical features with reference to 'chronic abdomen' which are common to all types of tumor: Subacute obstruction, loss of weight, and (iron-deficiency) anemia because of chronic blood loss.

Additional or alternative manifestations may vary; they are as follows.

– Intestinal obstruction because of intussusception.
– Overt GI bleeding leading to melena/frank rectal bleeding.
– Obstructive jaundice (in half of the cases of) duodenal carcinoma.
– Diarrhea in carcinoid of the jejunum/ileum.
– Carcinoid syndrome in few cases of usually the small intestinal carcinoid tumor.

Malabsorption syndrome

Malabsorption implies the inability to absorb sufficient nutrient owing to disease of the GIT. It is most commonly found because of dysfunction of the small intestine or the pancreas.

Clinical manifestation of malabsorption syndrome is referable to the underlying pathology.

Subacute obstruction develops, if the primary underlying pathology is obstructive.

Clinical manifestations referable to bacterial overgrowth itself are often nonspecific; they are:

– Malaise, weight loss.
– Nausea, vomiting, excessive borborygmi.
– Diarrhea, usually watery. It may result from fermentation of digested carbohydrates to lactic acid in the large intestine.
– Frank steatorrhea (bulky pale offensive stools which are difficult to flush)—Caused by malabsorption of fat.

Small intestinal tuberculosis (Ulcerative TB)

- Nonspecific symptoms of TB
  - Low-grade evening fever f/b night sweat
  - Loss of appetite
  - Loss of weight
- Specific features of 'early' stage of ulcerative TB, i.e.when the disease is confined only to mucosa in the form of ulcer/s
  - Vague colicky pain in abdomen
  - Chronic diarrhea/recurrent attacks of diarrhea not responding to usual treatment
  - Fetid odor to stools
  - Diffuse abdominal tenderness
- Feature of advanced stage, i.e. on formation of stricture/s
  - Subacute intestinal obstruction
- Additionally, may be associated with pulmonary TB

Free ebooks ==> www.Ebook777.com

HIV enteropathy

Presenting symptoms: Pain in abdomen, diarrhea and loss of weight.
Other symptoms: Fever, sore throat, arthralgia, lymphadenopathy, erythematous maculopapular rash.

Roundworms

Differentiating points: Palpable mass (worm bolus); the mass changes its position around the umbilicus.
Supporting points: – Fever.
– Vomiting of worms during the episode of acute pain, or earlier.

A pitfall
Vomiting of worm/s may suggest the diagnosis, but it is not pathognomonic of intestinal obstruction caused by roundworms.

Internal hernia

Recurrent attacks of colicky pain and abdominal distension. Rarely, a mass may be palpable.
However, in most of the patients symptoms are nonspecific and very few cases are diagnosed in the elective situation.

Chronic mesenteric ischemia

Generally, elderly women. Consequently, age-related disorders like diverticulosis may mislead!
Pain classically related to eating—Postprandial pain in abdomen—'Intestinal angina'.
Loss of weight as patient is afraid of eating.
Nausea, vomiting, diarrhea ±.
H/o numerous investigations having been carried out including occasionally, a negative laparotomy ±.
O/E: – Underweight.
– Epigastric bruit ±. However, in normal elderly, a bruit is not unusual even otherwise.

Peutz-Jeghers syndrome

– Attacks of intestinal colic.
– (Iron-deficiency) anemia due to chronic blood loss. Acute GI bleeding is less common than chronic blood loss.
– Pigmentation on the lips and buccal mucosa since infancy.

Eventually acute intestinal obstruction will occur due to intussusception or intraluminal obstruction by a large polyp.

Ileocecal tuberculosis (Hyperplastic TB)

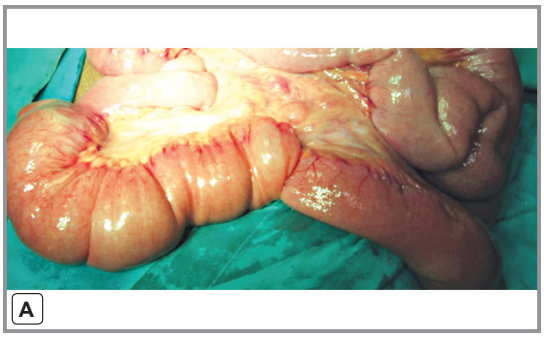
Nonspecific symptoms: Low-grade evening fever f/b night sweat, loss of appetite and loss of weight.
Specific manifestations
– Subacute intestinal obstruction.
± A lump in the RLQ - Higher and medial than the normal position of the cecum (because of fibrosis in the lymphatics along the ileocecal vessels).
- Slightly tender, mobile and firm.

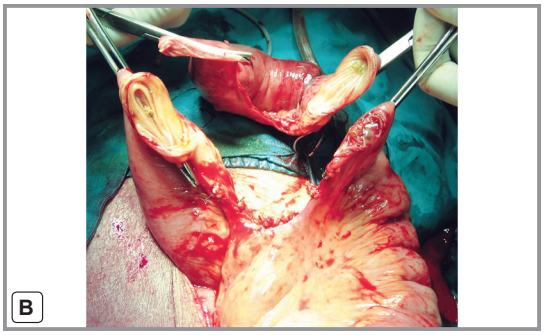
Amebiasis (Amebic colitis)

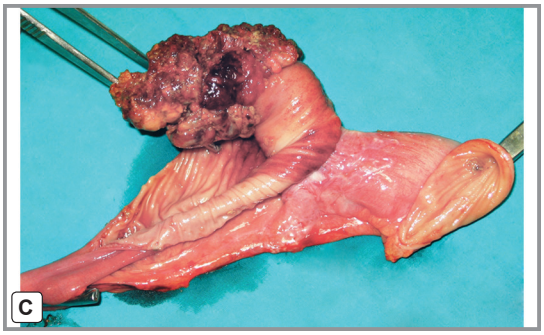
Few patients with amebiasis do ever present with true dysentery characterized by loose motions with blood and mucus.
Irregular bowel habits and loose motions with or without mucus are more frequently seen.
Mode/s of presentation:
– *The most common complaint* is pain in the epigastrium or in the lower quadrant/s.
– Dyspepsia-like symptoms.
– Irregular bowel habits, diarrhea with minor changes in the diet, and flatulence.
– (Curiously) constipation with passage of mucus in stools.

There are hardly any positive physical signs. Tenderness along the colon ±.
Notes
– Intestinal amebiasis mimics practically every chronic abdominal intervals.
– Amebiasis affects the colon. It rarely spreads proximal to IC junction.

www.Ebook777.com







Figs 33-4A to C (A) This is how an intussusception looks like during surgery **(B)** The irreducible intussusception resected **(C)** The resected specimen cut open to show the intussusceptum and the lead point (a stromal tumor as reported on histopathology)

– Most commonly affected segments are—cecum, ascending colon and sigmoid colon. Transverse colon and descending colon are less frequently involved.
– Amebic ulcers heal without fibrosis and consequently, stricture formation is exceedingly rare.
– Amebic ulcers may perforate.

Acute Nonspecific Ileocecal Mesenteric Lymphadenitis (ANIML): Refer to page 474.

Chronic intussusception → subacute intestinal obstruction.
– Bleeding PR may occur during an attack.
– A banana-shaped mass may be palpable per abdomen. The mass seems to change position on different occasions.
– It may advance sufficiently to be palpable on rectal examination.
– It may even protrude out of anus.

On such a background, frank manifestations of complete (acute) intestinal obstruction may occur when intussusception gets incarcerated.

Carcinoma of the cecum

Nonspecific manifestations:
– Dyspepsia.
– Severe iron-deficiency anemia unyielding to treatment, especially in men. In women, there are many other causes for the same viz. nutritional, multiple pregnancies, excessive menstrual losses, abnormal uterine bleeding, etc.
– Significant loss of weight.

Additional specific modes of presentation:
– A classic lump in the RLQ: Rapid progress, fixed, nodular surface, hard consistency.
– Progressive chronic distal small intestinal obstruction.

Free ebooks ==> www.Ebook777.com

- Intussusception. Before intussusception finally becomes irreducible to cause acute intestinal obstruction, it → recurrent attacks of acute pain in abdomen, with a lump being present at some times and smaller/absent at other because of spontaneous partial/complete reduction.
- Acute appendicitis.

Supporting features

- Age > 40.
- Melena.
- Low grade fever.

Appendicular dyspepsia

Differentiating points:

- The presence of a mild discomfort even during the so-called pain-free intervals.
- The onset of pain after a meal is extremely variable unlike in c/o peptic ulcer.
- Vomiting may be present but does not afford relief unlike in c/o gastric ulcer.
- Past h/o acute appendicitis often present.
- Distinct tenderness overlying the appendix.

Mucocele of appendix

Presents clinically as mild subacute appendicitis.

Cancer of the colon

Nonspecific manifestations:

- Dyspepsia.
- Severe (iron-deficiency) anemia unyielding to treatment, especially in men. In women, there are many other causes for the same viz. nutritional, multiple pregnancies, excessive menstrual losses, abnormal uterine bleeding, etc.
- Significant loss of weight.

Additional specific clinical points in c/o transverse colon lesions:

- Diarrhea alternating with constipation.
- A classic transverse colonic lump with differentiating points in favor of cancer: Rapid progress. Surface—nodular. Consistency—hard.

Cancer of the transverse colon may have to be differentiated from cancer of the stomach presenting with a lump.

Additional specific clinical points in c/o left sided lesions, i.e. of the descending and sigmoid colon:

- Progressive chronic obstruction.
- A recent change in bowel habit. Criteria: Irregularity, increasing difficulty, requirement of increasing doses of purgative, constipation alternating with diarrhea. The diarrhea is virtually spurious, i.e. it consists of rather mucus, in the presence of actual constipation.
- Bleeding PR especially apart from defecation, and tenesmus; these symptoms are particularly prominent with cancer of the rectum.
- Indentable/mouldable lump in abdomen because of fecal impaction.

Note: Basically, lumen of the left side of the colon is comparatively narrow, fecal content relatively solid and the type of lesion usually stricturous, i.e. obstructing.

Supporting features in general, in favor of CA colon:

- Age > 40.
- Melena.
- Low grade fever.

Idiopathic megacolon and megarectum:

- Pain in abdomen.
- Constipation.
- On per abdominal examination: Colon (usually the pelvic colon at least) is loaded with fecal masses.
- On DRE: Fecal impaction.

www.Ebook777.com

Notes: There are two forms of this conditions: 1. The symptoms dating back to childhood often with rectal impaction and soiling. This can be indistinguishable from congenital megacolon. And 2. Commencement of symptoms in later life.

Pneumatosis coli:

– Colicky pain in abdomen.
– Frequent bowel action, mucus in stools, bleeding PR.
– Large amounts of flatus.

Notes:

– A rare condition affecting both the large and the small intestine.
– Etiology is unknown, but there appears to be an association with COAD.
– Usually discovered incidentally on abdominal X-ray.
  Findings on plain X-ray: Gas-filled cysts in the submucosal and subserosal planes.
  Finding on Ba study: Filling defects.

Volvulus of the pelvic (sigmoid) colon

Chronic form → intermittent lower abdominal colicky pain and distension, which are relieved by passage of flatus and loose stool.

Supporting points:

– Middle aged/elderly patient.
– Previous h/o chronic constipation, laxative dependency.
– 'Severe' constipation, which is common in psychiatric patients.

Volvulus of the cecum

Basically it is a misnomer. Cecum alone is involved in only 10% cases of so-called cecal volvulus. Remaining 90% cases occur because of ileocolic torsion.

Chronic form → intermittent obstructive symptoms.

'Uncomplicated painful diverticular disease of the colon'

– Chronic intermittent lower abdominal pain mostly felt in the LLQ.
– A feeling of distension only relieved by passage of flatus.
– Patient is commonly constipated and produces pellet-like stools. Occasionally diarrhea.
– Usually no positive physical sign. In a thin-built individual however, a thickened loop of colon may be felt in LLQ.

Chronic ischemic colitis could be transient or stricturing.

Transient ischemic colitis:

– Middle age.
– Pain in abdomen of several days duration.
– Bleeding PR—almost invariable.
– Mild-to-moderate abdominal tenderness.
– Peripheral arterial occlusive disease ±.

Stricturing ischemic colitis:

– Chronic abdominal discomfort.
– Change in bowel habit.
– Bleeding PR ±.
– No positive physical sign.

In case of both the above conditions, additional clinical background of underlying cause may be present.

Causes of ischemic colitis:

– *Thrombosis – arterial or venous*: portal HT, malignant disease of the colon, arteriosclerosis, polycythemia vera, hyperviscosity syndrome (because of platelet abnormalities / high molecular weight dextran infusion).
– *Vasculitis*.

Free ebooks ==> www.Ebook777.com

– *Congestive heart failure*.
– *'Spontaneous'*.

Chronic amebic liver abscess: Refer to page 465.

TB of the liver

Symptoms are identical with those of amebic liver abscess.

Simple/Non-parasitic hepatic cysts

Clinical features are nonspecific:
– Pain in upper abdomen.
– Nondescript vomiting.
– Occasionally diarrhea.
– Non-tender smooth mass in the liver.

Remarks:
– Most of such cysts are asymptomatic and only become apparent when the cysts reach sufficient size to exert pressure on adjacent viscera.
– Jaundice is unusual.

Hydatid cyst

Differentiating features: Smooth rounded tense mass—in case of anteriorly located cyst.

Notes: Since the growth of the parasite is slow, many years elapse before the cyst reaches significant size. Palpable cysts are therefore rare in children. Pain, jaundice and ascites are uncommon and, in most patients, general health is good.

Metastatic disease of the liver

Clinical manifestations in the initial period:
– Pain in the abdomen and back.
– Severe flatulence and nausea → reduction in nutritional intake.
– Patient may appear physically fit.

Cachexia, jaundice, ascites develop *with progress of the disease*.

Notes:
– Many hepatic secondaries are asymptomatic.
– Patients with widespread involvement and expansion of the liver present with symptoms. In other words, by the time liver metastases become symptomatic, there is usually massive involvement.

Primary malignant tumor of the liver: Refer to page 555.

Hepatic hemangioma

Clinical manifestations:
– Pain - Dull in nature.
  - Acute posterior pleuritic pain lasting a few weeks suggests intratumoral thrombosis and inflammation.
– Dyspepsia.
– Palpable lump in abdomen, bruit (15% of cases).

Notes:
– Development of symptoms implies hemangioma of sufficiently large size (> 4 cm).
– In general, symptomatic tumor is more common in women. It may enlarge during pregnancy. Intratumoral thrombosis may be precipitated by oral contraception.

Mesenchymal hamartoma of the liver

– Children are more commonly affected.
– Present with expanding abdomen.
– O/E: A large lump in the abdomen.
– Vomiting: Because of displacement of stomach by the mass.
– Elevation of diaphragm → compression of the (right) lung.
– IVC obstruction with further expansion of the tumor.

www.Ebook777.com

Liver cell adenoma

– *Clinical features*: Pain in upper abdomen, and lump in abdomen.
– Children, postmenopausal women, and women who have been consuming high estrogen containing OC pills for over 4 years are more prone to develop liver cell adenoma.

Symptomatic hepatic artery aneurysm

There are *2 different modes of chronic presentation:*

1. Increasing nonspecific pain in RUQ, which may defy diagnosis.
2. – Pain resembling biliary colic.
   – Jaundice
   – Upper GI bleeding.

Supporting point: Middle-aged male patient.

Specific predisposing factors

– Trauma: Penetrating injuries of the liver, radiological interventions, needle biopsy, cholecystectomy, etc.
– Bacterial endocarditis.
– Collagen disorders particularly SLE.

Hepatic AV fistula

– Obscure recurrent GI bleeding with negative upper and lower GI endoscopy, because of sectorial portal HT accompanied by venous congestion of the related gut mucosa (stomach, duodenum, small intestine and colon).
– Portal HT and bleeding from esophageal varices. *Mechanism*: As collaterals develop over a period of years, the venous mucosal congestion (and obscure bleeding) subside but portal HT of the entire splenic bed eventually develops.

Chronic form of Budd-Chiari syndrome

Presents in a similar fashion to acute form (refer to chapter 34) but at a reduced tempo and with less evidence of hepatic failure.

Basically presents with pain in abdomen and vomiting.

Differentiating points:

– Ascites.
– Large tender hepatomegaly.
– Peripheral edema extending on to the thighs, if the vena cava becomes progressively obstructed.

Predisposing factors:

– Women.
– Oral contraception.
– Hematological disorders like hemolytic anemia, polycythemia, etc.
– Collagen disorders like rheumatoid arthritis, sarcoidosis, SLE.

Additional features of the underlying malignancy will be present in case, if the syndrome develops secondary to malignancy.

Chronic cholecystitis

Specific differentiating symptoms:

– Recurrent attacks of typical pain in epigastrium/RUQ, or atypically in LUQ.
  - Radiating to right infrascapular region.
  - Often persistent than intermittent.
– Qualitative dyspepsia for fatty food.
– Jaundice $\pm$. Jaundice often subsides after a few days but may persist as a major presenting symptom.

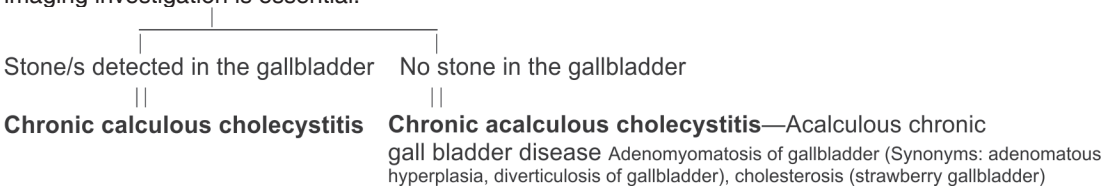
Nonspecific symptoms:

– Belching.
– Dyspepsia.
– Flatulence.

The only reliable sign infrequently found on physical examination is tenderness in the RUQ.

Free ebooks ==> www.Ebook777.com

More often than not, the clinical features of chronic cholecystitis are nonspecific.
As tenderness is the only reliable sign and that too being found infrequently, confirmation of the diagnosis by imaging investigation is essential.



Ductal stones (Stones in the common bile duct)

– Episodic upper abdominal pain and dyspepsia.
– Recurrent episodes of biliary colic accompanied by intermittent jaundice.

More severe/serious modes of presentation

– Progressive jaundice due to impaction of stone.
– Cholangitis.
– Pancreatitis.
– Secondary biliary cirrhosis and portal HT.

Although a few patients with ductal stones are asymptomatic, the majority present sooner or later with severe symptoms, and by and large incur a significant morbidity as the pathological potential of ductal stones is high.

Sclerosing cholangitis

– More commonly seen in males and usually presents in the 5th decade of age.
– Pain in the RUQ, jaundice, itching.
– Fever and attacks of rigors.
– Vague ill health, asthenia.
– Tender hepatomegaly.

Correlation between symptoms and progress of the disease is weak.
Rate of progression of the disease is variable and is influenced by the frequency of episodes of bacterial cholangitis and the development of cholangiocarcinoma, the diagnosis of which is often difficult in these patients.

(Recurrent) Pyogenic cholangitis

- Recurrent episodes of cholangitis.
- Debilitated patient.
- The disease may be further complicated by formation of hepatic abscess.
- Prevalent in South-east Asia.

Biliary Ascariasis

– Attacks of acute pain in the RUQ, vomiting and fever.
– Mild jaundice during the attacks of pain ±.
– Patients are generally younger than the age group in which gallstones are common.

Cystic disease of the biliary tract

– Cholestatic jaundice.
– Pain and lump in abdomen.
– Complications: Recurrent attacks of cholangitis and pancreatitis, biliary cirrhosis, portal vein thrombosis, cholangiocarcinoma, rupture of the cyst with biliary peritonitis.

High incidence in the Japanese.
Strong female predominance.
Usually presents in childhood.

www.Ebook777.com

Benign tumor of the gallbladder

→ Unexplained discomfort or pain in the RUQ.

Carcinoma of the gallbladder

Symptoms and signs referable to the biliary tract. – Refer to the table – 'step 2'.

Intrahepatic cholangiocarcinoma

– Mild pain in abdomen.
– Presentation with jaundice is uncommon.

Extrahepatic cholangiocarcinoma

– Dull pain in upper abdomen and progressive obstructive jaundice.
– Loss of weight, ascites and hepatomegaly ⇒ advanced disease.

Postcholecystectomy syndrome

Persistence of symptoms and signs referable to the biliary tract after cholecystectomy.

The complaints tend to be more troublesome when cholecystectomy has been performed in the absence of gallstones.

The syndrome excludes those patients whose symptoms are because of organic disease outside the biliary tract.

Postcholecystectomy syndrome usually reflects the failure of proper evaluation (clinical and investigative) prior to cholecystectomy.

Common causes of postcholecystectomy syndrome:

– Retained or recurrent stones.
– Gallbladder/cystic duct remnants.
– Bile duct strictures and other unrecognized iatrogenic injuries (choledochoduodenal fistula).
– Papillary stenosis.
– Biliary dyskinesia.

Pancreatitis

Basically, symptoms and signs, although nonspecific, referable to the pancreas.

For definition of pancreatitis, criteria of chronic pancreatitis, and notes on pancreatitis, refer to page 467.

*Causes of chronic pain/clinical circumstances associated with chronic **relapsing** pancreatitis:*

– Pancreatic intractable pain.
– Low grade cholangitis.
– CA pancreas.

Clinical manifestations:

– Pain, exacerbated by taking food and/or alcohol.
– Loss of weight resulting from loss of appetite produced by pain and malabsorption.

± Jaundice. – due to narrowing of the retropancreatic bile duct.

± A lump in epigastrium. Possible causes: A pseudocyt, CA head of the pancreas.

On physical examination, a hard consistency mass may be palpable in the epigastrium. However, it will not be palpable if the organ is shrunken and sclerosed.

(Surgical) Complications of chronic/chronic relapsing pancreatitis:

– Pain: Pancreatic intractable pain / Low-grade cholangitis / Fear or suspicion of CA: These conditions may be associated with chronic relapsing pancreatitis
– Suppurative cholangitis
– Obstructive jaundice
– Biliary cirrhosis
– Pancreatic cyst / pseudocyst
– Pancreatic ascites
– Pleural effusion

Free ebooks ==> www.Ebook777.com

– Fear / suspicion of CA
– Duodenal obstruction.

CA head of the pancreas

Main presenting feature is progressive obstructive jaundice (however, intermissions are not uncommon). Refer to chapter 37.

CA body and tail of the pancreas

– Pain: - In the epigastrium/back/either upper quadrant.
  - May be episodic or constant.
  - May be dull and vague.
  - Severe intensity suggests infiltration of the perineural lymphatics and the posterior parities.
  - May be related to meals, or otherwise.
  - Flexing the trunk forwards may lead to partial relief.
– Significant loss of weight.
– Hematemesis and melena are late features. Mechanism of GI bleeding: Mucosal invasion (gastric/duodenal) or portal HT.
– No abnormal physical finding in the early stage.

Notes:

– Diagnosis varies from the simple and clinically obvious to the most difficult and almost impossible.
– The initial symptoms and signs depend on the site and extent of the disease.
– The tumor itself is seldom palpable.

Sclerosing mesenteritis

– Recurrent pain in abdomen.
– Loss of weight.

Rare idiopathic condition. Tends to form masses that involve pancreas and small intestinal mesentery.

Intraperitoneal/Extraperitoneal abscesses

Basically, subacute inflammatory abdomen involving the region according to the location of the abscess, and hectic fever, nonspecific abdominal symptoms and general ill-health.

Differentiating points:

– The presence of predisposing factor, e.g. - Operative intervention with postoperative leak.
  - Primary infective/inflammatory focus like peritonitis, appendicits, diverticulitis, etc.
  - Accidental abdominal trauma.
– Anorexia and wasting (loss of weight).

Notes:

– When it develops after a laparotomy to deal with an infective pathology, condition of the patient initially improves temporarily, but after an interval of a few days or weeks symptoms of toxemia reappear. The condition deteriorates steadily and often rapidly.
– Swinging fever may be absent if the patient has been receiving antibiotics or steroid.
– In deep-seated abscess abdominal signs may be absent.
– Examples: - Subphrenic abscess.
  - Generalized peritonitis that has not been adequately treated predisposes to intraloop abscesses.
  - Perforated appendicitis predisposes to appendicular abscess.
  - A pelvic abscess may result from perforative appendicitis, perforative diverticulitis, and breakdown of a rectal anastomosis.

Chronic pseudocyst of the pancreas

Usually asymptomatic, and often, no recent attack of acute pancreatitis can be identified.
Past h/s/o attack/s of acute pancreatitis ±.
An ill-defined lump in the epigastrium.

www.Ebook777.com

Endometriosis
- Pain: Cyclical or constant.
- Deep dyspareunia.
- Pain on defecation if the pouch of Douglas is involved.
- Tender nodules in the pouch of Douglas.
- Uterus may be fixed in pelvis.

(Pelvic) **Adhesions**
- Dragging pain; usually constant but occasionally may be cyclical.
- Dyspareunia.
- Pain on defecation.
- H/o previous surgery common.

Pelvic congestion
- Worse at the end of day.
- Dyspareunia.
- Multiparity.

The existence of this condition is debated.

Ovarian mass
- Dull ache unless it undergoes torsion, when it will present with acute pain.
- Pressure symptoms related to bladder/bowel.
- A lump in the pelvis.

Endometrioma
- Mid-cycle pain.
- Subfertility and symptoms of endometriosis.
- Palpable mass.

Fibroids
- Menorrhagia.
- Pressure symptoms.
- Palpable uterus.

Adenomyosis
- Pain often central.
- Heavy painful periods.
- *US scan* may show mild fibroid change without definable fibroids.
- *Laparoscopy*: Negative, or mild endometriosis that does not resolve with surgery.

Prolapse
- *May not cause pain*, but causes dragging sensation and backache.
- Urinary hesitancy, fecal hesitancy.
- May have a bulge from introitus.
- On examination, prolapse is visible.

For differential diagnosis of renal diseases, refer to chapter 54.

Symptomatic AAA

Modes of presentation: Pain, distal embolization.

Constant *pain* in the abdomen or lower lumbar region.
Tenderness overlying the aneurysm (lump in abdomen).

Distal embolization → multiple small infarcted patches on the feet—described as 'trash foot'.

Free ebooks ==> www.Ebook777.com

A note
The major difficulty in a patient with pain and aneurysm is to determine whether the aneurysm is indeed the cause for pain. Possibility of renal disorder, musculoskeletal disorder, pancreatitis, peptic ulcer and MI should be rigorously investigated.

Idiopathic retroperitoneal fibrosis
- Average age at presentation 50–55 years, but the disease is documented in young (40s) and elderly individuals.

Clinical presentation is insidious, with a variable picture.
- The most common presentation: HT, obstructive uropathy (unilateral/bilateral) and renal failure.
- A nagging vague low backache.
- Edema of the scrotum and lower limbs because of venous obstruction.
- Intermittent ischemic pains in the lower limbs because of arterial obstruction.

Aortitis (Inflammatory aneurysm)
This has an established association with retroperitoneal fibrosis.

Visceroptosis
Women much more frequently affected than men.
H/o repeated pregnancies often present.
Peptic ulcer-like pain.
In standing position: – Bulging of the hypogastrium.
– Downward displacement of liver, spleen and kidneys evident on palpation.

Chronic functional pain (*synonyms:* Chronic gastrointestinal syndromes):
- NUD (**N**on-**U**lcer **D**yspepsia)
- IBS (**I**rritable **B**owel **S**yndrome)
- SOD (**S**phincter of **O**ddi **D**ysfunction)

Non-ulcer dyspepsia
Criterion: Chronic intermittent dyspepsia in a patient in whom an ulcer has been ruled out.
Cause could be organic (known or diagnosable) or psychosomatic.
Known/diagnosable specific organic causes: GERD, gastroparesis, postgastrectomy syndromes, intestinal angina, biliary colic and chronic pancreatitis.
Diagnosis is based on compatible symptomatology. There are no pathognomonic symptoms/signs.

Irritable Bowel Syndrome (IBS)
Abdominal discomfort or pain associated with defecation or a change in bowel habit and with an element of disordered defecation.
Characteristic features:
- Abdominal discomfort associated with more frequent bowel movements or loose bowel movements.
- Relief of discomfort with defecation.
- Increased mucus per rectum.
- Sense of incomplete evacuation.
- Abdominal bloating sensation with visible distension.
- Alternating constipation and diarrhea.

Supporting features:
- Age of onset < 50 years.
- Chronic fluctuating course. Stress-induced exacerbation of symptoms.
- Overall, nonprogressive.
- Weight - stable.
- Sleep is not disturbed by pain.

$\geq$ 3 characteristic features $\pm$ one or more of the supporting feature/s suggest the diagnosis of IBS.

O/E: Sigmoid colon loop may be tender. Otherwise, no obvious positive finding.

www.Ebook777.com

Notes:

– The course of IBS is highly variable, but essentially it is a chronic relapsing condition.
– *Severe tenderness, blood in the stool is unlikely to be due to IBS.*
– If it comes to distinguish IBS from uncomplicated diverticular disease of the colon, clinically it may not be possible. This is further confounded by the fact that both conditions may co-exist.

Sphincter of Oddi dysfunction

Benign, acalculous obstruction to the flow of bile and/or pancreatic fluid through the sphincter of Oddi which can result in pain, cholestasis or pancreatitis.

Clinical manifestations:

– Biliary-type pain with/without evidence of organic obstruction.
– Postcholecystectomy syndrome.
– Recurrent idiopathic pancreatitis.
– Papillary stenosis.

Classification applicable to postcholecystectomy syndrome:

Type I: – Unexplained biliary-type pain for > 6 months after cholecystectomy
and
– Abnormal liver enzymes on 2 occasions
– CBD dilatation (> 12 mm by ERCP)
– Delayed drainage of the CBD on ERCP (> 45 min)

Type II: – Unexplained biliary-type pain for > 6 months after cholecystectomy
and
– 1 or 2 out of the other 3 criteria (*vide supra*)

Type III: Unexplained biliary-type pain for > 6 months after cholecystectomy, without any other criteria (of those mentioned above).

Diagnosis of SOD:

– Based on clinical criteria.
– Numerous (other) causes of pancreaticobiliary type of pain must be ruled out beforehand.
– NUD and IBS also should be ruled out.

Free ebooks ==> www.Ebook777.com

34 ACUTE PAIN IN ABDOMEN (ACUTE ABDOMEN)

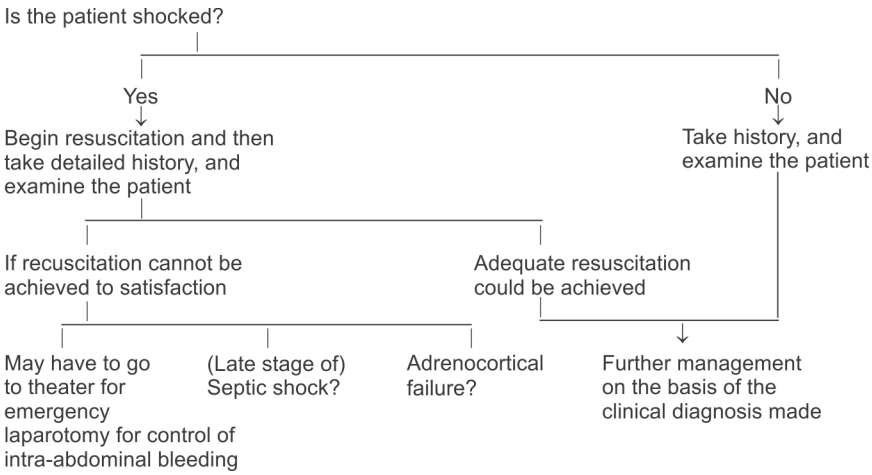
- 'Acute abdomen' is **defined as** the most severe, unbearable pain in abdomen of sudden onset in a previously well patient
- Trauma is excluded by convention as the cause is obvious and actual or potential life-threatening effects can be anticipated viz. bleeding, perforation of a hollow viscus, or both
- Pain being severe and unbearable, the patient with 'acute abdomen' usually presents within first 48 hours, though there are exceptions
- Pain in abdomen, especially the 'acute abdominal pain' is among the most frightening of all the symptoms, since the connotation to the patient experiencing it encompasses the entire gamut of catastrophes.

GENERAL REMARKS

1. From point of view of therapeutic implication, a case of acute abdomen may be classified into 'surgical' or 'non-surgical'. **'Surgical' does not necessarily mean** that an urgent laparotomy is always indicated. In certain cases of surgical acute abdomen, essentially conservative treatment is the mainstay of initial treatment, as for example in ureteric colic, acute pancreatitis, acute cholecystitis, etc. **Nevertheless**, in a large number of cases, an urgent laparotomy is indicated and hence, the need to differentiate surgical from medical cause of acute abdomen.
   One practical hint worth remembering: Pain lasting for 6 hours or more is most likely to be caused by a surgical condition.
2. Pain is a protective mechanism, which should not be masked by drugs (analgesic / sedative) unless and until definitive diagnosis is arrived at and definitive management planned.
3. Severity of pain may not denote severity of disease. Examples: (a) Severe colicky pain due to ureteric stone is not acutely life-threatening. (b) Severe fulminant fatal pancreatitis may be totally painless.
4. If severe pain continues for 6 hours after gastric decompression / simple enema / antispasmodic drug (depending on the case), then it may be advisable to operate.
5. Blunt abdominal trauma is notorious for producing delayed rupture of solid viscera (such as spleen / liver, etc.) with bleeding, or of hollow viscera with perforation (and peritonitis). **Even the mildest nausea, diarrhea or pain in abdomen after trauma therefore makes it essential that the patient be hospitalized** (and specialist opinion obtained)**. There should be no exception to this rule, no matter how trivial the trauma may appear to have been.**
6. **Spectrum of disease causing 'acute abdomen' of particular importance in tropical countries**: Amoebiasis, typhoid, abdominal TB, malaria, filariasis, schistosomiasis, abdominal crisis of sickle cell disease.
7. **The menaces**: If incorrectly treated, or untreated, death may result from one or combination of the following:
   - Sepsis
   - Acute fluid imbalance
   - Acute acid-base imbalance.

www.Ebook777.com

BASIC GUIDELINE



OUTLINE OF CASE-TAKING:

1. History and physical examination.
2. 'Provisional clinical diagnosis'.
3. **Appropriate** investigations. Commence appropriate treatment on the basis of provisional diagnosis made. Monitoring and revaluation if required.
4. 'Definitive diagnosis'.

HISTORY

Age
For causes of acute abdomen specific to the pediatric age-groups, refer to the pediatric section.

Gender
Women are more prone to calculous acute cholecystitis, diseases of the uterus and adnexa, and functional pain.

Residence
Urinary stone disease, filariasis are common in endemic areas.

Occupation
Lead poisoning in painters; arsenic poisoning in industry → chronic intermittent pain.

PAIN

- **Observe** as the history is unfolding: Does pain seem to be definitely present, OR Does it appear to be 'vague'?
- Is this the **first episode** of such pain, **OR** Is it a **recurrent** episode?

If recurrent,

  - How long does each episode last?
  - How much is the pain-free interval?
  - During the so-called pain-free interval, are you completely asymptomatic, OR Do you continue to have some discomfort / dyspepsia?
  - Are the durations of pain-episodes and pain-free intervals more-or-less constant, or are they variable?
  - Does the site of pain remain constant during each episode, or does it vary?
- **Duration** (of this episode)?

Free ebooks ==> www.Ebook777.com

- **Exact site**? - Observe as the patient points out the site. Does it appear to be segmental?

OR

Is it accurately located by pinpointing with a fingertip?

Is it vaguely located over a more diffuse area by putting the flat of a hand, or by moving around spread-out fingers?

– **In c/o pain in the RUQ / RLQ**:
  - Is it paining in this area right from the onset, OR Did the pain migrate (i.e. shift) from originally the epigastric / umbilical region?
  - Does the pain from epigastrium / RUQ radiate to the right lower back / the right shoulder?

– **In c/o pain in lumbar region**:
  - Do you feel more comfortable moving around, holding the flank?
  - Does the intensity of pain alter due to changing position?

Nature of pain?

OR

Colicky, OR continuous with fluctuating intensity? ⇓ Obstructive pathology of the upper urinary tract

Further details to be asked ↓

- Does it radiate to groin/vulva/testicle/penis/ inner thigh?
- Is it precipitated by running/jolting?
- Hematuria? if yes, then ↓
- Did pain come first, f/b hematuria? or
- Did hematuria come first f/b pain?
- Similar past history?

Examples of obstructive disorders
– Ureteric colic (*syn.* renal colic).
– Renal sone which may be obstructing the neck of calyx

Steady continuous? ⇓ Inflammatory pathology of the upper urinary tract

Further details to be asked ↓

- Intensity of pain—very severe or not so severe?

– Severe pain ⇒ inflammation involving the parenchyma. *Examples*: (Pyelo) nephritis, pyonephrosis, carbuncle of kidney, infarct of kidney perinephric abscess, mismatched blood transfusion, toxicity due to drugs like sulfa
– Less severe pain or, discomfort rather than pain ⇒ inlammation of mucusa.
Examples: pyelitis, pyelonephritis, ureteritis

– **In c/o pain in suprapubic region**

- Is it associated with **acute** retention of urine?
  i.e.
  – Is the intensity of pain—severe?
  – Is the pain continuous?
  – Suprapubic tender distention; dull on percussion

⇓

Pain due to acute retention of urine, which could occur due to various causes like enlarged prostate, stone, blood clot, acute cystitis, etc.

Not associated with acute retention of urine

- Does the pain radiate to distal urethra?
- Nature, intensity and constancy?
- H/o strangury?
- H/o ↑ed frequency, urgency?

– Intermittent discomfort
– Intensity of pain most severe when bladder is full, and relieved at least partially by voiding
– Strangury
– ↑ed frequency of micturition
– Urgency

⇒ most probably cystitis

Is it referred to the tip of penis? ⇓ Vesical stone / Trigonitis

www.Ebook777.com

- Is pain in the abdomen associated with pain in the back?
  – Exact site in the back? And other details?
  ↓
  a. If in the mid-back: Rhythmic or persistent? Does it decrease after food?
  b. If in the loin: Any urinary symptoms?
  c. Mid-sacral?
- Is the area of pain extending? – Extension of the area of pain may indicate spreading inflammation; e.g. spreading peritonitis.
- Was the mode of onset of pain abrupt?
- Did the intensity of pain become maximally severe almost at the onset, or did it take some time to become severe?
- Nature (= Character)—Colicky / constricting / burning / throbbing / twisting / shooting / dragging / vague?
- Can you walk comfortably with the body straight-upright, OR do you have to bend forward, hands clasping the abdomen while being able to walk only slowly?
- Did the pain initiate by jolting / running / traveling on a bumpy uneven road?
- Do you get some relief by
  – Assuming sitting position
  OR – Passing flatus / stools / taking enema
  OR – Applying heat?
- Do you get pain while passing urine?
  – Is the pain experienced at the end, or is it experienced throughout the act of micturition?
  – Is it referred to the tip of penis / labia majora / perineum / suprapubic region?
  – Nature? Is it scalding?
  – Does the pain disappear after passing urine?
- Is the pain related to periods in any way? For example,
  – Mid-menstrual
  – Before and during menstruation.

For INTERPRETATION OF, AND DISCUSSION ON PAIN, refer to chapter 33.

Anorexia?

The absence of anorexia probably rules out disorder of GIT (and its accessories).

Nausea?

Causes (relevant to 'acute abdomen')—disorders of stomach / gallbladder; or it could be psychogenic.

Vomiting?

- Frequency?
- Contents?
- Color?
- Odor?
- Is it 'effortless'?

INTERPRETATION OF, AND DISCUSSION ON VOMITING

- Vomiting is **usually absent** in diseases of anterior and posterior abdominal wall and diseases which give referred pain in abdomen
- **One or two episodes** of vomiting will occur in any case of acute abdomen due to intraperitoneal (i.e. visceral) disorders, intoxication, or psychogenic cause
- **Persistent vomiting** ⇒
  – Acute intestinal obstruction (Exception—distal colonic obstruction)
  – Acute inflammation of GI tract (Exception—localized inflammations like appendicitis, etc.)
- **Non-GIT causes of persistent vomiting**:
  – Normal pregnancy, and pregnancy-related disorders
  – Intracranial affections like meningitis, tumor, bleeding (in the newborn)

Free ebooks ==> www.Ebook777.com

- **Bilious vomiting** ⇒ intestinal obstruction with the level of obstruction being beyond the ampulla of Vater
- **'Feculent' vomiting = odor** of vomitus is dirty like feces ⇒ distal intestinal obstruction. Feculent vomiting does not literally mean vomiting of feces
- **'Effortless'** vomiting is seen in paralytic obstruction.

Vomiting of blood ('Hematemesis')?

Causes (relevant to 'abdominal pain'): Refer to page 387.

Absolute constipation?

- "Feels like passing flatus but is not able to do so, and feels that passage of flatus will relieve pain." - This statement may be taken as absolute constipation. Absolute constipation suggests complete intestinal obstruction.
- 'Technical' criterion of absolute constipation—'Not having passed feces **AND** flatus **for 24 hours**'.
- To conclude absolute constipation during the initial 24 hours after commencement of the pathological process leading to intestinal obstruction may be practically difficult as a normal person usually passes one stool per day (i.e. once in 24 hours) and also may be unaware of passage of small amounts of flatus normally. The situation may be even more difficult to assess in people who normally pass one stool in 48–72 hours [(and yet do not fit in the criterion of simple constipation (which by definition is '< 2 stools per week regardless of consistency and volume')].
- Practical situation demands early diagnosis in a case of acute abdomen. It is not prudent to wait for more than 24 hours to conclude or rule out absolute constipation! Hence, "feeling like passing flatus but inability to do so, and that, passage of flatus will relieve pain" may be taken as absolute constipation. This symptom may be supported by other symptoms / signs of intestinal obstruction. Additionally, it may be confirmed by passing a flatus tube twice with 'negative result' (both times, or) at least the second time.
- A pitfall: Many patients may pass feces / flatus after the onset of obstruction—this simply reflects evacuation of the intestine distal to the site of obstruction.

Distension of abdomen?

- How sooner or later did distension begin after the commencement of pain in abdomen?

Site:

- Is it confined to central region / right lumbar region / left lumbar region / along the periphery of the abdomen?
- If generalized, then did it initially develop in right or left lumbar region before becoming generalized?

INTERPRETATION OF, AND DISCUSSION ON DISTENSION

– Causes of distension occurring 'early' in the course of GI pathology: Distal small intestinal obstruction, colonic obstruction (both mechanical and pseudo-obstruction), gastric outlet obstruction
– Distension occurring 'late' in the course of GI pathology: Proximal small intestinal obstruction, late stages of perforation and inflammation (leading to peritoneal reaction and paralytic ileus)
– Other causes of distension: Bladder outflow obstruction, bleeding, tympanitis, uremia, hypokalemia.

Diarrhea?

- Frequency?
- Consistency—watery or semisolid?
- Amount?
- Is it associated with gripping pain in central abdomen?
- Does it contain mucus / pus / fat?
- Does it float in the pan?
- Is it foul-smelling?

DISCUSSION ON DIARRHEA

Criteria of diarrhea: > 3 semi-liquid stools per day.

Causes (relevant to acute abdomen): Enteritis, colitis, ileal position of appendicitis / similar such inflamed organ, may also occur in partial obstruction.

Abnormal bowel habit / bloating?

May suggest intermittent intestinal obstruction.

Fever?

- Grade (Degree)—'slight' or 'higher'?
- 'Hectic'?
- Rigors?

www.Ebook777.com

INTERPRETATION OF FEVER

- *'Slight'* fever may occur in many cases of acute abdomen—due to infection and/or dehydration
- *'Higher'* fever ⇒ infection
- *'Hectic'* fever ⇒ presence of pus, i.e. 'abscess'. [(Example—appendicular abscess (NOT appendicular mass)]
- *Causes of rigors*: Septicemia, cholangitis, pyelitis, onset of portal pyemia (may be secondary to acute appendicitis), acute retroperitoneal lymphangitis (filarial), malaria.

Did the body temperature fall to subnormal level?

This may suggest internal bleeding.

Are you feeling excessively thirsty?

- Have you been feeling excessively thirsty right from the onset of pain, or later on after vomiting many times?
  - Excessive thirst right from the onset ⇒ internal bleeding
  - Excessive thirst in the later stages of a disease with vomiting is experienced due to dehydration.

Did you faint? OR Do you feel you will faint on standing? OR Did you feel 'light headedness'?

⇒ vascular catastrophe.

Examples—acute rupture of ectopic pregnancy, dissecting aneurysm.

Increased frequency of micturition?

Causes:

- Pelvic peritonitis (provided other symptoms / signs agree).
- Urological causes (Refer to chapter 54).

Oliguria?

Causes:

- Dehydration due to intestinal obstruction / persistent vomiting from other causes
- Internal bleeding
- Urological causes (Refer to chapter 54).

Hematuria (Red / brown / stale tea-colored / smoky urine)?

Progress of the illness?

- Overall, how will you rate the progress of your illness – 'very rapid', or 'relatively gradual'?
  - Causes of 'very rapid' progress: Perforation / Inflammation (rupture of a viscus, acute pancreatitis, acute phlegmonous gastritis, acute retroperitoneal lymphangitis), torsion of a solid viscus, internal bleeding, strangulated intestinal obstruction, acute mesenteric ischemia
  - Rest of the acute abdominal conditions progress 'relatively gradually'

H/o **trauma during the previous week**?

Trauma might have been mild or forgotten

Particularly in a c/o blunt abdominal trauma however trivial it might appear externally, a high index of suspicion is essential to avoid missing of an intra-abdominal significant (potentially life-threatening) injury.

Any other significant illness present simultaneously / in the recent past?

- Acute MI / atrial fibrillation can predispose acute mesenteric ischemia due to embolism
- Acute MI may also → referred nonspecific pain in upper abdomen
- HT, IHD, CVD: The same atherosclerotic pathology responsible for these conditions may predispose to acute mesenteric ischemia due to thrombosis upon a stenotic lesion.
- Bleeding disorder may → shock due to internal bleeding / intestinal colic / acute lump in LQ
- Respiratory tract disease e.g. lobar pneumonia in the right base may → pain in the RLQ simulating acute appendicitis especially in children
- DM. Diabetic coma may → generalized peritonitis-like abdominal pain and physical findings
- Renal disease
- Jaundice.

Past history

- Similar past history?

  The presence of similar past history may qualify the case as recurrent acute pain (= chronic intermittent pain).

Free ebooks ==> www.Ebook777.com

- Postprandial abdominal pain ('intestinal angina'), loss of weight and diarrhea may ⇒ pre-existing chronic mesenteric ischemia on the background of which if acute mesenteric ischemia occurs, then the most probable pathology could be thrombosis.

Obstetric and gynecological history

- LMP?
- Amenorrhea?—Of how many months duration?
- Symptoms of the early months of pregnancy?
  - Amenorrhea
  - Nausea, tiredness and easy fatigability
  - Increased frequency of micturition
  - Engorgement of breasts
- Has the pregnancy occurred with an IUD in place?
- Has the pregnancy resulted from a failed postcoital contraception?
- Past h/o ectopic pregnancy?
- H/o 'spotting' OR bleeding equivalent of a menstrual period?
- Infertility?
- H/o tubal sterilization especially within the past 1–2 years?
- H/o tubal reconstructive surgery?
- Have you been using oral contraceptive?
- Have you got an IUD inserted?
- Abortion or delivery recently?
- Any interventional procedure recently like 'D and C', hysterosalpingography, etc?

DISCUSSION ON OBSTETRIC AND GYNECOLOGIC HISTORY

– *LMP*:
  - Gives an idea about whether the patient could be pregnant or otherwise
  - PID unrelated to pregnancy ('postmenstrual' PID) will commence soon after menstruation
  - Pain due to ruptured corpus luteum or ovarian cyst is experienced in the second half of a menstrual cycle
  - LMP helps to locate the time of mid-cycle pain
– Causes of bleeding in the 1st and 2nd trimester: Threatened abortion, ectopic pregnancy, placenta previa, cervical tumor.
– Oral contraceptive use may be implicated in acute mesenteric ischemia
– An IUD may predispose to PID. IUD also increases the risk of serious infection (PID) and also delays recovery
– Abortion and delivery may predispose to PID
– Interventional procedure upon the genital tract can predispose to PID, endometritis.

Treatment taken?

A treatment (particularly if inappropriate) may alter natural course of the disease, and consequently symptoms and signs.

Personal history

- Smoking?
  Diagnostic implication—smoking may → peptic ulcer disease, vascular disease.

GENERAL EXAMINATION

Is the patient fully conscious and well-oriented?

– The patient may have fallen asleep after a severe bout of pain has subsided but clear in mind, and alert when aroused
– (Persistent) Drowsiness is a bad sign.
  Causes of persistent drowsiness: Effect of a sedative drug, water and electrolyte imbalance, gangrene of intestine.

Posture?

– Lying on the back quietly, reluctant to make unnecessary movements suggests acute peritonitis
– Restless, tossing / rolling about in bed in agony, unable to find a comfortable position suggests colic (e.g. ureteric)

www.Ebook777.com

- FFD at hip joint may suggest iliacus hematoma or iliopsoas abscess
- Patient with cardiorespiratory embarrassment finds more comfort in sitting-up position.

Does the patient appear to be in distress due to pain?: Usually yes.

Eyes: Icterus?

Facial appearance?

- Clear and calm between the attacks of colic, and in mild infections
- Hippocratic (sunken eyes and drooping eyelids) ⇒ late stages of peritonitis / terminal phase of illness prior to death
- Anxious—in rest of almost all other intraperitoneal (visceral) causes of acute abdomen.
- Working of alae nasi may suggest respiratory disorder such as for example lobar pneumonia in the right base leading to pain in abdomen simulating acute appendicitis especially in a child.

Do the accessory muscles of respiration seem to be active?
That may suggest respiratory disorder such as for example lobar pneumonia in the right base leading to pain in abdomen simulating acute appendicitis especially in a child.

Apparent state of health?: The patient with acute abdomen usually looks sick.

Tongue?

- Dry ⇒ dehydration
- Dry and brown ⇒ acute intestinal obstruction / uremia / later stages of severe illness.

Skin: Temperature, mobility, turgor, and moisture?

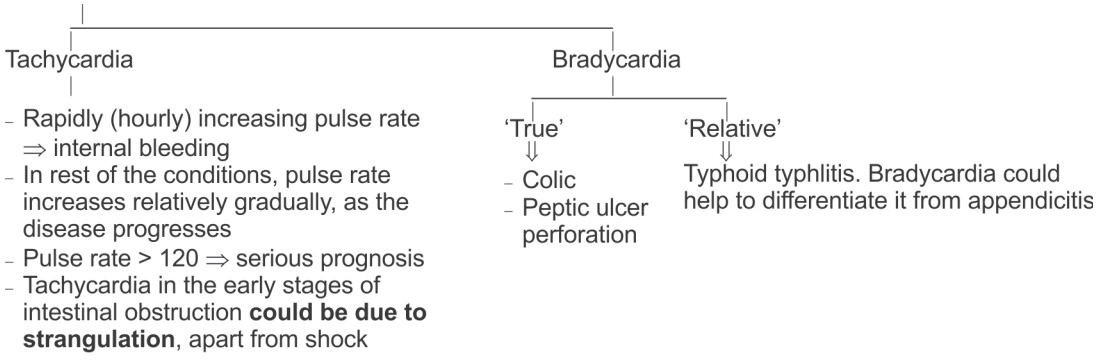
Dorsum of the hands: Venous filling?

Feet:

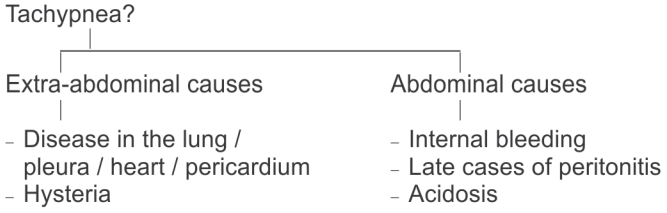
- Edema?
- Veins on the dorsum of the feet (with the patient lying in supine position)?

Body temperature?

Pulse rate



Respiratory rate



BP?

JVP?

Free ebooks ==> www.Ebook777.com

ABDOMINAL EXAMINATION

For discussion on examination-bed, position of the patient, and exposure of the part, refer to chapter 33.

INSPECTION

Methods

1. From top.
2. From side, i.e. tangential.
   - This often requires some bending even if the examiner has acquired sitting position by the bed-side. Tangential inspection helps to detect smaller bulges, and pulsations.

Movements—of the anterior abdominal wall associated with (normal) respiratory excursions

- Normal / diminished / absent?
- If diminished or absent—confined to a region, OR all over the abdomen?

Inference of diminished/absent movements depends basically on whether the abdomen is flat/scaphoid, or distended.
Diminished/absent movements in flat/scaphoid abdomen suggest irritation/inflammation of parietal peritoneum ('parietal peritonitis')
The anterior abdominal-wall movements may also falsely appear to have diminished/absent if the abdomen is severely distended. Severe distension tenses the anterior abdominal wall preventing its movement

- Diminished movements ⇒ 'lesser' degree of irritation of parietal peritoneum e.g. caused by blood (in the peritoneal cavity (hemoperitoneum)) OR in the earliest stage of peritonitis
- The absence of movements all over the abdomen ⇒ diffuse peritonitis, commonly due to perforation
- The absence of movements confined to a quadrant ⇒ acute inflammation / torsion / strangulation.

Contour

Scaphoid OR distended?
↓
- Does distension involve particular area?
- Is it 'severe'?
- What is its relation with vomiting?

Inference

- Scaphoid (or flat) in peptic perforation (in earlier stages), colics, torsion
- Causes of distension: (Distal) intestinal obstruction / colonic obstruction / gastric outlet obstruction, bladder outflow obstruction, bleeding, tympanitis, late stages of perforation and inflammation (leading to peritoneal reaction and paralytic ileus), uremia, hypokalemia
- **Predominant distension, with late vomiting** occurs in distal intestinal obstruction
- **Incessant vomiting with no or minimal distension** occurs in proximal intestinal obstruction.

For inference on fullness (distension) of abdomen, refer to chapter 33.

Skin

- Hyperpigmentation in the midline is seen in pregnancy
- Bluish discoloration in flanks—could be due to acute pancreatitis causing retroperitoneal bleeding
- Erythema ⇒ inflammation
- Bruising—could result from trauma.



Fig 34-1 Grey Turner's sign in acute pancreatitis

Umbilicus

- Transversely stretched?
- Pigmented?
- Periumbilical discoloration?

Inference

- Transverse stretching ⇒ a large amount of fluid in the peritoneal cavity, e.g. due to bleeding, peritonitis

www.Ebook777.com

- Pigmentation of umbilicus ⇒ internal bleeding / knuckle strangulation of intestine in a small umbilical hernia
- Periumbilical bluish discoloration ⇒ internal bleeding due to various causes including hemorrhagic pancreatitis
- Periumbilical yellow tinge ⇒ acute pancreatitis.

Visible peristalsis

- **Only rarely seen in acute intestinal obstruction**
- May be seen in subacute / chronic gastric / intestinal obstruction
- May be normally seen in thin-built individuals, and in patients with divarication of recti

'Ladder pattern' appearance = central abdominal distension with horizontal grooves—due to distended loops of obstructed small intestine. Seen in chronic obstruction, rather than in acute obstruction

Fullness (bulging) of intercostals spaces may be seen in large hepatic abscess in the right lobe near the lateral surface, and in subphrenic abscess.

PALPATION

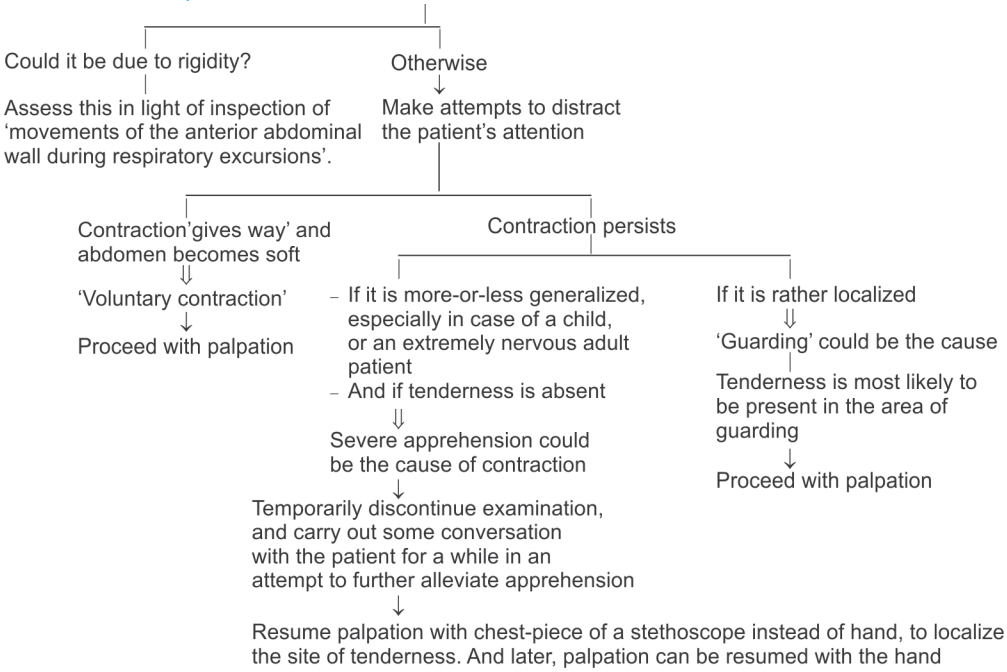
Position of the examiner: Sitting **comfortably** on the right side of the patient.

General Principles

1. *Selecting the area to begin palpation*
   Begin palpation in the region that is farthest from the site of pain or lump.
2. *To achieve a relaxed abdomen continuously* during palpation, it is essential to distract the patient's attention from abdominal examination. Keep talking to the patient frequently / as required. *The patient should not be allowed to* talk as talking necessitates contraction of the anterior abdominal wall muscles.

A. First round of palpation: Gentle 'superficial' palpation with flat of the fingers

1. Any **hyperaesthesia in well-defined areas**? viz. the shoulder / Boas's area / Sherren's triangle / lumbar area.
   For inference on hyperesthesia, refer to chapter 33.
2. Abdomen—**Soft, OR contracted? If contracted**



Free ebooks ==> www.Ebook777.com

3. **Tenderness** present or absent? If present—in which area / quadrant?
4. **Lump**?
5. (Gross) **Organomegaly**?

B. The next round of palpation: 'Deep' palpation

– This is carried out to obtain further details.

1. Tenderness

Localize the area of tenderness: Refer to chapter 33.

OTHER CLINICAL TESTS / SIGNS to determine the site of disease and/or the nature of pathology

a. **'Cough tenderness'**
- *Purpose*: Localization of inflammatory pathology e.g. appendicitis, diverticulitis, cholecystitis, etc.
- *Principle*: Movements associated with coughing may lead to irritation of the inflamed organ, which in turn may cause tenderness in the respective area / quadrant of the abdomen
- *Procedure*
  – Ask the patient to turn her / his face away (to avoid droplet infection)
  – Ask to cough and to point out the area of tenderness, if any
- *Practical observations to label the test as positive:*
  – If parietal peritonitis is well-established, then patient often anticipates pain, and either refuses, or expresses reluctance to cough
  – The act of coughing may be half-hearted
  – On coughing, the patient immediately winces and moves her / his hands in an attempt to protect the area affected by inflammatory pathology.

b. **Murphy's sign**
- *Purpose*: Demonstration of acute cholecystitis
- *Procedure*: Hook the costal margin gently with tips of fingers and ask the patient to breathe deeply
- *Inference*: Inspiratory 'catch' due to tenderness ⇒ positive sign.

c. **Rovsing's sign**
- Pressure in the left lumbar region leading to pain in the RLQ ⇒ positive sign for appendicitis
- *Principle:* Compression of descending colon generates a wave of increased intraluminal pressure passing backwards, in turn stretching the ostium of the inflamed appendix, causing pain
- *Significance:* The test is positive **even in retrocecal appendicitis**, where palpation in the RLQ may fail to elicit tenderness.

d. **RN Cooper's method** of eliciting tenderness in **acute appendicitis in obese patients**
- Ask the patient to lie in the left lateral position and then elicit tenderness in the RLQ
- In this position, the viscera overlying the appendix shift to the left, exposing the appendix to direct palpation. The abdominal wall is also relaxed to a certain extent by this position.

e. **Obturator test**
- Flexion and internal rotation of thigh (also flexing the leg at knee for convenience) leading to tenderness ⇒ positive sign for appendicitis
- *Principle:* The peculiar movement of thigh at the hip joint (described above) → stretching of the obturator internus, disturbing (hurting) the inflamed appendix if it lies on that muscle
- *Significance*: The test is positive in low-lying appendix, where palpation per abdomen may fail to elicit tenderness.

f. **Psoas test**
- Hyperextension of the thigh leading to tenderness ⇒ positive sign for appendicitis
- *Principle:* Hyperextension of thigh at the hip joint → stretching of the psoas, disturbing (hurting) the inflamed appendix if it lies on that muscle
- *Significance*: The test is positive in retrocolic appendicitis, where palpation per abdomen may fail to elicit tenderness.

g. **'Rebound tenderness'** ⇒ imminent gangrene of intestine in internal strangulation.
The test may also be positive in acute (gangrenous) appendicitis.

www.Ebook777.com

- Apply gentle but consistently increasing pressure in the previously localized area of tenderness, and then suddenly release the pressure
- Experiencing more pain while lifting off the pressure than when the pressure was being applied ⇒ positive test
- If other symptoms and signs clearly favoring the diagnosis of acute appendicitis are present, then avoid doing this test as it hurts the patient.

h. **Shifting tenderness**

- *Purpose*: To differentiate ANIML from acute appendicitis
- *Procedure*
  - After carefully noting (and better marking) the site of maximum tenderness in supine position, let the patient turn to lie on the left side for a few minutes
  - Locate the point of maximum tenderness again
- *Observe* whether the site of maximum tenderness moves (shifts) to the left of the original site
- *Inference*: Shifting tenderness suggests the diagnosis of ANIML rather than appendicitis.

Causes of localized tenderness

Acute inflammatory lesion, strangulation, torsion of an organ or a lump.

Causes of diffuse tenderness

– Those causes of peritonitis in which the pathological process is explosive, or the inflammatory response is minimal e.g. acute perforation of a peptic ulcer and typhoid-ulcer perforation respectively
– Internal bleeding.

2. Any **lump**?

If palpable, examine the details as described in chapter 35.

3. **Organomegaly**?

Liver, gallbladder, spleen, kidneys and distended urinary bladder.
For details, refer to chapters 33 and 35.

MEASUREMENT

Measure **abdominal girth at a fixed level** (such as umbilical), which can be repeated and then compared with previous readings (monitoring).

PERCUSSION

Aims of percussion:

– to determine, in combination with palpation, the size of organs viz. liver, spleen and kidneys
– to detect abnormal collection of fluid / gas
– to determine the structure (content) of a lump
– to detect a distended urinary bladder.

Obliteration of liver dullness in midaxillary line ⇒ free gas in the peritoneal cavity, e.g. in perforative peritonitis.
Principle: Free intraperitoneal gas tends to rise up and lie below the domes of the diaphragm. On the right side it diminishes (*syn*. obliterates) the liver dullness. The sign is not valid on the left side as the left side is normally resonant due to the presence of gas-filled stomach and colon.
Normally, superior border of the liver-dullness, in the mid-axillary line lies in the 7th IC space.

Fallacy:

– Emphysema of lung.
(– Anteriorly, the hepatic dullness may sometimes be obliterated by an anteriorly lying loop of intestine)

Shifting dullness

Free ebooks ==> www.Ebook777.com

This clinical test is done when there is dullness in the flanks. For clinical method, refer to chapter 33.
Causes of free fluid in the peritoneal cavity (relevant to acute abdomen):
– Internal bleeding
– Peritoneal exudate in perforation, strangulation, etc.
– Rarely, an acute abdominal condition supervening in a case of (pre-existing) ascites

Percussion to detect distended urinary bladder—refer to chapter 35.

AUSCULTATION

- Auscultation is done over—the stomach and intestines, the liver and spleen, and the aorta and its major branches
- Abnormal auscultatory sounds may be produced due to, basically 3 types of pathology:
  – Obstruction of the intestine → abnormal **peristaltic** sounds—hyperperistaltic / diminished / absent
  – Inflammation of solid organs like liver / spleen; perihepatitis, perisplenitis → **rubbing** sound. Refer to chapter 33
  – Stenosis of the aorta / its main branches (i.e. including renal arteries, etc.) → **murmur.**

  Normal peristaltic sounds
  – These are produced because of normal physiological contractions of the intestines, propagating a mixture of fluid and gas
  – Gurgling sounds, which start with a minimum intensity, increasing to moderate intensity and then stopping abruptly
  – Frequency: 1–2 / 15 seconds
  – Normal small intestinal peristaltic sounds are best heard over the periumbilical region and RLQ.

In intestinal colics, normal peristaltic sounds are present.

***Hyperactive** peristaltic sounds*
These are characterized by increased frequency, intensity and duration, synchronizing with increased peristaltic activity in the absence of mechanical obstruction and consequently without any pain or without severe pain. Examples—hunger pangs, enteritis.

Abnormal peristaltic sounds
1. Increased frequency, intensity and duration, having a cavernous resonance, synchronizing with each attack of severe colicky pain = '**hyperperistaltic** sounds' ⇒ mechanical intestinal obstruction.
2. **Diminished** sounds = sounds with decreased frequency, intensity and duration.
   – Diminished in a localized area ⇒ inflammation / torsion of an organ underneath
   – Diminished all over the abdomen ⇒ internal bleeding
   – Mechanism of diminished sounds – basically paralytic obstruction (in evolution).
3. Near-total absence of peristaltic sounds is called '**silent abdomen**'; heart and breath sounds may be surprisingly heard with a stethoscope placed on the abdomen. Silent abdomen suggests paralytic obstruction (established).
4. **High-pitched tinkling sounds** of constant intensity (*cf* increasing intensity of normal / hyperperistaltic sounds), synchronizing with bodily movement of the patient's trunk occur in paralytic obstruction. They occur due to spilling over of fluid from one distended coil to another on even slightest movement of the patient.

EXAMINATION OF GROINS AND EXTERNAL GENITALIA

1. **Pulsations of external iliac and femoral arteries**
   Absent / weak pulses on both sides ⇒
   – Disease of the aorta—abdominal or higher up
   – Rupture of a dissecting aneurysm
   – Abdominal angina.
   (Diseases of abdominal aorta may occasionally cause pain in abdomen.)
2. **Hernial sites**: Epigastric, umbilical and paraumbilical, LAPAROSCOPOIC PORT SITES, inguinal, FEMORAL,

www.Ebook777.com

OBTURATOR, GLUTEAL, SCIATIC.

Is there a swelling in the groin?

Yes ↓

Examine the usual attributes, with particular emphasis on the following points:
- Skin overlying the swelling?—Red, tense and glossy ⇒ strangulated hernia / inflamed hernia
- Temperature?:
  - ↑⇒ inflamed hernia / early strangulation
  - Cold in an established strangulated hernia
- Tenderness? ⇒ a complicated hernia
- Impulse-on-coughing is absent in a hernia complicated by obstruction/strangulation
- Does it feel tense?—A complicated hernia feels tense

No ↓

- Tenderness? The presence of tenderness, especially in an obese patient in whom a small hernia may not be visible / palpable, in suspected case of intestinal obstruction, may be due to obstructed or strangulated hernia
- Adducter spasm?—could be due to obturator hernia

3. **Scrotum**
   Causes of 'acute scrotum' which can cause pain in abdomen: Epididymo-orchitis, torsion of testicle.

PELVIC EXAMINATION

- In a female patient: Vaginal and rectal examination
- In a male patient: Rectal examination.

On PV/E:
- Tenderness?
- Any mass?

On DRE:
- Tenderness?
- Any mass palpable in the lumen?
- Any mass palpable through the wall?
- Blood on the fingerstall?

Clinical method for tenderness:
- Rectal tenderness is more difficult to gauge than abdominal tenderness
- Always start the assessment by pressing gently, directly backwards onto the sacrum. The reaction of the patient to this maneuver gives a baseline from which any more degree of tenderness can be gauged. It is also important to compare one side with the other.

Inference
- Tenderness high on the right side usually suggests 'pelvic appendicitis'
- A mass palpable in the lumen can be the apex of an intussusceptum
- A tumor in the pelvic colon may be palpable through the wall of the rectum
- Blood on the fingerstall ⇒ melena OR local pathology depending on the nature of blood
- The sign of 'emptiness' or 'ballooning' of rectum does not have much practical significance.

PALPATION IN SITTING POSITION (with hands locked and resting up on the head)

- This is possible only if the patient is not very sick.

Free ebooks ==> www.Ebook777.com

For clinical methods and other details, refer to chapter 33.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. On the whole, the presence of an individual symptom is by no means as important to diagnosis as is the presence of two or more in combination in a characteristic pattern.
2. **In old age**
   - Localization and severity of pain may blunt. Presentation may be atypical, **even with perforation of a viscus**
   - Cancer is more common over 70, than in those under 50. Hence, older patients even with apparently vague abdominal symptoms should be carefully assessed
   - Intra-abdominal inflammatory conditions such as diverticulitis may present with nonspecific symptoms such as acute confusion or anorexia and relatively little abdominal tenderness. The reasons for this are not clear.
3. Any pain in the pelvis may be gynecological in origin, and the complaints of disturbances of menstrual periods may strengthen the evidence.
4. A **golden rule**: Always consider, at an early stage, the possibility of an extra-abdominal cause responsible for pain in abdomen; even though most patients with acute abdominal symptoms have an intra-abdominal pathology.
5. **Persistent vomiting** in itself may produce pain in abdomen due to strain on anterior abdominal wall muscles.
6. The site of pain may not always correlate with a specific diseased organ. For examples (a) Esophagitis may produce pain in lumbar region/s. (b) Pain of pancreatitis may occur in any region of the abdomen. (c) Pain due to pyogenic liver abscess may not be necessarily localized to the RUQ.
7. **Pain from 'loin to groin'** may be caused apart from obstructive pathology of the ureter, by rupture of AAA.
8. Obstetric / gynecologic conditions which may lead to shock: acute rupture of ectopic pregnancy, ruptured corpus luteum / ovarian cyst, acute PID.
9. Abdominal distension can be a very difficult sign to assess, since the shape (i.e. protuberance) of the abdomen is of no much help if the clinician has not seen the patient when her / his abdomen was normal.
10. **Tenderness in a case of diffuse peritonitis** *v/s* **false tenderness in a hysterical patient:**
    - In diffuse peritonitis, peristaltic sounds are absent—'silent abdomen'
    - In hysterical patient, normal peristaltic sounds are present.
11. **Voluntary muscle contraction v/s 'guarding' v/s 'rigidity'**
    - *Voluntary abdominal-wall-muscle contraction* can occur in children due to apprehension or due to cold weather
    - Voluntary contraction may be overcome after gaining confidence of the patient by gentle palpation, and engaging the patient in conversation, distracting her / his attention
    - In-spite of above efforts, if the anterior abdominal wall does not relax, then 'guarding' may be considered as the cause of abdominal-wall-muscle contraction
    - '**Guarding**': Movements of a particular region of the anterior abdominal wall on respiration continue to occur, but the muscles go in spasm when palpation begins
    - Guarding ⇒ inflammation not of the parietal peritoneum, but inflammation of the visceral peritoneum, which allows gentle movements of the anterior abdominal wall related only to respiratory excursions, but not those which result from palpation
    - *Sometimes, guarding may be experienced in severe colicky pain*
    - Guarding is usually localized. However, some disorders can cause generalized guarding
    - *Causes of generalized guarding*: Severe form of ulcerative colitis, toxic dilatation of colon, acute retroperitoneal lymphangitis (filarial), rupture of corpus luteum or ovarian cyst.
    - **'Rigidity'**: Gentle movements of the anterior abdominal wall related even to respiratory excursions

www.Ebook777.com

are absent; the question of the abdominal-wall muscles yielding to the palpating hand does not even arise.

– Rigidity ⇒ irritation / inflammation of the underlying parietal peritoneum.
– Two aspects of rigidity need to be noted—degree and extent.
  a. The ***degree of rigidity*** depends on the type of irritant and the rapidity of progress of the condition.
  – *'Severe'* (practically board-like) rigidity occurs in peptic perforation and virulent infections
  – *'Moderate'* rigidity occurs in acute pancreatitis
  – *'Slight'* rigidity occurs in irritation caused by urine, blood, and in postoperative period.

  The classification into severe, moderate and slight is rather subjective. The abdomen with a severe rigidity if palpated once, will never be forgotten. Moderate and slight degrees of rigidity can then be compared and understood on that basis with relative ease.
  b. *Causes of localized rigidity*: Acute inflammatory lesions, gangrene, and torsion of solid organ / lump.
  Causes of diffuse rigidity: (i) Those causes of peritonitis, in which the process is explosive, or inflammatory response minimal e.g. acute perforation of a peptic ulcer and typhoid-ulcer perforation respectively; (ii) Internal bleeding.

Fallacies

a. *A false sense of the presence of rigidity* may be felt if there is severe abdominal distension, as it is difficult to depress the tensed up abdominal wall.
b. *Sometimes, rigidity may be absent even in the presence of a large amount of irritant fluid like pus within the peritoneal cavity*. Possible reasons may be as follows:
  i. Greater omentum may prevent the pus from coming in contact with parietal peritoneum.
  ii. The abdominal wall may be too weak to generate muscle spasm—e.g. in elderly and frail patients.
  iii. Severe toxemia.

Causes (and their differentiating features) **of rigidity other than parietal peritonitis**

Diffuse

– Acute retroperitoneal lymphangitis (filarial)
  - Patient is suddenly taken ill with acute pain in abdomen
  - High-grade fever with rigors
  - Repeated vomiting. Diarrhea ±
– Tetanus
  Need for differentiation arises as the patient with tetanus may experience pain in abdomen in addition to the presence of diffuse rigidity
  - Convulsions
  - Jaws and neck muscles also exhibit rigidity
  - Normal peristaltic sounds
– Psychological
  - Anticipation of pain
  - Nervousness
  - Shyness

  } Discontinue the examination temporarily. Carry out some conversation in an attempt to alleviate apprehension, and then resume examination.
– Spider bite → board like (i.e.severe) rigidity and exquisite tenderness as venom is neurotoxic
– Strychnine poisoning

Localized

– Injury/infectionof the abdominal wall muscles. Rigidity is confined to the area of the lesion.
– Irritation of lower intercostal nerves as for example in fracture of ribs → unilateral upper abdominal rigidity

12. An obstructed hernia is a common cause of acute intestinal obstruction.
The diagnosis of a hernia may be easily missed due to (a) Failure to examine the hernial sites as a result of hurriedly carried out incomplete abdominal examination, or shyness on the part of an especially female patient; and (b) Significant thickness of subcutaneous fat in a fatty patient. Even a large hernia may be missed in fatty females.
Hence, the hernial sites must be examined carefully not only for a swelling, but also for tenderness.

13. **Acute epididymo-orchitis and torsion of the testis → referred pain in lower abdomen**.

Free ebooks ==> www.Ebook777.com

If the external genitalia are not examined, then a case may be misdiagnosed as acute appendicitis / diverticulitis.

GENRAL OUTLINE of analysis to arrive at the diagnosis

- Does the disorder appear to be catastrophic?
- Does the cause of pain appear to be 'extra-abdominal' or 'abdominal'?

↓

In the anterior abdominal wall / intra-abdominal?

- Is this the 1st episode of pain OR is this a recurrent episode?
- Pathological nature and possible anatomical site of intra-abdominal disorder?
- Differential diagnosis in peculiar situations
- How much is the degree of homeostatic imbalance?
- Associated disorder?—closely related OR—otherwise?
- 'Residual problem'.

STEPWISE ANALYSIS OF DATA

Step 1

Does the disorder appear to be **catastrophic**? – if so, it needs an aggressive management approach.

Symptoms / signs suggestive of a catastrophic disorder

– (Excessive) Thirst from the onset
– Fainting
– Vomiting, excessive sweating, pallor and cold extremities ⇒ severe excruciating pain ⇒ acute serious life-threatening disease
– Prostration from the outset:
  - Perforation / inflammation. Examples—rupture of a viscus, acute pancreatitis, acute phlegmonous gastritis, acute retroperitoneal lymphangitis.
  - Torsion of a solid viscus or a lump
  - Internal bleeding. **Do not forget to r/o bleeding disorder.**
  - Strangulation / acute mesenteric ischemia.

Step 2

Does the cause of pain appear to be due to extra-abdominal disorder, or due to abdominal disorder?

Extra-abdominal *causes of pain*

a. **Referred** pain from the lungs / pleura / heart / spine / scrotum.
   Differentiating points:
   – No positive finding in the abdomen
   – The presence of signs in the respective organs.
b. **Metabolic**: Uremia, diabetic ketoacidosis, food hypersensitivity, porphyria.
c. **Hematological***: Sickle cell anemia, hemolytic anemia, leukemia, etc.
d. **Neurological***: Abdominal epilepsy, abdominal migraine, meningitis, etc.
e. **Intoxication due to extrinsic factors**: Drugs, filarial abdomen, malaria, bacterial infections, insect or snake poisoning, poisoning by arsenic / lead, etc.
f. **Collagen disorder**: Acute rheumatic fever, SLE, polyarteritis nodosa.

* Hematological and neurological causes are more common in children.

www.Ebook777.com

g. **Psychogenic**. The site of pain may be variable, complaints multiple, physical signs absent / vague.

Abdominal causes of pain

In the **anterior** abdominal **wall**

Abscess, hematoma†, myositis, spontaneous rupture of rectus, etc.

Differentiating points

- Vomiting is often absent.
- Pain ↑ed on contracting the anterior abdominal wall muscles.

Intra-abdominal: Could be intra-peritoneal or retroperitoneal, and each in turn may be visceral or non-visceral.

This is the most important group under the heading of 'Abdominal causes of acute pain.'

- Colic
- Acute intestinal obstruction**
- Inflammation (usually localized)
- Perforation (Perforative peritonitis)
  'Peritonism' = condition mimicking early stages of peritonitis
- Mesenteric vascular ischemia
- Torsion
- Internal bleeding
- Specific gynecological/obstetric causes
- Spectrum of disease causing 'acute abdomen' of particular importance in tropical countries.
- Pseudosurgical emergency, e.g. acute ascites

† A hematoma of the rectus muscle can occur after surprisingly mild trauma, so that the patient may not volunteer appropriate history, deeming it irrelevant.

** Virtually, acute intestinal obstruction is also a type of 'colic'. However, it is considered separately because it forms a major pathological entity by itself.

'Extra-abdominal' causes of pain must be considered at an early stage. They can be diagnosed without much difficulty if an index of suspicion is kept in mind, and confirmed by appropriate investigation.

Examples of extra-abdominal causes to be considered in specific clinical situations

The clinical situation	*A possible extra-abdominal cause of the pain*
1. Pain in epigastrium not fitting in any clearly defined surgical disorder	MI *Clues*: – Any symptom / sign pointing at cardiovascular dysfunction – Any suggestion of cardiogenic shock
2. Intestinal colic	Bleeding disorder
3. Severe colicky pain mimicking intestinal obstruction	– Poisoning due to metal, strychnine – Acute porphyria – Tabes dorsalis
4. Hypovolemic shock, and hence suggesting intraperitoneal bleeding	Bleeding disorder
5. Apparently acute appendicitis	Lobar pneumonia in the right base, especially in a child Physical signs in the chest may be mild *Clues* in favor of pneumonia: – Working of alae nasi and accessory muscles of respiration – A tinge of cyanosis

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

6. Apparent generalized peritonitis	– Fluid and electrolyte imbalance with paralytic ileus, nausea and vomiting – Diabetic coma – Adrenocortical failure
The clinical situation	*A possible extra-abdominal cause of the pain*
7. Lump in the lower quadrant possibly with FFD at the hip (due to a large iliacus hematoma as diagnosed by an imaging investigation)	Bleeding disorder
8. In general, when pinpointing a specific diagnosis is not possible	Back. Pain caused by irritation of lower thoracic and 1st lumbar nerves by trauma / tumor / inflammation of the spinal structures (cord / canal / column) or paraspinal muscles *Clue*: Any positive finding on examination of the back and nerves Hypoesthesia / hyperesthesia in the area of skin innervated by the nerve

In most of the cases of 'acute abdomen' however, the disease lies in the abdomen.

Note the chronological order of development of symptoms and signs.

Examples of some inferences.

- Persistent vomiting in itself may produce pain in abdomen due to strain on the anterior abdominal wall muscles
- Acute colicky pain, incessant (bilious) vomiting, 'mild-to-moderate' abdominal distension without absolute constipation (or absolute constipation developing very late) ⇒ proximal small intestinal (may be jejunal) obstruction
- Acute colicky pain, abdominal distension, absolute constipation without vomiting (or vomiting occurring very late) ⇒ distal colonic obstruction
- Pain f/b vomiting f/b fever occurs in acute appendicitis.

Step 3

Is this the 1st episode of pain OR recurrent episode?

In these cases, sometimes pain may be severe and at other times, it may not be so severe.

Causes of recurrent acute pain ('Recurrent acute pain' is synonymous with 'chronic intermittent pain'.)

Mechanical	***Inflammatory***	***Neurologic and metabolic***	***Poisoning***	***Miscellaneous***
– Obstructive pathology. Examples of intermittent intestinal obstruction: hernia, intussusception, adhesions, volvulus – Ampullary stenosis – Gallstones	– Recurrent (/ ? 'chronic') appendicitis – Inflammatory bowel disease – Chronic relapsing pancreatitis – Retroperitoneal lymphangitis (filarial) – Endometriosis / endometritis – Sclerosing mesenteritis – Familial Mediterranean fever	– Nerve-root compression / entrapment – Diabetic radiculopathy – Abdominal epilepsy – Abdominal migraine – Uremia – Acute intermittent porphyria	– Lead poisoning in painters – Arsenic poisoning in industry	– Functional abdominal pain (non-ulcer dyspepsia, irritable bowel syndrome, sphincter of Oddi dysfunction, etc.) – Chronic mesenteric ischemia, median arcuate ligament syndrome (- caused by isolated celiac artery compression) – Midcycle pain

Step 4

- **Pathological** nature and **possible anatomical site** of the **'intra-abdominal'** disorder?
- There are essentially 10 subgroups under this heading (the intra-abdominal causes of pain). Refer under

www.Ebook777.com

the step 2 above. It is necessary to differentiate one from the other to arrive at the diagnosis

- **It is often necessary to determine the nature of pathology first, and then think about the possible anatomical site of the disease.** This is because the progress of an acute intra-abdominal disease may be very rapid and the patient will have to be taken up for an urgent laparotomy without having a confirmation of the exact anatomical location of the disease; for example, in a c/o acute perforative peritonitis, the patient will usually go for an urgent laparotomy without knowing whether a perforation is in the stomach or in the intestine. Nevertheless, one should endeavor as far as possible to determine the anatomical site of disease in every case.
- Radiological examination, US scan and routine blood and urine investigation should be done at the least in every case as they greatly assist in arriving at and/or confirming the diagnosis.
- Radiological examination: – X-ray chest with diaphragm
  – X-ray abdomen with pelvis
  } Both in upright position as far as possible
  - Thus, X-ray of the entire trunk ensures that no radiologically diagnosable disease is missed
  - Upright position ensures detection of free gas under the diaphragm, and multiple air-fluid levels
  - X-ray chest will also help in preanesthetic evaluation.
- If patient is too sick to stand upright, then X-rays may be obtained in left lateral decubitus if perforative peritonitis is suspected or in supine position if intestinal obstruction is suspected.

A quick guide for BASIC DIFFERENTIATING FEATURES
(Excluding mesenteric vascular ischemia and specific obstetric/gynecological disorders)

Clinical differentiating features ↓	**Colic**	**Acute intestinal obstruction**	**Inflammation** (Usually localized)	**(Parietal) Peritonitis**	**Torsion**	**Internal bleeding**
Duration: Prolongation of acute pain ≥ 2 weeks	X	X	Possible	X	X	X
Shifting of pain	X	X	Possible, e.g. in acute appendicitis	X	X	X
Radiation of pain	**Y**	**Y**	May be possible, e.g. in acute cholecystitis	X	X	X
Extension of the area of pain	X	X	±	**Y** ⇒ spreading peritonitis	X	**Y**
Pain reaching maximally severe intensity almost at the onset	X	± + in case of • Acute progressive intussusception • Acute volvulus	± + in case of • Acute pancreatitis • Acute phlegmonous gastritis	± + in case of peptic perforation and enteric perforation	**Y**	X
Colicky pain	**Y** Exception: So-called 'gallbladder / biliary colic'	**Y**	X	X	X	X
Constricting pain	X	X	X	X	X	X
Twisting pain	X	Volvulus	X	X	**Y**	X

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

Clinical differentiating features ↓	**Colic**	**Acute intestinal obstruction**	**Inflammation** (Usually localized)	**(Parietal) Peritonitis**	**Torsion**	**Internal bleeding**
Jolting / running leading to precipitation of pain	Possible, e.g. in ureteric colic	X	Possible, e.g. in acute appendicitis	X	X	X
Incessant vomiting, later on becoming feculent	X	**Y** Exception: Early stage of colonic obstruction	X	X	X	X
Non-descript vomiting	±	**NA**	**Y**	**Y**	**Y**	**Y**
Absolute constipation	X	**Y** Exception: Proximal small intestinal obstruction	X	X	X	X
Mild fever	X	X	Possible, e.g. in acute appendicitis	X	X	X
Fever with rigors	X	X	Cholangitis, pyelonephritis, septicemia due to any other cause	X	X	X
Excessive thirst right from the beginning	X	X	X	X	X	**Y**
Fainting	X	Possible, e.g. in intussusception	X	X	X	**Y**
Excessive sweating, pallor, cold extremities	X	Possible, e.g. in intussusception	X	X	X	**Y**
Similar past history	Possible	Possible, especially, in postoperative adhesive obstruction, and volvulus which is not treated by definitive surgical treatment.	Possible	Usually unlikely	Possible	Usually unlikely
Lying on the back quietly	X	X	**Y**	**Y**	±	±
Restless; tossing or rolling about in bed in agony	**Y**	±	X	X	X	X
Subnormal temperature	X	X	X	X	X	**Y**

Contd...

www.Ebook777.com

Contd...

Clinical differentiating features ↓	Colic	Acute intestinal obstruction	Inflammation (Usually localized)	(Parietal) Peritonitis	Torsion	Internal bleeding
A rapid (hourly) increase in pulse-rate	X	X	X	X	X	**Y**
Bradycardia	**Y**	± (Tachycardia at a later stage with development of shock)	'Relative' bradycardia in typhoid typhlitis	Possible, e.g. in peptic perforation	X	X
Tachypnea	X	X	X	**Y** In late cases	X	X
Distension of abdomen	X	**Y** Exception: Proximal small intestinal obstruction	X	**X** at an earlier stage **Y** at the later stage of paralytic ileus	X	± When present, it is only to some extent
Tenderness	±	X Exceptions: 1. Strangulating obstruction 2. Obstruction complicated by perforation	**Y**	**Y**	**Y**	**Y**
Guarding	±	X	**Y**	**NA**	**Y**	**NA**
Rigidity	X	X, unless strangulating	±	**Y**	±	**Y**
Lump / Organomegaly	X	± Example: Intussusception	± Examples: Appendicular mass, liver abscess, etc.	NA Even if a lump is present, it may not be palpable due to rigidity	±	X
Hyperperistaltic auscultatory sounds	X	**Y**	X	X	X	X
Diminished peristaltic sounds in the affected area / quadrant	X	X	**Y**	X	**Y**	**Y**

'X' = No, i.e. not applicable. 'Y' = Yes, i.e. applicable.

- Further differentiation in each of the first 5 groups (i.e. colic ↔ torsion) should be done as far as possible. If that is not possible, then differentiation should be done **at least in 2 groups viz. the one requiring urgent surgery and the other not requiring urgent surgery**
- **In the absence of any e/o trauma, shock, depletion of extracellular fluid, or intestinal obstruction, conservative management is indicated in the first instance.**

COLICS

Differentiating features of colic in general

– Typically colicky (griping) in nature (which may radiate), sometimes severe enough to make the patient toss about in bed unable to find a comfortable position

Free ebooks ==> www.Ebook777.com

- May be relieved by pressure or application of warmth
- Belching, vomiting, retching ±
- Varying degrees of collapse ±

Diagnostic points of interest

- The site and cause of colic?
- Any moderate-to-severe degree of homeostatic imbalance? For example: Uterine tube colic just preceding (hypovolemic) shock suggests acute rupture of ectopic gestation.

Clinical points which may guide to arrive at the diagnosis

Age: Causes of colic are different in adults and children. For causes of colic in children, refer to chapter 74.

Gender. Common colics in **women**:

- During child bearing period: Uterine tube colic
- It is also common just before acute rupture of ectopic gestation
- Over 40, especially if fatty women: Biliary pain.

Site of pain:

- Umbilical / epigastric ⇒ intestinal / appendicular / pancreatic colic
- Umbilical / epigastric and backache ⇒ pancreatic colic
- RUQ ⇒ biliary colic
- Lumbar ⇒ renal colic
- Suprapubic ⇒ uterine, or uterine tube colic.

A pitfall

Splenomegaly may cause attacks of acute colicky pain (Mechanism—uncertain).

DIFFERENTIAL DIAGNOSIS OF COLICS

Acute intestinal colic

= colicky pain in central abdomen due to irritation of intestine associated with catarrhal enteritis.

Symptoms

- Diarrhea
- Intensity of griping pain is particularly more before passing a motion
- H/o 'improper' diet, i.e. unaccustomed / unusual / unhygienic
- Family history may be contributory, i.e. other members of family having dietary indiscretion of same type being suffering from the same symptoms around the same time period.

Sign: 'Hyperactive' peristaltic sounds on auscultation.

Note: **A bleeding disorder** can result in bleeding into the wall of intestine in turn leading to disturbance of mobility and colicky pain in abdomen, e.g. Henoch-Schönlein purpura. Hence, it is important to obtain carefully, a h/o bleeding disorder.

Appendicular colic

- Occurs in 'obstructive appendicitis'
- Visceral pain which initially occurs in the umbilical region (occasionally in epigastrium) is colicky in nature. As it is associated with vomiting, initial (~ 6–8 hours or so) clinical picture may suggest acute intestinal obstruction!

Acute biliary colic

- Severe colicky pain in RUQ, which may radiate to the right side of the back / right shoulder.
- Duration: 30 minutes to several hours
- Shivering ±
- Tenderness overlying the gallbladder usually present
- Jaundice ±
- Natural history: Merges into acute obstructive cholecystitis, OR resolves either spontaneously or as a result of analgesic / antispasmodic treatment.

www.Ebook777.com

Causes of uterine colic

– Normal labor pains
– Abnormal labor states: Premature labor, abortion, closing phases of a tubal pregnancy.

Uterine tube colic

Differentiating point: Colicky pain in suprapubic region in a woman of child-bearing age.

Physical findings on PV/E, when uterine tube colic occurs due to acute rupture of ectopic gestation:

– In the vaginal fornix: Tenderness and a mass on the affected side.
– Severe tenderness on rocking the cervix.

Ureteric (*syn.* **renal**) **colic**

Differentiating points

– The site of origin and radiation of pain: Typically 'loin to groin' (i.e. towards groin / scrotum / labium / inner thigh). Characteristically shown by the patient with the hand with outstretched thumb put obliquely on the flank (thumb resting on the loin and finger-tips pointing towards the groin).
  - Pain restricted to the loin ⇒ the stone just coming out of the pelvis
  - Pain radiating from loin to the lower quadrant of abdomen, and sometimes even down to the level of scrotum / labium ⇒ obstruction of the mid-ureter
  - Pain radiating along the urethra to the tip of penis or inner thigh ± symptoms of vesical irritability (increased frequency, urgency, suprapubic discomfort) ⇒ obstruction of the lower ureter
  - The attack of pain may be precipitated / aggravated by jolting / running
  - Pain may be followed by hematuria. Hematuria is slight
– Duration of an individual attack: Rarely > 8 hours
– Attacks of pain are repeated at longer or shorter intervals, until the stone is ejected into the bladder or becomes impacted in the ureter
– Strangury ⇒ stone impacted in the lower end of ureter
– Contributory past history: Similar attacks / passage of gravel or stone in urine / known proven c/o urolithiasis
– Temperature becomes subnormal, and pulse quickens as the attack progresses
– Guarding of the lateral abdominal muscles, but not as a rule of the rectus abdominis
– Tenderness in respective renal angle
– Testicle of the affected side may be drawn up.

C*auses and differentiating features* of **acute colicky pain in lumbar region**

1. *Calculous colic* (caused by a stone in the ureter)
2. *Crystal colic*: Caused by the passage of a shower of oxalate crystals, e.g. after eating excess of strawberries.
3. '*Clot colic*': Rare. May occur on the background of operated kidney. Hematuria occurs first, which is then followed by pain.
4. Acute colicky pain may also be encountered in hydronephrosis (Dietl's crisis), and pyonephrosis.
5. Splenomegaly also may cause colicky pain.

Notes on ureteral pain

– Ureteral pain is usually acute and secondary to obstruction which is of sudden onset.
– Ureteral pathology (irrespective of the nature, i.e. whether stone or tumor or anything else) that develops slowly and causes 'mild' (incomplete) obstruction does not cause pain
– *Mechanism/*s of ureteral pain:
  - Acute distension due to sudden obstruction
  - Hyperperistalsis to overcome the obstruction
  - Spasm due to irritative nature of pathology (usually stone).

***Fate* of a ureteric colic**

Free ebooks ==> www.Ebook777.com

Disappearance of pain
⇓
Obstructing element (usually a stone) passed off in the bladder, OR formation of a diverticulum into which the stone migrates

Colicky nature of pain replaced by dull ache
⇓
Impaction of stone in the ureter
- Impaction in the upper/mid-ureter → dull ache in the loin
- Impaction in the lower ureter → dull ache in the lower quadrant of abdomen

ACUTE INTESTINAL OBSTRUCTION

Diagnostic points of interest

- Type of obstruction—paralytic OR mechanical?
- In the case of mechanical obstruction:
  - Simple OR strangulated?
  - Does it affect small or large intestine, and further to that, what is the exact level?
- Degree of homeostatic imbalance?
- Cause of obstruction?

Clinical features	**Paralytic ileus**	**Mechanical obstruction**
Pain	Intensity—not severe Nature—tight diffuse discomfort With recovery of intestinal motility, colicky pain described as wind pain is experienced by the patient	– Severe colicky – After cessation of peristaltic activity, the colicky pain is replaced by constant diffuse pain, which is not usually severe
Vomiting	Effortless	Time of onset, character, etc. vary according to the level of obstruction
Distension of abdomen	Flaccid rather than gross	Gross. However, the time of onset, degree and location vary according to the level of obstruction
Peristaltic sounds	Absent, although occasionally high-pitched sounds may be heard	– Initially hyperperistaltic – As paralytic ileus sets in, they get diminished, and finally disappear

PRINCIPLES AND PITFALLS in a suspected case of acute intestinal obstruction

1. Clinical picture of small intestinal obstruction in **aged** patients could be secondary to pelvic peritonitis primarily resulting from acute appendicitis. In other words, acute appendicitis in aged patients may present with clinical picture of small intestinal obstruction.
2. **Initial (~ 6–8 hours) clinical picture of acute obstructive appendicitis** may be confused as intestinal obstruction.
3. If there is rigidity, and the absence of peristaltic sounds in a particular area / quadrant; ± tachycardia, then primarily it may be a case of localized perforative peritonitis, or adynamic segment of gangrenous gut.
   Causes of localized perforative peritonitis: Perforative appendicitis, perforative diverticulitis, suppurative or perforative cholecystitis, perforation of a visceral abscess. The intestinal loops trying to localize the pathological area become paralyzed, giving a picture of intestinal obstruction. However, this conclusion may be drawn only **provided strangulated obstruction can be ruled out**.
4. If a patient with a clinical picture of strangulating intestinal obstruction has a (groin) hernia, then keep in mind a possibility of strangulated 'W' shaped hernia.
5. In a case of **acute pancreatitis**, toxic exudate might spread in the layers of transverse mesocolon → spasm of transverse colon → clinical picture of intestinal obstruction.
6. **In case of acute mechanical intestinal obstruction of apparently uncertain origin**
   Have you checked **ALL** the **hernial sites,** particularly the femoral, umbilical and laparoscopic port sites?

www.Ebook777.com

A small hernia may be easily overlooked, especially if the patient is fat, and the abdominal symptoms and signs of intestinal obstruction are gross and overshadow a small tender lump in the groin.

- In an obese patient, a small hernia may be neither visible nor palpable. Tenderness at the known hernial site may be the only clue about obstructed / strangulated hernia as the cause of intestinal obstruction
- Not uncommonly, some patients are completely unaware that they have been suffering from a hernia! Even in such patients, a tender, irreducible, often tense swelling without impulse-on-coughing, or only tenderness at the known hernial site should be taken seriously, as that may be the only suggestive clue to the cause of intestinal obstruction
- Although tenderness indicates simple obstruction or strangulation, practically, strangulation should be considered in every such case unless proved otherwise (at operation)
- In *elderly thin-built (average body weight 35–40 kg) multiparous women*
- Intestinal obstruction could be due to obstructed obturator (pelvic floor) hernia
- Preoperative clinical diagnosis is difficult, as groin mass is rarely found on clinical examination.
- Even if a groin mass is present, it occurs in the region of saphenous opening, and distinguishing it from a femoral hernia is clinically not possible.

***Paralytic** obstruction ('ileus')*

Differentiating points: Refer to the table above.

Supporting point: Clinical situation predisposing to the development of paralytic obstruction.

***Causes** of paralytic obstruction*

- Overdistension of intestine in the natural course of untreated mechanical obstruction
- In the natural course of any untreated acute intra-abdominal disease like inflammation, peritonitis, torsion, etc.
- Septicemia due to focus of sepsis anywhere in the body
- Toxemia
- Mesenteric vascular occlusion
- Postlaparotomy
- Following any major / supramajor surgery in any region of the body
- Following severe trauma anywhere in the body
- Following child birth
- Electrolyte imbalance like hypokalemia, hypochloremia
- Hypoproteinemia
- Drug-induced, e.g. tricyclic antidepressants.

Mechanical simple obstruction

Cardinal symptoms

Pain, nausea and vomiting, absolute constipation, and distension.

- All 4 symptoms are not present in every case (unless a patient presents exceptionally late)
- Usually 3 out of 4 symptoms are present.

Points to note regarding the cardinal symptoms

1. Chronological order of the occurrence of symptoms will vary according to the level of obstruction.
   Rarely, profuse bilious vomiting and absolute constipation—both may be prominent features without noticeable delay between the 2 symptoms. This situation suggests obstruction involving both the (proximal) small intestine as well as the distal large intestine—an example: ileosigmoid knotting.
2. Severity and exact site of pain and distension may give an idea about the level of obstruction ± probable pathology.
   Examples: (a) Clear-cut colicky pain of severe intensity in the central abdomen suggests acute small intestinal obstruction. Pain in the RLQ (overlying distended cecum) rather than symmetrical central abdominal pain, and which may not be typically colicky and severe suggests large intestinal obstruction. (b) Absent or minimal distension v/s severe distension.
3. Change in the nature of the symptoms correlate with progress of pathology.

PAIN

Free ebooks ==> www.Ebook777.com

- **Severe colicky** pain coinciding with hyperperistaltic rushes (audible on auscultation)
- After cessation of peristaltic activity owing to increasing distension of the intestine, colicky pain is replaced by continuous diffuse pain, which is not usually severe
- **Development of continuous diffuse pain of severe intensity may indicate** the onset of strangulation.

NAUSEA AND VOMITING

- Early appearance of these symptoms ⇒ proximal small intestinal obstruction
- Relatively later appearance ⇒ distal obstruction, i.e. distal small intestinal / proximal colonic
- The absence of vomiting OR vomiting occurring very late (many hours / days after other symptoms have been established) ⇒ distal colonic obstruction. Even though complete obstruction of distal colon may have been present for several days, vomiting may not occur at all
- Changes in the character of vomitus with progress of obstruction: Initially yellowish (nonbilious) f/b bilious (dark green colored owing to the presence of bile) f/b feculent (dirty foul smelling owing to bacterial overgrowth in the obstructed intestine).

  (Feculent ≠ Fecal. True fecal vomiting can occur only in gastrocolic fistula.)

ABSOLUTE CONSTIPATION ⇒ complete obstruction.

DISTENSION: Onset, degree and location (in broad sense) will vary according to the level of obstruction.

Cardinal signs *of mechanical simple obstruction*

- Distended tympanic abdomen
  - In proximal small intestinal obstruction, distension may be absent; but when present, it will be delayed and minimal
  - In distal large intestinal obstruction in which peripheral distension occurs, may be followed by central distension if the ileocecal valve is incompetent, or basically competent valve gives way due to progressively increasing back-pressure from the colon
  - Central distension ⇒ distal small intestinal obstruction
  - Ladder-pattern type distension around the umbilicus ⇒ distal small intestinal obstruction. However, this may be seen in chronic obstruction, rather than in acute obstruction
  - Peripheral distension (in the beginning) ⇒ distal large intestinal obstruction
  - 'Severe' distension is encountered in distal small intestinal obstruction, mechanical obstruction of left colon, volvulus of pelvic (sigmoid) colon
- Tenderness—absent
- On auscultation
  - During initial stages—hyperperistaltic sounds
  - In later stages, with the development of paralytic ileus the peristaltic sounds get diminished, and finally disappear

Differentiating a rather proximal small intestinal obstruction from a rather distal large intestinal obstruction is not difficult. However, the intermediate territory of distal small intestine and proximal large intestine gives rise to a mixed clinical picture and consequently it may not be possible to differentiate between the two

The most important means of distinguishing small from large intestinal mechanical obstruction (and incidentally, both of these from paralytic obstruction) is plain X-ray of the abdomen

Causes of mechanical obstruction (especially applicable to adults)

	Small intestine	**Large intestine**
Extramural	– Adhesions – Hernia – Mass (neoplastic / inflammatory) in the vicinity causing external compression – Volvulus	– Volvulus – Mass (neoplastic / inflammatory) in the vicinity causing external compression – Hernia – Adhesions

www.Ebook777.com

Intramural	– TB – Tumor–benign, malignant (adenocarcinoma, carcinoid, lymphoma, mesenchymal tumors) – Intussusception – Regional enteritis – Radiation enteropathy	– Tumor—adenocarcinoma, other unusual cancers (SCC, carcinoid, lymphoma, sarcoma), polyp – TB – Diverticulitis – Regional enteritis – Ulcerative colitis – Pseudotumor caused by bilharzial granuloma—macroscopically indistinguishable from carcinoma
Intraluminal	– Bolus obstruction—gallstone ('gall stone ileus'), roundworms, trichobezoar, phytobezoar, food bolus – Polypoid tumor	– Fecal impaction – Polypoid tumor – Roundworms – Rarely gallstone, foreign body

(For causes of intestinal obstruction applicable to children, refer to the pediatric section.)

EXTRA-ABDOMINAL CAUSES to be considered leading to severe colicky pain mimicking that due to intestinal obstruction

Poisoning with lead

A blue line on the gums.

Acute intermittent porphyria

– Recurrent attacks of severe colicky pain in abdomen and neurologic and psychiatric abnormalities
– Both sensory and motor neuropathies occur, as may delirium, coma and seizure
– Attacks may be precipitated by barbiturates, anticonvulsants, alcohol, estrogens, contraceptives or prolonged fasting.

Confirmation of diagnosis—by demonstration of large amounts of urine porphobilinogens.

Abdominal tabes (Tabes dorsalis)

– Paroxysms of intense lightening pains
– Muscular in-coordination
– Disturbances of sensation
– Trophic disturbances especially of bones and joints.

DIFFERENTIAL DIAGNOSIS OF THE CAUSES OF ACUTE INTESTINAL (MECHANICAL) OBSTRUCTION

Intraperitoneal Adhesions → chronic adhesion syndromes or acute intestinal obstruction

Chronic adhesion syndromes: Chronic pain, or episodic subacute obstruction—refer to chapter 33

– *Acute intestinal obstruction* occurs almost always in the small intestine although the level of obstruction varies. Colonic obstruction due to adhesions is rare
– *Causes of intraperitoneal adhesions*: Abdominal surgery ('late postoperative adhesions'), peritonitis from any cause, sclerosing peritonitis (– usually drug-induced, e.g. certain β-blockers), inflammatory bowel disease, radiation enteritis.

Postoperative intestinal obstruction

– *Early (During the 1st 5 days)* obstruction may be caused by anastomotic edema, adhesive fibrinous matting, or kinking of intestinal loops. It is often incomplete
– *Obstruction occurring / persisting beyond 7 days of surgery* may be caused by organized bands or abscesses.

External hernia

Risk factors for development of complications in groin hernias

- In adults:
  – Old age
  – Short duration (< 1 year)
  – Femoral > oblique inguinal
  – Coexisting COAD
- In children:
  – Very young. Infants
  – Boys rather than girls

Free ebooks ==> www.Ebook777.com

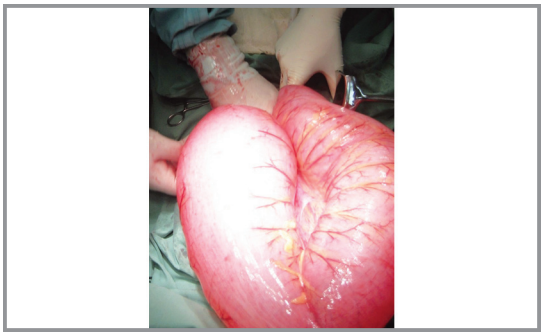


Fig 34-2 Appearance of acute volvulus of pelvic colon during surgery

  - Right side
- In general, any hernia with small neck and/or irreducibility is predisposed to obstruction.

Differentiating features of obstructed hernia

- Irreducibility
- Sudden onset of pain: Initially in the abdomen with features of acute intestinal obstruction. Subsequently, development of pain at the hernial site in addition to pain in the abdomen suggests onset of strangulation
- Tender, often tense swelling. No impulse-on-coughing
- In the absence of clinically demonstrable swelling, tenderness will be present at the least at the known hernial site.

Volvulus of pelvic (sigmoid) colon

Chronic form → intermittent lower abdominal colicky pain and distension, which are relieved by passage of flatus and loose stool

Acute form, basically presents as acute large intestinal obstruction. However, pain may not be typically colicky.

Differentiating points of acute volvulus

- Massive distension of abdomen
- Tympanic on percussion
- On DRE:
  - Rectum is empty (and maybe distended)
  - Blood on the finger-stall suggests gangrene

Supporting points in favor of acute volvulus:

- Middle aged / elderly
- Previous h/o chronic constipation, laxative dependency
- 'Severe' constipation (common in psychiatric patients).

Volvulus of cecum

Basically, it is a misnomer. Cecum alone is involved in only 10% cases of so-called cecal volvulus. Remaining 90% cases occur due to ileocolic torsion

- *Chronic* form presents with intermittent obstructive symptoms
- *Acute* form presents as acute distal small intestinal obstruction.

Volvulus of small intestine

- *Secondary* small intestinal volvulus develops due to rotation of a loop of small intestine around an adhesion, a band, an ileostomy or a colostomy
- *Primary* small intestinal volvulus results from malrotation of gut or congenital excessive mobility from loose fixation or long mesenteric attachments (volvulus of midgut in neonate).

Ileosigmoid knotting

- It is a type of acute volvulus in which knotting occurs between the loops of small intestine and sigmoid colon
- *Basically*, the patient presents with acute intestinal obstruction with shock, and rapid deterioration of

www.Ebook777.com

general condition.

Differentiating points

Profuse bilious vomiting and absolute constipation—both are prominent features without noticeable delay between the 2 symptoms.

Volvulus of stomach

Chronic gastric volvulus → chronic epigastric distress and vomiting.

Acute gastric volvulus → acute severe pain in abdomen.

Differentiating points of acute gastric volvulus

- Ineffectual retching
- Distension and tenderness in the epigastrium
- Shock follows rapidly.

Predisposing factors

- Stomach that has been pulled into diaphragmatic hernia / hiatal hernia / abdominal wall defect
- Large gastric tumor
- Connective tissue disorders.

Intramural pathologies of small/large intestine → chronic pain in abdomen and/or other clinical manifestations, rather than 'acute abdomen'.

Gallstone 'ileus'

Differentiating points

- Elderly
- H/o gallbladder disease
- Distal small intestinal obstruction (terminal ileum being the narrowest part of intestine).

Supporting point: The absence of more common causes of intestinal obstruction.

Variations in presentation

- H/o gallbladder disease may be absent
- Intestinal obstruction may occur at any level, including duodenum (as a result of cholecystoenteric fistula). Colonic obstruction can occur due to impaction of stone in the colon (reaching through cholecystocolic fistula).

Intestinal obstruction caused by roundworms

Differentiating points

A mass / masses (of worm bolus) may be palpable exhibiting the following characteristics. If abdomen is distended however, then these characteristics may not be appreciated clearly.

- Wriggling worms may be felt
- The mass changes its position around the umbilicus
- 5–10 cm in diameter
- Moderately tender
- Firm consistency.

Supporting points

- Fever. A characteristic toxemia
- Worms may be vomited or passed nasally during the episode of acute pain, or earlier.

A pitfall

- Vomiting of worms may suggest the diagnosis, but it is not pathognomonic of intestinal obstruction caused by roundworms.

Notes

Intestinal obstruction due to roundworms can occur in 4 ways: Refer to chapter 13.

Phytobezoars

Predisposing factors

- Ingestion of large amounts of high-fiber foods
- Inadequate mastication
- Previous gastric surgery causing hypo- or anacidity and loss of the antral pump mechanism.

Free ebooks ==> www.Ebook777.com

Intussusception (Refer to Fig 33-4)

Basically, acute mechanical small intestinal obstruction.

Differentiating points

– Sudden onset of vomiting
– Cramp-like pain.

Supporting points

– Rectal bleeding (⇒ strangulation)
– Abdominal mass (in ~ 60% of cases).

The condition is encountered most commonly in children. Refer to chapter 74.

Intestinal obstruction caused by intestinal tuberculosis

Exact preoperative clinical diagnosis of intestinal TB is difficult.

***Strangulating** obstruction*

The causes of mechanical obstruction, which are prone to progress to strangulation

– Hernia (both external and internal)
– Volvulus
– Intussusception
– Bands / adhesions.

Pathological basis of the clinical picture of strangulation is a combination of three elements.

1. Mechanical obturation of the lumen of the intestine.
2. Stagnation of blood in the affected loop of intestine, particularly if a large segment of intestine is involved leading to a decrease in blood volume which in turn leads to hypovolemic shock.
3. Peritonism followed later by peritonitis → septic shock.

Thus, there is an overlap of septic shock on hypovolemic shock.

Differentiating points **suggesting strangulation, in a suspected case of acute mechanical obstruction**

– Prostration
– Pain—continuous, diffuse, severe. *Continuous pain is the cardinal feature; it outweighs any lack of confirmatory evidence from physical signs or special investigations*
– Thirst
– Pallor
– Tachycardia
– Rigidity. **Exception—intussusception**. Reason—the strangulated loop lies inside the adjacent intestinal loop, thus preventing parietal peritonitis from occurring
– Tenderness
– Absence of peristaltic sounds—localized or generalized depending on the length of strangulated intestine, and duration of the illness.

Clinical data, which may give a clue to the probable cause of strangulating obstruction

– Age: 3 months to 3 years (although commonest is 3 to 9 months) ⇒ intussusception
– A k/c/o external hernia
– H/o previous abdominal operation ⇒ bands / adhesions
– '**Acute bleeding piles**' ⇒ internal strangulated hernia in paraduodenal fossa (acute obstruction of inferior mesenteric vein)
– **Tenderness at a hernial site even in the absence of a visible / palpable swelling, especially in an obese patient—could be due to strangulated hernia.**

BRIEF NOTES ON INTESTINAL OBSTRUCTION

Definition of intestinal obstruction: By convention, obstruction of the small or large intestine. The term does not include obstructive pathology of esophagus / stomach / proximal duodenum

In '**simple**' obstruction, blood supply of the obstructed intestine is intact

In '**strangulating**' obstruction, there is direct interference to the intestinal blood supply, most commonly caused by extrinsic compression

www.Ebook777.com

Acute obstruction:
- Mode of onset—sudden
- Clinical course (i.e. progress)—rapid
- Vomiting occurs early
- In general, caused by a small intestinal lesion, though it is not a rule

Chronic obstruction:
- Mode of onset—insidious
- Progressive constipation with late distension and vomiting
- In general, caused by a large intestinal lesion, though it is not a rule.

In practical terms, it is better to refer to small intestinal and large intestinal obstruction as such.
Large intestinal obstruction is more likely to require surgery, and carries much higher morbidity and mortality.
Subacute obstruction = incomplete obstruction of small / large intestine. → (Recurrent episodes of) abdominal colicky pain and distension, which are relieved by passage of liquid or semisolid stool
Intussusception = a segment of intestine invaginating itself into an adjacent segment
Volvulus = a twist of intestinal loop on its mesenteric pedicle
Closed loop obstruction = the obstructed segment (loop) is closed at both ends. Examples: 1. Vovulus. 2. Obstructed loop in a hernia. 3. Colonic obstruction in the presence of an intact ileocecal valve
Pseudo-obstruction = obstruction of small / large intestine in the absence of a mechanical cause or acute intra-abdominal disease.

Small intestinal pseudo-obstruction
- *Primary = idiopathic*. Familial hollow visceral myopathy, which is included in the primary category, is a particularly severe disorder which involves the smooth musculature of esophagus, entire GI tract including colon and often urinary bladder
- *Causes of secondary pseudo-obstruction*: Drug misuse (e.g. laxative abuse), certain systemic disorders e.g. DM, hypothyroidism, scleroderma, progressive systemic sclerosis, acute intermittent porphyria, Chaga's disease
- Clinical features of small intestinal pseudo-obstruction: Recurrent episodes of subacute obstruction.

Colonic pseudo-obstruction has 2 forms—acute and chronic.
Chronic: Could be primary, or secondary. Causes of secondary pseudo-obstruction: Motility disorder, DM, hypothyroidism, malignancy, psychosis, drug and laxative abuse
Acute colonic pseudo-obstruction = selective distension of the cecum and proximal colon with a sharp cut-off usually at the splenic flexure, and less frequently hepatic flexure and sigmoid (on a plain X-ray)
It is best considered as a localized form of adynamic ileus
Predisposing background: Pre-existing non-intestinal conditions requiring hospitalization e.g. severe trauma particularly to the spine, major surgery, sepsis, MI, severe renal disease, severe respiratory disease, widespread malignancy
Symptoms mimic acute large intestinal obstruction. Dominating feature is progressive abdominal distension. Constipation is not absolute. Patient will continue to pass small amount of flatus / liquid stool
Signs:
- Massive abdominal distension
- Tenderness in RLQ suggests compromised viability of cecum
- Peristaltic sounds—variable—either normal, or hyperperistaltic.

PARTICULAR PROBLEMS with a case of acute intestinal obstruction

1. **Small intestinal obstruction in a patient without an external hernia and without previous laparotomy**
   In this situation, there is a place for conservative measures in the first instance, but if definite improvement is not apparent at each reassessment there should be no hesitation in advising surgical treatment.
2. **Small intestinal subacute obstruction**
   Unlike large intestinal obstruction, which is usually insidious in onset and gradual in progress, small intestinal obstruction generally starts suddenly. Should the onset in fact be insidious, perhaps with short

Free ebooks ==> www.Ebook777.com

periods of colicky pain and nausea interspersed with symptomless intervals, the unwary clinician may persist with conservative measures for too long. Possible serious causes in such clinical situation could be gallstone ileus, or mesenteric vascular occlusion.

ACUTE INFLAMMATION

Differentiating features in general

- Duration—less than a week. However, prolongation of acute pain for a period of ~ 2 weeks or even more is possible
- Anorexia, nausea and a few bouts of vomiting. Vomiting is non-descript
- Fever
- Hyperesthesia ±
- Guarding. Sometimes, rigidity
- Lump ±
- Diminished peristaltic sounds in the area / quadrant affected.

Diagnostic points of interest

- Which organ / tissue / space seems to have been inflamed?
- Specific pathology? What is the stage in the natural history of the (untreated) disease? What is the cause—pathogenic agent?
- The degree of homeostatic imbalance?

The site of pain	*Organ / tissue / space*	*Pathology*
RUQ / epigastrium	Biliary tract Liver Pancreas Stomach Duodenum Subphrenic space Maldescended appendix	Cholecystitis, cholangitis Hepatitis, pyelephlebitis, abscess Rare causes: Infected cyst in the liver, Caroli's syndrome Pancreatitis Perigastric abscess Acute exacerbation of DU Subphrenic abscess Appendicitis
LUQ / epigastrium	Stomach Spleen Gallbladder Subphrenic space Colon	Acute phlegmonous gastritis Perisplenitis, splenic abscess Cholecystitis Subphrenic abscess Colitis
RLQ / suprapubic	Appendix Meckel's diverticulum Cecum Small intestine Lymph nodes Uterine tube Kidney Prostate	Appendicitis Meckel's diverticulitis Amebic typhlitis, typhoid typhlitis Regional enteritis Mesenteric lymphadenitis (ANIML), iliac lymphadenitis Salpingitis Pyelitis, perinephric abscess Prostatitis
LLQ / suprapubic	Colon Uterine tube Lymph nodes Kidney Prostate	Diverticulitis, colitis Salpingitis Iliac lymphadenitis Pyelitis, perinephric abscess Prostatitis
All over the abdomen	Colon Retroperitoneum	Severe form of ulcerative colitis Acute retroperitoneal lymphangitis

RUQ / epigastrium

www.Ebook777.com

Interpretation of important symptoms and signs

If there have been multiple episodes

– Complete absence of symptoms during pain-free intervals ⇒ peptic ulcer—acute exacerbation of DU
– The presence of a mild discomfort / dyspepsia even during the so-called pain-free intervals ⇒ acute cholecystitis
– If etiological factor does not seem to have been eradicated (on referring to the previous medical record), then think of specifically liver / pancreatic disease (though the same principle is applicable to other disorders as well, in general).

Pain

– Associated pain
  - In shoulder ⇒ subphrenic abscess / liver abscess / acute cholecystitis
  - In the mid-back—rhythmic, decreasing after food ⇒ penetrating peptic ulcer (acute exacerbation of DU)
  - In the mid-back—persistent ⇒ acute pancreatitis
– Radiating to right infrascapular region / shoulder ⇒ acute cholecystitis
– 'Pain reaching maximally severe intensity almost at the onset' ⇒ acute pancreatitis
– Burning pain ⇒ acute exacerbation of DU
– **'Ulcer-like' pain may be due to (apart from peptic ulcer,) recurrent pancreatitis**
– Throbbing pain ⇒ acute pyogenic abscess
– Pain relieved by assuming sitting position ⇒ pancreatitis.

Fever

– Hectic fever ⇒ abscess
– Fever with rigors ⇒ basically septicemia. Examples—pyelephlebitis, cholangitis

Progress

Very rapid progress ⇒ acute pancreatitis / acute phlegmonous gastritis / acute emphysematous cholecystitis.

Icterus ⇒ hepatic / biliary / pancreatic disease.

Fullness *(bulging) of intercostals spaces (middle / lower, on lateral aspect)* ⇒ a large hepatic abscess / subphrenic abscess.

Tenderness at

– The duodenal point ⇒ acute exacerbation of DU
– The Murphy's point ± Boas' point ⇒ acute cholecystitis
– The phrenic point ⇒ subphrenic abscess.

Lump could suggest phlegmonous cholecystitis / empyema of gallbladder / perigastric abscess.

Organomegaly

– Tender hepatomegaly ⇒ acute hepatitis / hepatic abscess / pyelephlebitis
– Non-tender 'hepatomegaly' (Non-tender liver palpable per abdomen being mistaken as hepatomegaly) may be due to a subphrenic abscess, which pushes the liver down
– Acute cholecystitis.

Acute (obstructive usually calculous**) cholecystitis**

Basically, acute inflammatory abdomen involving the RUQ.

Differentiating points:

– Pain radiating to right infrascapular region
– Tenderness at Murphy's point ± Boas' point
– Murphy's sign positive.

Supporting points:

– Recurrent attacks
– Jaundice (in ~ 25% cases). Cause of jaundice could be one of the following: Ductal stones (in only 12% of these cases) / reactive hepatitis / edema of the CBD.

Additional features which may support the diagnosis:

– Belching
– Dyspepsia
– Tender palpable mass (in ~ 25% cases). Cause of the mass could be one / more of the following: Omental phlegmon / empyema of gallbladder / abscess due to localized perforation / carcinoma of gallbladder, especially if the patient is elderly

Free ebooks ==> www.Ebook777.com

– Segmental spasm of the right rectus muscle may be mistaken for a gallbladder lump!

Atypical features:

– Pain may start in the LUQ, but at its most severe it is invariably on the right side
– Pain may radiate to the chest.

Notes on acute obstructive calculous cholecystitis:

– Known pre-existing gallbladder disease may be present or chronic symptoms over several months to years may precede the acute presentation. Alternatively, acute obstructive cholecystitis may be the first intimation of gallstone disease
– The presence of only pain and tenderness ⇒ mild cholecystitis
– Severe pain, fever, toxicity, (mild) abdominal distension, in addition to tenderness and guarding / rigidity ⇒ severe cholecystitis.

Acute acalculous cholecystitis

– Usually occurs in critically ill, elderly patients, but it can also occur in children
– Predisposing factor—(prolonged) serious illness; examples—depletion of blood volume, prolonged paralytic ileus, sepsis, starvation, multiple BTFs, morphine administration exceeding 6 days, intravenous hyperalimentation, etc.
– However, the condition may occur *de novo*, without the presence of any critical illness in the background
– Diagnosis is often difficult , especially in critically ill patients.

Acute (bacterial) **cholangitis**

Basically, acute inflammatory abdomen involving the RUQ

Differentiating points

– Intermittent high grade fever with (severe) rigors
– Jaundice
– Tender hepatomegaly, however, may be difficult to detect it due to tenderness and guarding.

Supporting point: Known to have a possible underlying cause, e.g. stone disease, instrumentation of the biliary tract, etc.

Notes

– Classical triad of pain-fever-jaundice is present in about 70% of cases
– Intensity of pain may be variable
– Marked tenderness ⇒ abscess formation (honeycomb liver)
– Hypotension ⇒ severe cholangitis, when renal failure is usually present
– Acute bacterial cholangitis is a serious life-threatening emergency caused by infection of an obstructed biliary tract
– *Causes*
  - Occluding stone in the CBD is the most common cause
  - Bile duct stricture
  - Tumor of the bile duct, head of the pancreas, periampullary tumor
  - Following instrumentation of the biliary tract e.g. following ERCP, PTC
  - Risk factors for the development of cholangitis following ERCP are the presence of fever before the procedure, and malignant biliary obstruction.
  - Parasitic infestations of the biliary tract. Example—round worm (Ascaris. lumbricoides)
  - Bilioenteric anastomosis, spontaneous bilioenteric fistula
  - Cystic disease of the biliary tract
  - Duodenal diverticula.

(Recurrent) Pyogenic cholangitis

Recurrent episodes of cholangitis
Debilitated patient
The disease may be further complicated by formation of hepatic abscess

www.Ebook777.com

Prevalent in South-East Asia.

Acute hepatitis

Basically, acute inflammatory abdomen involving the RUQ.

Differentiating points

– Dark-colored urine
– Tender hepatomegaly.

Supporting points

– Icterus
– Anorexia and nausea
– Pruritus, pale stools.

Causes of acute hepatitis: Refer to chapter 37.

Acute pylephlebitis

= septic inflammation of the wall of the portal vein or its major radicals.

Differentiating points

– Background of primary focus of sepsis like acute appendicitis, inflamed piles, etc.
– High-grade fever with rigors
– Tender hepatomegaly.

Acute pyogenic liver abscess(es)

Since most pyogenic abscesses are secondary to other infective processes, the clinical features may be dominated by the primary disorder.

Basically, acute inflammatory abdomen involving the RUQ, along with the following manifestations:

– Hectic fever, rigors, profuse sweating
– Anorexia, icterus
– SOB, pleuritic type chest pain, cough ±
– Fullness with pitting edema of IC spaces (in right lobe abscesses)
– Tender hepatomegaly.

Additional finding: Signs in lower lobes of the lungs.

Pitfalls

– Mode of onset is often subacute
– Symptoms may have been present for many weeks
– Pain may develop relatively late. Pain may not be necessarily localized to the RUQ
– Fever is the most frequent and consistent symptom
– Diaphragmatic involvement may result in pleuritic type chest pain, cough and SOB
– Diagnosis is often delayed for several days after admission
– Other ways of presentation: 'Sepsis', peritonitis due to rupture.

Notes

– Usually multiple
– The etiology is unexplained in majority of patients
– Increased incidence in elderly, diabetics and immunosuppressed
– In elderly, pyogenic liver abscess is commoner in females and the symptoms may be vague.

Causes

1. The commonest cause is obstruction of the biliary tract.
2. From primary focus of infection / infestation in the GIT. – Infection is carried along the portal vein.
3. Secondary to infection of hydatid cyst, septicemia / pyemia or faciocervical actinomycosis – Infection is carried along the hepatic artery.
4. From primary focus of infection in the umbilicus.
5. Direct extension of infection from a penetrating wound, subdiaphragmatic abscess, or empyema thoracis.

Amebic liver abscess (usually single)

Basically, acute / subacute inflammatory abdomen involving the RUQ, along with the following manifestations:

Free ebooks ==> www.Ebook777.com

- Low-grade fever, sweating
- Anorexia
- Icterus ±
- Fullness with pitting edema of IC spaces (in right lobe abscesses).

Differentiating points

- Progressively increasing pain in right hypochondrium and lower part of chest
- Continuous dull-aching. Aggravated by movements
- Tender hepatomegaly—varying from 2–15 cm below the costal margin.

Supporting points

- Diarrhea (in 50% of cases)
- Non-productive cough ⇒ abscess must have reached undersurface of the diaphragm
- Patient learns to support his enlarged liver with his hands while walking
- Patient residing in endemic area for amebic disease.

Predisposing factors

- Male gender
- Malnutrition
- Excessive alcohol consumption.

Pyogenic liver abscess	*v / s*	*Amoebic liver abscess*
– The clinical features may be dominated by the primary disorder		Occasionally, mode of onset may be insidious taking about 2 weeks or more. In such patients, significant weight-loss may occur
– Pain—not as common		Progressively increasing pain
– Fever—hectic		Fever—low-grade
– Jaundice—more common		Jaundice—less common

Acute pancreatitis

Basically, acute inflammatory abdomen involving the RUQ / epigastrium.

Differentiating points:

- Associated persistent pain in middle of the back
- Pain in the abdomen reaches maximally severe intensity almost at the onset
- Pain relieved by assuming sitting position
- Progress—very rapid
- Prostration.

Supporting points:

- Onset following excessive intake of food / alcohol
- Marked retching and troublesome hiccups. Retching may persist despite the stomach being kept empty by nasogastric aspiration
- Profound shock in severe cases; in fact patient may present with shock. Cause of collapse: Absorption of incompletely split products of proteins
- Abdominal distension—due to localized / diffuse paralytic ileus, or rarely due to ascites. (Shifting dullness ⇒ ascites.)
- Later in the course:
  - Epigastric mass forms which suggests pseudocyst or marked peripancreatic fat necrosis
  - Bluish discoloration of flanks / umbilicus occurs due to bleeding into fascial planes
- Rare manifestations:
  - Small red tender nodules on the skin—due to nodular fat necrosis in the skin
  - Polyarthritis / bone pains—due to fat necrosis
  - Thrombophlebitis of the leg veins.

www.Ebook777.com

– Obvious presence of predisposing clinical setting: alcoholism, gallstone disease, trauma, ERCP, unexplained shock following abdominal surgery, malnutrition, scorpion bite, etc.

Additional features which may be present:

– Icterus
– Diarrhea, hematemesis, melena
– Dyspnea, cyanosis—due to diminished excursion of diaphragm resulting from inflamed pancreas
– Abnormal chest signs—due to basal atelectasis / pneumonitis / left-sided or bilateral pleural effusion.

Remarks / pitfalls

1. Site of pain can be variable. It can occur or radiate anywhere in the abdomen.
2. Degree of pain may vary from mild to severe. Occasionally, even the severe fulminant fatal pancreatitis may be totally painless.
3. Physical findings may be minimal in striking contrast to the severity of symptoms. Patient may look well on one end or, gravely sick at the other extreme.
4. Toxic exudate might spread in the layers of transverse mesocolon → spasm of transverse colon → clinical picture of intestinal obstruction.
5. Rare causes of acute pancreatitis: Mumps, hyperparathyroidism, vascular disease, etc.
6. In certain percentage of patients cause may remain obscure ('idiopathic pancreatitis').

There are no pathognomonic symptoms and signs of pancreatitis, but pain is usually the cardinal symptom. *The diagnosis of acute pancreatitis is based on 'thinking about it as a possibility' and excluding other sources of pain in abdomen.*

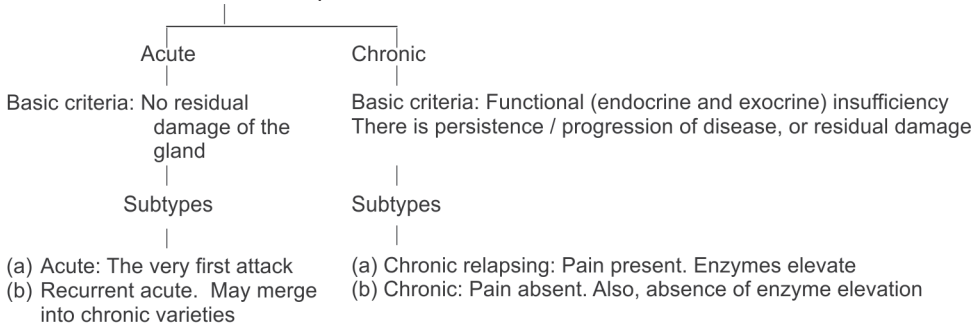
DD of acute pancreatitis

The clinical picture of acute pancreatitis tends to change with evolution of the disease, and consequently the differential diagnosis also tends to change with time.

– **2–3 hours** after the onset: Acute cholecystitis—indeed, acute cholecystitis may coexist with acute pancreatitis.
– **After 6–8 hours**: Perforated DU, acute appendicitis
– **After 2–3 days**: Intestinal obstruction (due to marked abdominal distension and ileus).
– **If patient presents with profound cardiovascular collapse**: MI / acute aortic dissection / ruptured aortic aneurysm / acute mesenteric ischemia / massive PE.

Notes

1. Inflammation and **autodigestion** of the pancreas is defined as pancreatitis.
2. *Basic clinical classification* of pancreatitis



An acute pancreatitis may be further classified according to severity into

a. Acute edematous = mild and self-limiting.
b. Acute fulminant—hemorrhagic / necrotizing / suppurative.
c. Acute persistent = subacute pancreatitis: Unresolving attack. Suspect development of complication.

– Edematous form can progress to fulminant form

Free ebooks ==> www.Ebook777.com

- Distinction, basically between edematous and fulminant, **may be impossible at the onset** of the disease (on the basis of clinical features and conventional laboratory investigations)
- A few scoring systems are in vogue for objective determination of the severity of acute pancreatitis such as Ranson, Glasgow, APACHE II.

3. Acute pancreatitis can mimic any other abdominal emergency. In fact, it can coexist with some such as acute cholecystitis.
4. It can occur at any age, but is rare in children and very young adults. In very young adults, it is usually associated with trauma, infections, parasites, drugs, or is hereditary.
5. In young adults < 40, alcohol is the most common cause. Alcoholic pancreatitis has a tendency to recur. First attack is usually severe and can prove to be fatal. Subsequent attacks tend to be milder. Eventually merges with chronic pancreatitis.
6. In middle-aged and older patients, biliary tract disease must be considered as the most probable etiological factor. 'Gallstone pancreatitis' tends to be severe. Frequently accompanied by serious complication in acute stage. Recurrence may be prevented by appropriate treatment of gallstones.
7. After recovery from one/more attack/s of acute pancreatitis, the case must be investigated to identify, and if possible, to eliminate the etiological factor. The need to identify surgically remediable problem, most commonly gallstones, is obvious.
8. **Complications** of acute pancreatitis

   A. *Acute / subacute and potentially life-threatening*
   - Shock
   - Pulmonary insufficiency
   - Pancreatic abscess(es) due to secondary infection of necrotic tissue in the pancreatic bed (i.e. infected pancreatic / peripancreatic necrosis). Presents 1 or 2 weeks after the initial attack has subsided
   - Bleeding resulting from pseudoaneurysm / s or sectorial portal HT
   - Hypocalcemia.

   B. *Other*
   - Pseudocyst formation
   - Biliary tract obstruction
   - Colonic stricture formation.

Acute perigastric abscess

Basically, acute inflammatory abdomen involving the RUQ / epigastrium, with a localized tender mass with cellulitis.

Supporting points:
- Hectic fever
- H/s/o peptic ulcer disease.

Acute exacerbation of (a chronic) **DU**

Basically, acute inflammatory abdomen involving the RUQ / epigastrium.

Differentiating points
- Burning pain
- A k/c/o peptic (duodenal) ulcer
- Tenderness at the duodenal point.

Supporting point: Associated rhythmic pain in the mid-back.

LUQ / epigastrium

Acute phlegmonous gastritis

Basically, acute inflammatory abdomen involving the LUQ / epigastrium, leading to severe progressive peritonitis.

www.Ebook777.com

Supporting points:
– Pre-exisiting gastric lesion
– Elderly and alcoholic patient.
It is usually an agonal event.

Acute perisplenitis
= capsular inflammatory irritation caused by splenic infarction.
Basically, acute inflammatory abdomen involving the LUQ, with fever, chills and malaise.
Supporting points
– Continuous pain
– Rubbing type of auscultatory sounds.
Additional features which may be present: Pain referred to the left shoulder, and left pleural effusion.
Predisposing factors
- Sickle cell disease
- Splenomegaly due to a congestive disorder such as CML or myelosclerosis
- Thromboembolic disorders such as AF, arterial embolic (atheromatous) disease, diabetes-associated microvascular disease, acute torsion of an ectopic (wandering) spleen
- Infection / infestation such as falciparum malaria, Q-fever, AIDS
- Severe necrotizing pancreatitis, pseudocyst of pancreas.

Clinical presentation may be variable
– Acute symptoms and signs may be severe, and acute life-threatening complication may develop like splenic rupture, abscess
OR – Symptoms may be minor
OR – The condition may be asymptomatic.

Splenic abscess
Basically, acute inflammatory abdomen involving the LUQ, with fever.
Differentiating point: Tender splenomegaly (50%).
Predisposing disorders
- Infection in the adjacent viscera e.g. diverticulitis
- Infection in remote areas causing metastatic abscess in the spleen, e.g. typhoid and paratyphoid fever, osteomyelitis, otitis media, puerperal sepsis, etc.
- Infarcted spleen
- Post-traumatic splenic hematoma.

Remarks
– In many cases no cause is detected
– Clinical features of splenic abscess are essentially nonspecific.

CONDITIONS COMMON TO BOTH UPPER QUADRANTS—subphrenic abscess, acute pyelonephritis.

Subphrenic abscess
Basically, acute inflammatory abdomen involving the RUQ / LUQ, with
– Hectic fever
– Non-specific abdominal symptoms and general ill-health
– Respiratory symptoms ±.
Differentiating points
– The presence of a predisposing factor, e.g.
  - Operative intervention with postoperative leak
  - Primary infective / inflammatory focus like perforated peptic ulcer, pancreatitis, cholecystitis, appendicitis, PID, etc.
  - Abdominal trauma

Free ebooks ==> www.Ebook777.com

- Anorexia and wasting (loss of weight)
- Fullness with pitting edema of the IC spaces
- Tenderness overlying 8th–11th ribs. In c/o subhepatic abscess—tenderness under the costal margin.

Supporting points

- Pain in the shoulder
- Tenderness at phrenic point
- Palpable (NOT palpably enlarged!) liver
- In c/o postlaparotomy status: No evidence of wound sepsis and rectal examination negative.

Additional features

- Signs in the lower lobe of the lung
- Pleural effusion / empyema.

Notes

- **Thoracic signs are more useful than abdominal ones**
- When it develops after a laparotomy to deal with an infective pathology, condition of the patient initially improves temporarily, but after an interval of a few days or weeks (often between 2nd and 3rd week post-laparotomy) symptoms of toxemia reappear. The condition deteriorates steadily and often rapidly
- Swinging fever may be absent if the patient has been receiving antibiotics or steroid
- Liver may not be palpable if fixed due to adhesions.

Acute pyelonephritis

Basically, acute inflammatory abdomen involving the flank and hypochondrium.

In a few cases, pain may simulate ureteric colic.

Differentiating points

- Female patient especially during childhood, at puberty, after intercourse, during pregnancy, or during menopause
- Prodromal symptoms—headache, lassitude and nausea
- Sudden mode of onset of pain and rigor and vomiting
- Fever—102–103°F, remitting
- Cystitis (frequency, urgency and scalding pain during micturition)
- Tenderness in hypochondrium and loin.

In the early stages, when it is imperative to make a correct diagnosis, the urine is clear macroscopically.

Severe pyelonephritis

- Repeated rigors and fever ≥ 104°F, *often without a corresponding rise in pulse rate*
- Vomiting, sweating and thirst
- Patient feels awful
- Enlarged kidney may become palpable after abdominal guarding has subsided
- Urine—scanty and highly concentrated.

In blood-borne infections the organisms and pus cells do not appear in the urine until the infection has spread from cortex to medulla—a matter of hours, or even days.

Severe bilateral pyelonephritis

In addition to the clinical features listed above, there are also likely to be manifestations of uremia.

Pyelonephritis of pregnancy

- Usually occurs between 4th and 6th months of gestation in women who have a past h/o recurrent UTI.
- In 90% of cases, right side alone is affected
- In some patients it runs a severe and protracted course and occasionally → abortion / premature delivery.

Periodic examinations of urine are necessary during the rest of the pregnancy, the puerperium and for several months afterwards because if the infection has not been eradicated, recrudescence is liable to occur.

www.Ebook777.com

Pyelonephritis associated with chronic urinary retention

Causes

1. Introduction of organisms during catheterization / instrumentation if aseptic precautions were not observed, or operation on the lower urinary tract.
2. May arise spontaneously.

In most cases, it is bilateral.

Notes on acute pyelonephritis

– Usually, but not necessarily always, it is an acute ascending pyogenic infection usually associated with lower UTI (obstruction and/or vesicoureteric reflux often being present)
– Large numbers of bacteria are found in the urine, several hundred thousand per ml in the acute phase
– Natural history: Healing / perinephric abscess / papillary necrosis / recurrence / chronic pyelonephritis.

RLQ / suprapubic

Inference from pain and tenderness

Pain

– Migrating from umbilical region → RLQ ⇒ appendicits / Meckel's diverticulitis
– Jolting or running precipitating the attack/s of pain ⇒ appendicitis.

Tenderness

– Localized at McBurney's point ⇒ appendicitis / Meckel's diverticulitis
– A little medial to McBurney's point ⇒ - Meckel's diveriticulitis / ANIML, particularly in children
  - Regional enteritis in adults
– Much more medially on either side and also in vaginal fornix ⇒ salpingitis
– In renal angle ⇒ pyelitis / perinephric abscess
– A little below the renal angle ⇒ retrocecal appendicitis.

Acute appendicitis

Basically, acute inflammatory abdomen involving the RLQ.

Differentiating points

– Pain migrating from umbilical (rarely epigastric (unexplainable)) region to RLQ
– Patient moves with caution. Finds it easier to stoop forwards. And while lying in bed, she/he is more comfortable with the right lower limb flexed
– Tenderness at McBurney's point
– Rovsing's sign positive.

Supporting points

– Pain precipitated by jolting / running / traveling on a bumpy uneven road
– Previous similar attack/s.

A pitfall

Pain may not shift to the RLQ. This fact may suggest retrocecal / retroileal / pelvic appendicitis. Explanation: Inflamed appendix in such positions cannot come in contact with parietal peritoneum to produce pain.

Notes

1. Acute appendicitis is essentially a clinical diagnosis and there is no laboratory / radiological investigation which is diagnostic of the condition.
2. Appendicitis is the commonest abdominal surgical emergency, and also the one with the most variable symptoms. The most important factor responsible for variability of symptoms is variability in the position of the appendix. Position means position of the body and tip. Position of the base is always constant.
3. Colicky pain (intitially felt in umbilical region) suggests obstructive appendicitis.
4. Anorexia, nausea, vomiting: These are successive gradations of the same mechanism viz. protective pylorospasm.
   Vomiting is of short duration, i.e. up to about 3/4 episodes and stops as soon as the stomach gets empty.
5. Fever is always mild, i.e. up to 101°F; however, fever may be high in children with appendicitis.
   A moderate / high-grade fever within 24 hours of the onset of pain virtually rules out the possibility of acute

Free ebooks ==> www.Ebook777.com

appendicitis.

Hectic, i.e. swinging fever later in the course of the disease, i.e. after about 3 days or so suggests the development of appendicular abscess.

6. Pulse rate is usually normal in 1st 6 hours. Later, it may rise commensurate with fever. Increasing pulse rate suggests progress towards complication.
7. The diagnosis of appendicular lump should be considered in a patient who presents 48 hours after the commencement of acute appendicitis even if the lump may not be palpable due the presence of guarding / rigidity.
8. In general, comparatively slow progress and 'mild' symptoms and signs may suggest 'catarrhal' appendicitis, whereas sudden mode of onset with colicky pain, 'severe' symptoms and signs, and rapid progress suggests 'obstructive' appendicitis.
9. In a c/o appendicitis
   - If patient develops hectic fever after about 3 days or so, then suspect appendicular abscess
   - If patient develops a high-grade fever and rigors and pain and tenderness in the RUQ, then suspect pyelephlebitis
   - If patient frequently asks for a bedpan, then suspect a pelvic abscess.

Special C/Ms depending on position of the appendix, age of the patient, obesity, and pregnancy:

***Retrocecal** appendicitis*

- Pain referred to the testicle and perhaps hematuria ⇒ inflamed appendix lying on the ureter
- Deep tenderness is experienced in the loin rather than in the RLQ, as cecum prevents the pressure exerted by the hand from reaching the inflamed appendix
- Fixed flexion at the hip ⇒ psoas spasm induced by inflamed appendix lying on the muscle. Hyperextension of the thigh → tenderness
- Rigidity in the RLQ is absent. ∴ An inflamed retrocecal appendix is called a 'silent appendix'.

Deep tenderness in the loin and urinary symptoms when present, makes it necessary to differentiate a retrocecal appendicitis from an acute renal disease. Clinically, positive Rovsing's sign suggests appendicitis.

***Pelvic** appendicitis* → symptoms due to irritation of bladder and rectum; however, in women bladder symptoms may be absent due to interposition of the uterus.

- Suprapubic discomfort
- Increased frequency of micturition, strangury
- Mucus in stools, diarrhea, tenesmus
- Deep tenderness just above and to the right of the symphysis pubis
- Tenderness on DRE
- Positive obturator test is suggestive.

Postileal (*syn.* retroileal) appendicitis accounts for the cases of 'missed appendix'.

- This poses the greatest difficulty in diagnosis as pain will not shift
- Diarrhea may occur due to irritation of small intestine
- Tenderness, if any, is ill-defined; may be present immediately to the right of the umbilicus.

***Maldescended** appendicitis*

Pain and tenderness in subhepatic region may lead to the mistaken diagnosis of cholecystitis.

In children

- Diagnosis may be difficult due to various reasons. Reliable history may not be available.
- Children react with pain in abdomen, to many non-surgical acute illnesses; for example acute enteritis, acute RTI, etc. As a matter of fact, acute appendicitis may be actually associated with these illnesses. Refer to chapter 73.
- If the diagnosis is in doubt, the best policy is to operate rather than to wait. However, in practice, mortality remains high especially in infants, mainly due to delay in arriving at the diagnosis.

In aged, i.e. above 75

- Temperature and pulse are often normal, and abdominal signs are 'mild'

www.Ebook777.com

– Lax abdominal wall fails to develop adequate rigidity even in the presence of gangrene, basically because of lack of energy / power due to old age
– Gangrene and perforation occur more frequently due to arteriosclerotic appendicular artery.

***In obese** patients*

Local signs may be diminished / totally obscured by obesity. If the possibility of acute appendicitis is thought of based on symptomatology, then it is better to operate relying only on symptoms.

During pregnancy

As enlarging uterus goes on pushing the appendix, the site of pain tends to be much higher and lateral.
The most important DD in pregnancy is acute pyelonephritis. Theoretically, it is discussed that urine—R and M examination may help to differentiate between the two. However, one should remember that a retrocecal appendicitis lying on the ureter might also give hematuria and pyuria. ∴ The best policy, when the diagnosis is in doubt, is to operate. Complicated appendicitis tends to induce abortion / premature labor.

DD of acute appendicitis as applicable most commonly in practice:

1. **Meckel's acute diverticulitis:** Symptom complex of Meckel's acute diverticulitis is very similar to that of acute appendicitis. Meckel's diverticulum is a rarity in itself, and to get it acutely inflamed is a further rare event.
2. **Amebic acute typhlitis:** In a real difficult situation to differentiate between the two, a therapeutic trial for amoebic typhlitis (with an appropriate injectable antiamoebic) is worth considering. Complete disappearance of symptoms and signs within an hour or two suggests the diagnosis of amoebic typhlitis.
3. **ANIML:** Patient seldom looks sick. Shifting tenderness is an important sign. If a doubt exists, it is better to operate. Some people believe that appendicectomy helps resolution of the condition.
4. **A 'leaking' DU:** The initial pain in epigastrium due to 'a slow' perforation of 'a small' peptic ulcer may mimic the initial visceral pain of acute appendicitis. Followed by sealing of small perforation, the irritant duodenal juices gravitating to the RLQ → continuous pain. Thus, on the whole the pain that shifts from the central region to the RLQ may lead to a mistaken diagnosis of acute appendicitis.
5. **In children—Viral gastroenteritis and ANIML:** Acute gastroenteritis usually → a generalized colicky pain that never becomes continuous and localized, but the associated vigorous vomiting or diarrhea may result in muscle tenderness that may happen to be worse on one side than the other.
6. **In adolescent girls and premenopausal women**, many of the acute pathological conditions pertaining to the internal genital organs and an occasional physiological condition like **mid-cycle pain** can mimic acute appendicitis, e.g. **acute salpingitis, TO abscess, twisted ovarian cyst, (subacute) rupture of ectopic pregnancy, etc**.
7. **In aged—a perforated cecal CA**, **obstructing CA of the distal colon.**
   There are a host of other diseases, which can mimic acute appendicitis, and it may not be possible to correctly diagnose them clinically preoperatively; for example diverticulitis, actinomycosis, regional enteritis and many more. Often in such a situation, the patient's abdomen is opened with an intention of appendicectomy. Sometimes during the operation and at other times only after the operation the true cause responsible for the pain becomes evident.

Appendicular abscess

Basically, acute inflammatory abdomen involving the RLQ / suprapubic region, with hectic fever, nonspecific abdominal symptoms and general ill-health.

Differentiating points

– Primarily, a c/o acute appendicitis
– **The mass will stop short of iliac crest / inguinal ligament by about a fingerbreadth**
– Fixed flexion at the hip if present will be mild

Supporting point: Tenderness, and guarding or rigidity

Note: Swinging fever may be absent if the patient has been receiving antibiotics or steroid.

Meckel's acute diverticulitis

Basically, acute inflammatory abdomen involving the RLQ

Free ebooks ==> www.Ebook777.com

Differentiating point: Pain persists in the central abdominal area without migrating to the RLQ
Supporting points:
– Previous similar attack / s
– Intermittent lower GI bleeding

Remark: Usually occurs in children and frequently a diagnosis of acute appendicitis is made.

Amebic acute typhlitis

Basically, acute inflammatory abdomen involving the RLQ
Differentiating point: Diffuse tenderness in the RLQ
Supporting point: Diarrhea.

Typhoid acute typhlitis

Basically, acute inflammatory abdomen involving the RLQ.
Differentiating points
– Remittent fever
– Diffuse tenderness in the RLQ
– Relative bradycardia.

Supporting point: Diarrhea.

Regional enteritis (Terminal ileitis)

Basically, acute inflammatory abdomen involving the RLQ
Differentiating points: Antecedent h/o abdominal cramping, diarrhea and weight loss
Supporting points:
– Diarrhea
– A doughy mass of inflamed ileum.

Notes:
– Relatively rare condition
– Resembles acute appendicitis except that it commences with diarrhea.

Acute nonspecific ileocecal mesenteric lymphadenitis (ANIML)

Basically, acute inflammatory abdomen involving the RLQ.
Differentiating points
– Child / a teenage girl
– General manifestations of viral infection: Headache, generalized body aches, running nose, periorbital puffiness, slight conjunctivitis, moderate lymphadenitis (cervical / axillary / inguinal)
– Fever ±, up to 104° F usually returning to normal within 48 hours
– Short attacks of central abdominal pain lasting from 10–30 minutes, associated with circumoral pallor. The attacks tend to come on when the patient is tired
– Despite pain and fever, the patient is often hungry
– Vomiting ±
– Patient does not look sick.
– Tenderness is greatest along the line of the mesentery
– 'Shifting tenderness'
– Self-limiting course of the illness – 3 to 4 days.

Note: Enlargement and even suppuration of the involved lymph nodes may occur. Organisms responsible may be yersinia, campylobacter, viruses, etc.

Acute tuberculous mesenteric lymphadenitis

Basically, acute inflammatory abdomen involving the RLQ
Clinically, it may not be possible to differentiate from acute appendicitis.

PARTICULAR PROBLEM

Only pain in the RLQ. NO positive sign including tenderness.

These patients are treated conservatively. Signs may develop later, the condition may become worse, and

www.Ebook777.com

operation may become necessarily. OR, they improve and are cured without ever knowing the final diagnosis.

LLQ / suprapubic

(Colonic) Acute diverticulitis

Basically, acute inflammatory abdomen involving the LLQ (± RLQ).

Differentiating points

– Age > 40
– Fever and malaise
– Sigmoid colon palpable, tender and thickened
– DRE may reveal tender mass.

Additional features:

– Diarrhea / constipation
– Tenderness may **also** be present on the right side—if the pelvic colon loop lies across the midline.

Notes:

– It is a rare condition
– It closely resembles acute appendicitis on the left side.

Painful diverticulosis

– Acute severe pain in the LLQ—due to excessive colonic segmentation
– Flatulence, distension and sensation of heaviness in the lower abdomen
– **NO** fever and malaise *cf* in acute diverticulitis.

PARTICULAR PROBLEM

Pain of obscure nature in the LLQ, i.e. when definite clinical diagnosis could not be made, and symptoms and signs gradually subsided.

The usual outcome in such a clinical scenario: The patient is investigated (by sigmoidoscopy / colonoscopy / Ba enema, etc.)

↓

A. If investigations yield negative result, the patient is reviewed after a month, and may be found asymptomatic. In these circumstances, the true diagnosis is never determined, but it is probable that some relatively unimportant condition such as torsion of an appendix epiploicae was responsible.
B. The investigations may reveal a diverticular disease or CA. Both the diseases may coexist. Hence, the undoubted presence of diverticula does not in itself rule out the possibility of CA.

CONDITIONS COMMON TO BOTH LOWER QUADRANTS

Including suprapubic region when pain involves both lower quadrants simultaneously

Acute iliac lymphadenitis (*Syns.* **Acute iliac adenitis. Iliac abscess)**

Basically, acute inflammatory abdomen involving a lower quadrant, and hectic fever.

Differentiating points:

– No muscle guarding
– Fixed tender lump in the inguinal area (just above the inguinal ligament)
– The mass extends up to the iliac crest, spine, inguinal ligament or in the femoral region.

Supporting points:

– Painful fixed flexion deformity at the hip joint
– Primary focus of infection in the lower limb (foot ↔ buttock) or perineum. Adenitis **ma**y occur **2 weeks** after the primary lesion has settled.

Acute pyomyositis of iliopsoas (*Syns.* **Iliopsoas abscess. Psoas abscess)**

Basically, acute inflammatory abdomen involving a lower quadrant, and hectic fever.

Differentiating points:

– No muscle guarding

Free ebooks ==> www.Ebook777.com

- Fixed tender lump in the inguinal area (just above the inguinal ligament)
- The mass extends up to the iliac crest, spine, inguinal ligament or in the femoral region
- Cross fluctuation between the two lumps across the inguinal ligament (when a lump is also present below the inguinal ligament).

Supporting point: Painful fixed flexion deformity at the hip joint.

Thus, it may be noted that physical findings in both acute iliac abscess and psoas abscess are almost the same; the differential diagnosis may be impossible clinically.

Iliacus / iliopsoas hematoma

Basically, acute inflammatory abdomen, and a lump involving a lower quadrant.

Differentiating points:

- No muscle guarding
- Fixed tender lump in the inguinal area (just above the inguinal ligament)
- The mass may extend up to the iliac crest, spine, inguinal ligament or in the femoral region.

Supporting points:

- Painful fixed flexion deformity at the hip joint
- Bleeding tendency.

Tuberculous psoas abscess

Differentiating points:

- Subacute illness
- H/s/o osteomyelitis of the spine (X-ray changes will be evident)
- Nonspecific symptoms of TB
- Fixed tender lump in the inguinal area (just above the inguinal ligament)
- The mass will extend up to the iliac crest, spine, inguinal ligament or in the femoral region
  Cross fluctuation between the two lumps across the inguinal ligament (when a lump is also present below the inguinal ligament)
- Expansile impulse-on-coughing.

Supporting point: Painful fixed flexion deformity at the hip joint.

Acute pyelitis

Basically, acute inflammatory abdomen involving a lower quadrant, and hectic fever with rigors.

Differentiating points:

- The presence of urinary symptoms
- Tenderness in renal angle.

Acute perinephric abscess

Basically, acute inflammatory abdomen involving a lower quadrant, and hectic fever.

Differentiating points:

- Tenderness in the renal angle
- Fullness—diffuse acute inflammatory lump—in the loin
- Obvious presence of primary source of infection like tuberculous osteomyelitis of vertebra.

Remarks:

- Local signs present early if infection starts in the lower part of perinephric fat
- Infection at the upper pole is masked by the lower ribs and signs in the loin are much less marked.

Notes:

Causes (primary sources) of perinephric abscess:

- Renal—cortical abscess, carbuncle, pyonephrosis
- Perinephric—hematoma, tuberculous osteomyelitis of vertebra, periureteral lymphangitis
- A distant focus of infection; infection of kidney occurring due to hematogenous spread.

DD of acute perinephric abscess

- Pyonephrosis

www.Ebook777.com

- Pyomyositis of abdominal wall or paraspinal muscles
- Osteomyelitis of the spine with spread to the paraspinal tissues
- Subphrenic abscess.

Acute PID

Basically, acute inflammatory abdomen involving usually both lower quadrants, although it may be dominant on one side.

Differentiating points:

- Pain following menstrual period, OR during the first week after abortion / delivery / instrumentation (IUCD insertion, termination, or surgery)
- Pain usually bilateral
- The presence of urogynecological symptoms like increased frequency of micturition, heavy / prolonged periods, vaginal discharge, dyspareunia
- Site of tenderness—quite medial to McBurney's point
- Lower abdominal tender mass ±.

On PV/E

- Discharge
- Inflamed cervix
- Cervical excitation positive, (i.e. cervical motion is tender)
- Tenderness in both vaginal fornices (adnexal tenderness).

Supporting points:

- Age < 35 years
- Recent sexual intercourse
- Change in partner.

Notes:

- Patient may not admit to all the symptoms, especially if she is a young unmarried girl
- Symptoms are usually mild, but can be severe with signs of peritonitis and septic shock
- Acute cases are often atypical, either because of mild disease or because it has been modified by previous treatment
- Clinical manifestations of pelvic sepsis are wide; they range from otherwise asymptomatic infertility to septic shock
- *Various clinical forms of PID*
  1. PID unrelated to pregnancy. Typically follows a period. This is the commonest form. PID mentioned without any specification conventionally means this form of PID. Such a patient may have an IUD inserted in her uterus.
  2. Postabortal PID (= Septic abortion).
  3. Infected obstructed labor.
  4. Puerperal PID (= Puerperal sepsis).
  5. Postcesarean PID.
- *Various pathological forms*
  1. Cervicitis.
  2. Endometritis.
  3. Salpingitis, pyosalpinx, hydrosalpinx.
  4. Salpingo-oophoritis, tubo-ovarian abscess.
  5. Pelvic peritonitis, pelvic abscess.
  6. Parametritis, abscess in the broad ligament.
  7. Septic thrombophlebitis of ovarian / uterine veins—serious. → septicemia with few local signs.

Septic abortion

= infection of retained products of conception. This in turn, → increased bleeding, and risk of uterine perforation.

- Patient becomes sick and febrile (> 100° F) during the first week after abortion
- Foul vaginal discharge, sometimes frank pus

Free ebooks ==> www.Ebook777.com

– Anemia, jaundice due to sepaticemia
– A h/o neglected spontaneous abortion or unskilled interference

If a h/o pain or fever precedes the vaginal bleeding, there is a possibility that interference with the pregnancy has occurred

Unfortunately, the patient may be so frightened that she will deny having tried to procure an abortion

Golden rule: Any acute pelvic inflammation in a woman of child-bearing age may be the result of an abortion

– Tachycardia of over 120 ⇒ the infection has probably spread beyond the uterus
– Bleeding from the cervical os
– Cervical excitation positive
– Lateral fornices—hot, tender, thickened and perhaps with a mass suggesting parametritis.

Puerperal sepsis with localized pelvic inflammation

– Abdomen distended
– Ill-defined tender mass arising out of the pelvis
– *On pelvic examination*
  - Vagina shows signs of recent child-birth or abortion. Infected lacerations ±
  - Cervix open and tender. May be drawn up behind the symphysis
  - Pouch of Douglas swollen, thickened
  - Uterus and appendages form a mass, which is difficult to define due to tenderness

Puerperal sepsis may lead to generalized peritonitis

A visible mass extends up to the umbilicus.

Infected obstructed labor / postcesarean section acute PID

Predisposing factors:

– Longer the labor lasts, greater the risk
– Dead baby
– Early rupture of membranes
– Inadequate aseptic precautions.

Pelvic abscess

Basically, acute inflammatory abdomen involving the hypogastrium ± one or both iliac fossae, and hectic fever, nonspecific abdominal symptoms and general ill-health.

Differentiating points:

– The presence of predisposing factor, for example:
  - Operative intervention with postoperative leak
  - Primary infective / inflammatory focus like PID, septic abortion, puerperal sepsis, appendicitis, perforated peptic ulcer etc.
  - Accidental abdominal trauma.
– Frequent stools, tenesmus, and mucus in stools
– Anorexia and wasting (loss of weight)
– Ill-defined tender mass in the suprapubic region
– On DRE:
  - In a male patient—a boggy tender mass above the prostate filling the rectovesical pouch. DO NOT expect fluctuation
  - In a female patient—a soft bulging lump in the pouch of Douglas. DO NOT expect fluctuation. Sometimes the mass is almost visible at the vulva
  - On bidigital palpation (with index finger in the rectum and thumb in the vagina), the two fingers will not meet.

Notes:

– PID is the commonest cause of a pelvic abscess
– A pelvic abscess can grow quite large without making a patient very sick or causing obvious signs. Hence, it is essential to perform frequent DR / PV examinations in a patient who is predisposed to develop and/or suspected to have developed a pelvic abscess to avoid missing the diagnosis

www.Ebook777.com

– Possible causes of bulging into the pouch of Douglas on DRE, other than pelvic abscess: 1. Hematocele associated with a chronic ectopic pregnancy. 2. An ovarian cyst. Some suspected cysts turn out to be post inflammatory collections of fluid (Postinflammatory pelvic pseudocysts).

Rectus sheath acute hematoma

Differentiating points:

– H/s/o a precipitating cause, e.g. 1. Strenuous exercise. 2. An elderly patient, particularly on anticoagulant treatment, having received a trivial injury
– Well-localized pain, tenderness ± a swelling in the lower quadrant
– Absence of GI upset.

Acute prostatitis. Refer to chapter 54.

PERITONISM

– Pain in abdomen is not the only presenting chief complaint. It is often associated with additional chief complaints
– Physical findings are like 'diffuse inflammatory abdomen', i.e. tenderness and guarding (or sometimes mild-to-moderate rigidity) almost all over the abdomen.

Severe ulcerative colitis

Usually starts de novo. Occasionally follows as a progression of mild or moderate form of ulcerative colitis.

*Basically, s*evere diarrhea and severe pain in abdomen and distension of abdomen, and fever.

Differentiating points:

– > 6 loose motions per day, tenemus, profuse rectal bleeding.

Supporting points:

– Anemia and hypoproteinemia
– Hypovolemia—shock, electrolyte imbalance and acidosis.

Toxic dilatation of colon

– Severe pain in abdomen in a k/c/o colitis of any nature, i.e. amebic / bacterial / pseudomembranous / ulcerative / Crohn's
– Severe systemic toxicity. Fever, tachycardia, marked fluid and electrolyte depletion, leukocytosis.
– Distension of abdomen
– Physical findings of acute inflammatory abdomen practically involving the whole abdomen
– Peristaltic sounds—absent
– Precipitating factor like
  - Administration of drug affecting intestinal motility like opiates, anticholinergic, etc.
  - Electrolyte / acid-bace imbalance, e.g. metabolic alkalosis, hypokalemia, etc.
– Findings on plain X-ray abdomen: Distension of colon. Loss of haustrations. Mucosal irregularities. Intramural air.

Notes:

– The entire colon may be affected but the transverse colon is predominantly involved
– Pathology: Transmural extension of the inflammatory process with deep ulcers reaching the serosa and vasculitis and inflammation of the myenteric and submucosal nerve plexuses. This leads to thinning of the bowel wall.

Acute necrotizing enteritis / enterocolitis

Basically, generalized acute inflammatory abdomen, though pain may be localized during the early stages.
And (Bloody) diarrhea and distension of abdomen.
As a matter of fact, the clinical picture may be a mix of intestinal obstruction and peritonitis. Possible reasons for the development of clinical picture of intestinal obstruction:

1. Enteritis = inflammation. Inflammation → irritation → colicky pain.
2. Paralytic obstruction must be setting in more rapidly resulting in other manifestations of intestinal obstruction developing rather earlier viz. distension of abdomen, bilious vomiting and absolute constipation.

Peristalsis:

Free ebooks ==> www.Ebook777.com

- Normal or even hyperactive in the initial stages
- Absent later as paralysis sets in.

Notes:

- This is a condition, which occurs apparently in the absence of any obvious cause
- Incidence more common during summer, and more prevalent in men than in women
- No age is exempt, but more common between 25 and 50.

(Acute) Gangrenous ischemic colitis

- Occurs almost always in elderly
- Clinical presentation with typical symptoms suggesting acute mesenteric occlusion
- Bloody diarrhea is a late sign
- Physical signs of generalized acute inflammatory abdomen usually on the left side. Patient is severely ill
- Additional clinical background of underlying cause may be present.

Causes of ischemic colitis:

- *Embolism* from atrial fibrillation / MI / atheromatous plaque in the aorta
- *Thrombosis*—arterial or venous: Portal HT, malignant disease of the colon, arteriosclerosis, polycythemia vera, hyperviscosity syndrome (due to platelet abnormalities / high molecular weight dextran infusion)
- *Low cardiac output states* (nonocclusive infarction)
- *Surgical injury* to vessels: Following aortic reconstruction with ligation of the inferior mesenteric artery, following resection of adjacent intestine
- *Vasculitis*
- '*Spontaneous*'.

Acute retroperitoneal lymphangitis (filarial)

- High-grade fever with rigors
- Repeated vomiting
- Diarrhea ±
- Distension ±
- Tenderness and guarding / rigidity all over
- Peristaltic sounds—diminished / absent.

Note: In more severe cases, the patient becomes highly toxic, apathetic and delirious. The patient may die before developing any localizing signs.

Ileosigmoid knotting

It is a type of acute volvulus in which knotting occurs between the loops of small intestine and sigmoid colon leading to acute intestinal obstruction, shock, and rapid deterioration of general condition.

Ruptured ovarian cyst

- In the majority of cases, rupture occurs spontaneously.

On pelvic examination:

- Tenderness may be localized to one fornix
- A mass may not be palpable as the cyst has ruptured.

ACUTE (PARIETAL) PERITONITIS

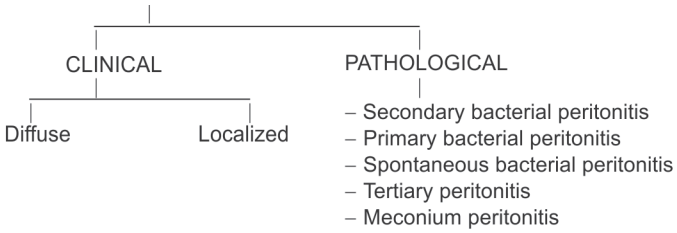
- = acute inflammation of parietal peritonium usually, but not necessarily resulting from perforation of a viscus (hollow / solid) through a pathological area (like an ulcer / necrosis / abscess, etc.)
- Sudden mode of onset and rapid progress implies that perforation of a hollow viscus must be assumed.

Differentiating physical findings in general

- The patient prefers to lie on the back quietly, reluctant to make unnecessary movements
- Cough tenderness
- Rigidity.

www.Ebook777.com

Classification of acute peritonitis



Whether peritonitis will remain localized to a quadrant, or spread to become diffuse will depend on the following factors:

1. The organ which perforates. Perforation of the 'main tube' of GIT such as stomach, duodenum, small intestine and large intestine usually will → diffuse peritonitis. Perforation of 'an accessory' (e.g. appendix, gallbladder, diverticulum, etc.) → localized peritonitis as perforation of these viscera follows a period of inflammation which promotes localization.
2. Mode of onset and progress of the pathology. 'Explosive' nature of pathology will tend to lead to diffuse peritonitis, e.g. peptic perforation.
3. Natural ability of the body to localize the pathology by various mechanisms.
4. Immunity of the host.

Common causes of diffuse peritonitis

– Peptic perforation—the commonest cause in adults
– Typhoid ('Enteric') perforation
– Dysenteric perforation
– Tubercular perforation
– 'Primary' peritonitis
– Any of the causes from 'localized peritonitis' group may → diffuse peritonitis. Predisposing factors: 1. Young children or elderly, due to short greater omentum. 2. Immunocompromised status due to any cause.

Other (less common) causes of diffuse peritonitis

– Severe form of clostridial (necrotizing) enterocolitis.
  - H/o consumption of improperly cooked food might be available
  - H/o severe colicky pain and diarrhea in the beginning might be available
– Gangrenous enteritis f/b perforative peritonitis caused by round worms.

Common causes of localized peritonitis

– Appendicitis
– Cholecystitis
– Diverticulitis (Meckel's / other).

In cases of localized parietal peritonitis, clinical features of inflammatory pathology will precede the development of physical signs of parietal peritonitis, by few days. The whole abdomen may not show characteristic signs of peritoneal irritation. In this situation, nomenclature used for the statement of diagnosis may vary depending on personal preference, though correlation with the underlying pathology will remain unchanged. For example: "Acute peritonitis in the RLQ most probably due to perforative / gangrenous appendicitis" may be expressed only as "acute perforative / gangrenous appendicitis".

Causes of severe rapidly oncoming upper abdominal peritonitis with prostration and marked toxicity

– Peptic perforative peritonitis
– Acute phlegmonous gastritis
– Acute emphysematous cholecystitis.

Medical causes to be considered leading to pain in abdomen mimicking that due to apparently generalized peritonitis:

– Fluid and electrolyte imbalance with paralytic ileus, nausea and vomiting

Free ebooks ==> www.Ebook777.com

– Diabetic coma
– Adrenocortical failure.

Peptic perforation during early stages

Basically, diffuse peritonitis (tenderness and rigidity almost all over the abdomen).

Differentiating points:

– Pain:
  - Sudden mode of onset (described as 'something having given way' or 'burst' within the abdomen)
  - In the epigastrium, very rapidly spreading all over
  - Maximally severe intensity reached almost at the outset
– Scaphoid abdomen
– 'Board-like' diffuse rigidity.

Supporting points:

– H/s/o chronic peptic ulcer / 'hyperacidity'
– Hematemesis (peptic perforation with bleeding)
– 'True' bradycardia
– 'Silent abdomen'.

Peptic perforation during later stages

Initial physical findings modify after about 24 hours or so, if the patient fails to receive appropriate treatment during that (i.e. early) period.

– Tachypnea
– Distension of abdomen develops. Mechanisms: Paralytic ileus setting in, and accumulation of exudative free fluid within the peritoneal cavity
– Rigidity may persist, though it may not remain as severe as 'board-like'.

Typhoid (Enteric) perforation

Differentiating points:

– H/o continuous fever for two weeks preceding acute pain in abdomen; f/b severe continuous pain of sudden mode of onset, and fall of temperature. (Perforation occurs anytime from the end of the 2nd week onwards of fever.)
– Pain begins in the lower abdomen
– Patient looks 'toxic'
– (Relative) Bradycardia is replaced by tachycardia
– Hypotension, if the patient is in the state of late (i.e. decompensated) septic shock
– Although diffuse, the signs are present mainly in the lower abdomen.

Pitfalls:

– A history typically s/o enteric fever may be absent
– Abdominal wall rigidity may not be marked due to asthenia.

C/Ms of an uncomplicated typhoid fever

– Continuous high-grade fever. Initially, fever might follow a 'step-ladder' pattern, although it is not essentially so. Fever is maximum during the 2nd week
– Vague dull abdominal pain
– Loose motions or constipation
– Distension of abdomen and tympani (= bell-like note on percussion)
– Toxemia
– Relative bradycardia
  ± Hepatosplenomegaly.
– The classical rash called 'rose spots' can be seen in only fair-skinned patients.
  The secret of diagnosis is by excluding other types of common fevers, i.e. for example, by the absence of headache, backache, bodyache and marked weakness—s/o influenza; sneezing, watering and blocking of nose—s/o common cold; unilateral chest pain which is increased on deep breathing / coughing, and cough

www.Ebook777.com

with / without expectoration—s/o pneumonia, etc.

Notes:

- Arriving at the diagnosis of typhoid enteric perforation early, may be difficult as several symptoms and signs of 'acute abdomen' are masked in many cases. Reasons: 1. Pre-existing dull pain in the abdomen, distention of typhoid meteorism and toxemia make the episode of perforation less dramatic, and the exact onset of perforation is difficult to pinpoint. 2. Abdominal wall rigidity is often less marked due to asthenia
- Toxemia is out of proportion to what could be attributed to peritonitis alone
- Rarely, ileoileal intussusception develops due to an inflamed Payer's patch, in addition to typhoid perforation
- Reports of various investigations may not be of sufficient help for various reasons in the management of this life-threatening urgent situation
- The diagnosis mainly rests on the clinical suspicion of perforation in a patient giving prior history s/o typhoid fever.

Dysenteric perforation (Amebic) → Gangrenous amoebic colitis / amebic acute perforative peritonitis.

Gangrenous amebic colitis = Acute fulminating form of amebic colitis.

Differentiating points:

- Dysentery for about 8–10 days before the development of acute abdomen. However, diarrhea may not be necessarily severe.
- Generalized tenderness and guarding; though more marked in the RLQ
- Vague lump ± — may suggest a mistaken diagnosis of acute appendicitis.

Further course:

- Rapid deterioration
- High-grade fever, toxemia and all other signs of frank generalized peritonitis.

Amoebic acute perforative peritonitis—due to perforation of amoebic ulcer/s of the colon.

- A h/o dysentery preceding the development of acute abdomen. However, diarrhea may not be necessarily severe
- Preoperative diagnosis of amebic ulcer perforation is not always possible.

Acute peritonitis due to rupture of an amebic liver abscess

- Such a comprehensive diagnosis may not always be possible clinically. Aspiration of reddish brown pus, if peritoneal tapping is done, is suggestive
- A prior indication of an amebic liver abscess may or may not be present. Sometimes, the patient may present for the first time straightway with peritonitis
- The event of rupture is generally spontaneous. Occasionally, it may be precipitated by abdominal trauma.

Tubercular perforation

- Exact preoperative clinical diagnosis of tubercular perforation as such is difficult
- Perforations located just proximal to the narrowed part of intestine lead to classical signs of acute peritonitis
- *A pitfall*: Rigidity is less marked if patient is asthenic due to widespread systemic tuberculosis.

Discussion on peritonitis with respect to pathological classification

Secondary bacterial peritonitis

This accounts for the majority of cases of acute peritonitis. When generalized and severe, it still carries an appreciable mortality.

Basically caused by:

- Infection / gangrene of (usually a hollow) viscus.
- Perforation of GIT through a pathological area (like an ulcer / necrosis / abscess, etc.) / anastomotic dehiscence / injury.

Most common causes in practice (excluding trauma): Acute appendicitis, perforated peptic ulcer, acute diverticulitis of colon, perforation of an intestinal tumor, inflammatory bowel disease, intestinal infarction, acute cholecystitis, anastomotic dehiscence.

Free ebooks ==> www.Ebook777.com

Primary bacterial peritonitis

= bacterial peritonitis developing in the absence of surgery / trauma or any (primary) intra-abdominal focus of infection.

- In many cases, clinical differentiation between primary and secondary bacterial peritonitis is difficult, if not impossible. In practice, most cases are explored surgically and the condition is diagnosed (retrospectively) because of the absence of a primary focus of infection
- Encountered in—children, adolescents and adult women
- *Infants and children* present with 'acute abdomen'. Physical signs indicate parietal peritonitis. Awareness is important as primary peritonitis is not a common condition, and hence it will be overlooked unless it is considered in the differential diagnosis
- In *adolescent girls / young women*, mild (nonperforated) appendicitis associated with a seropurulent peritonitis (retrospective, at operation) is considered to be primary peritonitis
- In *adult women,* it may follow child birth.

Spontaneous bacterial peritonitis

= bacterial infection of ascitic fluid in the absence of any intra-abdominal surgically treatable source of infection.

Encountered in:

- Chronic liver disease like cirrhosis with ascites, chronic active hepatitis
- Nephrotic syndrome: Largely encountered in children
- Patients on CAPD: Can also arise as a consequence of catheter-related infection (subcutaneous tunnel or catheter exit-site). Risk factors for catheter exit-site infection:
  - Short cuff-exit distance (< 2 cm)
  - A number of previously placed PD catheters
  - Low serum. albumin level (< 3.5 g%).
- Immunocompromized status
- *Single important predisposing factor in general*: Low ascitic protein concentration (< 1 g%) irrespective of the nature of underlying disease, whether hepatic or renal
- *Clinical manifestations* may develop insidiously, and signs of peritonitis may be minimal
- 30% of patients may not have symptoms / signs directly referable to the abdomen. Hence, a high index of suspicion must be kept in susceptible patients
- In cirrhotic patients, deterioration of LOC can be caused by hepatic coma, septic shock.

Tertiary peritonitis

= persistence or recurrence of intra-abdominal infection following apparently adequate treatment of primary or secondary peritonitis.

- This occurs in ICU patients
- Infection is diffuse and poorly localized
- In critically, ill patients it appears to be more reflection than a cause of adverse outcome.

Meconium peritonitis— neonatal peritonitis.

- Caused by antenatal extravasation of meconium into the peritoneal cavity leading to serious illness characterized by intraperitoneal calcification, dense inflammatory fibrosis with or without giant pseudocyst formation. In the majority of cases, no obvious cause can be found.

ACUTE MESENTERIC VASCULAR ISCHEMIA

Causes	*Essential pathology*
– Acute mesenteric ischemia de novo – Acute mesenteric ischemia upon chronic mesenteric ischemia	} Embolism / thrombosis / vasculitis, etc. of essentially the SMA
– Nonocclusive mesenteric ischemia	Splanchnic vasoconstriction
– Acute / subacute mesenteric vein thrombosis	Thrombosis

www.Ebook777.com

– Spontaneous	?

– Although the final common clinical picture may be quite similar in all, the subtle clinical features may vary according to the etiopathology.
– The condition is not common. It is usually diagnosed late. Mortality rates are high.

ACUTE MESENTERIC ISCHEMIA (DUE TO ARTERIAL OCCLUSION of essentially the SMA)

Causes

a. Embolism
   – The commonest source is the heart: Atrial fibrillation, MI, CHF, valvular heart disease. H/s/o these disorders may be available
   – Other possible source is the aorta: Aneurysm, atherosclerosis, iatrogenic (intra-arterial manipulations of catheters, wires, etc. during cardiac / peripheral arteriographic procedures).
b. Thrombosis
   – There is a formation of *in situ* thrombus on an underlying stenosis
   – *Immediate causes*: Low-flow state, sudden expansion of a plaque e.g. due to intraplaque bleeding.
c. Rare causes—aortic dissection, fibromuscular dysplasia, arteritis associated with various autoimmune disorders (like RA), oral contraceptive, etc.

Differentiating points:

– Prostration from the outset
– Pain:
   - In central abdomen, although may not be well-localized
   - Nature—colicky or constricting
   - Intensity—severe; out of proportion to physical findings.
   - a. Sudden (Abrupt) mode of onset on the background of a heart disease or recent arterial catheterization ⇒ acute mesenteric arterial embolism.
     b. Insidious mode of onset superimposed on a h/s/o chronic mesenteric ischemia ('intestinal angina') ⇒ mesenteric arterial thrombosis.

   A pitfall: Intensity of pain may be mild.
– O/E early in the course, no abnormality is detected.
  Thus, at this juncture the clinical scene is "severe pain in abdomen out of proportion to the physical findings".
– With time, physical findings s/o acute inflammatory abdomen f/b peritonitis develop.
– Physical findings s/o frank peritonitis suggest late disease indicative of irreversible intestinal ischemia.

Additional manifestation which may be present

– H/s/o acute embolic occlusion to other parts of the body (limbs for example) earlier or simultaneously.
– Minor GI bleeding. However, it's a late sign.

Notes

In general, simple laboratory and radiographic imaging studies cannot confirm or exclude the diagnosis of acute mesenteric ischemia. It is, therefore, essential to keep in mind the typical patterns of presentation for patients *who are subsequently found to have acute mesenteric ischemia*:

– Elderly
– Abrupt onset of severe constant pain that is not well-localized
– Vomiting or / and diarrhea ±
– A high index of suspicion is essential for early diagnosis.
– Arterioarterial emboli tend to be smaller; consequently, they tend to cause more localized ischemia.
– Diffuse bowel ischemia suggests mostly the cardiac source of emboli.

NONOCCLUSIVE MESENTERIC ISCHEMIA

Pathophysiology is incompletely understood although essentially it is splanchnic vasoconstriction.

Causes

Free ebooks ==> www.Ebook777.com

a. Primary.
b. Secondary to cardiopulmonary bypass, shock states (causing mesenteric vasoconstriction), the use of vasoactive medications (digoxin, ∝-adrenergic agents, vasopressin).

Causes of poor prognosis (high mortality): Delay in diagnosis, significant associated comorbidities.

Clinical diagnosis requires a high index of suspicion.

- The presence of a risk factor (refer to the causes above) should raise the suspicion
- Pain in abdomen may be absent in about 25% of cases
- Otherwise, the clinical presentation is similar to that of acute mesenteric ischemia
- Diffuse tenderness, rebound tenderness, rigidity are ominous signs

Occasionally, patients may present with shock or sepsis of unknown etiology.

MESENTERIC VEIN THROMBOSIS

- There is a broad range of clinical presentations
- Patients may be asymptomatic, mildly symptomatic, or present with acute catastrophic illness
- The condition is often benign but acute ischemia can be lethal
- Early and aggressive treatment may limit progression of thrombotic process
- The disorder usually involves the superior mesenteric vein and splenic vein, and less commonly inferior mesenteric vein and portal vein.

*The **clinical diagnosis***

- The clinical presentation of acute / subacute MVT depends on the location and extent of the splanchnic thrombus
- Suspect the disorder when acute / subacute abdominal symptoms develop in a patient with prior thrombotic episodes, hypercoagulable state, a documented coagulopathy or prior splenectomy.

Pain in abdomen

- Mode of onset—insidious or sudden
- Worsens progressively
- Generalized, nonspecific.
- *Abdominal distension* often present but tympany may be lacking due to mesenteric and bowel wall edema and to sequestrated intraluminal and intraperitoneal fluid.
- Duration of pain and distension: 1 week to 1 month; in some patients, may last even much longer.
- *Diarrhea*
- *GI bleeding*: Often occult. Gross bleeding suggests either advanced process or chronic form (variceal bleeding).
- Diagnosis can usually be made with a high index of suspicion, and a CECT / CEMRI.

Conditions associated with MVT (aetiology)

- Direct injury: Trauma, postsurgical (particularly postsplenectomy), intra-abdominal inflammatory states (pancreatitis, IBD, peritonitis, abscess)
- Local venous congestion or stasis: portal HT / cirrhosis of the liver, CHF, hypersplenism
- Hypercoagulable states. Previous episode of DVT / PE.
- *Natural history* is unknown, and often relates directly to the extent of thrombosis and underlying etiology However, prognosis may not be as ominous as in acute mesenteric ischemia. However, it left undiagnosed and untreated, there is a distinct mortality
- Primary acute mesenteric vein thrombosis causes less widespread ischemia → comparatively less mortality rates
- Outlook in cases secondary to visceral infection / PHT / systemic hypercoagulability is similar to mesenteric arterial occlusion.

TORSION

Differentiating features in general

- Pain:
  - Maximally severe intensity reached almost at the outset

www.Ebook777.com

- Nature—twisting
– Prostration from the outset
– Tender lump
– Guarding
– Diminished peristaltic sounds in the affected area.

Supporting feature: Similar past history ±.

Causes

1. Torsion of a normal structure e.g. omentum.
2. Torsion of an otherwise normal structure, but which is 'floating': Floating spleen, floating kidney.
3. Torsion of abnormal structure: Ovarian cyst, pedunculated fibroid of uterus.

Torsion of omentum

A rare emergency, and consequently is seldom diagnosed correctly. It is usually mistaken for appendicitis.

Torsion of ovarian cyst

Differentiating points:

– In a woman, lower abdominal unilateral pain suggesting torsion
– Prostration from the outset
– Tense, tender lump in lower part of abdomen
– On pelvic examination: An exquisitely tender mass to one side of the uterus
– If an ovarian cyst is known to have been present, the diagnosis is certain.

Notes:

– The torsion of a cyst often includes the uterine tube in the process
– Rarely, in young girls and in the elderly, the torsion also includes the body of the uterus
– *Ovarian hematoma* due to bleeding into an ovarian cyst occurs spontaneously, or following trauma
– The clinical presentation is similar to that of torsion, but the pain is less severe.

Torsion of uterine fibroid

– Clinical features are similar to those of torsion of ovarian cyst
– However, torsion of fibroid is rare.

INTERNAL BLEEDING

Differentiating features in general

– Excessive thirst and prostration right from beginning
– Fainting
– Subnormal body temperature
– Rapid (hourly) increase in pulse rate
– Tachypnea
– Rigidity present (but not board-like).

Pain in shoulder due to irritation of undersurface of diaphragm suggests considerable bleeding.

A pitfall: Fainting and nausea may occur even when the pain is not severe.

***Causes** of internal bleeding*

1. Vascular:
   – Rupture of a AAA
   – Rupture of other aneurysms like splenic, hepatic, etc.
   – Rupture of vascular lesion (hemangioma) of a solid organ like liver.
2. Gynecologic / obstetric:
   – Ruptured ectopic gestation
   – Ruptured Graafian follicle
   – Rupture of a vein over uterine fibroid.

Free ebooks ==> www.Ebook777.com

3. Spontaneous rupture of spleen.
4. Peptic perforation with bleeding.
5. Rupture of an SOL of solid organ like liver / spleen.
6. Hemorrhagic diseases.
7. From secondary peritoneal carcinomatosis.

Rupture of a AAA

– This is a clinical diagnosis based on the principle—"pain and collapse in a patient known to have a AAA = rupture of the aneurysm"
– Patients usually present with varying degrees of shock and even those who are normotensive usually give a h/o collapse or light-headedness with subsequent recovery.

Symptoms

Pain.

– Site:
  - Center of the abdomen / lower back
  - May mimic renal colic (loin to groin).
– Continuous
– H/o collapse or light-headedness with subsequent recovery ±
– May be a k/c/o AAA.

Specific sign (in addition to the general differentiating features s/o 'internal bleeding')

– A lump in the central abdomen which may be pulsatile
– However, the presence of an aneurysm can be difficult to detect clinically, especially in the obese.

Notes:

– Many aneurysms rupture posterolaterally into the retroperitoneum where tamponade combined with hypotension slows the rate of blood loss and may even temporarily seal the rupture. Anterior rupture into the peritoneal cavity usually causes devastating blood loss
– The presence of an aneurysm can be difficult to detect clinically, especially in the obese, and where there is clinical suspicion of rupture, an imaging investigation should be undertaken urgently to detect the presence of an aneurysm
– An imaging investigation cannot reliably confirm or exclude the diagnosis of rupture and should only be used to confirm the presence, and details of the aneurysm.

Acute rupture of ectopic pregnancy

Differentiating points:

– Woman of child-bearing age
– One or two missed periods
– A h/o slight blood-stained discharge ± i.e. vaginal bleeding from spotting to the equivalent of a menstrual period
– On vaginal examination
  - Bleeding from the cervical os
  - Cervix feels softer than normal
  - Gentle movement of cervix—tender
  - Tenderness in all fornices.

Other clinical features, one or more of which may or may not be present:

– Instead of missed period, abnormally light menstrual period
– Deciduous tissue may be passed vaginally and an erroneous diagnosis of spontaneous abortion made
– Transient symptoms of pregnancy may be experienced.

High-risk factors for development of ectopic pregnancy:

– Prior ectopic pregnancy

www.Ebook777.com

- Previous PID
- Pregnancy with an IUD in place
- Pregnancy resulting from failed postcoital contraception
- Tubal sterilization especially within the past 1–2 years
- H/o tubal reconstructive surgery
- Infertility.

Rupture of a lesion in the liver

Examples: Hepatic cystadenoma, liver cell adenoma, HCC.

Rupture of a lesion in the spleen

Examples: Splenic abscess, cyst, tumor.

SPECIFIC GYNECOLOGIC CAUSES of acute abdomen

Salpingitis and PID: Refer to page 476.

Torsion of ovarian cyst, uterine fibroid: Refer to page 487.

Rupture of corpus luteum or ovarian cyst

- In 2nd half of menstrual cycle
- The patient may be shocked; and prefers to lie still
- Generalized guarding.

Pain due to rupture of corpus luteum usually resolves in 24–48 hours.

US scan findings: Free fluid. May not show cyst; hence US scan alone may not be diagnostic.

Mid-cycle pain

- Severe but brief pain
- The patient prefers to lie still
- Guarding and tenderness
- Self-limiting. Settles in 24 hours.

Endometritis

- On the background of recent delivery, termination of pregnancy, or ERPC
- Bleeding PV without or with discharge
- Nausea
- Feverishness
- Febrile
- Cervical os may be open.

US scan of little help.

Uterine fibroid(s)

- Rarely a cause of pain.

Conditions when fibroids can cause pain:

1. Red degeneration during pregnancy.
2. Submucous fibroid may cause crampy period pain and bleeding.
3. Rarely, torsion.

(ACUTE) PAIN IN ABDOMEN IN A PREGNANT WOMAN

The diagnosis and management of acute pain in abdomen in pregnancy may give rise to anxiety for the

Free ebooks ==> www.Ebook777.com

surgeon

Modification of symptoms and signs may lead to delayed or incorrect diagnosis and, when combined with a reluctance to operate on a pregnant woman may result in increased morbidity / mortality

Anatomic and physiologic changes in pregnancy which modify symptoms/signs or make clinical assessment difficult:

1. Gravid uterus displaces abdominal organs cephalad and brings adnexal structures into abdomen.
2. Compression of IVC by gravid uterus may cause supine hypotension syndrome.
3. Increased plasma volume, decreased hematocrit, and generally lower BP make assessment of acute blood loss more difficult.
4. Relative leukokytosis makes evaluation of infection more difficult.

Two practical tips helpful for diagnosis:

1. The incidence of some conditions may change during pregnancy

- Conditions, the incidence of which decreases
  - Examples:– PID.
    – Peptic ulcer
- Conditions, the incidence of which increases
  - Examples: – UTI
    – Acute pancreatitis, although rare, occurs more often in pregnant than in non-pregnant women of the same age
    – Infarction of fibroid occurs almost exclusively during pregnancy

2. Classification

- General surgical emergencies
  - The pattern of symptoms and signs may get altered
    - Attacks of pain in the groin/lower abdomen during 2nd trimester. This has a physiological basis: Rapid uterine growth early in 2nd trimester causes tension and stretching of round ligaments, causing spasm with sudden movement or change in position
- Obstetric conditions peculiar to pregnancy
  - Physiological
  - Pathological
    - – Rupture of ectopic pregnancy
      – Infarction of uterine fibroid (red degeneration)
      – Rupture or torsion of corpus luteum cyst
      – Spontaneous abortion
      – Septic abortion
      – Placental abruption

CAUSES OF ACUTE LUMP IN ABDOMEN

Refer to chapter 35.

Spectrum of disease causing 'acute abdomen' of particular importance in TROPICAL COUNTRIES

1. **Malaria**: Refer to chapter 13.
2. **Amebiasis** → – Amebic acute typhlitis
   – Gangrenous amoebic colitis
   – Amebic acute perforative peritonitis.
3. **Typhoid**: Perforation of ulcer → peritonitis.
4. Abdominal **TB**
   ↓
   – Intestinal obstruction (often acute-on-chronic)
   – Acute tuberculous peritonitis due to

www.Ebook777.com

- Perforation/s located just proximal to the narrowed part of intestine
- Rupture of tuberculous mesenteric lymph node.

5. Schistosomiasis

a. *Schistosoma haematobium* → urinary tract disease and consequently various clinical manifestations depending on the pathology.

b. *Schistosoma mansoni* and *Schistosoma japonicum* →
- Dysentery during the onset of egg lying in a first infection
- In severe cases, ulceration and massive bleeding; this could be fatal
- Perforative peritonitis
- Stricture of colon.

6. Abdominal crisis of sickle cell disease →
- Acute abdominal pain
- Perforative peritonitis.

7. Gnathostomiasis

– Caused by a nematode *Gnathostoma spinigerum*
– Infestation usually follows ingestion of raw fish
→ single migratory subcutaneous swelling which may appear anywhere, including the abdominal wall, the chest, the face or hands
– Also, may → spontaneous perforation of the right side of the colon, hematuria, spontaneous pneumothorax.

DIFFERENTIAL DIAGNOSIS

Tenderness in the RLQ due to acute inflammatory pathology v/s distended cecum due to an obstructive lesion distally

– The presence of other evidences of peritoneal irritation ⇒ acute inflammatory pathology
– The absence of other evidences of peritoneal irritation ⇒ distended cecum due to an obstructive lesion distally.

Acute appendicitis v/s ANIML

– Pain: Complete freedom from pain in between attacks ⇒ ANIML rather than appendicitis
– Vomiting: Though vomiting may occur in ANIML, the presence of vomiting will favor the diagnosis of appendicitis
– Patient seldom looks sick in ANIML, but will almost always look sick in appendicitis.

Iliac abscess v/s intraperitoneal lump, e.g. appendicular mass

Iliac abscess will extend up to the iliac crest, spine, inguinal ligament or even in the femoral region, while appendicular mass stops short of iliac crest / inguinal ligament by about a fingerbreadth.

Iliac abscess v/s acute septic arthritis of the hip joint

Acute septic arthritis of the hip joint →
- Exquisitely painful and tender joint
- Moving the joint **even little in any direction** → severe tenderness
- 'Night cries'
- Tenderness on percussing the greater trochanter.

Differential diagnosis of lumbar pain (Differentiation of various conditions from renal pathology)

1. *Intraperitoneal pathology*:
– Patient preferring to lie motionless to minimize pain ⇒ intraperitoneal pathology
– Patient who may be more comfortable moving around and holding the flank is more likely to have a renal pathology.

2. *Radicular pain* (irritation of costal nerves, most commonly T_{10}-T_{12}):
– Hyperesthesia of the overlying skin, and alteration of intensity of pain due to changing position ⇒ radicular pain
– Intensity of pain arising from renal pathology should not alter with changing position. Moreover, if renal

Free ebooks ==> www.Ebook777.com

pain is caused by obstructive pathology, then the pain will be colicky in nature.

3. *Pain experienced during the pre-eruptive phase of herpes zoster involving any of the segments between T_{11} and L_2* may simulate pain of renal origin. Hyperesthesia of skin may favor the diagnosis against renal disease.

MONITORING AND REVALUATION

Monitoring of vital parameters

1. Development of tachycardia in a patient who has been 'conserved' ⇒ continued bleeding / uncontrolled sepsis; e.g. development of tachycardia in a conserved c/o acute appendicitis suggests that the conservative treatment is failing and that it may have to be abandoned and an urgent surgery will be required.
2. Relief of pain will suggest that the disease has subsided, or perforation / gangrene has taken place. Pulse rate will help to differentiate. Normal pulse rate ⇒ improvement of the condition, whereas tachycardia ⇒ deterioration (perforation or gangrene).
3. In a case of peritonitis, rising pulse rate ⇒ spreading peritonitis. Even a rise of 10 beats / minute is significant.

Monitoring of abdominal girth

Progressive increase of abdominal girth ⇒ deterioration.

Revaluation

– Revaluation should be carried out by the same clinician at 'frequent' intervals; frequency varying according to the case
– Look for evolution of new symptoms and signs
– Look for modification of existing symptoms and signs

→ Respond to the inference of revaluation.

An example: In a suspected case of perforative peritonitis: Lessening of the degree or disappearance of rigidity after a few hours may mean—(a) Improvement as in case of a very small perforation which soon seals spontaneously, or (b) Neutralization of irritant fluid by peritoneal exudate.

- Significant symptomatic improvement coupled with improvement of vital parameters suggests (a)
- The absence of symptomatic improvement, persistent tachycardia, and appearance of shifting dullness suggest (b).

RESIDUAL PROBLEM

= Persistence of pain without a sign suggesting threat to life

This situation may mean

or

The case does not qualify for acute abdomen

Manage as a c/o acute inflammatory abdomen

↓

Conservative management to begin with, for 24–48 hours

If no response,

↓

Laparotomy

The length of time to wait before deciding to operate depends on:

1. The severity of pain
2. Possibility of a medical cause
3. Reliability of the patient herself/himself. The opinion of a pediatrician or psychiatrist may be invaluable in appropriate cases.

www.Ebook777.com

35 LUMP IN ABDOMEN

DEFINITIONS

1. **Anterior abdominal wall**
   - Bounded superiorly by the costal margins; inferiorly by a line connecting each anterior superior iliac spine to pubic symphysis; and on each side by a vertical line through the anterior superior iliac spines.
   - Layers: Skin, subcutaneous tissue, muscles, transversalis fascia, extraperitoneal fat and parietal peritoneum.
2. **Posterior abdominal wall**
   - Composed principally of 3 sets of bones, 4 sets of muscles, and fat, fibrous tissue, nerves, vessels and lymph nodes.
   - Bones: Lumbar vertebrae, sacrum, and the wings of ilium.
   - Muscles: Posterior part of the diaphragm, psoas major, iliacus, and quadratus lumborum.
3. **Retroperitoneal lumps**
   The term is usually confined to the lumps arising from tissues (muscles, fat, fibrous tissue, nerves, vessels, lymph nodes, and developmental remnants) of this compartment but excluding origin from the retroperitoneal organs (pancreas, kidneys, ureters, and adrenal glands).

GENERAL REMARKS

1. A palpable abnormal lump in abdomen must be assumed to be due to a serious disease.

2. *Specific symptomatology* regarding lump in abdomen *can be divided in 3 groups*.
   a. Details of the lump itself.
   b. Symptoms of dysfunction. These can result from physical compression of critical anatomical structures in the vicinity, or from a specific physiological expression of disease (as for example in case of carcinoid tumor); or both. Hence, it is important to keep in mind that a physiological dysfunction could be primary or secondary. For example, vomiting may result from cause/s other than actual physical origin of lump from GI tract.
   c. Symptoms related to acute or chronic pain in abdomen.

3. Occasionally, histological examination of a mass in the cecum removed by right hemicolectomy may reveal that instead of carcinoma, it is tuberculosis, sarcoidosis, lymphosarcoma, etc. The disease found may require further specific treatment.

4. At the end of clinical case-taking, one of the following three possibilities can occur
   (a) can arrive at a confident clinical diagnosis.
   OR (b) a working diagnosis ('the most probable diagnosis') is made which is thought to be accurate.
   OR (c) the lump remains undiagnosed.

In a lump that remains undiagnosed, investigations should not be prolonged indefinitely; the patient must be subjected to diagnostic (cum therapeutic) laparoscopy/laparotomy at some time, and preferably while the lump is still amenable to treatment!

Free ebooks ==> www.Ebook777.com

HISTORY

Age

There are specific causes of abdominal lump in infancy and early childhood.

Chief complaints

- Document in *chronological order*.
- Verify the *exact site* and *duration*.
- Is it *pulsatile*?
- Is it, OR was it *reducible*?

Duration of the lump?

Mode of onset of the lump: Sudden / insidious?

How fast did it **progress**?—Rapidly over days
OR—Over weeks/months/years
OR—Is it stationary?

Is it associated with **severe pain**?

At this juncture, it will have become clear as to whether the lump is acute or chronic.

– Duration of 1–2 weeks, sudden mode of onset, rapid progress over days, and associated with severe pain ⇒ Acute/subacute lump.
– Duration > 3 weeks, insidious mode of onset, and progress over weeks or slow progress over months/years ⇒ Chronic lump.

Continue further history-taking accordingly.

Further HISTORY to be asked in a case of ACUTE/SUBACUTE LUMP

- Was this swelling reducible earlier, and the same having become suddenly irreducible and painful now?
  Pain
- Do you feel it in the abdominal wall, OR inside the abdomen?
- Exact site of pain? Observe as the patient is showing the site.

Inference from the above 2 details about pain, and further history to be taken accordingly

OR

If patient feels pain in the abdominal wall and if it is accurately located by pinpointing with a fingertip
⇓
Parietal swelling
↓

- Is the pain severe, and throbbing in nature?
- Hectic fever?
- H/s/o regional acute lymphadenitis?
- History of trauma (direct or indirect)?
- History of bleeding disorder?
- Are you on any anticoagulant medication?

If pain is experienced inside the abdomen, and if it is vaguely located over a more diffuse area by putting flat of one or both hands, or by moving around spread-out fingers
⇓
Intra-abdominal lump
(intraperitoneal or retroperitoneal)
↓
Take the case further as in 'acute abdomen' – Refer to chapter 34

- History of treatment taken: including—local application of ointment/herbal medicine/fomentation, etc?
  —Repeated manipulations?
- Is this the first episode or is this a recurrent episode?
  If recurrent, details of the previous episode/s?

Further HISTORY to be asked in a case of CHRONIC LUMP

A. *Symptoms which may give clue about general pathological nature of the lump*

- Is this the **first episode or** is this a **recurrent** lump?
  If recurrent, details of the previous episodes?
- Any **recent increase in size** of the lump which was practically stationary for a long time?
- Did it **regress** any time during the course?
- Does it ever **fluctuate** in size?

www.Ebook777.com

- **Fever**?

  Causes—Dehydration, chronic infection, occult intra-abdominal abscess, autoimmune disorder, hematologic malignancy like lymphoma.
- **Loss of appetite**?
  - Nonspecific (i.e. for all foods), OR qualitative (i.e. for particular type of food such as meat/fatty food etc.)?
  - Mild or severe?

  Inference: - Nonspecific anorexia ⇒ TB, malignancy in general.
  - Nonspecific anorexia especially if severe ⇒ malignancy.
  - Anorexia particularly for meat may suggest CA stomach.
  - Anorexia particularly for fatty food may suggest gallbladder disorder.
- **('Significant') Loss of weight**?

  Causes (relevant to abdominal disease)—Effect of persistent vomiting and diarrhea; disorders of esophagus/stomach/small intestine/liver/gallbladder/pancreas (esophageal obstructive disease, gastric ulcer, 'abdominal angina', hepatitis, cirrhosis, chronic pancreatitis); worm infestation; TB; malignancy; malabsorption syndrome.

  However, some patients with cancer of stomach/large intestine may maintain their weight surprisingly until advanced stage.

B. *Nonspecific symptoms which may give clue about the tissue/organ of origin of the lump*

- '**Indigestion**'? ⇒ GIT disorder.
- (**Excessive**) **Belching and eructations**?

  Causes (relevant to 'chronic abdomen')—Disorders of the esophagogastric junction/stomach/gallbladder; psychogenic.
- **Nausea**?

  Causes (relevant to 'chronic abdomen')—Disorders of stomach/gallbladder; psychogenic.

  Nausea should not be taken lightly. It could be an early symptom of CA stomach.
- **Vomiting**?

  Frequency, amount, color, contents, relation to eating?

  Causes (relevant to 'chronic abdomen')—Disorders of stomach-duodenum/gallbladder; (subacute/chronic) intestinal obstruction; 'chronic'! appendicitis, renal lithiasis, pregnancy and pregnancy-related disorders, psychogenic.
- Episodes of **vomiting of blood**? (**Hematemesis**)

  Causes (relevant to 'chronic abdomen')—Chronic peptic ulcer; esophagogastric varices (due to cirrhosis/chronic pancreatitis/pancreatic tumor); GERD complicated by Barrett's ulcer penetrating into esophageal branch of aorta; ingested (especially sharp) foreign body (either leading to esophageal mucosal injury or penetrating into aorta); aortic aneurysm forming fistulous communication with esophagus.
- Black or brown colored stools of tarry consistency? (**Melena**)

  Cause: GI bleeding from anywhere proximal to sigmoid colon (inclusive of sigmoid colon).
- **(Persistent) wind-ball movement** and **'borborygmi'**? ⇒ subacute/chronic intestinal obstruction.

  (Occasional wind-ball movement may be experienced in an otherwise normal individual with 'indigestion'.)
- **Diarrhea**?

  Frequency, amount, consistency (watery or semisolid); is it associated with gripping pain in central abdomen; does it contain mucus/pus/fat; does it float in the pan; is it foul-smelling?

  Causes (relevant to 'chronic abdomen')—Disorders of small intestine/large intestine/pancreas; malabsorption syndromes.
- **Constipation**?

  Causes (relevant to 'chronic abdomen')—Greatly reduced food intake (including effect of persistent vomiting); (subacute/chronic) intestinal obstruction; uremia; psychogenic.
- **Abnormal bowel habits / bloating**?—may suggest irritable bowel syndrome, intermittent intestinal obstruction.
- **Dark colored urine, pale stools and itching**? = Features suggestive of obstructive jaundice.
- Present OR recent past history of **jaundice**?

  Causes (relevant to 'chronic abdomen')—Disorders of liver/biliary tract.

C. *Specific symptoms which may give clue about the tissue/organ of origin of the lump*

- Dysphagia? ⇒ Esophagus / esophagogastric junction.

Free ebooks ==> www.Ebook777.com

- Classical symptoms of peptic ulcer? ⇒ Gastrodeudenum.
- Colicky pain ± fullness particularly in the flanks relieved by passing flatus/stool?
  Passage of excessive mucus and/or blood per rectum?
  Passage of altered blood mixed with mucus/pus (especially apart from defecation), and tenesmus?
  Recent alteration of bowel habit?
  } ⇒ Colon/Rectum.
- Fatigue, malaise, myalgia, arthralgia, headache?: Although more or less 'nonspecific', these symptoms ⇒ Hepatitis.
  Jaundice? ⇒ active liver disease.
  Forgetfulness, confusion, poor concentration, personality changes? ⇒ advanced hepatic dysfunction.
- Increased/decreased frequency of micturition, nocturia, urgency, urge incontinence?
  Hesitancy, poor flow, feeling of incomplete emptying, after-dribbling, urethral discharge?
  Pain while passing urine, strangury, pain in the perineum, pain during defecation?
  Hematuria (Red/Brown / stale tea-colored / smoky urine), pyuria, phosphaturia?
  Passage of gravel/stone, fecal matter/gas, or worms?
  Incontinence, enuresis, hematospermia?
  } ⇒ Urinary tract.

D. If **pain** is experienced, ask the details as for 'chronic abdomen' – Refer to chapter 33.

E. H/o **treatment taken** so far and response to it?

ABDOMINAL EXAMINATION (including examination of lump)

INSPECTION

- Movements of the anterior abdominal wall associated with respiratory excursions?
- Contour?
- Asymmetry?
- Visible lump? Note the features of the lump as usual.
- Skin?
- Dilated veins?
- Umbilicus?
- Visible peristalsis?
- Pulsations? – The lump must be demonstrably expansile, i.e. while the anterior surface moves forwards, the right margin of the mass should move to the right, and the left margin to the left.
- Fullness (bulging) of intercostals spaces?

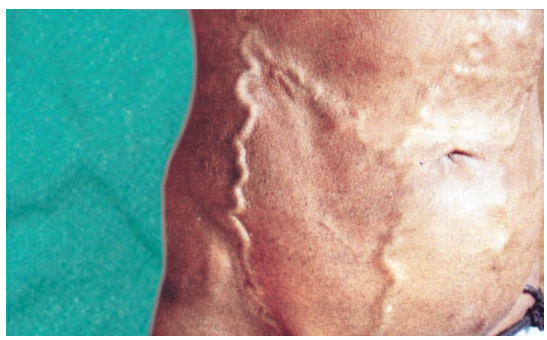


Fig 35-1 Dilated veins due to IVC obstruction

PALPATION

A. First round of palpation—**Gentle 'superficial' palpation** with **flat of the fingers**

1. Hyperesthesia?
2. Abdomen soft, OR contracted?
3. Tenderness?
4. Lump?
5. (Gross) Organomegaly?

B. Next round of palpation—**'Deep' palpation**

www.Ebook777.com

1. Localization of tenderness: Refer to chapter 33.
2. Lump

OR

Circumscribed

Fallacies to be kept in mind, especially in thin patients with lax abdominal wall

- Contracted segment of rectus abdominis
- Prominent sacral promontory
- Fecal masses in the (pelvic) colon
- Gaseous distension of certain viscera like stomach, cecum
- Aorta

Apparently huge, occupying almost the whole abdomen?

Fallacies to be kept in mind

- Fat → deep accentuation of umbilicus
- Fetus: pregnant uterus is easy to diagnose
- Fluid (ascites) →
  - Bulging mainly in the flanks, bulging in the anteroposterior diameter being only slight
  - Transverse stretching of the umbilicus
  - Shifting dullness at the least
- Feces are indentable
- Flatus gives hyperresonant note on percussion

Details of the lump as usual

- Temperature?
- Tenderness?
- Number?
- Site and extent?
  - Does it extend right up/down to the iliac crest / spine / inguinal ligament / femoral region?
  - Site and extent may suggest connection with an organ or a tissue normally situated in that area. Fallacies to be kept in mind: Malrotation of gut, 'malplacement' of viscera such as testicle, kidney; transposition of viscera, visceroptosis, abnormally mobile otherwise normal viscera (gallbladder, spleen, kidney, etc.).
- Margin: - Well-defined OR diffuse?
  - Sharp OR rounded?
- Shape: Does it resemble a particular organ, AND Is it regular (hemispherical, ovoid), OR irregular?
- Size?
- *Keep the palpating hand upon the lump steady for a while and concentrate to feel for the following 3 findings* viz. vascular thrill, pulsation, and hardening of the lump under the palpating hand.
  - Remember that it is normal to be able to feel the pulsations of the aorta in the epigastrium.
  - Hardening (due to contraction) of the lump suggests plain muscle on the surface of the lump which in turn suggests its connection with (practically origin from) either uterus or intestine.
- Impulse-on-coughing? Examples: Hernia (reducible/irreducible), iliopsosas abscess, lumbar abscess. Impulse in case of abscess is rather vague and not convincingly expansile unlike in a hernia.
- Mobility: The direction of mobility is tested during spontaneous (deep) respiration, and the extent of mobility is tested by additional maneuver.

a. <u>Direction of mobility during spontaneous (deep) respiration</u>: Is it craniocaudal, or anteroposterior?

Craniocaudal mobility ⇓

A lump originating from an organ or a tissue in contact with the diaphragm, OR a huge upper abdominal lump in close contact with one of these organs/tissues: liver, stomach, spleen, gallbladder, porta hepatis, lesser omentum, hepatic or splenic flexure of the colon, kidney, lesser sac

Anteroposterior mobility ⇓

Swelling is likely to be in connection with the anterior abdominal wall

Clinical method

Place (lateral border of the) hand parallel to and 'a little away' from the inferior margin of the lump. Press the hand lightly with 'just appropriate' pressure. Ask the patient to take deep breath. If the lump

Free ebooks ==> www.Ebook777.com

moves down (caudally), then it (i.e. its inferior margin) will touch the hand.

b. *Clinical Method* to test the extent of mobility

Grasp the lump with one hand or both hands, and move it in all possible directions—side-to-side, craniocaudal, oblique, and from before backwards (i.e. anteroposteriorly).

Fallacies

i. As the lump is grasped from outside, i.e. including the anterior abdominal wall, movement of the anterior abdominal wall may mistakenly appear as mobility of the lump.

ii. Especially while eliciting side-to-side mobility, when the lump is pushed to make it move, compression of the lump may occur rather than its bodily movement. *Simultaneous movement of both the lateral borders confirms that the lump as a whole is mobile.*

Observations and inferences

i. Freely mobile lump

⇓

– A lump originating from those organs, which are naturally mobile by virtue of having a mesentery, or the mesenteries themselves. Examples—small intestine/its mesentery; transverse colon/transverse mesocolon; pelvic colon/pelvic mesocolon; greater omentum itself; an organ sometimes having unnaturally long mesentery such as ascending and descending colon, or gallbladder.
– Organs sometimes with unnaturally long pedicle. Examples—kidney, spleen.
– Lumps known to have long pedicles. An example—ovarian lump. Also, the degree of mobility is proportional to the length of the pedicle.

ii. Axes in which mobility is free and restricted

– Lump exhibiting more side-to-side mobility as compared to craniocaudal mobility ⇒ the lump possibly related to small intestine/its mesentery.
– Lump exhibiting more craniocaudal mobility as compared to side-to-side mobility ⇒ the lump related to transverse colon/transverse mesocolon, or greater omentum.

iii. The absence of mobility (i.e. 'fixed' lump)

⇓

– Retroperitoneal lump (usually other than the kidney).
– Could be intraperitoneal lump (which initially might have been mobile), having got fixed to the posterior abdominal wall due to postinflammatory adhesions (suggesting inflammatory pathology), or infiltration (suggesting malignancy).

– Anatomical plane and fixity? – This is tested by straight leg-raising test.

www.Ebook777.com

The lump becoming prominent
or/and
The lump remaining palpable to the same extent as before raising the legs
⇓
Lump in the anterior abdominal wall
Exceptions: urachal cyst, abscess in the falciform ligament
Differentiating features in favour of these swellings:
(i) They are easily and superficially palpable
(ii) They move forward along with the anterior abdominal wall during inspiration

The lump becoming less prominent /less palpable/impalpable
⇓
Intraperitoneal / Retroperitoneal lump

- Immobile lump is most likely to be retroperitoneal.
- A lump in the iliac fossa that is intraperitoneal (e.g. an appendicular mass) does not reach up to the iliac crest. Clinically, there is always a gap of at least a finger-breadth between the lateral border of the lump and the anterior superior iliac spine + adjoining portion of the iliac crest
- A bulge in the upper part of the femoral triangle ± cross fluctuation (in the portions of the lump above and below the inguinal ligament) ⇒ a retroperitoneal lump (e.g. iliac/iliopsoas abscess)
- An additional swelling if present in the gluteal region, may suggest iliac bone swelling

- Surface: Smooth, OR Uneven (nodular/irregular)?
  Nodularity ⇒ tumor (benign/malignant) / lymphadenopathy.
  Irregular ⇒ malignancy.
- Consistency: Soft / Firm / Hard / Soft-to-firm / Firm-to-hard / Crepitus?
- Fluctuation?
- Cross-fluctuation - in c/o two cystic swellings apparently communicating with each other?
- Transillumination?
- Moulding? Examples of indentable lumps: dermoid cyst, colon loaded with formed feces.
- Compressibility?
- Reducibility? If reducible, what is the site, size and shape of the defect through which the swelling appears-disappears?
- Surrounding area?

3. Organomegaly
- Liver
- Gallbladder
- Spleen
- Kidneys/Perinephric mass.
- Distended urinary bladder. In adult, normal bladder is neither palpable nor percussable.
  It may be worth attempting palpation of bladder in children and thin-built adults.
  Clinical method: Lift up the lumbar spine with one hand while pressing the other hand into the midline of the lower abdomen.

4. Hernia of the linea alba?
5. Feel for pulsations of the aorta and iliac arteries.
6. Feel for para-aortic enlarged lymph nodes.
7. 'Succussion splash'?
8. Determine the direction of bloodflow in dilated veins, if any.
9. Measurements.

PERCUSSION

1. Determine, in combination with palpation, the size of normal organs viz. liver, spleen and kidneys.
2. Determine the structure (content) of the lump.

Free ebooks ==> www.Ebook777.com

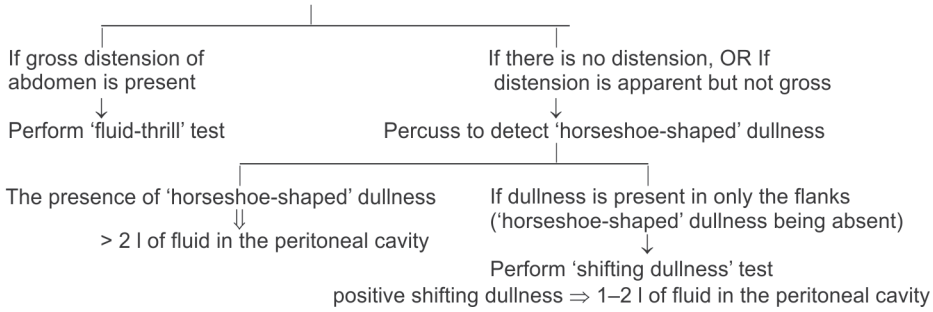
a. Percuss over the lump/distended portion of the abdomen.
   Observation: Resonant OR Dull OR Mixed note (i.e. partly resonant and partly dull)?
   – Resonant note ⇒ lump/coils of intestine filled with gas.
   – Dull note ⇒ lump of solid consistency / lump filled with fluid.
   – **Is the dullness in continuity OR not, with that of the liver or spleen** (as applicable in a given case)?
   – Does the dull note on the posterior aspect merge with paravertebral dullness?
     - Normally, there is always a zone of resonance between the liver/spleen dullness and the paravertebral dullness.
     - Dullness due to renal enlargement always merges with paravertebral dullness with no resonant band in between.
   – A band of resonance overlying (the anterior surface of) the lump which otherwise has a dull note suggests that a loop of intestine must be attached to the lump. Examples—basically a renal lump, a lump in the small intestinal mesentery, or a lump in the mesocolon.

b. 'Hydatid thrill' and 'After-thrill' for hydatid cyst.
   Principle
   A peculiar trembling or vibratory sensation is created due to floating daughter cysts striking against the endocyst.
   Procedure
   – Place 3 fingers upon the lump. The middle finger (pleximeter) is placed firmly while the other 2 fingers (the watching fingers) are placed lightly to ascertain their sensitivity to vibrations.
   – Percuss over the pleximeter.
   – Try to feel for thrill. After feeling the thrill, do not lift the fingers off immediately; instead continue to place them on the swelling for a while for feeling 'after-thrill'.
   'After-thrill' is felt due to continued strikes against the cyst wall by the freely floating daughter cysts set in motion by percussion, even after percussion of the pleximeter is stopped.

3. Percussion is better than palpation for detecting a distended bladder.
   Clinical Method
   Step 1. Begin percussion in the midline from just below the xiphisternum towards the symphysis pubis. Note and mark the point where dull note is perceived. Start percussion again from the same point in the epigastrium and proceed below, but now away from the midline by a few degrees. Keep repeating, but each time progress more and more away from the midline, towards the mid-inguinal point. Repeat the same procedure in the other half. Each time note and mark the points where the note changes to become dull.
   Step 2. Begin percussion from right anterior superior iliac spine towards the symphysis pubis. Note and mark the point where dull note is perceived. Start percussion again from the same point, i.e. anterior superior iliac spine and proceed medially, but now more superiorly by a few degrees. Keep repeating, but each time progress more and more towards umbilicus.
   Step 3. Same as step 2 taking the left anterior superior iliac spine.
   Joining all the marked points will give the border of the distended bladder.
   Notes
   – In adult, normal bladder is neither palpable nor percussable.
   – Bladder can be percussed when it contains at least 150 mL of urine.
   – At a volume of about 500 mL, distended bladder becomes visible in thin-built patients.

4. Detect abnormal collection of fluid/gas in the peritoneal cavity.

www.Ebook777.com



Dullness may also shift in case of a mobile cyst (i.e. cyst with a long pedicle)!
Differentiating feature in favor of cyst (against free fluid): Apart from the fact that the dullness produced by a cyst will have convex borders on all sides, on change of posture, the site of dullness may shift *but the shape remains the same*.

Test to differentiate massive ascites from very large cyst
Place the edge of a ruler over abdomen just above the anterior superior iliac spines and press it firmly backwards.
Transmission of aortic pulsations to the fingers holding the ruler ⇒ cyst.
Ascitic fluid does not transmit aortic pulsations.

AUSCULTATION

- Listen for abnormal peristaltic sounds.
- Listen for abnormal sounds overlying the liver and spleen.
- Listen for bruit overlying the aorta/its main branches.
- Determine the size of (enlarged) stomach – Auscultation with scraping.

PALPATION IN SITTING POSITION with hands locked and resting up on the head (For details, refer to chapter 33)

- Feel for gallbladder tenderness.
- Feel for localized tenderness in rest of the abdomen.
- Any lump palpable now, which was not palpable earlier when the patient was being examined in lying-down position?
- If a lump/organomegaly was noticed earlier, are there any changes in the physical findings with change in position?
- Palpate the kidney.

TRANSILLUMINATION OF LUMP IN THE FLANK particularly in neonates and young children (such lumps are often renal in origin)

Procedure: – Position a flashlight or fiberoptic light source posteriorly against the costovertebral angle.
– Observe from the anterior aspect.

Inference: – A dull reddish glow ⇒ fluid-filled mass. Examples – cyst, hydronephrosis.
The absence of transillumination ⇒ solid mass. However, keep in mind the causes of false negative transillumination.

EXAMINATION OF GROINS AND EXTERNAL GENITALIA

- External iliac and Femoral artery pulsations.
- Inguinal lymph nodes.
- Hernial sites.
- Scrotum.

PELVIC EXAMINATION

- In a male patient: DRE.
- In a female patient: Vaginal examination and DRE.
  - Bimanual palpation is very useful to differentiate lump arising from the uterus, from nonuterine lumps.

Free ebooks ==> www.Ebook777.com

Movement of the lump on moving the uterus suggests origin of the lump from the uterus, e.g. fibroid, tumor arising from the body of the uterus.

- Lump moving independently from the uterus suggests lump originating from tube/ovary/related structure, but not the uterus.

RE-EXAMINE AFTER EMPTYING A HOLLOW VISCUS

For example – after emptying the stomach/urinary bladder.
If the lump is thought to be a distended urinary bladder, re-examine after emptying the bladder (by asking the patient to void, or by catheterization if patient cannot void due to any reason).
Disappearance of lump suggests that the lump was due to distended urinary bladder.
A large diverticulum of urinary bladder may still remain palpable even after micturition. Explanation: The wall of such a diverticulum contains no muscle. ∴ the act of micturition may even make it larger by transferring urine from the contracting bladder to the diverticulum!

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. In neonates 50% of all abdominal lumps are renal in origin.
2. *Causes of abdominal lump in infancy and early childhood*
   - Wilm's tumor of kidney, unilateral polycystic kidney, congenital hydroureter and hydronephrosis.
   - Neuroblastoma of adrenal and sympathetic chain.
   - Ovarian teratoma.
   - Retroperitoneal teratoma.
   - Duplication of ileum.
   - Lymphosarcoma.
   - Hydrometrocolpos in female infants.
   - Choledochal cyst.
   - Retroperitoneal lipoma.
3. As compared to adults, malignant tumors in infants show more rapid local growth due to embryonic nature of tissue, but anemia, loss of weight and cachexia are much less marked.
4. Recurrence and metastasis after removal usually occur within a time period equivalent to the age of infant at the time of operation plus nine months (Collin's law).
5. Symptoms of dysfunction can result from physical compression of critical anatomical structures in the vicinity, or from a specific physiological expression of disease (as for example in case of carcinoid tumor), or both.
6. An exceptionally extraordinarily large but lax (by virtue of fluid being under minimal tension) cystic swelling (e.g. an ovarian cyst, a bladder diverticulum, etc.) may be mistaken for ascites.
7. Multiple lumps in the abdomen usually suggest TB or malignancy.
8. Those lumps which move with respiration are more likely to be diagnosable from clinical signs than those which do not.
9. Apart from the guidance given by overall mobility, it is unlikely that physical examination will help further in the diagnosis of the lumps that do not move with respiration.

www.Ebook777.com

10. Sometimes, pathological nature of a lump may give a clue regarding possible anatomical organ of origin.
11. "A single point cannot make or refute a diagnosis": If a particular point in the data (symptom/sign) goes strongly against the apparently most probable diagnosis, then review the case ignoring that point, and then if that same diagnosis still appears to hold true, then that diagnosis should be accepted in the given case. The particular point in data may be (a) vitiated in history by the patient or in sign by the clinician, OR (b) an unusual manifestation of that disease in the given case.
12. A clinical triad of sudden high output cardiac failure, lower limb ischemia or venous engorgement, and a thrill or murmur over the abdominal mass is diagnostic of an aortocaval fistula.
13. **Organs/tissues from which huge lumps can arise**
    - Liver: Hydatid cyst, secondary carcinoid tumor, etc.
    - Spleen: Congestive splenomegaly, storage disease, tropical splenomegaly, chronic leukemia, myelofibrosis.
    - Stomach: Fibrosarcoma – The rarest and the least malignant tumor. It can attain immense size, and in a female patient, it is liable to be mistaken for an ovarian cyst.
    - Kidney: Hydronephrosis.
    - Ovary: Ovarian cyst.
    - Fat: Lipoma.

Free ebooks ==> www.Ebook777.com

STEP-WISE ANALYSIS OF DATA

Step 1

Acute? or **Chronic?**

Step 2

Anatomical plane, tissue/organ of origin, and pathological nature of the lump?
(Causes of acute abdominal lump)

- **Strangulated Hernia**
- **In the anterior abdominal wall**
  - Pyogenic abscess
  - Hematoma
    - Spontaneous—Bleeding disorder?
    - Rupture of inferior epigastric vessels
  - Rupture of rectus muscle due to straining
    - Tetanus
    - Zenker's degeneration in typhoid
- **Intraperitoneal**
  - Acute inflammation
  - Distension of a viscus
  - Torsion of an organ
  - Displacement of an organ
  - Venous thrombosis
  - Extravasation of blood/urine
- **Retroperitoneal**
  - Iliac abscess
  - Psoas myositis/abscess
  - Iliacus/Iliopsoas hematoma
  - Lymphadenitis / Lymph node abscess
  - Phlebitis of iliac veins

Step 2

Anatomical plane, tissue/organ of origin of the lump?

- **Reducible (hernia)**
  - Occurs through a defect in the parietal wall.
- **Parietal**
- **Intra-abdominal**
  - May be intra-peritoneal or retroperitoneal
    - Visceral / Non-visceral
    - Bony / Soft tissue

Specific

1. A bony swelling arising from pubis
2. Irreducible/Obstructed/Strangulated hernia
3. 'Subumbilical' acute/acute-on-chronic cellulitis/pyogenic abscess
4. In relation to the umbilicus (apart from hernia)
5. In connection to the rectus (sheath): Hematoma, abscess, desmoid tumor
6. In connection with V-I duct OR urachus
7. An intra-abdominal abscess burrowing through the anterior abdominal wall and resembling a parietal abscess
8. Lumbar (cold) abscess resulting from tuberculous osteomyelitis of the spine

General

A. *Clinical clues/tests useful for determining the anatomical plane, tissue/organ of origin of the lump*

- Disturbance of function, if any.
- Exact site and extent.
- Shape.
- Vascular thrill, Pulsations.
- Impulse-on-coughing.
- Movement on respiratory excursions.
- Extent of mobility on active maneuver.
- Straight leg-raising test.
- Consistency.
- Hearing of a bruit/murmur/hum on auscultation.

B. CLUES TO SUGGEST THAT THE SWELLING IS SITUATED IN THE ANTERIOR ABDOMINAL WALL

1. *Impulse-on-coughing* ⇒ essentially an uncomplicated hernia.

www.Ebook777.com

2. Anteroposterior mobility *with respiratory excursions*.
3. *On straight leg raising test*
   - The lump becomes prominent or/and remains palpable to the same extent as before raising the legs.
   - Exceptions: Swellings arising from falciform ligament, V-I duct, urachus, and a complicated hernia.
     Explanation: The swellings arising from falciform ligament, V-I duct and urachus lie deeper to peritoneum, thus virtually in the peritoneal cavity. Hence, they may get less prominent on straight leg raising. These swellings are preferably included in the group of anterior abdominal wall swellings as they are practically fixed to the posterior surface of the anterior abdominal wall even if they lie deeper to peritoneum. They move anteroposteriorly with respiratory excursions.

C. CLUES TO SUGGEST RETROPERITONEAL LOCATION OF THE LUMP

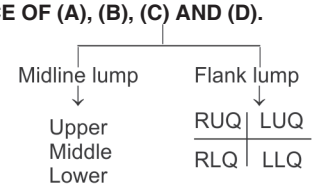
- The absence of GIT and urinary symptoms.
- Immobility.
- In case of a lump in the iliac fossa such as iliac abscess, the lump will extend up to iliac crest.
- Lower limb edema, ascites, dilated abdominal wall veins and varicocele suggest advanced pathology.

D. IN CASE OF INTRAPERITONEAL/RETROPERITONEAL LUMP: ORGAN/TISSUE OF ORIGIN?

Principles

1. A lump may arise from (a) any of the **structures that are normally present in that region**, OR (b) any other **structure abnormally present in that region**.
   Examples of abnormal positions of organs
   - Undescended **enlarged** testicle in the lower quadrant.
   - Liver, gallbladder and splenic lumps may be encountered in lower abdomen.
   - Floating kidney in the lower quadrant.
   - Horseshoe kidney in lower midline / front of the sacrum.
   - Cecum in RUQ.
   - In visceroptosis, stomach, flexures of colon and other parts of the colon may descend much lower in the abdomen.
   - Situs inversus.
   - Accessory spleen, ectopic pancreatic tissue, endometrial tissue.
2. (According to location) – Is the lump located in the **Midline / Flank**?
   'Midline' lump = lump in the midline extending more or less equally on either side, and flanks are empty.
   'Flank' lump = lump mainly situated in (one) flank, and *usually* does not extend beyond the midline.

E. COMING TO A CONCLUSION BY COMBINING THE INFERENCE OF (A), (B), (C) AND (D).



UPPER MIDLINE LUMP

Hernia	*Specific parietal swelling*	*Intraperitoneal / retroperitoneal lump*

Free ebooks ==> www.Ebook777.com

– Epigastric – Incisional	– A swelling in connection with falciform ligament – Cold abscess arising from caries of the ribs (commonly) or spine (rarely) – Basically a hepatic/subphrenic/perigastric abscess burrowing through the anterior abdominal wall and resembling a parietal abscess (virtually a collar-stud abscess)	– *Vascular*: Aorta/major branches – Moving craniocaudally with respiration: Stomach, liver, porta hepatis, lesser omentum – Moving anteroposteriorly with respiration: In connection with falciform ligament – Pancreas, Lesser omentum – Lesser sac, Subdiaphragmatic space

MIDDLE MIDLINE LUMP

Hernia	*Specific parietal swelling*	*Intraperitoneal / retroperitoneal lump*
– Umbilical – Paraumbilical – Incisional	– 'Subumbilical' acute/acute-on-chronic cellulitis/abscess: (a) de novo, (b) Infected enterocyst or urachal cyst – In relation to the umbilicus (apart from hernia): (a) Bud, (b) Granuloma, (c) Endometrioma, (d) Malignant tumor – In connection to the rectus (sheath): (a) Hematoma, (b) Abscess, (c) Desmoid tumor. – In connection with V-I duct: Enterocyst – In connection with urachus: Urachal cyst	– *Vascular*: Aorta / major branches – Moving craniocaudally with respiration: Stomach, liver, porta hepatis, lesser omentum – Moving anteroposteriorly with respiration: In connection with urachus, e.g. cyst, abscess – Pancreas, transverse colon/transverse mesocolon, greater omentum, small intestine/its mesentery, lymph nodes – *Abnormally present organ*—Horseshoe kidney

LOWER MIDLINE LUMP

Hernia	*Specific parietal swelling*	*Intraperitoneal / retroperitoneal lump*
Incisional	– Disease of the pubic bone like tumor, osteitis, etc. – 'Infraumbilical' acute/ acute-on-chronic cellulitis/ abscess: (a) de novo, (b) Infected enterocyst or urachal cyst – In relation to the rectus (sheath): (a) Hematoma, (b) Abscess, (c) Desmoid tumor – In connection with urachus: Urachal cyst	– Could be pelvic (emerging out of the true pelvis) or abdominal (i.e. nonpelvic) If the lower border is not palpable, i.e. if it is not possible to 'get below the lump' ⇒ Pelvic lump – (Otherwise normal) Transverse colon/transverse mesocolon/ greater omentum may be present in the lower abdomen, if mesocolon is excessively long – Urinary bladder, swelling in connection with urachus – Uterus, uterine tubes, broad ligament, ovary – Pouch of Douglas – Presacral connective tissue (Examples – teratoma, cystic hygroma) – Wall of the pelvis – Rarely prostate, rectum – *Abnormally present organ*—Horseshoe kidney (in front of the sacrum), greatly distended or visceroptotic stomach

RUQ LUMP

Hernia	*Specific parietal swelling*	*Intraperitoneal / retroperitoneal lump*

www.Ebook777.com

Incisional	– Cold abscess arising from caries of the ribs (commonly) or spine (rarely) – Basically a hepatic / subphrenic / perigastric abscess burrowing through the anterior abdominal wall and resembling a parietal abscess	– Moving cranio-caudally with respiration: Liver, gallbladder, stomach, hepatic flexure of colon – Ascending colon, kidney, suprarenal, head of pancreas, retroperitoneal connective tissue – Subdiaphragmatic space – *Abnormally present structure*—Cecum

LUQ LUMP

Hernia	*Specific parietal swelling*	*Intraperitoneal / retroperitoneal lump*
Incisional	Same as in the RUQ	– Moving craniocaudally with respiration: Stomach, spleen, splenic flexure, liver – Descending colon, kidney, suprarenal, pancreas, retroperitoneal connective tissue – Subdiaphragmatic space, lesser sac *A pitfall*: Occasionally, a mass in the left lobe of liver projects downwards and is palpable only to the left of the midline while the RUQ feels normal. In this circumstance liver may easily be mistaken for spleen. Particular difficulty may be experienced when the spleen and liver are simultaneously palpable

RLQ LUMP

Hernia	*Specific parietal swelling*	*Intraperitoneal / retroperitoneal lump*
– Inguinal – Spigelian	– An iliac abscess (pyogenic)/appendicular abscess burrowing through the anterior abdominal wall and resembling a parietal abscess – Inguinal swelling (apart from hernia) – In connection with peritoneum	– Peritoneum – Ileocecal junction (terminal ileum, cecum and appendix), mesenteric lymph nodes – A large diverticulum of urinary bladder: The lump does not disappear on micturition! – Ovary, uterine tube, broad ligament, pedunculated fibroid of uterus – Ascending colon, kidney, retroperitoneal connective tissue including lymph nodes, iliopsoas muscle, iliac artery, iliac bone – *Abnormally present structures*—Undescended **enlarged** testis, floating kidney, liver, gallbladder

LLQ LUMP

Hernia	*Specific parietal swelling*	*Intraperitoneal / retroperitoneal lump*
– Inguinal – Spigelian	Same as in the RLQ	– Peritoneum – Sigmoid colon/its mesocolon – A large diverticulum of urinary bladder: The lump does not disappear on micturition! – Ovary, uterine tube, broad ligament, pedunculated fibroid of uterus – Descending colon, kidney, retroperitoneal connective tissue including lymph nodes, iliopsoas muscle, iliac artery, iliac bone – *Abnormally present structures*—Undescended **enlarged** testis, floating kidney, floating spleen

Free ebooks ==> www.Ebook777.com

F. CRITERIA FOR INDIVIDUAL ORGAN LUMPS

Note: All the classic criteria may not be apparent in every case.

Hepatomegaly / localized hepatic lump

– Intraperitoneal lump in the RUQ (can be in upper midline).
– In close contact with the anterior abdominal wall. Fingers cannot be insinuated between the lump and the costal margin. Upper border—not palpable.
– Sharp (inferior) margin.
– Shape conforms to that of the liver.
– Moves craniocaudally with respiratory excursions.
– Dull note on percussion, and dull note of the lump continuous with the liver dullness.

And symptoms and (additional) signs of liver dysfunction.

Note: Surface and consistency vary according to pathology.

Causes of normal liver palpable below the costal margin:

– In infants.
– Altered local anatomy: Ptosis of the liver / Low diaphragm / Rickety deformity of the chest.
– Some pathology pushing the normal liver downward: Subdiaphragmatic abscess / Pleural effusion.

Gallbladder lump

– Intraperitoneal lump in the RUQ, extending towards the umbilicus.
– In close contact with the anterior abdominal wall.
– Margin – rounded.
– Shape – globular / ovoid.
– Moves craniocaudally with respiratory excursions.
– 'Pendulum movement, i.e. the lower pole moves freely across than the upper pole (applicable only if the lump is large).
– Dull note on percussion, and dull note of the lump continuous with the liver dullness.

+ Symptoms and (additional) signs suggestive of gallbladder disease.

Splenomegaly / Splenic lump

– Intraperitoneal lump in the LUQ, extending towards the umbilicus, and subsequently RLQ with oblique long axis.
– In close contact with the anterior abdominal wall. Fingers cannot be insinuated between the lump and the costal margin. Upper border—not palpable.
– Sharp (anterior) border with a notch.
– Moves with respiration.
– Dull note on percussion.
– (Differentiating point against renal lump: Splenomegaly does not cause fullness in the renal angle).

± Symptoms and (additional) signs suggestive of the underlying disorder causing splenomegaly.

Symptoms due to splenomegaly per se

– Pain: - Nature – chronic dragging or attacks of acute colicky pain.
  - may be experienced only when lying on one side.
– Abdominal discomfort.
– Early satiety.

Important point to note

The symptoms of splenomegaly itself (irrespective of cause) do not correlate with size, but in practice they tend to be marked only when the spleen becomes massively enlarged since most are due largely to mechanical displacement of adjacent organs and weight of the congested spleen.

A pitfall

Occasionally, a mass in the left lobe of liver projects downwards and is palpable only to the left of the midline while the RUQ feels normal. In this circumstance liver may easily be mistaken for spleen. Particular difficulty may be experienced when the spleen and liver are simultaneously palpable.

www.Ebook777.com

Gastric lump (practically pyloric)
– Intraperitoneal.
– Visible peristalsis (in chronic obstructive pathology).
– Margin – rounded.
– Shape – like that of greater curvature (applicable in c/o distension caused by chronic obstructive pathology).
– Moves (moderately) craniocaudally with respiratory excursions.
– Moves slightly across the axis of the stomach.
– Succussion splash (in chronic obstructive pathology).
– Resonant note on percussion upon a distended stomach.
+ Symptoms and (additional) signs suggestive of gastric disease.

Ileocecal lump
– Intraperitoneal lump in the RLQ.
– Shape – vertically ovoid, or round.
– Contractions (of smooth muscles) palpable (rarely).
– Slightly mobile (if no adhesions/infiltration).
± Symptoms and (additional) signs suggestive of ileocecal disease (including appendicular).

Transverse colon lump
– Intraperitoneal lump in the middle midline (umbilical region); occasionally in the upper and rarely in the lower midline.
– Upper border—felt separately from the liver by a band of resonance in between.
– No movement with respiration.
– More freely mobile in craniocaudal axis as compared to transverse axis.
± Symptoms and (additional) signs suggestive of large intestinal disorder/obstruction.

Sigmoid colon lump
– Intraperitoneal lump in the LLQ.
– Margin – rounded.
– Moderately mobile.
± Symptoms and (additional) signs suggestive of large intestinal disorder/obstruction.

Lump arising from **head of the pancreas**
– A lump situated above and to the right of the umbilicus.
– Upper border—felt separately from the liver by a band of resonance in between.
– No movement with respiration. Fixed.
± Symptoms and (additional) signs suggestive of pancreatic disorder.



Fig 35-2 Chronic retention of urine

Greater omental lump
– Intraperitoneal lump (usually) below the umbilicus.
– No movement with respiration.
– Otherwise, freely mobile. However, more freely mobile in craniocaudal axis as compared to transverse axis.

Mesenteric lump
– Intraperitoneal lump (usually) below the umbilicus.
– Margins – rounded.
– No movement with respiration.
– Otherwise, freely mobile. However, more freely mobile in the direction of the axis that is at right angles to the long axis of the root of mesentery, whereas extent of mobility is comparatively less along the long axis

Free ebooks ==> www.Ebook777.com

of the root of mesentery.
– A zone of resonance around, and classically, a belt of resonance across the cyst (applicable to a cyst of course).

Renal lump
– Visible fullness in the renal angle (For this, the kidney has to be at least 'moderately' enlarged).
– A lump in the loin.
– Vertically ovoid.
– Rounded margin.
– Part of the upper pole **may** be felt on deep inspiration.
– Slight downward movement with respiration.
– Ballottable.
± Symptoms and (additional) signs suggestive of urinary tract disorder.

Retroperitoneal lump, (other than renal/pancreatic)
– Site: - Connective tissue lumps, adrenal tumors are usually located in one of the flanks.
- Midline lump/s extending more on one side than the other, with nodular surface ⇒ para-aortic lymphadenopathy.
– Fullness posteriorly in the loin, but not exactly in the renal angle.
– Margin – rounded.
– Mobility – absent.
– Area of resonance in front.
– No symptoms primarily pertaining to GIT and urinary tract.

Pelvic lump in general
– Site—hypogastrium.
– Upper and lateral borders palpable, but the lower border not palpable, even by pushing the lump upwards, i.e. it is not possible to 'get below the lump'.
– Mobility – more from side-to-side and downward. Upper pole moves more than the lower part ('pendulum movement').
– Dull note on percussion.
– Palpable bimanually (on DRE/PV/E).

Distended urinary bladder
– Basically a globular pelvic lump in the midline just above the pubis, and may reach up to the umbilicus.
– Tender.
– Feels 'elastic'.
– Pressure induces a desire for micturition.
– Dull on percussion.
– Disappears on catheterization. Hence, if imaging diagnostic facilities are not available, catheterization must be insisted upon before attempting to make a clinical diagnosis.

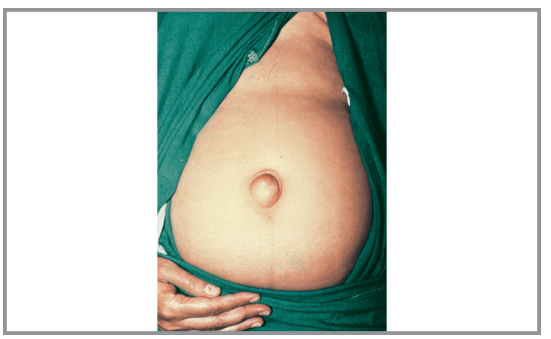


Fig 35-3 A small paraumbilical hernia

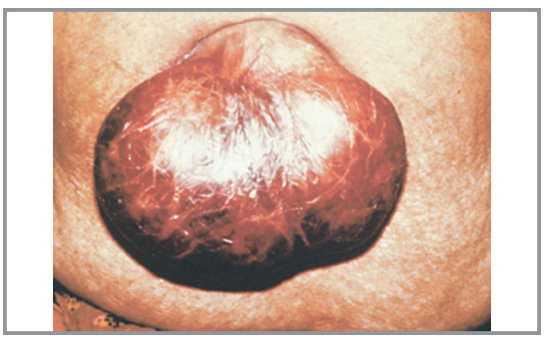


Fig 35-4 A paraumbilical hernia of 30 years duration!

www.Ebook777.com

A pitfall
Chronic retention may be associated with a distended bladder even though the patient may give h/o passing urine regularly (which in fact is due to overflow incontinence). Patient may be ignorant about the condition, and may look cheerful even with the bladder distended up to the umbilicus.

Urinary bladder lump
Basically a pelvic lump in the midline just above the pubis.
Does not disappear on catheterization.
Examples – tumor, (huge) stone, a large diverticulum.

Uterine lump
Basically a globular pelvic lump in the midline just above the pubis.
– Consistency much harder than that of a distended bladder.
– Contraction of involuntary muscles may be felt.
– On PV/E – the lump moves with the movements of cervix, and vice versa.

Broad ligament lump
Basically a pelvic lump; remains on one side of the midline.
– Not mobile.
– (Fullness in the fornix).
– If huge, then distance between the umbilicus and the anterior superior iliac spine is increased on the affected side.

Ovarian lump
Basically a pelvic lump; remains on one side of the midline.
– May be tender.
– Freely mobile.
– Distance between the umbilicus and the anterior superior iliac spine is increased on the affected side.
– Moves independently of the uterus.
– **May be wholly abdominal.**

Step 3

Pathological nature of the chronic lump?—Causes of lump, **including differential diagnosis of common disorders is discussed below.**

PARIETAL SWELLINGS

ABDOMINAL WALL HERNIA

– A h/o reducible/irreducible swelling usually with an insidious mode of onset.
– H/o exciting factor viz raised intra-abdominal pressure either continuously or intermittently.
  Occupation involving strenuous work / 'chronic' constipation / 'chronic' straining-type of difficulty in micturition / a big lump in the abdomen / ascites / chronic bronchitis.
– Expansile impulse-on-coughing.
– Peristalsis (visible across the skin) in case of an enterocele.
– Hernia may be controlled on occlusion of the defect;—the test applicable only to a reducible hernia.

Epigastric hernia
Differentiating points
– A small nodule in the midline, which is more prominent in standing position.
– Impulse-on-coughing often absent (suggesting herniation of only extraperitoneal pad of fat).
Supporting point: Strong muscular laborer.
Notes
– Usually encountered in males > 40.
– Can be multiple.

Free ebooks ==> www.Ebook777.com

- Mostly otherwise asymptomatic.
- A small number of patients present with vague upper abdominal symptoms in whom repair of the hernia provides relief.
- The hernias larger than 2 cm have the potential of strangulation.

Paraumbilical hernia

- More common in females, and largely confined to obese patients. It can occur in children.
- The defect lies just above or below the umbilicus.
- The earliest clinical manifestation is deformity of the umbilical button.
- The hernia may enlarge, but the neck remains dangerously small so that the risk of strangulation is ever present.
- Chronicity leads to firm adhesions between the sac and the contents so that almost all large hernias are irreducible.

Infantile (simple) umbilical hernia

- When the infant lies quietly, the only abnormality noted is redundancy of umbilical skin, but on crying or straining, intestine fills the hernia and the swelling becomes considerably larger, more tense and bluish beneath the thin shiny skin.
- The content (intestine) can be easily reduced, often with an audible gurgle.
- What is the size of the defect at the age of 1 year?
  - < 1 cm suggests that eventual spontaneous closure is very likely.
  - 1–2 cm suggests that closure is less likely, but it is still reasonable to wait until 3 years of age.
  - > 2 cm suggests that spontaneous closure will almost certainly not occur, and surgery at any time in the 2nd or 3rd year is reasonable.

Complications: 1. Incarceration / obstruction. Strangulation is virtually unknown.
2. Very rarely, rupture with partial evisceration, necessitating urgent intervention. A

Practical points which must be explained to the parents

- The hernia getting tense when the baby cries is not necessarily an indication of pain in the hernia.
- In the 1st 3–4 months of life the bulge may actually increase a little, but thereafter diminution can be expected. Maximum resolution occurs in the first year, and the process continues to a lesser degree in the subsequent years.
- The skin usually does not rupture. The thin skin of the first 4 weeks following birth becomes gradually thicker.

The worst umbilical defect is 'exomphalos'; it is frequently associated with other congenital defects.

Adult umbilical hernia

- Develops in patients with refractory ascites due to chronic liver disease.
- Possible complication: Incarceration / obstruction / strangulation.

Problems with respect to management

1. Marked atrophy of the abdominal muscles. B C
2. Large high-pressure veins in the abdominal wall.
3. Possible complication of infection of ascitic fluid in the postoperative period.

Paraumbilical hernia	*v/s*	*Umbilical hernia*
– A defect in the linea alba is separate from but adjacent to the umbilicus		The defect is in the umbilical cicatrix itself
– The defect is transverse slit/ellipse-like, with sharp edges		The defect is round-shaped, and the edge is blunt

Incisional hernia

- Symptoms: - Discomfort, pain.
  - Cosmetic disfigurement.
- The minimum covering of the hernia could be only the peritoneum and the skin.
- Usually it has a wide neck. Reduces readily on lying down and reappears readily on standing.
- Risk of complication – generally low. Nevertheless, can get complicated if it has been present for a long

www.Ebook777.com

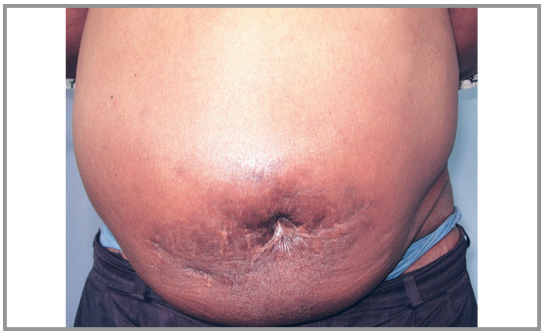


Fig 35-7 A large central incisional hernia, recurred thrice!

duration.

– The term 'ventral hernia' should be restricted to incisional hernia arising in abdominal midline operative wounds.
– *Risk factors for the development of an incisional hernia*
  - Age > 70, obesity, especially morbid obesity; anemia, jaundice, malignancy, COAD, steroid dependence.
  - Type of incision: longitudinal more prone than transverse; incision > 18 cm; multiple incisions.
  - Operation for peritonitis.
  - Factors operative in the postoperative period: wound or chest infection, distension, 'burst abdomen'.



Fig 35-5 A large incisional hernia – Got incarcerated due to exceptionally long duration of many years. Note skin ulceration resulting from pressure necrosis

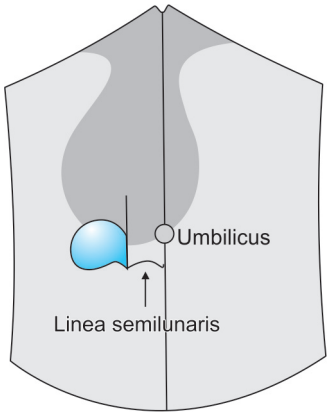
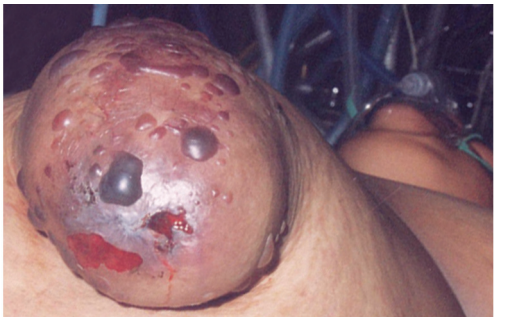
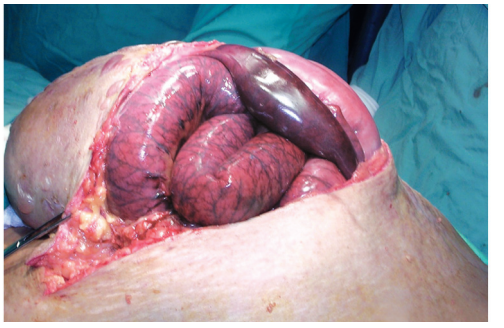


Fig 35-8 Spigelian hernia

Free ebooks ==> www.Ebook777.com







Figs 35-6A to C A strangulated huge incisional hernia

Large central incisional hernia (usually recurrent)
= a type of massive midline abdominal wall defect.
– Size of the defect could be ≥ 6 × 14 cm.

Causes of a massive midline abdominal wall defect other than a large central incisional (recurrent or otherwise) hernia
– Removal of infected synthetic mesh material.
– Removal of split-thickness skin graft and dense abdominal wall cicatrix.
– Trauma.
– Abdominal wall sepsis.
– Parastomal hernia.
– Abdominal wall tumor resection.

Parastomal hernia (= a specific type of basically incisional hernia)
– The term includes hernia related to colostomy, ileostomy, urinary conduit.
– It can occur many years after creation of a stoma.
– More likely to occur in elderly, and in patients with other abdominal wall herniae.
Modes of presentation:
– A bulge.
– Ill-fitting bag.
– Leakage.
– Acute, usually small intestinal obstruction.
Port-site hernia (= a specific type of basically incisional hernia)
Risk factors:
– Size of port > 10 mm.

www.Ebook777.com

– Obesity.
– Inadequate fascial closure.

In obese patients the small hernia can be easily overlooked as a cause of (small) intestinal obstruction.

Spigelian hernia

Clinical presentation is variable, according to the contents and the size/type of herniation.

– Pain is the most common symptom; intensity and nature may be variable.
– Some patients present acutely with a tender irreducible mass or intestinal obstruction.

Spigelian hernial orifice lies in the aponeurosis of oblique muscles of the anterior abdominal wall at the lateral edge of the rectus sheath, below the linea semilunaris. The defect is usually quite small and, if it lies in the upper part of this region, it is easily distinguished from the external inguinal ring; the lower it lies however, the more likely is confusion to arise (with an inguinal hernia).

Lumbar hernia

Gains exit through the lumbar triangle.
DD: Refer to chapter 27.

UMBILICAL SWELLINGS other than hernia

Umbilical bud (*Synonym* Umbilical **adenoma/polyp/raspberry tumor/enteroteratoma**)—Refer to chapter 81.
Umbilical granuloma—Refer to chapter 81.

Endometrioma

– Women between 20 and 45.
– At each period, the umbilicus becomes painful, and bleeds, and the mass between the folds of the umbilicus becomes more apparent.
– May be associated with endometriomas in the uterus/ovaries.

Pathologic anatomy

Ectopic endometrial glands occupy the same plane in the dermis as the sudoriferous glands, and open on to the surface in the same way.

Umbilical malignant tumor

– *Secondary carcinoma*: It is always a late manifestation of malignant disease.
– Rarely, *a primary malignant tumor* may occur like squamous cell CA, malignant melanoma.

'SUBUMBILICAL' SWELLINGS

Enterocystoma / Urachal cyst

Move anteroposteriorly with respiratory excursions.
Patient usually presents when the swelling gets complicated by acute cellulitis/pyogenic abscess.

RECTUS SHEATH SWELLINGS

Rectus sheath haematoma

Basically, an acute swelling with well-localized pain, following a precipitating cause, ± mild fever.

Precipitating causes

1. Surgery (particularly like laparoscopic).
2. Indirect trauma such as strenuous exercise, sudden strain, or paroxysm of coughing.
3. An elderly patient, particularly on anticoagulant treatment, having received a trivial injury.

Differentiating points:

– Tender swelling in the anterior abdominal wall (invariably) located on one side below the umbilicus.
– ~ 5 cm in diameter, though much larger hematomas may be encountered.

Notes:

– A spontaneous hematoma (i.e. the one that follows indirect trauma) can be misdiagnosed for a vascular tumor.
– Hematoma develops from rupture of an inferior epigastric vessel (usually artery).

Free ebooks ==> www.Ebook777.com

Abdominal desmoid tumor

- Parous woman.
- Site – anterior abdominal wall (anterior rectus sheath), usually inferior to the level of umbilicus.
- The lump: Craniocaudally ovoid. Restricted mobility. Consistency – firm or hard.

LUMBAR (COLD) ABSCESS

- Diffuse hemispherical swelling.
- Impulse-on-coughing present (but the impulse is rather vague).
- May be partially reducible.
- Features of chronic osteomyelitis of spine: deformity, rigidity, tenderness.
- X-ray of the spine may show features of tuberculous osteomyelitis.

May have to be differentiated from a lumbar hernia.	*Lumbar hernia*	*Lumbar cold abscess*
– Impulse-on-coughing	Expansile	Rather vague
– Reducibility	Completely reducible, unless complicated	May be only partially reducible

LIVER

'Key points' to be considered for ARRIVING AT THE DIAGNOSIS of hepatomegaly:

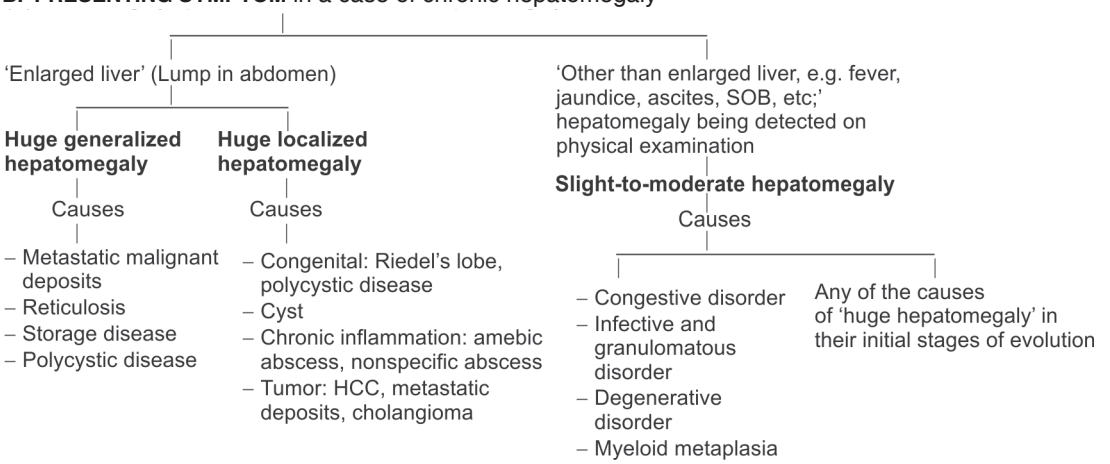
- Acute OR chronic?
- Presenting symptom – 'lump in abdomen' (due to enlarged liver), OR other than lump in abdomen?
- Morphology of hepatomegaly – huge, generalized, localized or otherwise?
- Is generalized hepatomegaly associated with jaundice or not?
- Surface of the enlarged liver?
- Hepatomegaly alone OR hepatosplenomegaly? Causes of hepatosplenomegaly are more specific.

Clues obtained from these key clinical points can be combined in various ways to arrive at the diagnosis, as discussed below.

A. CAUSES OF ACUTE HEPATOMEGALY

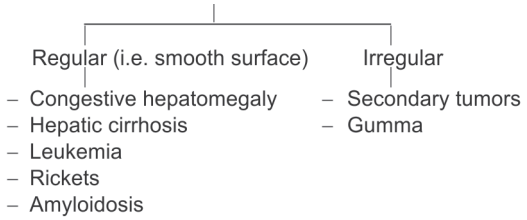
- Acute inflammation.
- Acute (Rapid) congestion.

B. PRESENTING SYMPTOM in a case of chronic hepatomegaly

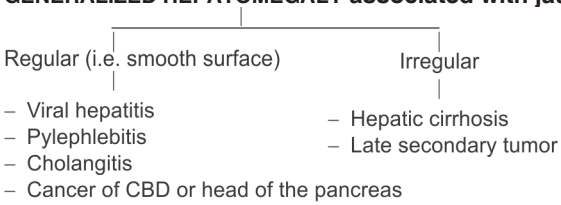


C. GENERALIZED HEPATOMEGALY WITHOUT JAUNDICE

www.Ebook777.com



GENERALIZED HEPATOMEGALY associated with jaundice



D. SURFACE OF THE ENLARGED LIVER

- **Smooth uniform** surface ⇒ liver engorged with blood or bile, or diffuse infiltration.
  - Engorgement with blood refers to congestive hepatomegaly.
  - Engorgement with bile refers to jaundice.
  - Examples of infiltration: fatty infiltration, reticuloses, some unusual diseases of unknown aetiology in which granulomatous lesions develop with a proliferation of histiocytes. The diagnosis of these conditions is confirmed on liver biopsy.
- **Finely nodular surface** suggests biliary cirrhosis.
- **Coarsely nodular surface** suggests alcoholic cirrhosis or multiple secondary deposits.
  - A h/o chronic (heavy) alcoholism / stigmata of liver failure ⇒ alcoholic cirrhosis.
  - Replacement of liver tissue by malignant deposits has to proceed almost to completion before hepatic failure results.

Further differentiation of each individual condition causing chronic hepatomegaly can be done by interrogative history, repeat physical examination and investigation.

DIFFERENTIAL DIAGNOSIS of common disorders

Acute hepatitis

Basically, acute inflammatory abdomen involving the RUQ, ± pain in the back, shoulder.

- Fever.
- Symptoms of hepatic dysfunction: - Anorexia, fatigue, malaise, myalgia, arthralgia, headache.
  - Jaundice.
- Coughing during deep inspiration.
- Gray complexion.
- Relative bradycardia.

Differentiating points

- Anorexia, nausea.
- Dark urine.
- Jaundice: - Marked in viral hepatitis and yellow fever. Slight in rest of the conditions.
- Hepatomegaly: - Localized in case of abscess. Slight generalized in rest of the conditions.

Supporting points

- Pruritus.
- Pale stools.
- The etiological background whenever apparent.

Causes of acute hepatitis

Free ebooks ==> www.Ebook777.com

1. Alcohol.
2. Drug-induced: – Hypersensitivity hepatitis: Sulfonamide, Largactil, PAS, Methyltestosterone, Butazolidin.
   – Toxic hepatitis: Halothane, Arsenic and phosphorous poisoning.
3. Hepatitis associated with systemic infections: Typhoid, pneumonia, septicemia, brucellosis.
4. Viral hepatitis: – A and E—Transmitted enterally. Self-limited.
   – B, C and D—Transmitted parenterally. May prolong and progress to chronic disease.
   – Infectious mononucleosis.
   – Yellow fever.
5. Amebic hepatitis/abscess.
6. Bacterial hepatitis: suppurative pylephlebitis, suppurative cholangitis.
7. Parasitic hepatitis: liver flukes.

Acute pyogenic liver abscess(es)

Refer to chapter 34.

Chronic pyogenic liver abscess (usually single)

– Tender hepatomegaly in a patient complaining of fever and discomfort/pain in abdomen.
– Mode of onset: insidious, or even silent (cryptogenic abscess).
– Clinical features of primary infective focus commonly in intestinal/biliary tract ±.
– The etiology is unexplained in half of the patients.
– Elderly are more prone.

Amebic liver abscess

Can be acute or chronic.

Refer to chapter 34.

Acute pylephlebitis

= Septic inflammation of the wall of the portal vein or its major radicals.

Basically, tender hepatomegaly.

Differentiating points

– Background of primary focus of sepsis like acute appendicitis, inflamed piles, etc.
– High grade fever with rigors.

Acute congestive hepatomegaly

Basically,

– Pain in the RUQ.
– Tender slight generalized hepatomegaly.
– Dependent edema.
– All other criteria of acute hepatitis *absent*.

Differentiating points

– Prominent and engorged neck veins
– Basal crepitations in the lungs
– Evidence of primary cardiac ailment

} ⇒ congestive cardiac failure.

– In acute Budd-Chiari syndrome, heart may be absolutely normal.

Causes of acute congestive hepatomegaly: 1. Congestive cardiac failure. 2. Acute Budd-Chiari syndrome.

Acute form of Budd-Chiari syndrome

Basically present with severe acute pain in abdomen and vomiting.

Differentiating points

– Ascites.
– Large tender hepatomegaly.

Predisposing factors

– Women.
– Oral contraception.

www.Ebook777.com

– Hematological disorders like hemolytic anemia, polycythemia, etc.
– Collagen disorders like rheumatoid arthritis, sarcoidosis, SLE.

Additional features of the underlying malignancy may be present in case if the syndrome occurs secondary to malignancy.

The condition may progress rapidly over a few weeks to hepatic failure, coma and death.

Riedel's lobe

Likely to be mistaken for an enlarged gallbladder.
Riedel's lobe is more wide and flat in contour, and lacks the spherical outline of a distended gallbladder.

Hydatid cyst

H/o often an otherwise asymptomatic lump in upper abdomen for anything from 6 months to a few years.
No constitutional disturbances except for occasional allergic episodes like itching, rash, edema, etc.
Mild jaundice $\pm \Rightarrow$ involvement of biliary radicles.
Pain and fever may suggest secondary infection.

Differentiating points

Characteristics of the lump:

– Spherical.
– Surface—smooth.
– Consistency—firm.
– Fluctuation $\pm$.
– 'Hydatid thrill' = Peculiar trembling/vibratory sensation felt on **palpation** of superficially located cysts; not possible in deep-seated cysts.
– 'After-thrill'.
– Translucent. More superficially situated cysts are particularly brilliantly translucent.

Supporting points:

– H/o attacks of urticaria $\pm$.
– Patient staying in the area known to be endemic for hydatid disease.

Notes:

– Patients may remain symptomless for years. They generally seek medical consultation when they experience discomfort or pain in the region of the lump.
– Can attain huge size.
– An infected cyst may simulate amebic liver abscess or even a clinical picture of 'severe acute abdomen'.

A solitary traumatic cyst

– Symptoms are delayed weeks or months after the trauma.
– Pain in the abdomen, frequently referred to the shoulder.
– Lump.
– Jaundice $\pm$.

Congenital polycystic disease

– Slowly progressing generalized irregular hepatomegaly in adult life.
– May be associated with polycystic kidneys.
– Causes of death: - Renal or hepatic failure.
  - Complications of portal HT.

Gumma

Enlargement is seldom as great as in a case of carcinoma.
Jaundice and ascites usually do not occur.

Hypertrophic type of cirrhosis

– Characteristics of the enlarged liver: Surface—irregular. Consistency – firm.
– Manifestations of portal HT: - H/o one or more episodes of hematemesis.
  - Ascites.
  - 'Caput medusae' around the umbilicus.

Free ebooks ==> www.Ebook777.com

- Jaundice ±.

Primary malignant tumor of the liver

Clinical manifestations:

– A lump in abdomen which produces dragging sensation or painful discomfort in the RUQ on exercise.
– General symptoms s/o chronic liver disease viz. anorexia, nausea, vomiting, fever, loss of weight, weakness.
– Jaundice and peripheral stigmata of chronic liver disease.
– Rarely, change in bowel habit.
– Ascites.
– Hepatomegaly.
– Bruit (10% cases).

Infrequent features: hypoglycemia, hypercalcemia, hyperlipidemia, hyperthyroidism.

Notes:

– **Due to fever, in tropical countries, the tumor may be mistaken for an amebic abscess.**
– ∝-1-fetoprotein (AFP) may be present in the plasma long before clinical features appear.

SPLEEN

The size of a normal spleen: ~ 12 × 7 cm.

The spleen is inaccessible to palpation when normal in size and shape.

Causes of splenomegaly can be classified in various ways.

A. Clinical classification: Acute, or Chronic.

Causes of acute splenomegaly

1. Acute Infections: viral hepatitis, infectious mononucleosis, typhoid, toxoplasmosis, typhus fever, tularemia, cytomegalovirus, septicemia, brucellosis, SBE, abscess.
2. Acute (Rapid) congestion: infarct, torsion, rupture.

B. *Causes of* **huge splenomegaly**

– Congestive splenomegaly.
– Storage disease.
– Malaria, kala-azar, schistosomiasis, trypanosomiasis.
– Chronic leukemia.
– Myelofibrosis.

C. Associated with **hepatomegaly**: Refer to page 523.

D. Associated with **generalized lymphadenopathy**

– AIDS, TB, infectious mononucleosis, syphilis, sarcoidosis.
– Leukemia, lymphosarcoma.

E. Splenomegaly **of surgical interest**

1. *Causes of splenomegaly of surgical importance ('Definite indications for splenectomy in appropriate situations')*
   – Tumor of the spleen, usually lymphosarcoma.
   – Splenic abscess.
   – Hydatid cyst.
   – Bleeding gastric varices due to sinistral portal HT (splenic vein thrombosis).
2. *'Hematological' causes of splenomegaly in which splenectomy is considered selectively ('Desirable indications for splenectomy')*
   – Hereditary spherocytosis/elliptocytosis.
   – Immune (idiopathic) thrombocytopenic purpura.
   – AIDS related thrombocytopenic purpura.
   – Autoimmune hemolytic anemia.
   – Sickling syndromes (sickle cell disease and sickle-beta thalassemia)
3. *'Debatable indications for splenectomy'*
   – Nonparasitic splenic cysts.

www.Ebook777.com

- Thalassemia syndromes.
- Lymphosarcoma with specific cytopenia or pancytopenia.
- Thrombotic thrombocytopenic purpura.
- Myeloproliferative disorders.

F. Etiological classification

1. *Congestive*
   - Intrahepatic portal HT: cirrhosis, Wilson's disease, hemochromatosis, congenital hepatic fibrosis.
   - Prehepatic portal HT: portal vein thrombosis/obstruction/cavernoma/atresia.
   - Posthepatic: Budd-Chiari syndrome, congestive cardiac failure.
   - Segmental (*syn*s. left sided, sectorial) portal HT: splenic vein occlusion by pancreatitis, pancreatic tumor, pancreatic pseudocyst, splenic artery aneurysm, and in infants who have had umbilical vein catheterization.
2. *Cystic*
   Cysts—congenital, traumatic, parasitic (hydatid), pseudocyst.
3. *Inflammatory and granulomatous*
   Splenic abscess, systemic lupus, rheumatic fever, serum sickness, sarcoidosis, etc.
4. *Neoplastic*
   - Hematological malignancy
   - Primary intrinsic malignancy: lymphosarcoma, plasmacytoma, fibrosarcoma, angiosarcoma.
   - Secondary intrinsic malignancy: carcinoma, melanoma.
   - Benign: hamartoma, fibroma, hemangioma, lymphangioma.
5. *Storage disease*. Common in children.
   Hemochromatosis, amyloidosis, Gaucher's disease, Wilson's disease, histiocytosis, etc.
6. *Infective diseases*
   - Acute: viral hepatitis, infectious mononucleosis, typhoid, toxoplasmosis, typhus fever, tularemia, cytomegalovirus, septicemia, SBE, brucellosis, abscess.
   - Subacute: AIDS, TB, malaria, SBE, brucellosis, leishmaniasis, trypanosomiasis, histoplasmosis.
   - Chronic: fungal disease, syphilis, bacterial endocarditis, tuberculous cold abscess.
7. *Deficiency diseases*
   Malnutrition, rickets.
8. *Hematological diseases*. → marked anemia.
   - Hemolytic disorder: hereditary cell membrane defects, autoimmune hemolytic states, thalassemia, sickle cell disease, hemoglobin C disease.
   - Myeloproliferative disorder: myeloid metaplasia (secondary cancer in bones, multiple myeloma, myelofibrosis, marble bone disease, thalassemia), polycythemia vera, essential thrombocythemia.
   - Thrombocytopenic purpura.
   - Miscellaneous: primary splenic hyperplasia, megaloblastic anemia, iron deficiency.
9. *Collagen diseases*
   RA, Still's disease.
10. *Miscellaneous*
    Hyperthyroidism, splenic mastocytosis, etc.

REMARKS

- There are numerous causes of splenomegaly; the relative incidence varies in different parts of the world. It is beyond the scope of this book to deal with the DD of all of them.
- Significant correlation between splenic size and blood counts is not always clinically significant. In general, the bigger the spleen the worst the cytopenia (from hypersplenism) although there are exceptions to this, e.g. marked thrombocytopenia/neutropenia in immune (idiopathic) thrombocytopenic purpura, where the

Free ebooks ==> www.Ebook777.com

splenomegaly is invariably mild; so much so that **gross splenomegaly suggests that the diagnosis is not ITP.**

– The importance of splenomegaly from surgical perspective revolves around decision-making of splenectomy.
– In 'surgical' causes of splenomegaly, the decision of splenectomy when indicated and appropriate, can be taken by the surgeon alone. Examples—splenic abscess, hydatid cyst, and bleeding gastric varices due to sinistral portal HT, etc.
– In 'hematological' causes of splenomegaly, the decision of splenectomy is primarily taken by the hematologist, pediatric physician or general physician.

For differential diagnosis of surgical causes of acute splenomegaly, refer to chapter 34.

Differentiation of each individual condition causing splenomegaly can be done by interrogative history, repeat physical examination, and investigation.

In the **clinical approach to a case of splenomegaly** and the subsequent DD that follows, only those cases are discussed below in which surgery may have to play a role in the overall treatment.

An important theme in the approach to arrive at the diagnosis is to identify **the major clinical scenario/'pattern'/presentation.** A patient of splenomegaly may fit into one of the following groups/ patterns.

- Pallor ± jaundice.
- Abnormal bleeding in the form of bruising/purpura/petechiae/ecchymoses, etc.
- Pancytopenia.
- Segmental portal hypertension.
- Acute/chronic pain the LUQ.
- Otherwise asymptomatic.

A. PALLOR ± JAUNDICE ⇒ hemolysis.

www.Ebook777.com

Single episode on the background of another major disease that is known to cause hemolysis

An example: extensive burns

Intermittent episodes/Chronic persistent problem

- (Undue) Fatigue. Pallor. - Unresponsive to iron therapy
- ± Dark coloured urine, jaundice
- ± Severe crises of RBC destruction which may require BTs. (Sudden onset of fever, pain in abdomen, nausea, vomiting, and extreme pallor, followed by increased jaundice)
- ± Gall-stone disease
- ± Chronic leg ulcers

Are these episodes known to be precipitated by certain chemicals/drugs?

Yes ⇓ Acquired hemolytic anemia

No

- (Usually) Children/adolescents
- ± Frontal bossing, malar prominence (due to extramedullary erythropoiesis)
- ± Signs of congestive heart failure (in crises, due to anemia)
- Hepatosplenomegaly ± (instead of only splenomegaly)

⇓

Congenital hereditary spherocytosis/elliptocytosis

- Middle-aged women
- Consistent presence of mild anaemia on which severe haemolytic crises are superimposed

⇓

(Acquired) autoimmune hemolytic anemia.

- Coombs' test positive (usually, but not invariably).
- Factor to be evaluated before deciding on splenectomy: Response to steroid therapy

Factors to be considered for evaluation before deciding on splenectomy:
- Degree of impairment of health in general
- Severe haemolytic crises have occurred or not
- Gallstone disease present or absent

B. – **BRUISING / PURPURA / PETECHIAE / ECCHYMOSES** – more prominent in dependent areas

– **SPONTANEOUS BLEEDING FROM MUCOUS MEMBRANES** – epistaxis / menorrhagia / prolonged bleeding from minor wounds

} ⇒ thrombocytopenic purpura

– Rarely bleeding from GIT/urinary tract, hemarthrosis and intracranial bleeding. Thrombocytopenic purpura may be immune related (ITP) or secondary.

Acute ITP
- Age: 18 months—6 years.
- Duration of purpura: 1–2 weeks.
- Short h/o preceding viral illness.
- NO other disease.
- 'Steroid facies' if already on treatment.
- Spontaneous remission ±.

Chronic ITP
- Duration of purpura > 6 months.

Free ebooks ==> www.Ebook777.com

– Remissions and relapses lasting several years.
– Response to steroid therapy – poor.

Notes:

– Spleen is palpable (mild splenomegaly) in only 25% of cases.
Gross splenomegaly suggests that the diagnosis is not ITP.
– Mild thrombocytopenia = PLC 150000–50000 / mm³.
– Severe thrombocytopenia = PLC 50000–10000 / mm³.
– Serious thrombocytopenia = PLC < 10000 / mm³.

C. PANCYTOPENIA IN THE PRESENCE OF AN APPARENTLY NORMAL BONE MARROW AND THE ABSENCE OF AN AUTOIMMUNE DISORDER ⇒ secondary hypersplenism.

↓

– Pallor ± jaundice.
– Bruising / purpura / petechiae / ecchymoses.
– Frequent infection, i.e. frequent/persistent/serious infection due to usual/unusual (opportunistic) microbes.

Causes of secondary hypersplenism:

– Portal HT
– Infective, e.g. malaria.
– Inflammatory, e.g. rheumatoid arthritis.
– Myeloproliferative and lymphoproliferative disorders.

Splenectomy should not be taken lightly.

Factors to be considered before deciding on splenectomy:

i. Prognosis of the underlying cause of hypersplenism.
ii. – The degree of cytopenia.
– Incidence of recurrent infection.
iii. – The extent of splenomegaly.
– The amount of discomfort caused by splenomegaly.
– The potential difficulty of the surgical procedure. The removal of a grossly enlarged spleen carries appreciable morbidity and mortality.

A note on hypersplenism

Hypersplenism is *defined as* a syndrome of *all* conditions of splenomegaly combined with decreased amounts of one or more circulating blood elements.

Classifications

i. Primary – caused by hematological disease. There is a work hypertrophy of the spleen due to the constant exposure of the spleen's phagocytic machinery to abnormal cells.
ii. Secondary = enlargement of spleen secondary to some other disease. Destruction of normal blood elements occurs because of a primary lymphoreticular process.

D. HEMATEMESIS FROM GASTRIC VARICES WITH NORMAL OR GOOD LIVER FUNCTION ⇒ segmental portal HT.

As esophageal varices may be absent, central portal pressures normal and gastric vessels easily overlooked at endoscopy, this condition can be easily missed. This condition is often under-recognized.

Causes of segmental portal HT: – following either acute or chronic pancreatitis; pancreatic pseudocyst.
– from direct extension of a pancreatic tumor.
– in infants who have had umbilical vein catheterization.
– Splenic artery aneurysm.

E. ACUTE INFLAMMATORY ABDOMEN INVOLVING THE LUQ ⇒ splenic infarct, abscess. Refer to chapter 34.

F. PAIN IN THE LUQ that may radiate to the back/shoulder and dull aching or dragging in nature **WITHOUT ANY OTHER SYMPTOM**:—could be due to splenic cyst.

G. OTHERWISE ASYMPTOMATIC

Splenomegaly may be due to splenic cyst discovered fortuitously.

'UNDIAGNOSED SPLENOMEGALY'

= the cause of splenomegaly remains unknown in spite of reasonable efforts

www.Ebook777.com

If the organ is minimally enlarged and the patient otherwise healthy
↓
Keep under careful FU

– If symptomatic
OR – spleen is noticeably enlarging
OR – has already significantly enlarged at presentation
↓
Diagnostic splenectomy may be considered
– Many such patients will be found to have an occult lymphosarcoma.
– Otherwise, keep the patient under careful surveillance as a lymphosarcoma may develop years later

HEPATOSPLENOMEGALY

Pathological Process	Common Conditions	Rare Conditions
Congestion	Portal HT	-
Storage disease	Storage disease	-
Chronic infections and granulomas	Malaria, Kala-azar, schistosomiasis	TB, syphilis, sarcoidosis
Blood disease	-	Hemolytic anemias
Reticulosis	Hodgkin's disease, leukemia	-
Myeloid metaplasia	Myeloid metaplasia	-

STOMACH

Examples of gastric disorders which may present with a lump in abdomen: Malignant tumor (carcinoma, sarcoma), benign tumor (leiomyoma, neurofibroma, neurilemmoma, etc. – all are rare), perigastric abscess due to chronic perforation, gumma (rare), bezoars.

Carcinoma of the stomach

Basically, a gastric lump.

Differentiating points

– Progress—rapid.
– Margins—ill-defined.
– Surface—irregular (nodular).
– Consistency – hard.

Supporting point: Male > 40.

Also refer to chapter 33.

Benign tumor, when large enough to produce a lump in abdomen.

Basically, a gastric lump.

Differentiating points

– Progress – slow.
– Surface – smooth.

Supporting point: Low-grade fever.

Notes

Benign tumors tend to produce a different set of symptoms on protruding towards the lumen, e.g. hematemesis/melena, indigestion, pain in epigastrium.

Perigastric abscess

Basically, a gastric lump, initially beginning as an acute illness which later attains chronicity. Long history of dyspepsia.

Differentiating points:

– Tender

Free ebooks ==> www.Ebook777.com

– Fixed
– Surface—irregular.

Supporting points:

– Middle age
– Past history suggestive of peptic ulcer.

Trichobezoar

Basically, a gastric lump in a psychiatric (female) patient, who may give a h/o swallowing (her own) hair.

Differentiating points:

– Margin – well-defined.
– Shape – that of the stomach ('J').
– Mobile.
– Surface – smooth.

Gastric outlet obstruction

Refer to chapter 33.

CHPS (Congenital Hypertrophic Pyloric Stenosis)

Basically, a neonate or an infant up to the age of 3 months presenting with non-bile-stained vomiting (characteristically forceful and projectile), but otherwise feeding well.

Refer to chapter 78.

SMALL INTESTINE

Examples of small intestinal disorders which may present with a lump in abdomen: Tumor, regional enteritis, enterocystoma (in relation to a remnant of V-I duct).

Tumor

Clinical manifestations common to all types of tumor of the small intestine: Subacute obstruction, loss of weight and (iron-deficiency) anemia due to chronic blood loss.

Additional or alternative manifestations may vary:

– Intestinal obstruction due to intussusception.
– Overt GI bleeding leading to melena/frank rectal bleeding.
– Obstructive jaundice in half of the cases of duodenal carcinoma.
– Diarrhea in carcinoid of the jejunum/ileum.
– Carcinoid syndrome in few cases of usually the small intestinal carcinoid tumor.

Regional enteritis (Crohn's disease)

– Abdominal cramping pain, diarrea and weight loss.
– A doughy mass of inflamed ileum ±.

ILEOCECAL REGION

Examples of ileocecal region disorders which may present with a lump in abdomen: Carcinoma of the cecum, appendicular mass, hyperplastic ileocecal TB, ameboma, actinomycosis, impacted ball of round worms, regional enteritis, chronic intussusception.

CA-Cecum

Basically, h/s/o disorder of ileocecal junction; in particular progressive chronic distal small intestinal obstruction.

Refer to chapter 33.

Appendicular mass

Basically, an intra-abdominal lump in the RLQ forming about 48 hours after the commencement of an attack of acute appendicitis.

– If there is significant guarding or rigidity, then a lump cannot be palpated. In such a situation, the diagnosis of appendicular mass must be considered on the basis of assumption of the natural history of acute appendicitis. Provided possibility of abscess is ruled out, conservative treatment is instituted in such a patient.

  If the patient responds well, then within about 24 hours or so, pain, tenderness and guarding/rigidity reduce

www.Ebook777.com

enough to allow palpation of the lump.
– No evidence of chronic intestinal obstruction.

Differentiating points in the initial period
– Tenderness.
– Guarding.
– The lump: Ill-defined. Fixed. Surface – smooth. Consistency – firm.

Supporting points
– Slight psoas spasm.
– Fever not hectic.

Differentiating points in the later period
– Tenderness decreases.
– The lump becomes better defined and slightly mobile.

The margin should be marked with a skin-pencil with date on day 1 of presentation, and subsequently every alternate day or so, so that the rate of regress can be judged well clinically.

Notes
– Appendicular mass is composed of greater omentum, edematous cecal wall and edematous coils of ileum matted together with lymph. In the midst lies a perforated or otherwise inflamed appendix.
– 5th day onwards, the lump either enlarges and heads towards formation of an abscess, or gradually resolves as inflammation subsides.

Hyperplastic ileocecal TB

Basically,
– A **long h**/o vague pain in the RLQ.
– H/s/o disorder of ileocecal junction; in particular manifestations of distal small intestinal incomplete subacute obstruction.
– *Nonspecific symptoms*: Low-grade evening fever f/b night sweat, loss of appetite and (significant) loss of weight.
– Vague ill-health.
– An ileocecal lump.

Differentiating points:
– The lump is situated **slightly higher and medial** than the normal position of the cecum (due to fibrosis in the lymphatics along the ileocecal vessels).
– The lump is slightly tender, mobile, and firm.

Supporting points:
– Younger age.
– Nonspecific symptoms of TB.
– E/o simultaneous presence of TB elsewhere, e.g. lungs, lymph nodes, etc.

Note: Sometimes the diagnostic problem can be quite perplexing.

Ameboma

Differentiating point: Marked response to therapeutic trial with injectable antiamebic medicine.

Supporting point: Previous h/o amebic dysentery.

Notes
– Ameboma may be indistinguishable from CA cecum.
– While the absence of amebae in the stool does not rule out ameboma, the presence of amebae in the stool does not necessarily prove the diagnosis of ameboma.

Actinomycosis

There are 2 clinical types:

1. ~ 3 weeks or so following appendectomy for perforated appendicitis, a mass develops in the RLQ.
Differentiating points:
– Lump—fixed and indurated.

Free ebooks ==> www.Ebook777.com

Soon commences to discharge. The purulent discharge is initially thin and watery; later due to secondary infection, it becomes thicker and odorous.
- Multiple discharging sinuses.
- Discharge from the sinuses contains yellow-colored granules of the size of pinhead. (Collect pus in a clear test tube and observe in a powerful light to see the granules.)
- Later, fistulae may develop.

Notes:
- Rare.
- Usually follows perforated appendicitis.
- Infection spreads mainly to the psoas, abdominal wall and adjacent organs.
- Spread of infection by the portal venous system results in multiple intercommunicating loculated abscesses (honeycomb liver).

2. The second clinical type is seen usually in young adult males.
- Vague pain in abdomen.
- Loss of weight.
- Fever occasionally.
- Usually no obstructive symptoms.
- Anemia.
- Slightly tender, firm mass.

In most instances, laparotomy is performed with one of the following findings:
- An abscess.
- The mass is found to be densely adherent to the posterior abdominal wall, and irremovable; necessitating an ileo-transverse bypass.

Impacted ball of round worms
- Subacute intestinal obstruction.
- The mass seems to change position on different occasions.
- A h/o passing round worms in stool and/or in vomitus.

Level of impaction: usually in the distal portion of ileum.

Regional enteritis (Crohn's disease)

Basically, inflammatory abdomen involving the RLQ, and anorexia, fever, loss of weight and malaise.

Differentiating points:
- *Antecedent* h/o abdominal cramping, diarrhea and weight loss.
- The abscess may track into the pelvis behind the inguinal ligament and present as a tender groin mass (psoas abscess).

Supporting points:
- Diarrhea.
- A doughy mass of inflamed ileum.

Notes:
- Relatively rare condition.
- Resembles acute appendicitis except that it commences with diarrhea.
- The abscess in the RLQ may result from one of the 2 mechanisms: 1. Intestinal perforation at the site of a deep fissuring ulcer or proximal to a stricture. 2. Rupture of inflamed regional lymph node without perforation of the intestine.

Chronic ileocecal / ileocecocolic intussusception
- Subacute intestinal obstruction.
- Bleeding PR may occur during an attack.

www.Ebook777.com

– A banana-shaped mass may be palpable per abdomen. – The mass seems to change position on different occasions.
– It may advance sufficiently to be palpable on rectal examination.
– It may even protrude out of anus.

On such a background, frank manifestations of complete (acute) intestinal obstruction may occur when the intussusception gets incarcerated.

COLON

Examples of colonic disorders which may present with a lump in abdomen: Carcinoma, diverticulitis, ameboma, hyperplastic TB, regional enteritis (Crohn's disease), chronic intussusception.

Cancer of the colon

Refer to chapter 33.

Chronic diverticulitis

→ tumor-like mass, which may be mistaken for a carcinoma.

Supporting points in favor of diverticulitis:

– Recurrent inflammatory attacks.
– Comparatively good health otherwise.

GREATER OMENTUM

Examples of greater omentum lumps: Lipoma, myxoma, malignant tumor, rolled-up omentum – due to tuberculous peritonitis.

MESENTERY

Examples of mesenteric lumps: (Mesenteric) Cyst, (pancreatic) pseudocyst, lipoma, fibroma, fibromyxoma, lymphosarcoma, metastatic tumor, lymphadenopathy.

GALLBLADDER

Mucocele of the gallbladder

Basically, a painless lump in the RUQ.

Supporting points

– Elderly.
– Previous h/o acute pain in abdomen indicative of biliary colic or mild acute cholecystitis.

The lump can attain enormous size.

Empyema of the gallbladder (Suppurative cholecystitis)

A complication of acute cholecystitis.

– A tender lump in the RUQ.
– Usually affects elderly patients in whom systemic signs including fever and leukocytosis may be minimal.

Distended gallbladder

Could be the result of a distal tumor of CBD, often in a jaundiced patient.

PANCREAS

(Retrogastric) Pseudocyst

– A lump in the epigastrium as large as a melon.
– Transmitted pulsations.
– Fixed.
– Often tense, hence fluctuation cannot be elicited.

EXTRAHEPATIC BILIARY TRACT EXCLUDING THE GALLBLADDER

Choledochal cyst

Symptoms other than lump in abdomen: Pain, cholestatic jaundice.

Complications: Recurrent cholangitis and pancreatitis, hepatic abscess formation, calculous disease, biliary

Free ebooks ==> www.Ebook777.com

cirrhosis, rupture of the cyst with biliary peritonitis (rare), portal vein thrombosis, and cholangiocarcinoma.

Malignancy (Extrahepatic cholangiocarcinoma)

– Progressive obstructive jaundice.
– Distended gallbladder (in case of distal tumors).
– Dull pain in the upper abdomen.
– Acute cholangitis / acute cholecystitis.
– Weight loss is not evident until the disease is advanced and is usually accompanied by evidence of hepatic involvement and ascites.

URINARY BLADDER

Examples of urinary bladder disorders which may present with a lump in abdomen: Distended bladder, diverticulum, massive papilloma, carcinoma, a large stone.

UTERUS

Uterine conditions (other than normal pregnancy) which may present with a lump in abdomen: Cryptomenorrhea, fibroids including subserous pedunculated fibroids, malignancy, pyometra.

(Normal) Pregnancy

– Amenorrhea in a woman of child-bearing age.
– Smooth uniform enlargement.
+ Classical symptoms and signs of pregnancy.

Fibroid

– Age above 30, but before menopause.
– Menorrhagia of some standing.
– Irregular enlargement of the uterus. Exception: submucous fibroid.

Note: a fibroid projecting laterally can be confused with an ovarian mass.

UTERINE TUBE

Subacute/Chronic rupture of ectopic pregnancy

– A lump on the side of the uterus or behind it.
– H/o missed period on last 2 or 3 occasions.

OVARY AND BROAD LIGAMENT

Examples of ovarian / broad ligament disorders which may present with a lump in abdomen: Ovarian cyst, broad ligament cyst, lipoma, fibroma, dermoid, malignant tumor, Krukenberg tumor, chronic abscess (Tubo-Ovarian mass).

Ovarian cyst / tumor

– Menstruation – normal or scanty depending on the amount of ovarian destruction.

Basically, a pelvic lump.

– Bulging of the abdomen in anteroposterior diameter, concavity in the flanks being maintained.
– Initially it may be situated on one side of midline, but eventually comes to lie in the midline.
– Freely mobile in all directions.
– On percussion: dull note anteriorly and resonance in the flanks.
– Dullness will have convex borders on all sides. On change of posture, the site of dullness may shift *but the shape remains the same*.

Points which may help to differentiate cyst and tumor

– Cyst tends to be smooth, and soft/firm in consistency.
– Tumor tends to be rather nodular (though finely, but not as smooth as cyst), and 'firmer'/hard in consistency.

Broad ligament cyst

Tends to displace the uterus on the opposite side.
Can be felt bimanually – per abdomen and per vaginum.

www.Ebook777.com

Usually not as big as an ovarian cyst.

T-O mass

Results from chronic salpingitis and oophoritis.

– Young age.
– Situated in the midline, or more often on one or the other side of the midline.
– Preceded by an attack of PID.

PERITONEAL CAVITY

Examples of disorders of peritoneal cavity which may present with a lump in abdomen: Localized tuberculous ascites, pseudomyxoma peritonei, abscess, talc/foreign body granuloma, endometriosis, lithopedion, loose bodies from appendices epiploicae.

SUBDIAPHRAGMATIC SPACES

An example of disorder of subdiaphragmatic space which may present with a lump in abdomen: Chronic abscess.

LESSER SAC

Examples of disorders of lesser sac which may present with a lump in abdomen: Pseudocyst of pancreas, chronic abscess. True cyst of the pancreas is rare.

Chronic psedocyst of pancreas

– A rounded smooth lump.
– Otherwise usually asymptomatic.
– Often, no recent attack of acute pancreatitis can be identified.

Acute pseudocysts (acute peripancreatic effusion) follow an established acute attack of pancreatitis.

KIDNEY

It is usually difficult to distinguish between the benign and malignant lumps, unless there are other clear-cut clues about malignant nature by virtue of regional or systemic spread of the disease.

Exception – In neonates and young children, cystic lumps can frequently be identified by transillumination.

'Key points' to be considered for ARRIVING AT THE DIAGNOSIS of renal lump

1. **Unilateral / Bilateral**?
   Causes of bilateral renal lump
   – Primarily polycystic kidneys.
   – Hydronephrosis due to obstruction of the lower urinary tract (bladder and below).
   – Otherwise usually a unilaterally occurring pathology affecting both sides simultaneously.
2. **Any peculiar chief complaint related to the lump**?
   For example – 'Acute dragging pain ± hematuria'—suggests floating kidney.
   – 'Painless hematuria' – suggests renal cell carcinoma / TB.
3. **Any symptom/sign in addition to the presence of a (renal) lump**?
   If present, what is the nature of the associated symptoms?
   – indicative of acute/chronic sepsis, or indicative of malignancy?
4. **On physical examination** – Are there differentiating features in favor of any specific pathology?

Floating kidney

H/o (acute) dragging pain ± Hematuria.

Differentiating points:

– Freely mobile kidney, which can be pushed in its normal location with a peculiar sickening sensation.
– Upper border of the kidney is palpable.

Size of the kidney: normal or may be slightly enlarged.

Sometimes patient may become neurasthenic on knowing the fact of the presence of a floating kidney.

Adult polycystic kidney

Free ebooks ==> www.Ebook777.com

Basically,

– Renal lump/s otherwise often without any additional symptom/sign, manifesting after 40 years of age.
– Pain in the loin ±. *Causes/mechanisms of pain*: Drag on the pedicle of a heavy organ OR due to tension within the cysts.
– Hypertension ±. Why some patients escape this complication is not clear.
– Hematuria ±. Usually 'moderate' episodic, recurring at varying intervals; each episode lasting for a few days. *Mechanism of hematuria*: Overdistension of one of the cysts, and its rupture in renal pelvis.

Differentiating points:

– Bilateral; however, **one side may be more advanced and bigger than the other** causing confusion in clinical diagnosis.
– Lobulated surface.
– Uremia.
– Abundant urine of low specific gravity (≤ 1.010) containing a slight trace of albumin, but neither casts nor cells.

Notes:

– Hereditary. Autosomal dominant trait.
– NOT easily demonstrable on IVU before the late teens.
– May be associated with congenital polycystic disease of the liver/pancreas/lungs.
– The most common complication: pyelonephritis.

Unilateral multicystic kidney

Basically, a renal lump.

The kidney is found to be functionless on functional investigation.

Hydronephrosis (Unilateral)

– Asymptomatic to begin with. Subsequently, a renal lump forms.
– Dull aching or a sense of weight in the loin may be brought on by alcohol or excessive fluid intake.
– Acute colicky pain ± (exact explanation not known).
– Hematuria ±.
– Manifestation of the primary cause of hydronephrosis may be absent. However, a previous h/o colic may point to the renal stone as a possible cause.

Differentiating points:

– Enlarged kidney with smooth or lobulated surface.
– Fluctuation ±.
– Fluid thrill in large hydronephrosis.
– Intermittent passage of large amount of urine with disappearance of the lump (Dietl's crisis).

A greatly enlarged kidney suggests obstruction at the level of PUJ.

Causes of PUJ obstruction:

1. Idiopathic – due to incomplete canalization of the ureter during fetal development. Patients may present at any age from prenatal period to old age, though they usually present during childhood.
2. Congenital kinking.
3. 'Lower pole crossing vessel'.
4. Stone.
5. Previous surgery.

A note on **bilateral hydronephross**

1. Symptoms of bilateral hydronephross caused by *a lower urinary tract obstruction* are overshadowed by those of the causative lesion. There is little to call attention to the hydronephross except, perhaps, a dull ache in the loins.
   The kidneys are unlikely to be palpable because before the hydronehross became large enough to be felt, renal failure will have occurred.
2. 'Hydronephrosis caused by bilateral *upper urinary tract obstruction*' is rare. Symptoms may be bilateral, but

www.Ebook777.com

more frequently are on one side, an investigation revealing hydronephrosis on the other side (incidentally).

3. Dilatation of the ureters and the renal pelves occur in every *pregnancy*. The right side is affected to a much greater extent. The extent of dilatation reaches its peak between the 5th and 6th months; then remains stationary until after delivery. Involution occurs within 2–12 weeks. *Significance of hydronephrosis during pregnancy*: increased liability to infection. Exceptionally, the hydronephrosis persists after pregnancy is over.

Pyonephrosis

Basically,

– A painful and tender renal lump with toxemia ± manifestations of the primary causative condition, e.g. hydronephrosis / acute pyelonephritis / renal stone.
– Acute colicky pain ± (Explanation of colicky nature of pain is not known).
– Fever of 99–100° F suggests open pyonephrosis, whereas a high-grade fever with rigors suggests closed pyonephrosis.
– A large lump suggests complicated hydronephrosis.
– Anemia.

Supporting symptoms are those which are s/o cystitis; sometimes these may be the chief complaints.

Notes:

– *In acute cases*, toxemia is marked.
– *In chronic cases*, toxemia (fever) and local tenderness may be minimal, but still the lump may remain a little tender.
– Pyonephrosis = the kidney is converted into a multilocular sac containing pus or purulent urine, with a varying amounts of renal parenchyma in its walls.

Perinephric abscess

Dull pain in the renal angle and constitutional disturbances (may be pyrexia of apparently unknown origin).

Differentiating points:

– Diffuse lump – tender, irregular and fixed.
– Guarding of the lateral abdominal wall muscles.
– Scoliosis of the lumbar spine with concavity towards the affected side.
  Bending the lumbar spine towards the opposite side is often painful, but similar movement towards the affected side is painless.
– Pitting on pressure overlying the loin.
– Manifestation of the primary causative condition.

Notes:

– If suppuration commences in the lower part of the perinephric fat, local signs develop early.
– If suppuration commences/is confined to the upper part of the perinephric fat, local signs are absent or minimal.
– As a rule, no pus or organisms are present in the urine.
– The most common causes: hematogenous, extension of a renal cortical abscess, extension of a local extrarenal infection (e.g. appendicular abscess), and ascending infection via periureteral lymphatics.
– Other causes: infection of a perirenal hematoma, perforation of the renal capsule from undue delay in operating upon a pyonephrosis or a renal carbuncle, advanced tuberculous pyonephrosis or tuberculous osteomyelitis of a nearby vertebra.

Carbuncle of the kidney

Clinical findings are similar to those of perinephric abscess.

Mechanisms of development of carbuncle of the kidney:

1. Hematogenous spread of *Staphylococcus aureus* from the primary source in a cutaneous lesion such as a boil, carbuncle, or an abscess.
2. Secondary infection of a small hematoma that might have resulted from a blow.

TB of the kidney

Basically,

Free ebooks ==> www.Ebook777.com

A renal lump ± vague discomfort/pain in the loin in a young adult.
Refer to chapter 54.

(In an adult patient,) **A tumor of the kidney, most probably a RCC** (Another possibility, clinically: Oncocytoma)
Basically, a lump (often painful) in the loin that is almost always associated with total hematuria.
Intermittent hematuria with clot colic is typically the first symptom.
Nonspecific symptoms: nausea, loss of weight, night sweats, bowel disturbance.
Paraneoplastic syndrome: Fever, muscle pains, neurological symptoms, pathological fracture, hypercalcemia, erythrocytosis.
Differentiating points:
- Nodular surface.
- Fixed, (in later stage).
- Hard consistency.
- Evidence of metastatic spread, in later stage.

Supporting point:
Varicocele of relatively short duration particularly on the left side.
Notes
- Probably originates from proximal tubular cells in the cortex of the kidney.
- < 10% of patients have all the 3 symptoms viz hematuria, pain, and lump.
  By the time a patient has the classical triad of hematuria, pain and a palpable renal tumor, she/he nearly always has metastases.
- Sometimes detected incidentally by imaging investigation for unrelated symptoms.
- Nonspecific changes in liver enzymes may not be necessarily due to metastatic disease. They resolve on removing the primary tumor.
- Oncocytoma is a benign tumor which can be differentiated from RCC only on histological examination.
  (Other possibilities: clear cell sarcoma, rhabdoid sarcoma)

Childhood malignant tumor of the kidney, most probably a nephroblastoma (Wilm's tumor)
Basically, a young child, more usually an infant presenting with a large lump in abdomen, otherwise asymptomatic.
Additional symptoms:
- Hematuria ± (late if at all) – Hematuria is an ominous symptom; it denotes bursting of a previously encapsulated tumor into the renal pelvis. Poor prognosis.
- *Non-specific symptoms*: pain in abdomen, anorexia, fever, failure to thrive.
- *Paraneoplastic syndrome*: e.g. hypercalcemia, erythrocytosis (from uncontrolled erythropoietin production).

Differentiating points:
- (May be) fixed.
- Nodular surface.
- Hard consistency.

Notes:
- Nephroblastoma (Wilms' tumor): Derived from embryonic metanephric blastema. Usually presents before the age of 6 years. The growth is rapid, however, invasion of the renal pelvis occurs late, and so hematuria is seen only in few cases. A large abdominal mass is the cardinal sign. Metastatic disease is almost always confined to the lung and/or liver.
- **Clear cell sarcoma:** Higher rate of metastasis to bone and brain.
- **Rhabdoid sarcoma:** Presents in the 1st 2 years of life.
- **Congenital mesoblastic nephroma** presents under 3 months of age.
- *Most important DD* (a tumor of nonrenal origin): Retroperitoneal neuroblastoma. – tends to metastasize to bones. Otherwise, there is no difference between nephroblastoma and retroperitoneal neuroblastoma with reference to clinical manifestations and treatment; attempts at differentiation are only of academic interest.

www.Ebook777.com

36 DISTENSION OF ABDOMEN

Causes of distension of abdomen

- Fat, Fetus → Physiological.
- Fluid: – Free fluid (Ascites)
  – Loculated fluid: - Distended bladder
  - A large cyst
  – Hemoperitoneum
- Food (Retained undigested), Feces, Flatus → ⇒ Gastrointestinal obstruction
- A huge lump.

→ Pathological.

Physical findings differentiating some of the causes of distention of abdomen

– *Fat* → deep accentuation of the umbilicus
– *Pregnant uterus* is easy to diagnose
– *Ascites* → - Bulging mainly in the flanks, bulging in the anteroposterior diameter being only slight
  - Transverse stretching of the umbilicus
  - Shifting dullness at the least
  - May be associated with - Umbilical herniation
    - Penile and scrotal edema
    - Pleural effusion
– *Hemoperitoneum* → the signs primarily of the underlying cause that led to hemoperitoneum
  Examples: - Hypovolemic shock ⇒ frank severe bleeding
  - Peritoneal irritation ⇒ hemorrhagic exudate from an inflamed organ, such as for example severe hemorrhagic/necrotizing pancreatitis
  - Increase in abdominal girth due to increase in ascitic fluid and hypochromic microcytic anemia ⇒ bleeding peritoneal carcinomatosis
– Feces are indentable
– Flatus gives hyperresonant note on percussion.

A CLINICAL APPROACH TO DIAGNOSIS

A. Associated clinical background usually leads to the diagnosis.
B. Acute/subacute, or Chronic?

Free ebooks ==> www.Ebook777.com

(1) **Acute/subacute**

Following some major event or a disease ⇓	Occurring otherwise
Hemoperitoneum *Examples of causes of hemoperitoneum* – Following diagnostic (± therapeutic) percutaneous intervention like liver biopsy, etc. – Following trauma – Following surgery, i.e. postoperative complication – Following severe hemorrhagic/necrotizing pancreatitis	– Over-eating, constipation – Acute irritant gastritis/food poisoning – Spontaneous haemoperitoneum—Refer to chapter 34 – Acute gastric obstruction (e.g. due to volvulus) – Acute mechanical intestinal (large/small) obstruction – Paralytic obstruction. – Acute Budd-Chiari syndrome/acute VOD } → rapidly oncoming ascites – Acute thrombosis of the portal vein – Severe ulcerative colitis – Toxic dilatation of colon – Megacolon: congenital, idiopathic, Chaga's disease – Acute ascites – ARF – Acute retention of urine – Constrictive pericarditis

(2) **Chronic**

Stationary ⇓	Fluctuating	Essentially progressive ⇓	Uncertain ⇓
Obesity	1. If related to feeding, then it could be the earliest symptom s/o gastric outlet obstruction. 2. Flatulent dyspepsia ⇒ – Gall bladder disease – Colonic disease	– Pregnancy – Gastric outlet obstruction – Intestinal obstruction (essentially though not invariably, colonic) – Megacolon: congenital, idiopathic, Chaga's disease – A lump in abdomen – Ascites	? Chronic retention of urine

C. In evaluation of ascites, diagnostic paracentesis (50–100 mL) is essential.

ASCITES

Definition: Accumulation of free fluid within the peritoneal cavity
Positive shifting dullness ⇒ 1–2 L of fluid. positive horse-shoe shaped dullness ⇒ > 2 L
In other words, clinically detectable ascites suggests the presence of no less than several hundred mL of fluid within the peritoneal cavity.

Causes of ascites

Disease of the peritoneum	Disease not involving the peritoneum
– *Peritonitis*:- Granulomatous (TB, sarcoidosis) - Fungal - Parasitic – *Tumor*: - Mesothelioma - Pseudomyxoma peritonei - Secondary peritoneal carcinomatosis – *Vasculitis* – *Miscellaneous*: Whipple's disease, familial Mediterranean fever, endometriosis, starch peritonitis	– Cirrhosis – CHF – Chronic Budd-Chiari syndrome – ARF – Hypoalbuminemia (nephritic syndrome, protein-loosing enteropathy, malnutrition) – Miscellaneous: myxedema, ovarian disease, pancreatic disease, chylous ascites, constrictive pericarditis – Urinary ascites may follow an undiagnosed intraperitoneal bladder rupture. *Sequelae*: peritonitis/intra-abdominal abscess / respiratory difficulty

www.Ebook777.com

Most common causes (90%) of ascites: TB, tumor, cirrhosis, and CHF.

Complications of ascites:

1. Spontaneous bacterial peritonitis.
2. Hepatorenal syndrome: May be spontaneous or precipitated by bleeding, excessive diuresis, paracentesis or drugs (aminoglycosides, NSAIDs).
3. Pleural effusion.
4. Mesenteric venous thrombosis.
5. Hernia particularly umbilical.

Representative fluid characteristics

Cause	*Appearance*	*Proteins g%*	*Cell count per mm³* *RBCs*	*WBCs*	*Other*
TB peritonitis	Clear, straw-colored, hemorrhagic or chylous	> 2.5	Occasionally high	> 1000 (> 70% lymphocytes)	AFB staining, and culture may be attempted, however, ascitic fluid does not contain tubercle bacilli
Bacterial peritonitis	Turbid or purulent	> 2.5	Low	> 250 polymorphs	Positive Gram's stain, culture
Tumor	Straw-colored, hemorrhagic, mucinous or chylous	> 2.5	Often high	> 1000 (50% lymphocytes)	Positive cytology
Cirrhosis	Straw-colored	< 2.5	Low	< 250	-
CHF	Straw-colored	Variable	Low	< 1000 (mesothelial)	-
Pancreatitis	Turbid, hemorrhagic or chylous	> 2.5	Variable	Variable	↑ amylase

Acute ascites

Very occasionally, ascites may rapidly develop in a patient with cirrhosis, and may mimic 'acute abdomen' due to associated presence of pain.

– Probable mechanism of pain: Stretching of the parietal peritoneum. Pain is the culprit symptom which misleads to think about acute abdomen
– Distension of abdomen comes on suddenly and progresses rapidly
– Patient is likely to be a k/c/o cirrhosis, with stigmata of chronic liver disease.

Acute form of Budd-Chiari syndrome

Basically, presentation with severe acute pain in abdomen and vomiting.

Differentiating points

– Ascites
– Large tender hepatomegaly.

Predisposing factors

– Women
– Oral contraception
– Hematological disorders like hemolytic anemia, polycythemia, etc.
– Collagen disorders like rheumatoid arthritis, sarcoidosis, SLE.

Additional features of the underlying malignancy may be present in case if the syndrome occurs secondary to malignancy.

The condition may progress rapidly over a few weeks to hepatic failure, coma and death.

Free ebooks ==> www.Ebook777.com

Chylous ascites

→ Rapid development of ascites.

Tapping reveals chyle with high fat and protein content. Repeated tapping → loss of body proteins.

It usually results from intraperitoneal rupture of chyle-laden mesenteric or retroperitoneal lymphatics or from exudation of chyle into the peritoneal cavity. It is essential to exclude malignancy in the abdomen (by imaging investigations).

Causes of chylous ascites: – Tuberculous or malignant disease of retroperitoneal lymph nodes
– Injury to thoracic ducts
– Filariasis.

Idiopathic megacolon and megarectum

– Pain in abdomen
– Constipation
– On per abdominal examination: Colon (usually the pelvic colon at least) is loaded with fecal masses
– On DRE: Fecal impaction is detected.

The patients can be divided into 2 main groups.

1. The symptoms dating back to childhood often with rectal impaction and soiling. This can be indistinguishable from congenital megacolon.
2. Commencement of symptoms in later life.
   The underlying cause of this condition is unknown.

www.Ebook777.com

37 JAUNDICE

Jaundice is **defined as** yellow discoloration of skin, sclerae, and mucous membranes due to increased levels of serum bilirubin concentration.

Normal serum bilirubin level is up to 1.5 mg% (17 μmol/L).
Jaundice becomes clinically evident only when serum bilirubin exceeds 3 mg% (40 μmol/L).

Jaundice is the most visible manifestation of liver and biliary tract disease.

Jaundice occurring during infancy and childhood, and jaundice occurring for the first time in an adult are two quite different situations. The differences between these situations are with respect to the underlying causes, and practical management.

GENERAL REMARKS

1. As elastic tissue-fibers have special affinity for bilirubin, the sclera and the skin of face and chest manifest early yellow pigmentation.
2. Areas of edema and vitiligo do not show yellow pigmentation.
3. Properly taken history and physical examination allows a correct diagnosis to be made in > 75% of patients.
4. In a case of jaundice there is usually overlapping of at least 2 groups, because the obstruction produced by disease at hepatocellular level can be very severe, and because long-standing extra-hepatic obstruction damages liver cells and results in the secondary factors which are supposed to be characteristic of hepatocellular disease. In other words, **the diagnostic accuracy of biochemical tests in the individual case is limited.** These tests should be done, but their results should not decide management unless they corroborate other strong evidence which points to the cause of jaundice.
5. In a case of surgical (obstructive) jaundice, stones may be present in a normal-sized CBD. There is ~ 5–10% incidence of ductal stones with CBD of 5 mm diameter.
   Diameter of normal CBD: Anatomically 4–10 mm (average of 7 mm).
   Criteria of dilated CBD: – On ultrasound scan: > 7 mm
   – On contrast study like cholangiography: > 10 mm.
6. In the management of a case of jaundice, there should be a close liaison between physician and surgeon from an early stage.

BASIC PATHOPHYSIOLOGY

Bilirubin is an insoluble waste product; and for excretion, it needs to be made water-soluble. This transformation is called 'bilirubin metabolism'. Bilirubin metabolism and elimination is a multi-step process. Various mechanisms and consequently the types of jaundice can be attributed to failure of different steps in metabolism and elimination of bilirubin.

A brief review of steps in the metabolism of bilirubin	*Possible abnormalities leading to jaundice*
Bilirubin	Excessive production → Hemolytic jaundice
↓	
Uptake by hepatocytes	Congenital hyperbilirubinemias

Free ebooks ==> www.Ebook777.com

↓

Conjugation with glucuronide – → conjugated bilirubin

↓

Secretion into bile canaliculi – Congenital hyperbilirubinemias
– Hepatocellular jaundice

↓

Downward flow through biliary tree Cholestatic jaundice

Enterohepatic circulation of bile

In health, 95% of bile acids entering the intestine are reabsorbed. The major fraction is reabsorbed from the terminal ileum; the terminal ileum is very important segment of the small intestine.

In obstructive type of jaundice, the enterohepatic circulation is affected; 3 consequences result from this impairment.

1. Malabsorption of fats and fat-soluble vitamins such as vitamin K, which in turn → its own consequences - 'steatorrhea', and bleeding tendency due to prolongation of PT.
2. Diarrhea due to dehydroxylation and deconjugation of bile salts by the colonic bacteria.
3. Increased cholesterol secretion in bile that becomes supersaturated.

TYPES OF JAUNDICE

Jaundice can be classified in various ways as follows:

– Classification based on pathophysiology: Hemolytic, benign familial congenital hyperbilirubinemia, hepatocellular, obstructive.
– Classification based on conventional terms used in clinical practice: Medical, surgical, 'malignant'.
– Jaundice in special circumstances: Postoperative jaundice, jaundice during pregnancy, jaundice during infancy and childhood, continued hyperbilirubinemia long after an attack of acute hepatitis, and idiopathic hyperbilirubinemia.
– 'Pseudo-jaundice'.

A. Hemolytic jaundice

A hemolytic disease → increased production of bilirubin with consequent increase in its blood-level. The primary hemolytic disease and resultant anemia also impair liver function to some extent diminishing the capacity to remove the excess of bilirubin. Small thrombi of inspissated bile and in chronic cases, bile stones may obstruct bile passages. Thus, in hemolytic jaundice, though the major pathological event is that of increased production of bilirubin, hepatocellular dysfunction and obstructive element also may come into force. This fact must be remembered while interpreting the blood reports of LFTs.

Causes of hemolytic jaundice: Congenital defects in RBCs like spherocytosis, elliptocytosis, sickling, etc. a large variety of infections, parasitic infestations like malaria, poisons like snake venom, etc. immunological such as following BTF, paroxysmal hemoglobinuria, etc. secondary to reticulosis, metastatic carcinomatosis, etc. after operative or accidental trauma (hematoma formation → a pigment load that exceeds hepatic excretory capacity); idiopathic.

B. Benign familial congenital hyperbilirubinemias

Features	Gilbert syndrome	Dubin-Johnson syndrome	Rotor's syndrome
– Plasma bilirubin concentration (in mg%)	≤ 3, Essentially all unconjugated	Usually < 7 (Range 1–25), ~ half conjugated	Usually < 7, ~ half conjugated
– Other distinguishing features	↓ bilirubin concentration with phenobarbital	↑ bilirubin concentration with estrogens	–
– Treatment	None necessary	No specific treatment. Avoid estrogen	None available

www.Ebook777.com

C. Hepatocellular jaundice = jaundice due to liver disease

Basically, normal amount of bilirubin load is not taken up by the diseased hepatocytes leading to unconjugated hyperbilirubinemia. However, conjugated hyperbilirubinemia is also present. This is explained on the basis of obstruction due to inflammatory edema, and necrosis resulting in rupture of bile canaliculi. Jaundice indicates substantive encroachment of liver function and hepatic reserve.

Causes of hepatocellular jaundice: Acute, chronic, and intrahepatic cholestasis.

1. ***Acute***

   Jaundice reflects the severity of the underlying condition.

2. ***Chronic***

   Unless the dysfunction is advanced, serum bilirubin level is lower than in the setting of acute hepatitis.

   Jaundice carries a poor prognosis.

   Causes: Chronic hepatitis, chronic Wilson's disease, hemochromatosis (arthritis + deep pigmentation + DM), autoimmune hepatitis (arthralgia, spider naevi, splenomegaly), cirrhosis, $\propto$1 antitrypsin deficiency (COPD is associated).

3. ***Liver diseases with prominent cholestasis***

   Some of the hepatocellular diseases have prominent cholestatic element, leading to what is called 'intrahepatic cholestasis'. It may often be difficult to distinguish it clinically from 'extrahepatic cholestatic jaundice' (i.e. jaundice due to large bile duct obstruction).

   a. Drug toxicity
      - Erythromycin, chlorpropamide, chlorpromazine, methimazole, etc.
      - Arthralgias, skin rash
      - Resolve within several months.

   b. Infiltrative diseases of the liver
      - The list of causes can be very long. Few examples—TB, sarcoidosis, lymphoma (particularly Hodgkin's), extensive tumors, etc.
      - Hepatosplenomegaly.

   c. Cholestasis with minimal histopathological abnormalities

      Recurrent episodes of jaundice + pruritus lasting upto several months, separated by periods of clinical remission.

      Causes: OC pills (Jaundice occurs within 2 months. Resolves promptly after discontinuation of pills), anabolic steroids (similar to OC pills), TPN, bacterial infections (Sepsis-related cholestasis in critically ill patient may be extremely difficult to distinguish from obstruction of bile ducts).

   d. Primary biliary cirrhosis
      - Predominantly occurs in women. Age of onset: ~ 40 years
      - Variable course, but ultimately ends up with liver failure
      - Serum bilirubin > 10 mg% $\Rightarrow$ extremely poor prognosis
      - Etiology of the condition is unknown.

D. 'Medical' jaundice

This is so called because surgery does not play a primary role in the treatment. Examples: Hemolytic jaundice, benign familial congenital hyperbilirubinemia, hepatocellular jaundice.

E. 'Surgical' jaundice (*synonyms* '**obstructive'** jaundice) = Jaundice due to **large bile duct obstruction**

Surgery is often (but not necessarily always) indicated, and should be executed after appropriate preoperative management and without undue delay (should be treated within 2–3 weeks of diagnosis). Undue delay of more than 3–4 weeks $\rightarrow$ increased morbidity/mortality. If biliary obstruction continues for long time, then liver cells degenerate. Increased serum bile salts $\rightarrow$ pruritus, depression of CNS, bradycardia and low systolic BP.

Causes of large bile duct obstruction

1. *In the lumen*
   - Ductal stones, i.e. choledocholithiasis. This is the most common cause.
   - Parasites, e.g. ascaris (round worms).

Free ebooks ==> www.Ebook777.com

2. *In the wall* (intrinsic diseases of bile ducts)
   - Congenital disorders, e.g. atresia
   - Tumor, e.g. cholangiocarcinoma, periampullary tumor
   - Primary sclerosing cholangitis
   - Edema of the wall due to infection-inflammation of an adjacent organ / tissue
   - Spasm of ampulla of Vater due to allergy/ulceration/psychogenic factor
   - Iatrogenic stricture—following surgical injury to bile duct / hepatic artery
   - Cholangitis following hepatic arterial infusion of chemotherapeutic medicine
   - AIDS cholangiopathy.

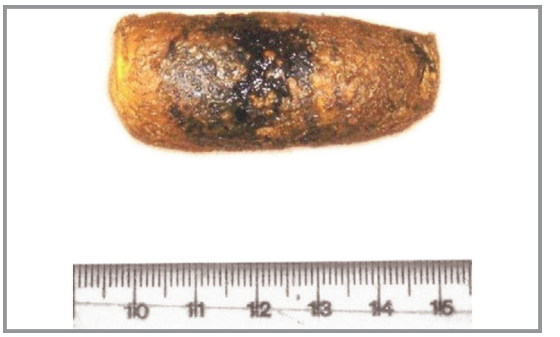
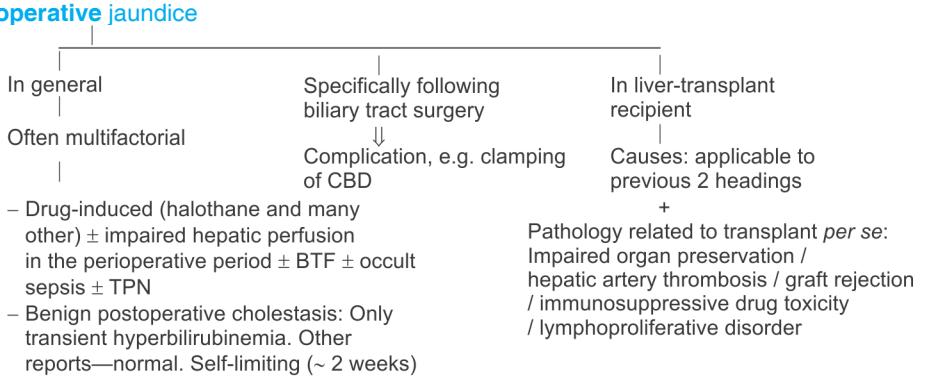


Fig 37-1 A (large) stone removed from CBD

3. *Outside the wall* (essentially extrinsic compression)
   - Neoplastic or inflammatory disease of the surrounding viscera like pancreas/lymph nodes/liver, e.g. CA head of the pancreas, chronic pancreatitis, HCC
   - Any SOL in the vicinity/porta hepatis, e.g. lymphadenopathy, vascular pathology like portal cavernoma/aneurysm
   - Torsion due to adhesion.
4. Primary duodenal carcinoma (50%).

F. 'Malignant' jaundice = jaundice specifically caused by a malignant disease—biliary, pancreatic or liver cancer. The presence of jaundice in association with liver tumors indicates extensive involvement of liver parenchyma, indicating incurability.

G. Postoperative jaundice



H. Jaundice during pregnancy

1. *Intrahepatic cholestasis*
   - Typically in 3rd trimester.
   - Main presentation—pruritus. Jaundice is infrequent.
   - Resolves within 2 weeks after pregnancy.
   - Tends to recur with subsequent pregnancies.
2. *Acute fatty liver*
   - Serious.
   - Occurs in 3rd trimester.
   - Pain in abdomen, nausea, encephalopathy.
   - Treatment: Prompt delivery; otherwise it may prove fatal.

www.Ebook777.com

I. Jaundice **during infancy and childhood**: Refer to chapter 80.

J. 'Pseudojaundice'

= yellow discoloration of skin with normal sclera and normal level of serum bilirubin.
Causes: Carotiderma (excessive carrots in diet), pernicious anemia, picric acid painting (malingering).

OUTLINE OF CLINICAL STRATEGY IN A JAUNDICED PATIENT

1. History and physical examination.
2. Key investigations: Blood, urine, and ultrasound scan of abdomen.
3. Evaluation of the investigation-reports to arrive at a 'provisional clinical diagnosis'.
4. Additional investigations, if required.
5. 'Definitive diagnosis'.

HISTORY

- **Age**?
- **Occupation**?
- Is this the **first episode** or Is this **a recurrent episode**? If recurrent, how may recurrences in the past, and details?
- **Duration**?
  If it is an acute case, also ask
  ↓
  – Swinging fever with rigors, f/b sweating?
  – Prostration?
  – Acute pain in the RUQ of abdomen?
  – **Contact with a jaundiced patient** within the previous 2 months?
- **Progress**?
  – Very rapid
  OR - 'Moderately' rapid
  OR - Intermittent
  OR - Slow and persistent, without remission?
- Is the **urine** 'light' **colored** OR high colored?
- Did you ingest raw shellfish/wild mushrooms?
- Do you get **undue fatigue**?
  Does your **anemia respond to iron therapy**?
- Do you get attacks of sudden onset of fever, pain in abdomen, nausea, vomiting, and extreme pallor, followed by increased jaundice?
  Were you transfused with blood during such attacks? (- for **severe crises of RBC destruction**)
- H/o **leg ulcers**?
- Has there been alteration of **color of stools**? – Pale / clay-colored?
  – Silvery?
- Do you get **itching**?
- **Pain**
  – Intermittent or constant (Continuous)?
  |
  If intermittent, – How long does each episode last?
  – How much is the pain-free interval?
  – During the so-called pain-free interval, are you completely asymptomatic, or do you continue to have some discomfort / dyspepsia?
  – Are the durations of pain-episodes and pain-free intervals more-or-less constant, or Are they variable?
  – Does the site of pain remain constant during each episode, or does it vary?

Free ebooks ==> www.Ebook777.com

- Duration of pain?
- Exact site of pain?
- In c/o pain in the RUQ / epigastrium: Does it radiate to the right lower back / right shoulder?
- Nature (Character)—colicky / constricting / burning / dragging / throbbing / twisting / shooting / vague?
- Does the pain initiate or get aggravated by lying down flat?
- Have you noticed any particular relieving factor such as assuming sitting position?

- Any h/o **gallstone disease**? – Present / Past; Treated / Untreated; Details?
- Any h/o **biliary-tract surgery** in the past?
- **Anorexia**? Is it **intense**?
- **Nausea**?
- How much **weight** did you loose over the past 6 weeks?
  (Does the loss of weight appear to be 'marked'?)
- Do you feel **weak**?
- Do you consume **alcohol** regularly?—How much and for how many years?
- Are you on any **(long-term) medication**?
- Chronic ingestion of **excess of vitamin A**?
- Recent past h/o – **Transfusion of blood or blood products**?
  – **Legal or illicit injection with hypodermic needle**?
  – **Unprotected sexual intercourse**?
- H/o pain **and swelling** of joints? (- for arthritis)
- Are you **diabetic**?
  Details: How long?
  On what treatment?
- **Skin rash**?
- **COPD**?
- Have you been suffering from **TB / sarcoidosis / lymphoma (particularly Hodgkin's), extensive tumors**?
- **OC pills**—Details.
- **Anabolic steroids**—Details.
- H/o **Hematemesis/melena**—Details.

INTERPRETATION OF SYMPTOMS

- Older **age** ⇒ more likelihood of malignant disease
- **Occupation**: Health-care workers are predisposed to hepatitis B/C/D. Industrial occupation with exposure to vinyl chloride may → cirrhosis
- High-grade **fever** with rigors and also if accompanied by pain ⇒ cholangitis
- H/o **contact with a jaundiced patient** in the recent past could be s/o (acute) viral hepatitis A/E
- **'Light' colored urine** ⇒ acholuric (hemolytic) jaundice
- **Undue fatigue and pallor** ⇒ anemia, which is often unresponsive to iron therapy
- **Crises of RBC destruction** } Suggest hemolytic disorder
- **Leg ulcers**
- **Pale / clay-colored stool, and itching** ⇒ cholestatic jaundice
- **Silvery** appearance of stools is due to a combination of steatorrhea and altered blood. ⇒ periampullary tumor.

Pain

– The presence of a mild discomfort even during the so-called pain-free intervals ⇒ possibility of gall-bladder disease.
– Pain in the upper abdomen/RUQ may suggest surgical disease such as cholangitis particularly due to cholelithiasis.
– Radiation of pain from epigastrium/RUQ → right side of the back (infra-scapular) or right shoulder ⇒ disease of gall bladder/bile ducts.
– Associated persistent pain in the mid-back could be due to pancreatitis.
– Pain caused by disease of gall bladder/CBD is nearly always continuous.
– Pain relieved by assuming sitting position ⇒ pancreatic disease.
– Pain relieved by applying heat ⇒ spasmodic pain caused by colic.

www.Ebook777.com

An obvious h/o **gallstone disease** may suggest surgical jaundice.
Previous biliary-tract surgery ⇒ more likelihood of a surgical jaundice.

Alcohol
– Alcohol may → hepatitis
– A daily intake in excess of 60–80 gm for men and 20 gm for women over a period of 10–15 years incurs a high risk of developing cirrhosis of liver. However, not all alcoholics become cirrhotic.

Long-term medication
- Erythromycin, chlorpropamide, chlorpromazine, methimazole, etc. may lead to drug toxicity
- **Chronic ingestion of excess of vitamin A** may → cirrhosis
- Recent past h/o **transfusion of blood or blood products / legal or illicit injection with hypodermic needle / unprotected sexual intercourse** could be s/o viral hepatitis B/C/D
- **Arthritis and DM** may suggest hemochromatosis
- **Pains in the joints alone** without swelling of joints = **arthralgia** ⇒ autoimmune hepatitis, drug toxicity
- Arthralgia + skin rash ⇒ drug toxicity
- **Recent onset of DM** is an important clinical feature of pancreatic cancer
- **COPD** may suggest ∝1 antitrypsin deficiency
- Diseases like TB / sarcoidosis / lymphoma (particularly Hodgkin's), extensive tumors, etc. → infiltrative disease of the liver → intrahepatic cholestatic jaundice
- **OC pills**, **anabolic steroids** → intrahepatic cholestatic jaundice.

In **GENERAL EXAMINATION**, look for the following specific findings

– Does the patient look toxic?
– Depth of jaundice—deep or 'light'?
– Deep pigmentation ⇒ Hemochromatosis.
– Scratch marks—due to itching—indicate cholestatic jaundice.
– Skin rash ⇒ Drug toxicity.
– Spider naevi (> 3).
– Palmar erythema.
– Leg ulcers may suggest hemolytic jaundice.

ABDOMINAL EXAMINATION

For clinical method of abdominal examination refer to chapters 33, 34 and 35.
Look for the following specific findings.

– Scar on the abdominal wall
– Tenderness
– Lump (*other than hepatomegaly*)

} May suggest surgical jaundice.

– Prominent veins.
– Ascites.
– Splenomegaly.
– Tender hepatomegaly on the background of rapidly oncoming ascites may be indicative of hepatic veno-occlusive disease.

ARRIVING AT A PRELIMINARY CLINICAL IMPRESSION

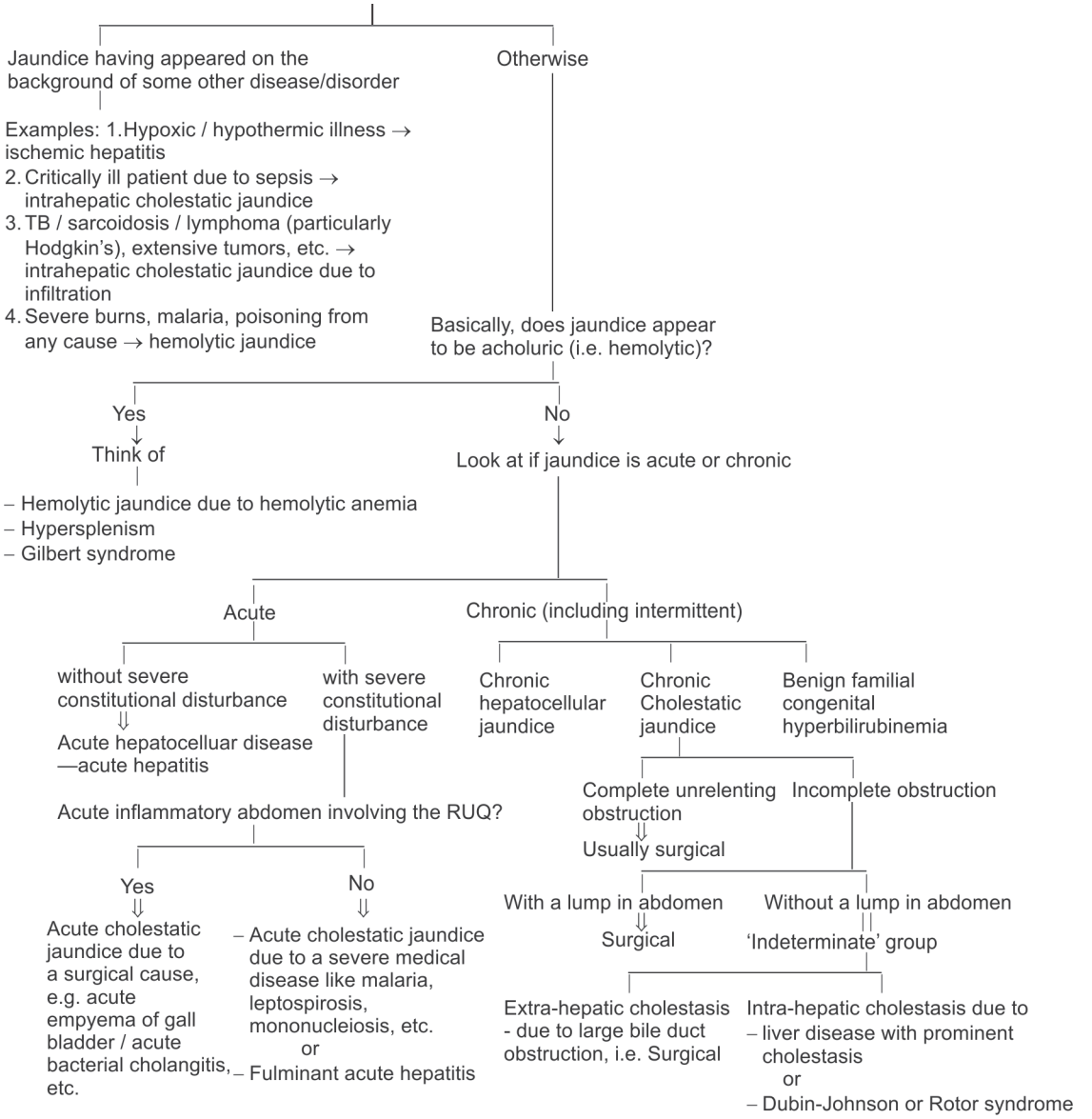
PRINCIPLES AND PITFALLS

1. **The absence of pain does not necessarily rule out surgical jaundice.**
2. Sepsis-related cholestasis in critically ill patient may be difficult to distinguish from obstruction of bile ducts.
3. In a case of jaundice there is usually overlapping of at least 2 groups.

Free ebooks ==> www.Ebook777.com

STEPWISE ANALYSIS OF DATA

From history and physical examination



Hemolytic jaundice

Differentiating points

- Undue fatigue and anemia not responsive to iron therapy.
- Urine—not high colored.
- The absence of pain, pale stools and itching.
- 'Depth' of jaundice—mild.

www.Ebook777.com

Supporting points
- Young age.
- Basic causative disorder/disease pointing to hemolysis may be present.
- Leg ulcers.
- Splenomegaly, or hepatosplenomegaly.
- Lymphadenopathy may be present if reticulosis is the cause.

Differentiating / supporting points	**Hepatocellular** (*syn. 'hepatogenous'*) **jaundice**		**(Chronic) Obstructive jaundice**	
	Acute	**Chronic**	**Incomplete**	**Complete**
Previous h/o jaundice	Rare	Rare	Frequent	Nothing particular
Duration	2–4 weeks	4–6 weeks	> 6 weeks	> 6 months
Progress	Very rapid	'Moderate'	Intermittent	Usually persistent, without remission
Clay colored stools	±	–	Intermittent	+
Pain in abdomen	Ache	Absent	Usual	Slight, steady and boaring
Present/past h/o gallstone disease or cholecystitis	–	–	+	Nothing particular
H/o biliary tract surgery in the past	–	–	±	±
Anorexia	+	+	Occasional	Intense
Nausea	+	+	Occasional	Occasional
Loss of weight	–	Marked; but weight increases in ascites	Frequent	Marked
Fatigue	+	+	Occasional	Occasional
H/o hepatotoxic drug ingestion	±	±	–	–
H/o hematemesis / melena	Absent	Present	Absent	Occasional
Toxic look	+	+	–	–
Depth of jaundice	'Moderate'	'Mild'	'Moderate' and variable	Intense
Spider nevi (> 3) and palmar erythema	Rare	Present	Absent	Absent
Porto-systemic anastomoses	Absent	+	Absent	Absent
Tenderness (in RUQ)	+	Absent	+	Absent
Palpably enlarged gallbladder	–	–	–	±
Splenomegaly	Absent	Present	Absent	Absent
Ascites	Absent	Present	Absent	±

Free ebooks ==> www.Ebook777.com

FBC (including peripheral blood smear)

– Anemia may suggest occult blood loss
– A low WBC / platelet counts may suggest hypersplenism
– Eosinophilia ⇒ granulomatous chronic infiltrative disease like sarcoidosis/parasitic disease / drug toxicity.

S. bilirubin

In chronic hepatocellular dysfunction, s. bilirubin is lower than in acute hepatocellular injury, unless the chronic disease is advanced.

S. transaminases

Increased levels ⇒ hepatocellular damage.

– In alcoholic liver disease: S. AST is < 10 times normal, and > S. ALT at least by 2 times
– Predominant elevation of S. transaminases in comparison with S. alkaline phosphatase ⇒ intrinsic hepatocellular disease.

A pitfall

In transient biliary obstruction due to choledocholithiasis, S. transaminases levels may be transiently but dramatically elevated exceeding 10–20 times normal.

S. alkaline phosphatase

– Cholestasis → increased formation of alkaline phosphatase by hepatocytes (reasons not clear), and regurgitation into plasma
– The degree of the excess may indicate the site of obstruction
– 'A marked rise' may suggest extrahepatic obstruction (surgical jaundice)
– 'Lesser' degrees of rise may suggest intra-hepatic cholestasis.

In a c/o obstructive jaundice, a rise in S. alkaline phosphatase precedes that of bilirubin and its fall is more gradual once obstruction is relieved.

In c/o fulminant Wilson's disease

- S. alkaline phosphatase : S. bilirubin ratio is < 2
- S. AST : S. ALT ratio is > 4, particularly when S. transaminases levels are elevated to < 10 times normal.

An atypical circumstance

Alcoholic hepatitis manifesting with pain, fever and leucocytosis may be difficult to distinguish from obstruction of bile ducts. S. alkaline phosphatase occasionally increased > S. transaminases levels. In such situation, points going in favor of alcoholic hepatitis are: Known alcoholic. Typical relation between AST and ALT.

S. albumin

Reduced level ⇒ hepatogenous jaundice.

PT

= a measure of plasma activity of certain coagulation factors (proteins) synthesized in the liver.

Prolonged PT ⇒ impaired synthesis of these coagulation factors, or deficiency of vitamin K (an essential cofactor in the further chain of clotting).

Vitamin K absorption requires an intact enterohepatic circulation (hence, an unobstructed biliary tree for intestinal absorption) of bile acids.

Urine for bile salts, bile pigments, and urobilinogen level.

– Urobilinogen level is increased in hemolytic jaundice.
– In a c/o complete unrelenting obstruction, examine every specimen of urine for urobilinogen over 24 hours. The absence of urobilinogen in every specimen of urine over 24 hours confirms completeness of obstruction.

www.Ebook777.com

S. bilirubin level	*S. transaminase (ALT, AST) levels*	*S. alkaline phosphatase & 5' nucleotidase levels*	*Urine bile pigments*	*Urine urobilinogen*	*Type of jaundice*
↑.	N	N	Absent	–	Benign familial congenital hyperbilirubinemia
↑. Unconjugated	-	Cholestatic component may develop with prolonged/recurrent hemolysis	Absent	Present	Acholuric
↑. Predominantly conjugated	↑	N	Uncertain	Present	Hepatocellular (Hepatogenous)
↑. Conjugated	N / ↑ ↑ may suggest intrahepatic cholestasis	↑	Present	Absent	Cholestatic

↑ = Increased, N = Normal

US scan of abdomen

This is the 1st line imaging investigation.

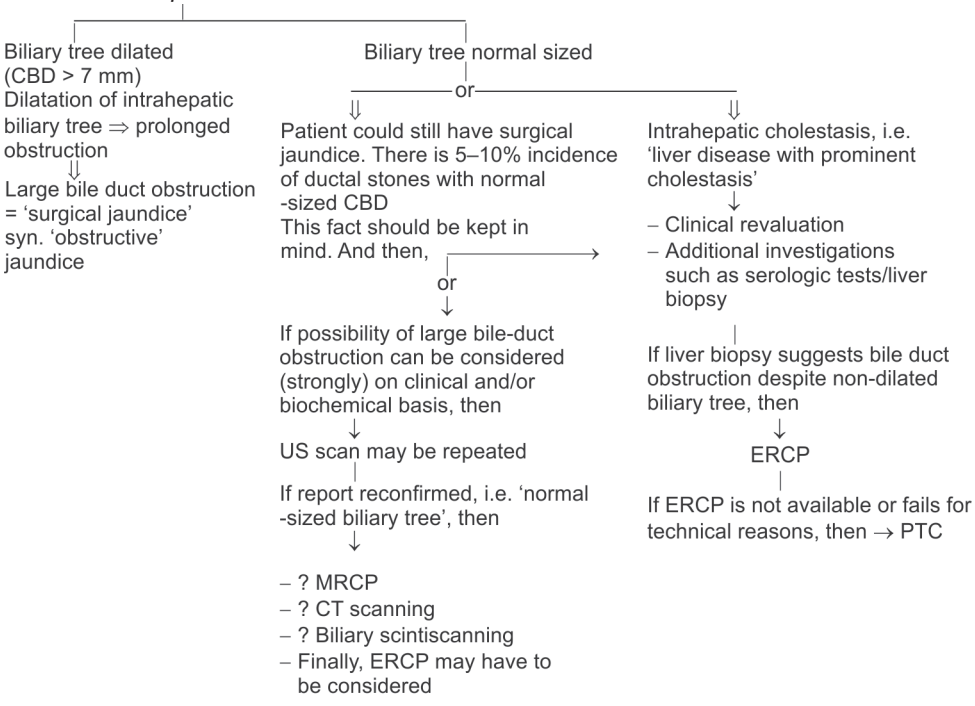
Look for: Dilatation of bile duct (> 7 mm), gall stones, gall bladder disease, pancreatic disease, hepatic parenchymal disease.

Additional diagnostic utility: Guided FNAC of a mass lesion can be performed.

Disadvantages

1. US scanning can't give accurate information on the **exact site and extent** of the pathology; ERC and/or PTC may be indicated for the same.
2. Technical difficulty in case of gaseous distension of upper abdomen, obesity, ascites, etc. Alternative: CT / MRI.
3. Ultrasound scanning is operator-dependant.

Evaluation of report



Free ebooks ==> www.Ebook777.com

ACHOLURIC JAUNDICE

- Get hematological investigations for demonstration of increased hemolysis
- Is the spleen palpably enlarged?
- Abnormally marked hemolysis of normal RBCs, in the presence of a palpably enlarged spleen suggests hypersplenism
- Think of Gilbert syndrome if RBCs and spleen are normal.
- **A low Hb level** | Sufficient evidence of hemolysis, if a reason
- **Increased reticulocyte count** | for increased hemolysis can be demonstrated

Examples of causes of increased hemolysis: Congenital spherocytosis, elliptocytosis, thalassemia, pernicious anemia, acquired hemolytic anemias.

Gilbert syndrome

- This is one of the causes of neonatal jaundice, however it may present in adult life
- A young adult usually presents without a clear-cut history of jaundice since birth
- Jaundice is usually intermittent (reason not clear)
- Clinical condition is usually mild; no need to investigate further, but in case of doubt, a needle biopsy of liver may be performed.

Possible role of surgery in acholuric jaundice: 1. Splenectomy for hypersplenism, 2. Cholecystectomy for (pigment) stones in gallbladder.

ACUTE HEPATITIS

- Anorexia, nausea, vomiting and tender hepatomegaly
- Characterized by hepatocellular jaundice
- *Etiology*—varied
- The most common causes: Viruses, drugs, alcohol.

Natural history

- In majority of cases the disease is mild and self-limiting. Full recovery. No long-term sequelae
- In some cases proceeds to acute fulminant liver failure (massive liver cell necrosis)
- Chronic carrier state. This may be associated with normal liver function or with the development of chronic liver disease.

1. **Viral** hepatitis
   - A, E - transmitted enterally. Self-limited
   - B, C, delta, G—transmitted parenterally (blood/blood products, needles, tattooing, unprotected sexual intercourse, mothers to babies, aerosol (dental treatment)). May prolong and progress to chronic disease
   - Other viruses: Epstein-Barr, cytomegalovirus, yellow fever virus, Ebola and Marburg (→ African hemorrhagic fever).
2. **Drug-induced** hepatitis
   Few examples—halothane, INH, phenytoin, methyldopa, etc.
3. **Alcoholic** hepatitis
   - May be mild and virtually asymptomatic
   - Edema and ascites ±
   - In severe cases, encephalopathy ensues
   - Recurrent attacks of acute hepatitis may be superimposed on cirrhosis
   - 'Typical' levels of serum transaminases.
4. **Wilson's disease**
   Initially clinically indistinguishable from acute viral hepatitis.
   - Age < 40
   - K-F rings on ophthalmologic examination
   - Disproportionate hyperbilirubinemia

www.Ebook777.com

- In c/o fulminant clinical manifestation:
  - S. alkaline phosphatase : S. bilirubin ratio - < 2
  - S. AST : S. ALT ratio - > 4, particularly when S. transaminases levels are elevated to < 10 times normal
- Confirmation of diagnosis: Liver biopsy.

5. **Ischemic** hepatitis
   Occurs on the background of some other hypoxic/hypotensive illness. Rapidly oncoming ascites and hepatomegaly may suggest hepatic veno-occlusive disease.
6. **Bacterial** hepatitis—may progress to form pyogenic liver abscess(es).
7. **Amoebic** hepatitis—may progress to form an abscess.

CHRONIC HEPATITIS

Chronic Hepatitis includes 3 syndromes all of which are characterized by hepatomegaly and persistent abnormality of LFTs.

1. **Chronic persistent** hepatitis
   Pathology: Continued inflammation of the portal tracts without necrosis, and normal architecture after an attack of acute hepatitis.
   Transaminases levels remain mildly elevated; other liver biochemistry is normal.
   Natural history
   - Prognosis good in majority of cases
   - Some cases progress to chronic active hepatitis and cirrhosis.
2. **Chronic active** hepatitis
   Usually follows hepatitis B, C or D, but may also be drug/alcohol induced.
   Pathology: Inflammation of the hepatic lobules is progressive for up to 6 months. Inflammation extends beyond the confines of the portal tracts and there is accompanying piecemeal necrosis.
   LFTs are abnormal: S. bilirubin, AST, ALT, S. alkaline phosphatase and γ-glutamyl transpeptidase levels are raised.
   Natural history
   - Often progresses to cirrhosis and liver failure
   - High risk of developing HCC.
3. **Autoimmune chronic active** hepatitis
   - Hyperglobulinemia, non-organ-specific autoantibodies
   - Associated with other autoimmune disorders, e.g. RA, ulcerative colitis, Hashimoto thyroiditis.

CHRONIC CHOLESTATIC JAUNDICE

Further diagnostic point of interest: What is the site and extent of obstruction?

- Obstruction can be extra-hepatic ('surgical' jaundice) or intra-hepatic ('medical' jaundice)
- A clinician can face a very difficult problem if the site of obstruction is uncertain. On one hand, the failure to relive obstruction results inevitably in death, a tragedy which could have been avoided by removing or bypassing the obstruction. On the other hand however, if cholestasis is intrahepatic and hence not amenable to surgery, then the patient's condition may be adversely affected by the (unnecessary) trauma of surgery.

1. **Complete unrelenting obstruction**

*Although hepatocellular disease **can** produce complete obstruction, such a phase rarely lasts more than a few days.*

The best way to detect completeness of obstruction is to examine every specimen of urine for urobilinogen over 24 hours. The absence of urobilinogen in every specimen confirms complete obstruction.
Additional imaging investigations are necessary to determine the exact site and extent of the extrahepatic obstructing pathology. Eventually, a laparotomy may be required.

Free ebooks ==> www.Ebook777.com

2. **Incomplete obstruction with a lump in abdomen** (excluding hepatomegaly; any cause of cholestatic jaundice may produce sufficient turgidity in the liver to render the organ palpable).

The (palpable) lump may be enlarged gall bladder or other than enlarged gall bladder (such as for example metastatic lymph nodes in porta hepatis causing obstruction of the biliary tree). If gall bladder is palpably enlarged, then the pathology (which in itself may not be palpable) could be a tumor obstructing the distal CBD rather than a calculous disease (as per Courvoisier's dictum).

3. **'Indeterminate' group**

This group consists of cases without palpable lump in abdomen (excluding hepatomegaly), and probably without gross constitutional disturbance; and the course of the illness therefore being described as more or less indeterminate.

Possible causes of jaundice in this group: Large bile duct obstruction, or liver disease with prominent cholestasis or benign familial congenital conjugated hyperbilirubinemia (Dubin-Johnson or Rotor syndrome).

If PT is prolonged

↓

Injection vitamin K 10 mg IM/IV once a day × 3 days

PT becomes normal **within 36 hours**	PT remains prolonged
⇓	⇓
Cholestatic jaundice likely to be due to large bile duct obstruction	Cholestatic jaundice more likely to be due to intrahepatic cholestatic pathology

Principles of management if the problem remains unresolved

↓

'Expectant line of treatment' for about a month. Investigate though to try to shorten the period of uncertainty as much as possible.

↓

Patient may improve, or may reach the stage of complete unrelenting obstruction.

↓

Indication for laparotomy becomes clear. However, by that stage considerable damage to the liver may have been produced by the prolonged period of cholestasis

ACUTE CHOLESTATIC JAUNDICE

It is associated with **constitutional disturbances**.

– Swinging fever with rigors
– Sweating
– Prostration
– Tense and tender hepatomegaly

} Common to many different types of infection of the liver.

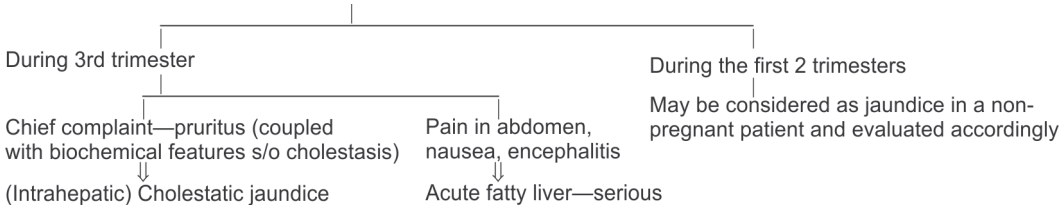
These patients present with acute illness

Essentially surgical	Essentially medical
Presentation: Acute inflammatory abdomen involving the RUQ	↓
↓	– Malaria
– Acute bacterial cholangitis	– Infectious mononucleiosis
– Recurrent pyogenic cholangitis	– Leptospirosis/relapsing fever:
– Acute pyogenic liver abscess	- At an early stage of the disease: spirochetes may be found in the blood
– Acute pylephlebitis	- At a later stage: Antibodies to these organisms are found
– Amoebic liver abscess	– TB
– Subphrenic abscess	– Syphilis
– Acute calculous cholecystitis	– Yellow fever
– Acute pancreatitis	– Shock

www.Ebook777.com

In a few patients the investigations reveal no specific cause and fail to substantiate a diagnosis of septicemia. This is a serious situation with a high mortality. Mainstay of treatment in such a case: Combination of powerful antibiotics. Should the patient improve, the possibility of gallstones being responsible for septicemia should be urgently investigated.

ARRIVING AT A DIAGNOSIS OF JAUNDICE DURING PREGNANCY



NOTES ON LIVER, BILIARY AND PANCREATIC DISORDERS WITH REFERENCE TO JAUNDICE

LEVER FAILURE

The hallmark symptom of liver failure is diminished LOC or coma. Hence, it is also referred to as hepatic encephalopathy.
Coma always indicates severe decompensation of liver function.

Acute (fulminant) liver failure

Severe encephalopathy occurring within 8 weeks of the onset of illness.
Pathology: Acute massive hepatocellular necrosis in a previously normal liver.
Causes: Viral infection, drugs, poisoning, severe shock.
More common in females irrespective of etiology.
Multisystem involvement

- Bleeding due to failure of clotting
- Shock due to loss of peripheral vascular resistance
- Coma due to cerebral edema
- Renal impairment
- Extreme susceptibility to serious respiratory infections due to severe depression of immune system
- Acute pancreatitis.

Prognosis: Bad despite newer methods of hepatic support and liver transplantation.

Chronic liver failure (*Syn.* End-stage liver disease)
Arises against a background of cirrhosis.

- Severe mental impairment
- Episodes of varieceal bleeding from portal HT
- Muscle wasting
- Fluid and salt retention → edema, ascites, pleural effusion
- Bleeding tendency
- Splenomegaly
- Thrombocytopenia.

CIRRHOSIS

Cirrhosis is the end result of hepatocyte death. Cirrhosis needs to be distinguished from hepatic fibrosis.
Causes / types of cirrhosis

- Posthepatitic (viral hepatitis B/C, alcoholic, etc.)
- Cryptogenic: cause unknown or cannot be established.

Free ebooks ==> www.Ebook777.com

Severity of the disease and *prognosis* can be judged on the basis of clinical assessment and biochemical profile. Severity scores are available for objective documentation.

PORTAL HYPERTENSION

The vast majority of cases arise against a background of cirrhosis.
Not all patients with chronic liver disease develop portal HT.

- Varieceal bleeding. A serious situation. Sequelae: Hypovolemia leading to further deterioration of the hepatic and renal function, and encephalopathy
- Fluid and salt retension → edema, ascites
- Hypersplenism.

SPECTRUM OF SYMPTOMATIC GALLSTONE DISEASE

The symptomatology and pathogenic potential of gallstones vary widely. Silent (asymptomatic) gallstones heavily outnumber the symptomatic ones.
When symptomatic, gallstones can cause:

- Chronic cholecystitis. Jaundice is rare.
- Acute biliary colic / Acute obstructive cholecystitis. Jaundice is seen in 20–40% of these cases
- Large bile duct obstruction. Jaundice in 100% of cases
- Cholangitis and septicemia. Jaundice in 100% of cases
- Acute gallstone-associated pancreatitis. Jaundice in 50% of cases
- Biliary fistulous disease
- Gallstone ileus
- CA gallbladder.

DUCTAL STONES

Pathogenic potentials of ductal stones: Jaundice, cholangitis, acute gallstone-associated pancreatitis.
Clinical terms used to describe ductal stones may be based on etiology and the way they get diagnosed.

A. Based on etiology

1. **Secondary** = stones migrated from the gallbladder through a patent cystic duct
2. **Primary** = stones which arise *de novo* within the bile ducts
   Predisposing factors:
   - Subclinical obstruction at the distal end of the CBD
   - Foreign body within the CBD like a non-absorbable suture, metal clip, etc. Patient presents several months after an uneventful surgery (e.g cholecystectomy) with jaundice and/or cholangitis.

B. Based on the way they get diagnosed

1. **Unsuspected** = stones detected accidentally on intraoperative cholangiography during a cholecystectomy.
   - Incidence: 5–10% of patients undergoing cholecystectomy
   - LFTs—normal although some patients may have a mild elevation of S. alkaline phosphatase that is either missed or attributed to other causes.
2. **Retained** (*Syn.* **Missed**) = stones that present or are diagnosed within 2 years of the intervention (surgical or endoscopic). Most are, in fact, detected much earlier.
   Missed stones indicate that the intervention failed to achieve complete ductal clearance.
3. **Recurrent** = stones presenting at least 2 years after (the first) intervention. They tend to be primary ductal stones. Almost always associated with significant dilatation of the CBD, indicating a fibrotic or dysfunctional terminal segment of the CBD.

www.Ebook777.com

PERIAMPULLARY CANCER

= cancer of the distal CBD / ampulla of Vater / periampullary duodenum (within 1 cm of papilla).
Ways of presentation other than obstructive jaundice:
– Pancreatitis
– Silvery appearance of stools due to a combination of steatorrhea and altered blood.
It is **important to distinguish between pancreatic adenocarcinoma and periampullary tumors** because of their vastly different prognosis. Clinical picture of both however, is dominated by jaundice (obstructive) and itching.

CHOLANGIOCARCINOMA

Classification according to the anatomical site of origin
– Intrahepatic: Often multicentric. Classified with primary liver tumors
– Proximal: From right/left hepatic ducts, confluence, and proximal common hepatic duct
– Middle: From distal common hepatic duct, cystic duct and proximal CBD.
Most of these tumors are scirrhous. Some can be very acellular, such that histological confirmation may be difficult.
Radiological feature: Stricture with proximal dilatation.

BENIGN BILE DUCT STRICTURE

Causes
– Most common—iatrogenic injury during cholecystectomy. Consequences of stricture: Recurrent cholangitis, hepatic fibrosis, secondary biliary cirrhosis, and portal HT
– Parasitic infestation
– Pseudotumor of chronic pancreatitis. This situation usually poses a diagnostic problem, as it may be very difficult to exclude an associated pancreatic cancer even on histology
– Sclerosing cholangitis—multiple strictures are the hallmark. An obscure disorder of uncertain etiology. It has a well-established association with ulcerative colitis.
  Affection of small intrahepatic radicles is called pericholangitis.

PRIMARY MALIGNANT TUMORS OF THE LIVER

Commonest—hepatocellular carcinoma (HCC). Other varieties are rare, e.g. hepatic cholangiocarcinoma.
Clinical manifestations of **HCC**
– In early stages—asymptomatic
– Dragging sensation / painful discomfort in RUQ
– General symptoms s/o a chronic liver disease: Anorexia, nausea, vomiting, weakness, jaundice, etc.
– Peripheral stigmata of chronic liver disease ±
– Hepatomegaly / hepatic mass. Bruit ±
– Ascites—may be blood-stained
– Acute presentation due to rupture → (Massive) hemoperitoneum.
Investigative parameters supporting the diagnosis of HCC
– Abnormal LFTs
– S. ∝-fetoprotein level–elevated in only 60–70% of cases. Estimation of the level is useful in screening high-risk group patients (cirrhotics for example), and follow-up cases after resection of tumor
– Hypoglycemia, hypercalcemia, hyperlipidemia, hyperthyroidism
– CT/MRI can detect tumor only of > 1 cm in diameter.

Free ebooks ==> www.Ebook777.com

HEPATIC METASTASES

More common than primary malignant tumors.
Common primary sites: GIT, pancreas, lung, and breast.

Clinical classification

- Occult = present but undetectable at the time of resection of the primary. Detected at subsequent follow-up
- Asymptomatic = known to be present but with no symptoms or jaundice. S. alkaline phosphatase or γ-glutamyl transpeptidase may be elevated. May be potentially curable
- Symptomatic: Pain in RUQ, overt clinical jaundice/ascites, hepatomegaly. Incurable.

Morphological classification

- Discretely nodular: single or multiple; unilateral or bilateral. Only this type is potentially curable by surgical resection
- Miliary = widespread small seedling deposits
- Diffusely confluent – involves multiple segments or lobes.

www.Ebook777.com

38 PORTAL HYPERTENSION

Normal portal venous blood pressure: 5–15 cm of saline.
While no value is absolute, portal hypertension may be assumed when the portal vein pressure exceeds 20 cm of saline.

Causes of portal HT

1. *Extrahepatic inflow obstruction*
- Congenital malformation of portal vein like atresia, etc.
- Portal vein thrombosis (causes: neonatal sepsis, pylephlebitis, trauma, tumor).
- Splenic vein thrombosis (→ sectorial portal HT).
- Portal vein compression, e.g. by enlarged lymph nodes, pancreatic disease, biliary tract tumors.

2. *Intrahepatic obstruction*
- Presinusoidal: periportal fibrosis and schistosomiasis.
- Postsinusoidal: cirrhosis (alcoholic, nutritional, postnecrotic, biliary), veno-occlusive disease, hemochromatosis, Wilson's disease, congenital hepatic fibrosis.

3. *Extrahepatic outflow obstruction*
- Hepatic vein thrombosis, Budd-Chiari syndrome, veno-occlusive disease.
- Tricuspid incompetence, right heart failure.

4. *Increased blood flow into portal venous system (No obstruction)*
Hepatic and splenic arterioportal fistulae. This is a rare cause of portal HT.

By far the most common cause of portal HT is postsinusoidal and results from cirrhosis of liver.
Not all patients develop portal HT as a result of their chronic liver disease. Furthermore, only about one-half of the patients with gastroesophageal varices ever suffer from GI bleeding.
Portal HT sometimes develops in patients with liver disease prior to the onset of cirrhosis. This has been attributed to intrahepatic arterioportal anastomoses, hyperdynamic circulation and the accumulation of vasoactive humoral factors which alter the resistance to flow.

Patients with portal HT caused by extrahepatic outflow obstruction rarely present with bleeding varices, but suffer with intractable ascites, painful hepatomegaly and rapidly deteriorating liver function.

In sectorial portal HT, the varices affect the short gastric and gastroepiploic veins.

Obstruction to portal venous flow is followed by enlargement of natural portosystemic communications, as well as by the development of new collateral channels at surgically constructed mucocutaneous junctions (colostomy, ileostomy).
Rarely, portal venous blood is shunted away from the liver by an enlargement of the umbilical vein and may be detected by a venous hum in the midline (Cruveilhier-Baumgarten syndrome).
Though there is a risk of variceal bleeding from the ileum, colon and hemorrhoidal areas, the major risk of bleeding is from the esophagus and stomach.
In the stomach, there is venous engorgement of the gastric mucosa with a tendency to erosive gastritis and a widespread diffuse bleeding (portal hypertensive gastropathy). Most patients suffering from this condition will have portal venous pressures in excess of 30 cm of saline.

Free ebooks ==> www.Ebook777.com

Colonic varices may occasionally be seen on sigmoidoscopy though not as commonly as may be expected. Nor is the caput medusae or periumbilical plexus of veins at all common. It is most prominent in patients who develop a small umbilical hernia into which an omental plug provides the portal blood flow.

Bleeding from periumbilical and retroperitoneal collaterals is troublesome during abdominal surgery.

3 clinical syndromes can be attributed to portal HT: Ascites, hypersplenism and GI bleeding.

– The development of fluid retention and ascites is extremely common, and its incidence correlates with the degree of advancement (stage) of the disease.
– Progressive enlargement of the spleen occurs as a result of vascular engorgement and associated hypertrophy, subsequently leading to development of hypersplenism. Hypersplenism results in anemia, leucopenia and thrombocytopenia. Only rarely are these features sufficient to produce major symptoms but they do lead to general debility. **After portal vein decompression, hypersplenism improves in approximately 50% of patients.**

Children with extrahepatic portal HT commonly bleed when suffering from URTI.

Patients with portal HT usually present to a surgeon because of active uncontrollable bleeding from varices (most commonly gastroesophageal), or for consideration of elective surgery for varices that have been resistant to non-surgical treatment.

Severity of cirrhosis, when responsible for portal HT, may be best assessed by using objective scoring systems.

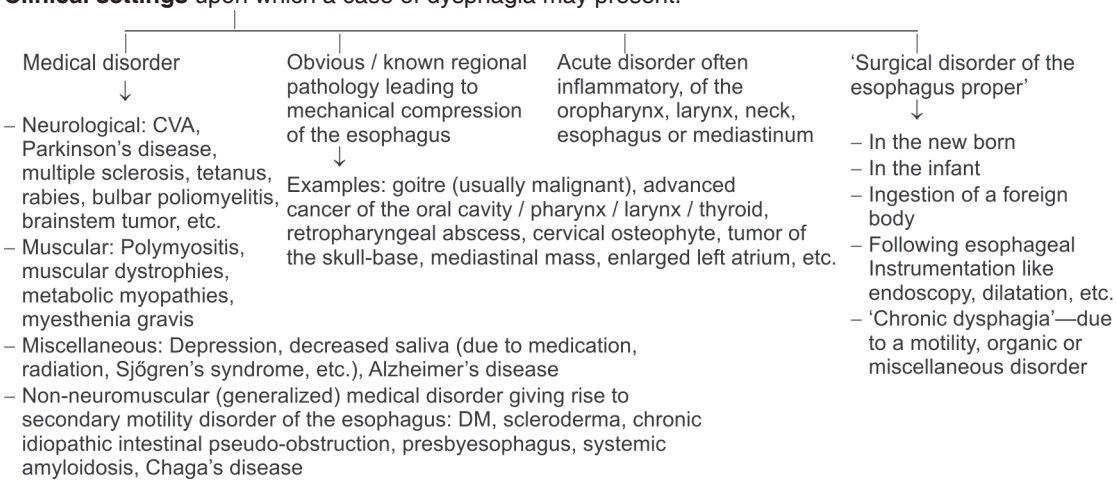
www.Ebook777.com

39 DIFFICULTY IN SWALLOWING AND OTHER SYMPTOMS OF ESOPHAGEAL DISORDER

'Dysphagia' is **defined** as a difficulty in swallowing, with or without pain.
It is a continum, bracketed by 'half-noticed hesitation/sticking of food on its journey to the stomach' on one hand, to 'inability to swallow even saliva' indicating total blockage on the other.

GENERAL REMARKS

1. **Dysphagia is the 'signature symptom' of esophageal disorder**. However, dysphagia may also be caused by nonesophageal disorders.
2. **Clinical settings** upon which a case of dysphagia may present.



One must be careful to distinguish dysphagia from disturbances resulting from neuromuscular incoordination (e.g. palatal paralysis) during swallowing. Coughing, choking, bronchospasm resulting from neuromuscular incoordination may be misinterpreted by the patient as difficulty in swallowing.

3. In obstructive dysphagia, symptom begins when ~ 25% of the lumen is compromised. And by the time patient presents, 50% of the lumen is compromised.
4. A patient with heightened awareness of swallowing events almost amounting to dysphagia may have normal radiological and motility studies, while a patient may not be aware for a long time, of a peptic stricture that may have taken years to develop. **20% of patients with peptic stricture are unaware of antecedent reflux.**
5. By the time dysphagia is recognized for what it is, half of the patients with cancer have metastases.
6. **Long duration of esophageal symptoms does not necessarily exclude malignancy**, as for example when cancer complicates pre-existing non-malignant stricture/achalasia.
7. Gastroesophageal reflux is a common condition which may coexist with carcinoma of the lower esophagus. (Hence, the insistence on several biopsy specimens during endoscopy).

Free ebooks ==> www.Ebook777.com

8. Dysphagia must always be taken seriously, and early diagnosis is mandatory.
9. Esophagus is not amenable to physical examination. Hence, 'history-taking' plays extremely important role in clinical evaluation of esophageal disease, and that must be supported by appropriate investigation.

Symptoms suggestive of esophageal disorder

- Dysphagia
- Excessive belching
- Difficult/unable to belch
- Odynophagia = Painful swallowing
- Present/past h/o ingestion of a foreign body
- Heartburn = Feeling of warmth behind lower part of the sternum
- Vanishing heartburn = Spontaneous disappearance of heartburn
- Regurgitation = Passive return of swallowed material to the pharynx/mouth when stomach / esophagus is overfilled / obstructed / hypotonic / valveless
- Waterbrash = A sudden filling of the mouth with clear, frothy salty fluid
- Pain in the chest—esophageal angina
- Aspiration and respiratory symptoms
- Globus = Sensation of a substernal lump
- 'Vomiting'
- Hematemesis (gross 'major' bleeding).

DYSPHAGIA

HISTORY

Step 1

- **Acute** or **Chronic**?

History to be asked in **AN ACUTE CASE**

- Did you (recently) swallow any foreign body?—Refer to chapter 62.
- Did you ingest a corrosive?—Refer to chapter 61.
- Did you recently undergo esophageal instrumentation like esophagoscopy, dilatation of an esophageal stricture, etc?—Refer to chapter 61.
- H/o acute (severe) pain in the mouth/throat?

History to be asked in **A CHRONIC CASE**
Description in patient's own wordings should be documented.

- What is **the exact nature of difficulty**?
  - Do you get nasal regurgitation, suffocation, coughing while attempting to swallow, i.e. while maneuvering a bolus through the pharynx?
    Does the voice have a nasal twang?
  - Is it globus that really bothers you rather than actual difficulty in swallowing?
  - Virtual feeling of hesitation/sticking of food on its journey to the stomach?
  - Do you get excessive secretion of saliva and a constant desire to swallow?

  ⇒ Obstructive disease of the esophagus proper.

Further history to be asked in a c/o **OBSTRUCTIVE DISEASE OF THE ESOPHAGUS PROPER**

- Duration?
- Mode of onset?
- Is it intermittent (in entirety or its severity)
  or
  Is it persistent and progressive
  or
  Is it stationary, or of variable progress?

www.Ebook777.com

- If progressive: Did it progress rapidly over weeks/few months, or did it progress gradually over years?
- If intermittent: Are episodes precipitated by emotional stress / too hot or cold liquids?
- What do you find easy to swallow—liquids or solids?
  Confirm by counter questioning: "So that means you find ….. more difficult to swallow than ….. Is that right?"
  In case of difficulty in swallowing both liquids as well as solids: Can you swallow saliva?

Another way to ask

- Can you swallow solids?
  |
  If yes, then
  ↓
- Do you find difficulty in swallowing only liquids?
- Can the difficulty be overcome by repeated swallowings / sipping of fluid / breath-holding / expiration against closed glottis?
- 'Intensity of dysphagia'—slight or severe?
- At what level do you think the food sticks? I.e. what is your perception of the level of obstruction?
- H/o **cough**?
  - Does it begin while attempting to swallow each gulp, or practically with the first or second sip of water?
    Is it associated with nasal regurgitation?
    Is it associated with suffocation/choking?
  - OR, does cough occur later on after meal is over?
  - Is the cough persistent?
- H/o hiccoughs?
- H/o **vomiting** (**virtually regurgitation**)?
  - Is it preceded by nausea?

  'Vomiting' that is not preceded by nausea, and which is effortless virtually means regurgitation.

  Further details of regurgitation to be asked
  - Amount of regurgitate?
  - Nature of regurgitate: Consistency, color, odor, blood, bile, food taken previously?
  - Is there a frequent regurgitation of mucoid material between feeds?
- H/o **pain**?
  - Is the swallowing painful? = 'Odynophagia' ⇒ oropharyngeal disorder, usually of inflammatory nature.
  - Do you get heartburn, i.e. feeling of warmth behind the lower part of sternum?
  - Do you get substernal pain? (Also refer to 'esophageal angina').
    - Does it remain limited to the midline?
    - Does it radiate between the shoulder blades/either lumbar regions?
    - Do you wake up due to pain from a sound sleep?
    - Intensity?
    - Nature?
    - Does the pain initiate or get aggravated by consuming particular type of food such as hot (chilly)/spicy/oily/fatty, or stooping forwards, or lying down flat?
    - Have you noticed any particular relieving factor?—such as belching or assuming sitting position or taking antacid or even NTG?
  - Do you experience constant deep boaring back pain?

  If patient's voice has become hoarse
- Did hoarseness follow dysphagia, or did it precede dysphagia?

At the end of this step 1, the case of obstructive disease of esophagus may fit in one of the 3 groups:
- 'Dysphagia suggestive of motility disorder'
- 'Dysphagia suggestive of organic disorder'

Free ebooks ==> www.Ebook777.com

– Indeterminate, i.e. 'not fitting in any of the 2 classical groups'.

INTERPRETATION OF SYMPTOMS OF STEP 1

Importance of gender

– Cardiospasm, carcinoma of hypopharynx, globus, and P-V syndrome is more common in females.
– Cancer of larynx and esophagus (except that of hypopharynx) is more common in males.

Nasal regurgitation, suffocation, coughing while **manoeuvering the bolus through pharynx**, and nasal twang in the voice ⇒ neuromuscular disturbance.

Acute (severe) pain in the mouth/throat ⇒ acute oropharyngeal disorder, usually of inflammatory nature.

Excessive secretion of saliva and a constant desire to swallow ⇒ obstructive disease of the esophagus and nothing more particular than that.

Diagnosis should not be based only on the **duration**, although by a general rule, dysphagia of only weeks/few months duration should arouse a strong suspicion of cancer. **However, long duration does not necessarily exclude cancer**, as for example when cancer complicates pre-existing non-malignant stricture or achalasia.

Persistent and progressive dysphagia usually ⇒ organic (mechanical) disorder.

Dysphagia for solids ⇒ significant disease, irrespective of the nature—whether organic (mechanical) or functional (motility disorder).

Intermittency of dysphagia in entirety or its severity usually ⇒ motility disorder. However, exceptionally, mechanical disorders like benign tumor or uncomplicated massive incarcerated hiatus hernia can lead to intermittent mild dysphagia.

Dysphagia for liquids only or more for liquids than for solids ⇒ more likely to be a motility disorder like achalasia, or laryngeal disorder.

If difficulty in swallowing can be overcome by repeated acts of swallowing / sipping of fluid / breath-holding / expiration against closed glottis ⇒ motility disorder. These maneuvers increase intra-esophageal pressure thereby helping to overcome the functional resistance.

Inability to swallow saliva, and constant drooling ⇒ total dysphagia.

Precipitation of episodes by emotional stress or liquids at extremes of temperature ⇒ motility disorder.

Intensity: – Slight in hiatus hernia, PV syndrome
– Can be severe in rest of the disorders such as achalsia cardia, stricture, cancer.

Coughing while attempting to swallow each gulp, or practically with the first or second sip of water essentially suggests neuromuscular disorder or upper aero-digestive disorder (pharyngeal/laryngeal).

Bout of coughing associated with nasal regurgitation ⇒ neuromuscular disorder.

Bout of coughing associated with suffocation/choking ⇒ neuromuscular disorder or tracheo-esophageal or broncho-esophageal (malignant) fistula.

Cough occurring later on after meal is over ⇒ regurgitation from a lower esophageal obstruction, or pharyngeal pouch.

Persistent cough ⇒ mediastinal mass / midesophageal lesion.

Regurgitation may suggest organic obstruction or late (advanced) achalasia cardia. Regurgitation is absent in DES (Diffuse Esophageal Spasm) and early achalasia cardia.

Pain:

If the patient is suffering from CA esophagus, then constant deep boaring back pain may suggest incurability.

For discussion on 'esophageal angina' refer to page 572.

Hoarseness of voice following dysphagia ⇒ infiltration of recurrent laryngeal nerve by cancer.

Hoarseness preceding dysphagia occurs in laryngeal lesions and thyroid cancer.

Sudden mode of onset of dysphagia may suggest previously silent pre-existing organic obstruction.

Step 2

Aim: Further differentiation to determine the most probable disease.

If MOTILITY DISORDER is suspected, ask for the following symptoms.

- Have you lost weight?
- Can you belch?
- Do you get pain in the chest?—Details: Exact site with extension/shifting/radiation, intensity, nature, timing, precipitating-aggravating-relieving factors, sweating?
- H/o regurgitation?

If ORGANIC DISORDER is suspected, ask for the following symptoms.

- How fast is it progressing? or Is it stationary?
- How about appetite?
- Have you lost 'significant' weight?

www.Ebook777.com

- Any antecedent h/s/o GERD?: (Postural) Heartburn, regurgitation, odynophagia and water brash.
- Any antecedent h/o ingestion of any corrosive a few weeks or more before developing dysphagia?

INTERPRETATION OF SYMPTOMS OF STEP 2

- Rapid progress over weeks / months ⇒ malignancy
- Gradual progress over many years ⇒ peptic stricture
- Progress of a corrosive stricture is variable
- Anorexia and 'significant' loss of weight strongly go in favor of malignant stricture. They are not the features of benign stricture.

At this juncture, possibility of a particular diagnosis or differential diagnosis about common disorders should become clear viz classic achalasia, diffuse esophageal spasm, vigorous achalasia, cancer, peptic stricture and corrosive stricture.

However, to make history-taking complete, the following questions may be asked for other organic disorders.

• Dyspepsia? • Breathlessness with meals? • Waning of symptoms of GERD, if they were present earlier? • (Iron-deficiency) Anemia?	Massive incarcerated (otherwise uncomplicated) hiatus hernia.
• Vague retrosternal pressure? • 'Spluttering' on swallowing? • Progressively increasing airway obstruction (inspiratory stridor)? • Regurgitation of a fleshy tumor from the mouth; appearing and disappearing?	These are various modes of presentation of a benign tumour, apart from dysphagia (dysphagia could be either intermittent and mild, or persistent).
• During meals, do you get bouts of coughing and spluttering, and/or gurgling noises during swallowing? • Do you take long to eat (even a small meal)? • Do you get regurgitation of non-acid material? • Do you get (constant) throat irritation?	Pharyngoesophageal diverticulum.

- Ask about history of various medical diseases, which can lead to secondary motility disorders.

Step 3

Aim: to determine complication.

- H/o breathlessness, wheezing, cough, expectoration of mucopurulent material, and fever?
  These pulmonary symptoms suggest aspiration pneumonia / bronchiectasis / lung abscess.

GENERAL EXAMINATION should be carried out in details.

EXAMINATION OF THE NECK

Specifically look for a goitre/thyroid cancer, laryngeal disease, or any other swelling in the neck.

EXAMINATION OF THE MOUTH AND PHARYNX

Specifically look for xerostomia, large growth of tongue/tonsil/pharynx, and retropharyngeal abscess.

IT IS WORTH ASKING THE PATIENT TO DRINK A MOUTHFUL OF WATER and:

1. Observe virtually.
   a. The presence of **dysphagia** will get **confirmed**. And, **probable level of obstruction** may be judged by asking the patient to point out the level of obstruction as s/he perceives while swallowing.
   b. **Difficulty in maneuvering the bolus,** i.e. nasal regurgitation, suffocation, coughing suggests a neuromuscular disorder rather than esophageal disorder.
2. Auscultate at the xiphoid cartilage for gurgle (as the patient drinks a mouthful of water).
   Normal duration of gurgle: 6 seconds.
   Duration of > 6 seconds ⇒ esophageal obstruction.

SYSTEMIC EXAMINATION: In the usual manner.

Free ebooks ==> www.Ebook777.com

PRINCIPLES AND PITFALLS

1. The process of arriving at the diagnosis essentially involves 3 steps: (a) Looking at the background ('clinical setting') on which the case is being evaluated, (b) Differentiating between esophageal and non-esophageal disorders, and (c) If esophagus seems to be the diseased organ, then fixing the pathological nature of the esophageal disease.
2. Occasionally, virtually chronic cause of dysphagia may present as an acute case and pose difficulty in diagnosis.
3. Bout of coughing, choking, bronchospasm on drinking/eating occur in the following situations.
   a. A neuromuscular disorder.
   b. Truly dysphagic (adult) patient who is very weak—due to long-standing dysphagia with consequent starvation.
   c. In neonates with congenital deformities preventing swallowing.
4. All patients with chronic dysphagia should be assumed to have malignant disease even if certain symptoms suggest a benign disorder. **Long duration does not necessarily rule out cancer**.
5. Hoarseness of voice from the beginning, coughing provoked by swallowing, and dysphagia more for liquids than for solids ⇒ laryngeal lesion.
6. **Nutritional depletion and avitaminosis**.
   Esophagus can be narrowed to a diameter of 3 or 4 mm and if the pathology is benign the patient may remain in good health. On the other hand a cancer that barely encroaches on the lumen may be associated with wasting. Almost from the onset of dysphagia, esophageal cancer proclaims its presence by a haunting visceral aversion to food, not explained by limitation of swallowing.
7. Weight-loss may be seen also in achalasia, but anorexia is less prominent. Weight gain following correction of the problem is the rule.

STEPWISE ANALYSIS OF DATA

Look at the **'clinical background'**.
(Refer to point number 2 under 'General Remarks' at the outset of the chapter.)

www.Ebook777.com

After having ruled out medical cause or an obvious regional pathology, evaluate the case further as under.

Specific cause of dysphagia in the new born

Is the baby 'Frothy (often) and in respiratory distress'?

- Saliva bubbling and dribbling from mouth, hence called 'frothy baby'.
- Prematurity / maternal polyhydramnios?
- Do the attempts at feeding result in coughing, choking and cyanosis?

↓

This clinical picture suggests esophageal atresia

Specific causes of dysphagia in infants

Congenital stenosis, ingestion of foreign body, dysphagia lusoria, (congenital) achalasia cardia, idiopathic dilatation of esophagus associated with megacolon, short esophagus, hiatus hernia.

In older children / adults

↓

Is the dysphagia **acute OR chronic?**

Causes of acute dysphagia

(a) *Acute inflammatory disorders* of the mouth / pharynx / larynx / neck / esophagus / mediastinum
- Acute tonsillitis, acute peritonsillar abscess (quincy), stomatitis
- Acute pharyngitis, diphtheria, acute retropharyngeal abscess
- Acute laryngitis
- Acute cellulitis in the neck e.g. lymphadenitis, parapharyngeal abscess, thyroiditis, etc.
- Acute esophagitis
- Acute mediastinitis

(b) *Following recent ingestion of a foreign body or corrosive*

(c) *Following instrumentation*, e.g. endoscopy / dilatation of a stricture. Odynophagia ± pain worsened by flexion of the neck, ± fever ⇒ ? Perforation of cervical esophagus.

(d) *In the absence of any antecedent situation*: Sudden mode of onset, abrupt and total dysphagia ± coughing, retching, anxiety, and drooling of saliva; episode of acute dysphagia probably having been triggered by inadequately chewed food suggests previously silent pre-existing organic obstruction

Causes of chronic dysphagia

(a) *Disorders of the mouth / pharynx / larynx*
- A large growth in the oral cavity e.g. of tongue, tonsil, etc. xerostomia.
- A tumor in the pharynx, chronic retropharyngeal abscess, Plummer-Vinson syndrome, globus hystericus, pharyngeal pouch, a foreign body in the pharynx
- Tuberculous laryngitis

(b) *Disorders of the esophagus*

Primary motility disorder
- Classic achalasia cardia
- Diffuse esophageal spasm
- Vigorous achalasia cardia
- Nutcracker esophagus
- Nonspecific motility disorders

Secondary motility disorder

Often associated with non-neurological (generalized) medical disorder
- DM
- Scleroderma
- Chronic idiopathic intestinal pseudo-obstruction
- Presbyesophagus
- Systemic amyloidosis
- Chaga's disease

Organic disorder
- (– Congenital atresia in the new-born)
- Cancer
- Corrosive stricture
- Peptic stricture
- Hiatal hernia
- Benign tumor
- Diverticulum
- Collagen disorder
- Shatzki's ring
- Dysphagia lusoria
- Dysphagia aortica
- Enlarged left atrium
- Mediastinal tumor
- Ingested foreign body

Miscellaneous
- Plummer-Vinson syndrome
- GERD
- Alcoholism

Free ebooks ==> www.Ebook777.com

Analysis of chronic dysphagia suggestive of **motility disorder**

- Age 20–40; may occur even in childhood
- Loss of weight
- Regurgitation

⇓

Classic achalasia cardia

- Pain in the chest indistinguishable from cardiac pain
- Loss of weight and regurgitation uncommon

⇓

Diffuse esophageal spasm (DES)

Manifestations of both - classic achalasia cardia + DES

||

Vigorous achalasia cardia

Analysis of chronic dysphagia suggestive of **organic disorder**

- Age (usually) > 40
- Shorter duration (in weeks or months)
- Persistent and progressive. Progress—rapid
- Anorexia
- 'Significant' loss of weight

⇓

CA—esophagus, or CA—cardiac end of stomach.

- Long duration* (in years)
- Very gradual progress
- Antecedent h/s/o GERD ±
- Loss of weight: uncommon

⇓

Peptic stricture

Following corrosive injury

⇓

Corrosive stricture
Progress may be variable

*Long duration does not necessarily exclude cancer.

Chronic dysphagia **not fitting in either** classical motility or organic disorder

Every c/o dysphagia over 40 years of age should be considered to be due to CA unless proved otherwise

Causes to be considered other than cancer

- GERD
- Alcoholic neuritis
- Foreign body ingested **long time** ago
- Plummer-Vinson syndrome
- Scleroderma
- Esophageal Crohn's disease

Hysteria

only provided the possibilities under the previous 2 headings have been ruled out, and all appropriate investigations are absolutely normal

Possible level of obstruction?

a. This may be judged from the h/o the perception of the level of obstruction by the patient herself / himself.

In the neck ⇓ Actual level of pathology can be anywhere along the length of the esophagus

Lower down the neck ⇓ Actual level of pathology will coincide with, or may be lower down than the level of perception of dysphagia
Pathology will NOT be in the cervical esophagus

b. Food sticking in the throat, and the patient starting to cough with first or second sip of water ⇒ obstruction in the lower pharynx or upper esophagus.
c. Persistent cough, SOB and (other) chest symptoms ⇒ mid-esophageal obstruction.
d. The presence of pain and regurgitation ⇒ obstruction in the mid/lower esophagus.
e. Hiccoughs ⇒ obstruction in the terminal esophagus.
f. Discomfort and burning in the region of xiphoid cartilage provoked by stooping, and relieved by antacids ⇒ lesion of the cardioesophageal junction.

Complication?

- Undernutrition.
- Aspiration pneumonia / bronchiectasis / lung abscess.
- Tracheo-esophageal or broncho-esophageal (malignant) fistula.

Associated disease?

Example: Plummer-Vinson syndrome.

www.Ebook777.com

DIFFERENTIAL DIAGNOSIS

CA esophagus

– Age > 40
– Dysphagia:
  - 'Shorter' duration, i.e. in weeks/few months. However, long duration does not necessarily exclude cancer
  - Persistent and progressive. Progress—rapid
  - Character of the triggering bolus: Initially, solid food.
– Anorexia
– (Significant) Loss of weight
– Regurgitation
– Repetitive aspiration pneumonia / bronchiectasis / lung abscess
– Bouts of coughing and choking may suggest tracheoesophageal / bronchoesophageal fistula
– Symptoms s/o distant mets ±.

Peptic stricture

– Age—usually above 60; otherwise, it may occur at any age including infancy
– Dysphagia:
  - Duration—in years
  - Usually progressive. Progress—very gradual
– Antecedent h/s/o GERD. However, 20% of patients with peptic stricture are unaware of antecedent reflux
– Weight loss—**uncommon.**

Malignant obstruction v/s peptic stricture

– Anorexia and significant weight loss are pathognomonic of cancer, when the duration of dysphagia is < 3 months in a patient over 40 years of age
– Development of peptic stricture may take years, and alteration in appetite and weight are less likely.

Corrosive stricture

Antecedent h/o ingestion of a corrosive a few weeks before the development of dysphagia.

Classification based on investigative evaluation

– Minimum diminution in the size of the lumen = Mild stricture
– Lumen size—50% of normal = Moderate stricture
– > 50% compromise of the lumen = Severe stricture
– Lumen completely obstructed, and the stricture can't be dilated = Severe undilatable stricture.

Massive incarcerated (otherwise uncomplicated) hiatus hernia

– Mild dysphagia
– Dyspepsia
– Breathlessness with meals
– Waning of symptoms of GERD, if they were present earlier
– Iron-deficiency anemia.

Dysphagia lusoria

– Presents after 1 year of age with feeding problems
– Stridor; worsened by flexion of the neck.

Benign tumor

Various possible modes of presentation:

1. Intermittent mild dysphagia, OR persistent dysphagia.
2. Vague retrosternal pressure.
3. 'Spluttering' on swallowing in case of a tumor lying just inferior to cricopharynx.
4. Progressively increasing airway obstruction (inspiratory stridor) in case of a tumor lying at the level of thoracic inlet.

Free ebooks ==> www.Ebook777.com

5. Repetitive episodes of aspiration pneumonia.
6. Regurgitation of a fleshy tumor from the mouth; appearing and disappearing. In this case there is a serious risk of acute laryngeal obstruction.
7. Gastrointestinal bleeding.
   – High index of suspicion is essential to think about a possible diagnosis of benign tumor
   – > 85% tumors are asymptomatic, and detected incidentally, if at all, during radiological or endoscopic examination of the esophagus.

Pharyngoesophageal diverticulum

– Late middle age / elderly
– Dysphagia:
  - Perceived in the cervical region
  - Difficulty in transfer of food from pharynx to esophagus
  - Progressive increase in severity
– During meals:
  - Bouts of coughing and spluttering
  - Gurgling noises during swallowing
– Long period to eat even a small meal
– Regurgitation of non-acid material
– (Constant) Throat irritation
– Hoarseness of voice
– Halitosis
– Pulmonary symptoms due to recurrent 'aspiration'.

Classic achalasia cardia

– Age: 20–40. However, can occur in childhood
– Dysphagia:
  - Duration ≥ 2 years prior to presentation
  - Character of the triggering bolus—In the early period, liquids only
  - Dysphagia typical of motility disorder, i.e. (a) Difficulty in swallowing can be overcome by repeated acts of swallowing / sipping of fluid / breath-holding / expiration against closed glottis. And (b) Aggravated by emotional stress / liquids at extremes of temperature
  - Intermittent with variable severity, **or can be progressive**.

± – Weight-loss
– There may be inability to belch
– Odynophagia
– Spontaneous episodes of pain in the chest—may be simulating angina pectoris

With development of moderate dilatation of the esophagus:

– Dysphagia **may** get less severe
– Development of spontaneous / postural regurgitation of foamy mucoid saliva, and later, of undigested food
– Heartburn (caused by acid fermentation products of retained food)
– Persistent eructation of foul smell
– Halitosis.

Symptoms suggestive of advanced disease:

– Loss of weight
– Pulmonary symptoms due to repetitive aspiration pneumonia / bronchiectasis / lung abscess: Breathlessness, wheezing, cough, expectoration of mucopurulent material, fever.

DES, i.e. diffuse esophageal spasm

– Emotional personality
– Dysphagia typical of motility disorder (Refer to above under 'classic achalasia)
– Chest pain
  - With eating, or spontaneously
  - May be indistinguishable from cardiac chest pain. And may be relieved by nitrates!

www.Ebook777.com

– Heartburn
– Loss of weight is uncommon.

Vigorous achalasia cardia

= Features of both classic achalasia + DES.
Chest pain is a more prominent feature.

DES v/s achalasia

Symptoms in favor of DES:

1. Triggering bolus may be any—liquids and solids.
2. Chest pain (often indistinguishable from cardiac origin). Chest pain is uncommon in achalasia.
3. Usually no loss of weight.

Nutcracker esophagus

It is a manometrically defined disorder of high-amplitude peristaltic contractions in patients with (non-cardiac) chest pain and/or dysphagia (essentially typical of motility disorder).
Natural history: May progress to achalasia/DES.

Nonspecific motility disorders

These are a group of disorders in which, patient presents with a variety of esophageal symptoms not meeting the specific criteria of specific disorders like achalasia, DES, nutcracker esophagus.

Plummer-Vinson syndrome

– Postmenopausal women
– Iron-deficiency anemia
– Glossitis, angular stomatitis
– Dry skin and eyes
– Koilonychia
– Spleenomegaly.

Dysphagia is thought to result more from esophageal spasm associated with inflamed atrophic mucosa than partial obstruction due to esophageal (flimsy) webs.

GERD (**G**astro**E**sophageal **R**eflux **D**isease)

There are different mechanisms causing dysphagia at different stages of the disease as it progresses.

1. In the stage of esophagitis: Spasm / edema. Remits with improvement of esophagitis consequent on treatment.
2. Secondary motility disorder.
3. Dysphagia resistant to treatment ⇒ formation of stricture.

DeMeester's scores for gradation of GERD *wrt* dysphagia:

– Occasional episodes of dysphagia ⇒ Minimal = Grade 1 dysphagia.
– Difficulty in swallowing requiring fluids to clear the obstructive feeling ⇒ Moderate = Grade 2 dysphagia.
– Episode of meat impaction requiring medical treatment ⇒ Severe = Grade 3 dysphagia.

Tumor of the skull-base

– Any skull-base tumor can extend anteriorly leading to displacement and obstruction of the pharynx, in turn leading to dysphagia
– The classical example = cranial chordoma—an uncommon tumor which arises predominantly from the clivus, just behind the pharynx
– *Additional symptoms* other than dysphagia: Dysarthria, vertigo, lower cranial nerve palsies, hyper-reflexia or ataxia.

Osteophytes

Points to be considered before holding osteophytes responsible for dysphagia: **1**. Cervical spondylosis is very common; 95% of people over the age of 60 years have some radiological evidence of it. **2**. Unless the anterior osteophytes are gross they are unlikely to be responsible for dysphagia. **3**. It is necessary to exclude all other potential causes of dysphagia.

Free ebooks ==> www.Ebook777.com

BELCHING

HISTORY

- Do you get excessive belching?
- Do you find it difficult or unable to belch?

Basically, excessive belching may be viewed as an indicator of increased voluntary or involuntary dry swallows taken to clear the esophagus, which in turn may suggest an obstruction of some kind.
Some patients with achalasia find it difficult to belch. *Mechanism*: Esophagus is overstretched already, and incapable of moving a pocket of gas through either sphincter.

ODYNOPHAGIA

HISTORY

- Duration?
- Did you swallow a bolus of food that was large / inadequately chewed, sharp / pointed / hard in consistency?
- 'Sore' throat?
- H/s/o GERD?
- Did you consume tablets of vitamin C (Ascorbic acid) / tetracycline / quinidine / KCl?
- Are you on any CT / RT?
- Did you swallow any foreign body? If so, size and shape of the object? Was the object sharp / pointed? Was the swallowed foreign body extracted? In that case, did odynophagia start after that?
- Did you ingest any corrosive?

Common causes of odynophagia

1. 'Sore' throat. Causes: (usually acute) inflammatory conditions in the mouth/pharynx/larynx. Examples: Glossitis, tonsillitis, pharyngitis, laryngitis, quincy (acute peritonsillar abscess), retropharyngeal abscess, etc.
2. Severe ulcerative esophagitis secondary to GERD.
3. Pill-induced esophagitis: Vitamin C (Ascorbic acid), tetracycline, quinidine, KCl, etc.
4. Radiation esophagitis.
5. **Odynophagia beginning even many hours after ingestion of a sharp foreign body or an endoscopy or endoscopic extraction of a foreign body must not be neglected** as it may be due to esophageal perforation.
6. Corrosive injury to the esophagus.
7. Infectious esophagitis.
8. CA esophagus.

HEARTBURN

Heartburn may be referred to various sites thereby necessitating it to be differentiated from pain of IHD.

HISTORY

- Do you get heartburn / Does heartburn exacerbate
  - On stooping (bending) / lying down?
  - Following large, fatty or spicy food?
  - On consuming chocolate / alcohol / fruit juices / coffee?
- Do you feel better
  - After sitting up or by taking antacid?
  - On avoiding the initiating / exacerbating drink or food item?

www.Ebook777.com

- Does it interfere with your daily activities? (⇒ severe, i.e. Grade 3 heartburn as per DeMeester's scoring system)

Note

– The pathognomonic hallmark of heartburn caused by gastroesophageal reflux is the initiation or exacerbation of pain on stooping (bending) or lying down, i.e. **postural heartburn**
– Heartburn is an expression of mucosal burn from refluxed acid-pepsin.

VANISHING HEARTBURN

HISTORY

- Have you been taking / have you taken appropriate treatment for heartburn as advised?
- Do you get fluctuations in heartburn?
- Does heartburn seem to have disappeared spontaneously?

Possible causes (after true remission as a response to appropriate treatment having been ruled out)

1. Occasionally in Barret's esophagus, development of adenocarcinoma that is too small to cause dysphagia, but big enough to block reflux.
2. Development of a peptic stricture.

In practice, the clinical clue of vanishing heartburn is a retrospective one.

REGURGITATION

A telltale history: Awakening from sound sleep with a paroxysm of coughing and choking that is relieved by sitting up and swallowing a glass of water taking an antacid mixture.

HISTORY

- What time does it occur?
  – At night?
    OR
  – During the day? Is it aggravated by stooping?
- What's the interval between eating and regurgitation?
  – Instantaneous / just few hours—usually suggests esophageal disease
    OR
  – Several hours / a day ⇒ gastric disease
- Physical characteristics, i.e. nature of the regurgitate?
  – Clear and frothy ⇒ esophageal disease
  – Blood-streaked nature basically ⇒ erosion / ulceration of mucosa, i.e. ulcerative esophagitis
  – Blood-streaked mucus and food may suggest CA esophagus
  – Green ⇒ gastric disease
  – Tasting acidic and bitter ⇒ gastric disease.
- Does it occur occasionally or very often?
  Occasional episode ⇒ minimum, i.e. Grade 1 regurgitation as per DeMeester's scoring system.
- Is it predictable on position or straining?
  ⇒ moderate, i.e. Grade 2 regurgitation as per DeMeester's scoring system.

Notes

– Regurgitation complicated by aspiration and respiratory symptoms ⇒ severe, i.e. Grade 3 regurgitation as per DeMeester's scoring system
– Regurgitation is caused by a patulous esophagogastric junction, with or without a hiatus hernia.

Free ebooks ==> www.Ebook777.com

WATERBRASH

It is caused by excessive salivation triggered by GE reflux.

PAIN (ESOPHAGEAL ANGINA)

Pain or distress in the chest caused by GE reflux is often clinically indistinguishable from cardiac angina.
Descriptor: 'squeezing' / 'heavy pressure' / 'tightness'. Can be severe. May follow exertion / emotional event. May be associated with perspiration!
Cause: Gastroesophageal reflux (in ~ 10–20% of cases).

HISTORY

- Duration?
- Intermittent or constant?
  If intermittent,
  - When do you get it?
  - What is the relation with swallowing?
  - Do you wake up from a sound sleep due to pain?
- Where exactly does it pain? Does it remain confined to the midline?
- Does it radiate between the shoulder blades/either lumbar regions?
- Intensity of pain?
- Nature (Character) of pain?
- Does the pain initiate or get aggravated by
  - Consuming particular type of food such as hot (chilly) / spicy / oily / fatty?
  - Stooping forwards?
  - Lying down flat?
- Have you noticed any particular relieving factor?—such as belching / assuming sitting position / taking antacid or even NTG / after regurgitation?

Features suggesting esophagus as the source of pain rather than the heart or other viscera:
- **Duration**—in days
- Pain occurs immediately after swallowing
- Pain awakens the patient from sound sleep
- Site—**substernal, maximum at the site of obstruction, limited to the midline** and **radiating to the interscapular** area / either **lumbar regions. However, can spread across the chest and radiate to neck, epigastrium!**
- **Relieving factors**: Belching / sitting-up / after regurgitation / antacid / swallow of a teaspoonful of viscus lignocaine / sublingual NGT, within 8–10 minutes - i.e. slower as compared to cardiac angina (within 2–3 minutes).
- The presence of **other associated symptoms** suggestive of esophageal disorder: The most important—**dysphagia**. **Other symptoms of reflux, when particularly posturally aggravated.**

A NOTE ON GERD (GastroEsophageal Reflux Disease)

GERD may be **defined as** a syndrome of excessive (i.e. in excess of physiological) gastroesophageal reflux. Dietary factors are important in the etiology of this disease.
In many patients the symptoms are mild and intermittent with no evidence of gross mucosal damage (= non-erosive reflux disease). In some patients however, (erosive) esophagitis develops with its consequences viz ulceration, stricture, columnar metaplasia and dysplasia in the metaplastic segment.

www.Ebook777.com

Clinical presentations

Typical	Atypical
– Heartburn	– (Esophageal) chest pain
– Regurgitation	– Pulmonary symptoms including asthma
– Waterbrash	– Laryngeal symptoms: Persistent dry cough, changes in voice, choking episodes
– Dysphagia	– Palatal and dental erosions
– Pain on swallowing hot / spicy food	– Chronic hypochromic microcytic anemia due to occult blood loss
	– Rarely, gross hematemesis

The symptoms may be aggravated after large meals, by spicy meals, by meals/snacks containing onions, peppermint, chocolate, coffee, caffeine, alcohol.
The symptoms may also be aggravated by activities which increase the intra-abdominal pressure, or by posture which aids gravity-induced reflux, e.g. bending, lying down flat (hence, the symptoms can be especially severe at night).

Clinical situations *(Guideline statements of diagnosis)*

1. **Non-erosive GERD without disabling symptoms.**
   – Symptom score (DeMeester's): H - , R - , D - .
   – Endoscopy: No erosion.
2. **GERD without disabling symptoms.**
   – Symptom score (DeMeester's): H - , R - , D - .
   – Endoscopy not done as yet / Endoscopic grade …… (as per Savary-Miller / LA classification).
3. **GERD with disabling symptoms, 'adequately'** (subjective) **controlled on medical treatment.**
   – Symptom score (DeMeester's): H - , R - , D - .
   – Endoscopic grade …… (as per Savary-Miller / LA classification).
4. **GERD with persistent disabling symptoms, 'inadequately'** (subjective) **controlled on medical treatment.**
   – Symptom score (DeMeester's): H - , R - , D - .
   – Endoscopic grade …… (as per Savary-Miller / LA classification).
5. **GERD complicated** by - Ulcerative peptic esophagitis / (Peptic) Stricture / Chronic ulcer (Barrett) in columnar-lined esophagus / Chronic aspiration / Hiatal hernia.
6. **Post-gastric surgery GER.**

A NOTE ON NON-REFLUX ESOPHAGITIS

Type	*Causative agent*	*Comment*
– Corrosive	Corrosives	Refer to chapter 61
– Infective	Candidosis Herpes	Predisposing factors: Chronic illness, immunocompromized status, antibiotic therapy, etc.
– Drug-induced	Antibiotics, NSAIDs, etc.	Symptoms: Heartburn / chest pain / odynophagia 4–6 hours after ingestion of medication
		Patients with a pre-existing esophageal disorder are at a higher risk
– Radiation	Radiation to mediastinum ± CT	Resulting ulcer and stricture may simulate recurrence of tumor
– AIDS	Fungi / viruses / bacteria	Deep ulcers may develop which may indeed progress to bronchoesophageal fistulae
– Other: Aphthous esophagitis, Crohn's disease, NG-tube-induced esophagitis, bullous dermatoses, etc.		

Free ebooks ==> www.Ebook777.com

ASPIRATION AND RESPIRATORY SYMPTOMS

– Mere hoarseness of voice may suggest aspiration of minor quantity of neutral (in terms of pH) esophageal content, and fleeting contact of contents with larynx.
– Cough, stridor, recurrent attacks of pneumonia, progressive pulmonary fibrosis and respiratory insufficiency ⇒ Repetitive nocturnal inhalation of obstructed esophageal contents, gastric contents or contents of a diverticulum.

GLOBUS

= Increased tone of pharyngeal muscles with the sensation of a lump deep inside the throat, and constriction. When the sensation is present a short period after eating or when fasting, it is called 'globus hystericus'—a neurotic symptom in patients with emotional instability.
The symptom may require a thorough (investigative) evaluation to rule out organic disorder. However, a common sense may be exercised in deciding which patient really needs a thorough evaluation.

www.Ebook777.com

40 VOMITING OF BLOOD—HEMATEMESIS

Hematemesis is **defined** as vomiting of blood, either fresh (bright red) or altered ('coffee grounds').
It **indicates gross 'major' bleeding** from the upper GI tract (proximal to ligament of Treitz) which should be taken as **a serious and urgent matter**, and the patient **must be hospitalized** even if vomiting is stopped by the time patient seeks consultation.

GENERAL REMARKS

1. **Most common causes** (> 90%): chronic peptic ulcer, drug-induced (NSAID) acute erosive gastritis, esophago-gastric varices.
2. By the time patients reach hospital 1/3rd will have stopped bleeding, 1/3rd will be oozing and 1/3rd still bleeding massively.
3. Practical clinical situations

Already an 'inpatient' being under treatment for some other disorder such as

- # base of the skull (middle / anterior fossa)
- Patients admitted in ICU ('stress')
- Medical conditions: Uremia, prolonged jaundice, coagulopathy, patient on anticoagulant treatment, malignant HT, SLE, etc.

Brought in emergency department as 'a case of vomiting of blood'

- Reassure the patient and relative
- Quick (partial) general examination
- Record and monitor vital parameters

- No episode of vomiting 'for a few hours' before the time of consultation
  and
- Patient hemodynamically stable

⇓

Bleeding apparently stopped, or 'slow' oozing might be continuing

↓

Patient must be hospitalized even then
Reasons: (a) Patient could be in compensated shock. (b) Rebleeding may occur any time.

Patient in hypovolemic shock

↓

- Commence the management of shock
- Hospitalize

HISTORY

Aims

1. R/o the possibility of hemoptysis / epistaxis / oropharyngeal source of bleeding (and consequently vomiting of swallowed blood) at the outset.
2. Focus on the most common causes first viz chronic peptic ulcer, NSAID-induced acute erosive gastritis and esophago-gastric varices.

Free ebooks ==> www.Ebook777.com

- *To r/o hemoptysis*:
  - Are you sure—you vomited blood, or else, did you cough out frothy bright red blood preceded by repeated bouts of coughing?
  - Is the blood mixed with sputum or food debris?
- *To r/o epistaxis*:
  Did you bleed through your nose, some of which you probably might have swallowed before vomiting out blood?

Further history to be taken for hematemesis proper, hemoptysis / epistaxis / oropharyngeal source of bleeding having been ruled out.

- Duration?
- Frequency?
- When did you vomit last?
- Is the blood bright red
  OR
  Does the vomitus look like 'coffee grounds'?
- Did you pass black tarry stools? If so, since when and how many times?
- Is this the 1st episode or a recurrent episode?
  If recurrent,
  - How many times did you suffer earlier?
  - When was the last episode?
  - What was the diagnosis made earlier?
  - Details of management then, especially *wrt* endoscopy.

H/s/o **chronic peptic ulcer**

- Do you get periodic pains in upper abdomen since a few months/years?
- Have you noticed that the periods of exacerbations are particularly associated with periods of stress / smoking / dietary or alcoholic indiscretions?
- What time do you get the pain, i.e. on empty stomach / early morning / on taking food?
- What are the relieving factors—food, or vomiting?
- How is your appetite? Is it so that you have a good appetite, but you are afraid of eating due to fear of pain?
- How about your weight—have you put on weight or have you lost weight?
- Do you usually consume excessively chilly and spicy diet?
- Do you have your food at irregular timings?
- In case of the absence of any of the classical clinical features (as above): Do you have at least a vague discomfort in the upper abdomen?
- Did you recently consume aspirin / ibuprofen / indomethacin or any other NSAID?

Basic h/s/o **esophagogastric varices**

- Was the (1st) major episode of bleeding preceded a few hours earlier by a mouthful of bright red bleeding?
- Distension of abdomen (due to ascites)?
  If there is no distension of abdomen at present, then • Did you have distension of abdomen? Are you on a diuretic?
- Lump in the abdomen (due to splenomegaly)?

a. H/s/o **cirrhosis**

- Past h/o jaundice (particularly of longer duration)?
- H/o fatigue, malaise, myalgia, arthralgia, headache?
- Fever, forgetfulness, confusion, poor concentration, personality changes?
- Ask the accompanying relative: of late, has his/her intellectual level and / or LOC been impaired?
- H/o heavy consumption of alcohol (> 50 units / wk) over many years regularly?

b. Recent past or present ongoing h/s/o **pancreatitis / chronic pancreaticobiliary disease / pancreatic tumor**?

www.Ebook777.com

c. H/o consumption of **OC pills?**

- Are you on anticoagulant/fibrinolytic / steroid treatment?
- Did the episode of bleeding follow persistent vomiting/retching?
- H/o ingestion of a foreign body?
- Have you been having heartburn, regurgitation?
- H/s/o CA-esophagus? (– Refer to chapter 39.)
- Have you been having a pulsatile lump in the abdomen?
- How long has NG/gastrostomy tube been in totally? (if relevant).

INTERPRETATION OF SYMPTOMS, AND DISCUSSION

– **# of base of the skull** (middle / anterior fossa): Bleeding occurs from the #-site; the blood trickled in the throat may be swallowed and later vomited out

– **Age and gender**: Risk of drug-induced acute erosive gastritis is highest in elderly females and particularly with aspirin

– **Duration and frequency** logically give an idea about the **severity of bleeding**

– Practical conclusion from color of vomited blood: **Bright red blood may be considered to be due to massive loss of blood** and consequently more serious**, whereas** '**coffee grounds**' **may be considered to be due to 'bleeding of comparatively lesser severity'.**

Source of bleeding—whether esophageal or gastroduodenal, cannot be determined only on the basis of whether the vomited blood is bright red or in the form of 'coffee grounds'. However, for the sake of discussion, following arguments can be made.

- 'Bright red blood' logically tends to imply severe bleeding. Source of bleeding is more likely to be esophagus. However, it may be gastroduodenal if loss of blood is massive
- 'Coffee grounds': Mechanism: Blood gets altered by reacting with gastric acid, which in turn suggests that blood could accumulate within the stomach for some time so that reaction with acid was possible. 'Coffee grounds' suggest 'lesser' bleeding. Source of bleeding is more likely to be stomach/duodenum, though it can be esophageal

– **Melena** associated with hematemesis suggests **massive loss of blood**

– Basically, chronic periodic epigastric pain; with periods of exacerbations particularly being associated with periods of stress / smoking / dietary or alcoholic indiscretions suggests chronic peptic ulcer

Excessively chilly and spicy diet and irregular feeding habit indicate possibility of chronic peptic ulcer disease.

All patients may not have classical clinical features of peptic ulcer. Some patients may be totally asymptomatic with active ulcer, and may present straight with complication as the first evidence of peptic ulcer disease. Consequently, **chronic peptic ulcer as a cause of hematemesis cannot be ruled out on the basis of the absence of symptoms** of peptic ulcer disease.

– Patients with **esophagogastric variceal bleeding** leading to hematemesis may have preceding warning bleed of a mouthful of bright red blood a few hours before major bleeding.

a. Most common cause of portal HT is cirrhosis. However, it must be remembered that **sometimes it can develop in patients with liver disease prior to the onset of cirrhosis**.
   - Fatigue, malaise, myalgia, arthralgia, headache: These symptoms are s/o a chronic liver disease
   - Fever, forgetfulness, confusion, poor concentration, personality changes ⇒ advanced cirrhosis
   - Impairment of intellectual level and LOC ⇒ hepatic encephalopathy
   - A daily intake in excess of 60–80 gm for men and 20 gm for women over a period of 10–15 years incurs a high-risk of developing cirrhosis of liver.

   However, not all alcoholics become cirrhotic.

b. Pancreatic / pancreatic obiliary diseases may lead to splenic vein thrombosis, which in turn can → portal HT.

c. OC pills may lead to thrombosis of hepatic veins, which in turn can → portal HT.

Free ebooks ==> www.Ebook777.com

- **Hematemesis following persistent vomiting / retching** is indicative of Mallory-Weiss syndrome. Usually, though not always necessarily, it is induced by excessive intake of alcohol.
- **Ingestion of a foreign body** can lead to bleeding from either a mucosal injury or aortic penetrating injury.
- Barrett's ulcer may complicate GERD. Penetration of a Barrett's ulcer into an esophageal branch of aorta can lead to hematemesis.
- Aortic infiltration by an esophageal cancer can lead to hematemesis
- A pulsatile lump in the abdomen suggests aortic aneurysm: Fistulous communication with aortic aneurysm can lead to hematemesis
- **Long-term placement of NG / gastrostomy tube** (for feeding purpose) may lead to an ulcer, which may bleed.

GENERAL EXAMINATION: In usual manner.

In SYSTEMIC EXAMINATION, specifically look for the following points:

- Abdominal distension due to ascites
- Physical signs s/o chronic liver disease
- Physical signs s/o portal HT
- Scar of previous laparotomy for a peptic ulcer disease
- Aortic aneurysm.

At this juncture, provisional clinical diagnosis can usually be made.

|

Further course

↓

- Continue monitoring
- Organize appropriate investigations (including UGI endoscopy-diagnostic ± therapeutic).

Evaluate the investigation reports. At this juncture, definite diagnosis will usually be made.

Less common causes of hematemesis

A. Swallowed blood: from nasopharyngeal / oropharyngeal source.

B. Esophageal

1. Reflux esophagitis.
2. Mallory-Weiss syndrome—usually, though not always necessarily, induced by excessive intake of alcohol.
3. Ingestion of a foreign body can lead to bleeding from either a mucosal injury or aortic penetrating injury.
4. Penetration of a Barrett's ulcer into an esophageal branch of aorta.
5. Angiodysplasia.
6. Aortic infiltration by an esophageal cancer.
7. Fistulous communication with aortic aneurysm.

C. Gastric

1. 'Stress' ulcers.
2. Long-term insertion of a NG tube (or less commonly gastrostomy tube).
3. Discrete ulcer at the hiatal rim / diffuse erosions within a massive incarcerated hiatus hernia.
4. Angiodysplasia.
5. Rupture of a large tortuous submucosal artery normally found in the body of the stomach within 6 cm of the esophagogastric junction (Dieulafoy syndrome).

D. Duodenal

1. Diverticulum.
2. Cancer.
3. Cholecystoduodenal fistula.

www.Ebook777.com

E. Isolated splenic vein thrombosis (without portal vein thrombosis)
→ splenomegaly and sectorial (*synonym* left-sided portal venous hypertension).
The most common presentation is bleeding gastric varices with normal or good liver function. *As esophageal varices may be absent, central portal pressures normal and gastric vessels easily overlooked at endoscopy, this condition can be easily missed.*
Causes of isolated splenic vein thrombosis: Acute or chronic pancreatitis; direct extension of a pancreatic tumor; in infants who have had umbilical vein catheterization.

F. Hemobilia.

G. Coagulopathies / side effect of anticoagulant/fibrinolytic treatment.

H. Acute febrile disease, e.g. malignant variola, scarlet fever.

I. Miscellaneous disorders: Malignant HT, vasculitis, prolonged jaundice, uraemia, connective tissue disorders.

Free ebooks ==> www.Ebook777.com

41 ANORECTAL PAIN, BLEEDING PER RECTUM AND OTHER MANIFESTATIONS OF ANORECTAL DISORDER

Anorectal symptoms

- Pain.
- Bleeding.
- Discharge.
- Itching.
- Difficulties with defecation.
- Constipation.
- Diarrhea/Dysentery.
- Diarrhea alternating with constipation.
- Alteration of bowel habit.
- SCOPA (**S**omething **C**oming **O**ut **P**er **A**nus/Rectum)
- External swelling in the anal/perianal region.
- Intestinal obstruction (Rare).
- Fever.
- Fecal incontinence.

GENERAL REMARKS

1. Symptoms due to anorectal disorders overlap with those due to intestinal (often large and occasionally small) disorders, and so obtaining a full gastrointestinal history is essential.
2. *Classification of GI bleeding*
   a. From a clinical standpoint in accordance with presentation
   ↓
   - Chronic occult blood loss → iron-deficiency anemia. Patient is unaware of bleeding as such. Fecal occult blood test is +ve.
   - Overt minor episodes of bleeding: This relates most commonly to colorectal disease, e.g. fissure, cancer, piles, polyp, IBD etc.
   - Acute massive bleeding → hematemesis/melena/fresh bleeding PR.
   - Recurrent obscure GI bleeding – acute or chronic. Although fortunately rare, the management is problematic, challenging.

   b. Conventional classification

Upper GI bleeding ↓	Lower GI bleeding ↓
– Chronic occult blood loss from gastric cancer, peptic ulcer, reflux esophagitis etc.	– Chronic occult blood loss commonly from cancer of (right) colon
– Hematemesis	– Trivial bleeding PR
– Recurrent obscure hematemesis (Rare)	– Overt minor episodes of bleeding PR
	– Acute massive bleeding PR
	– Recurrent obscure bleeding PR

www.Ebook777.com

The classification into upper/lower GI bleeding, although in established usage, is not entirely satisfactory, as it omits a group of disorders, albeit less common, causing bleeding from the middle GI tract (DJ junction ↔ proximal transverse colon).

In the upper/lower GI classification, right colonic bleeding lesions are grouped with lower GI bleeding.

3. *Causes of bleeding PR by anatomical site*

Anal	Rectal	Colonic	Upper GI
– Piles – Fissure – Perianal abscess / fistula – Cancer – Injury	– Polyp – Cancer – Proctitis – Prolapse – Injury	– Polyp – Cancer – Colitis (idiopathic / infective / infestative / ischemic) – Diverticular disease – Angiodysplasia	– Peptic ulcer – Meckel's diverticulum (ectopic ulceration) – Small intestinal vascular lesion – Small intestinal infection / infestation

4. *Mechanism of bleeding in some common disorders, with respect to pathology*:

- Piles bleed because of trauma to the engorged vascular cushions.
- Colitis and proctitis bleed from mucosal ulcerations.
- Tumors bleed from dilated, fragile neoplastic vessels.
- Diverticula bleed from erosion of vessel at their mouth.

HISTORY

- **Pain**?
  - Exact site? Can it be pinpointed with a finger-tip OR does it appear to be deep-seated and ill-defined?
  - Duration?

    In c/o long duration – Is the pain constantly present or is it intermittent?
  - Mode of onset?
  - Progress and Regress of this episode and previous episodes if any?
  - Is the pain continuously present throughout the day, OR Does the pain start during/soon after defecation?

    When related to defecation, how long does it last?
  - Intensity?
  - Nature?
  - Urological symptoms?
- **'Bleeding per rectum'**?
  - Duration?

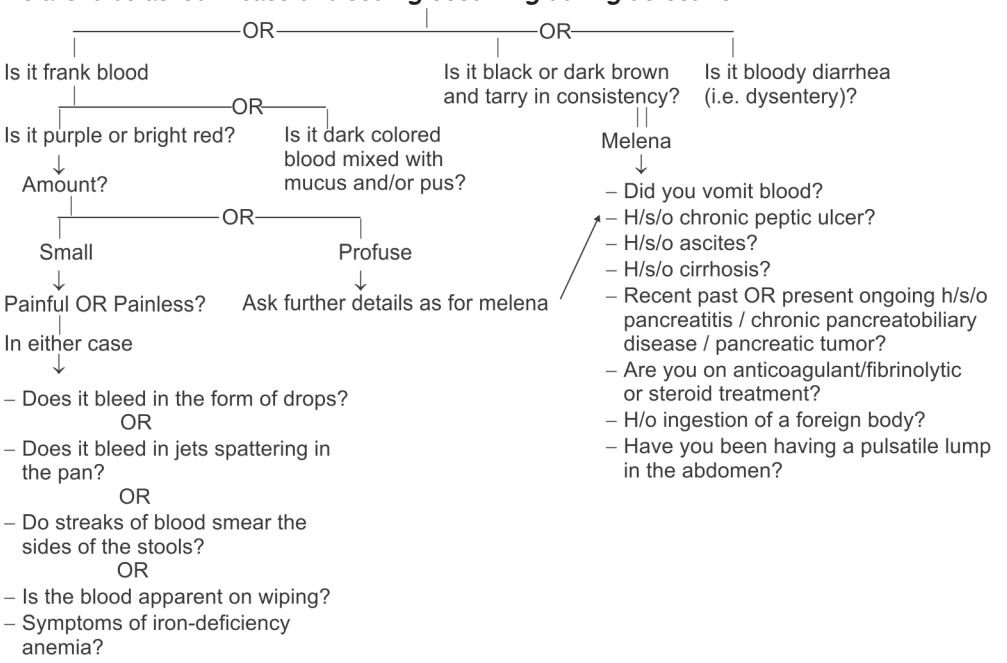
    In c/o long duration – Is the problem constantly present or is it intermittent?
  - Progress and Regress of this episode and previous episodes if any?
  - Does it occur during defecation?

    OR

    Does it occur apart from defecation (i.e. occurring even without trying to pass stools, with staining of underclothes)?
  - Amount: Massive, trivial or moderate?
  - Did you get any injury in or through the perineum?

Free ebooks ==> www.Ebook777.com

*Details to be asked in case of **bleeding occurring during defecation***



*Details to be asked for **ulcerative colitis***

– How much is the quantity of blood passed – Small, moderate or profuse? Profuse bleeding occurs in severe colitis.
– Frequency of diarrhea? – Less than 4 ⇒ mild colitis.
4 to 6 ⇒ moderate colitis.
More than 6 constituting severe diarrhea ⇒ severe colitis.
– Constitutional symptoms?
– Are you suffering from OR Did you suffer earlier from arthritis?
– Did you suffer from development of acute painful nodules under the skin, particularly in the shins?
– H/s/o uveitis?
– How about appetite? Appetite is lost in moderate and severe colitis.
– How about weight? Weight is lost in moderate and severe colitis.
– H/s/o iron-deficiency anemia? Iron-deficiency anemia occurs in moderate and severe colitis.
– How has been the response to treatment (if applicable)? Mild and moderate disease responds (well) to treatment.

Bleeding/blood-stained discharge occurring apart from defecation ⇒ the site of lesion usually beyond (i.e. distal to) the anal sphincter (anal verge), or surrounding area.

- **Discharge**?
  = Discharge apart from defecation i.e. occurring even without trying to pass stools leading to staining of underclothes.
  – Does discharge come out of the anal canal?
  OR
  Does it come from a site away from the anal canal?
  – Duration?
  In c/o long duration – Is the problem constantly present or is it intermittent?
  – Mode of onset?
  – Progress and Regress of this episode and previous episodes if any?

www.Ebook777.com

  - Nature - Blood-stained
    OR - Essentially purulent—thick / thin?
    OR - Mucoid?
  - In case when essentially blood-stained—have you been suffering from prolapsed piles / prolapsed rectum / chronic anal fissure?
  - In case when essentially purulent and also recurrent—does it occur from the same spot each time, or from different spots?
  - Foul smelling?
- **Itching**?
  - Do you have children or other members in the family with similar history? Think of enterobial (thread worm) infestation.
  - Are you suffering from any anorectal disorder?
  - Do you have incontinence—may be even a minor leakage of stools – liquid/solid?
- **Difficulties with defecation**
  - **Frequency** of defecation?
  - **Consistency**—hard/Semi liquid/Liquid (watery)/Tarry/Greasy? Does the greasy stool float on the water?
  - **Color**—dark brown/Black/Pale/Clay-colored?
  - Do you pass **mucus, pus**? **Color** of the mucus?
  - Do you experience painful and ineffective attempts of defecation requiring straining (i.e. **tenesmus**)?
  - **Do you have to strain** to pass stools?
  - Do you get a sensation of fullness in the rectum and a feeling that the bowel has not been completely emptied after defecation (i.e. **dyschezia**)? The rectum indeed is loaded with feces after defecation.
  - Do you have to go to the toilet several times a day but only actually defecating once or twice?
- **Constipation**?
  - Duration?

If chronic/habitual ↓
- Diarrhea alternating with constipation?
- Pencil-shaped or pipe-stem-shaped stools?
- Pellet-like stools?
- Pain while passing stools?
- Previous h/s/o colitis, diverticulitis?
- Past h/o surgery(ies) upon the anorectum?
- H/o DM?
- H/o Parkinson's disease / spinal cord injury / hypothyroidism / any CTD?
- Have you been consuming any medicine/s?
- H/o ingestion/insertion of a foreign body?

Unusual, although this point may have to be asked occasionally particularly in c/o a child, psychiatric patient, or a prisoner

If acute ↓
- Is it associated with acute severe pain in abdomen?
- Have you been passing flatus at least during the last 24 hours? If not, then ↓
  Do you feel like passing flatus but are not able to do so and that passage of flatus will relieve the pain in abdomen?
- Distension of abdomen?

Otherwise ↓
might be a transient non-specific episode of constipation.

- **Diarrhea/dysentery**?
  Is it associated with griping pain in abdomen?
- **Diarrhea alternating with constipation**?
- **Alteration of bowel habit**
  Criteria of alteration of bowel habit: The development of a specific alteration in addition to irregularity, constipation and requirement of increasing doses of purgative *in a person who has been having a regular bowel habit*.
  - Have you developed irregular bowel habit of late?
  - How often do you pass stools in a week?
  - Have you been experiencing increasing difficulty with the problem of constipation?

Free ebooks ==> www.Ebook777.com

- Do you have to take a purgative to overcome the problem? Has the requirement of dose of purgative been going up?
- Sensation of fullness in the rectum?
- Do you feel that the bowel has not been completely emptied after defecation?
- Diarrhea alternating with constipation; diarrhea occurring particularly in the morning?
- Pencil-shaped or pipe-stem-shaped stools?

Ask duration and progress for each of the above symptoms.

- **SCOPA**?

  = a palpably enlarged abnormal mass which is reducible; however, it may become irreducible if it has been present for a long time.
  - Duration?
  - Mode of onset?
  - Progress and Regress of this episode and previous episodes if any?
  - Does it reduce spontaneously after completion of defecation?

    OR

    Does it need to be manually reduced?
  - Is it painful?
- H/o any **external swelling**?

  = a swelling which has been present constantly ever since it appeared.
  - Duration?
  - Mode of onset?
  - Progress / Regress?
- H/s/o **intestinal obstruction**—chronic / acute-on-chronic?
- **Fever**?
- Fecal **incontinence**?
  - Do you get incontinence of solid matter, liquid or gas?
  - Do you have to wear pads? – The number of pads required gives an idea about the quantity-severity of incontinence.
  - Is the incontinence experienced rarely, sometimes, usually, or always?
  - Has the resultant disability compelled you to alter your occupation/life-style?
  - Did the problem follow childbirth?
  - Did the problem follow an anorectal surgery?
  - Is it associated with urinary incontinence? ⇒ neurological cause.

  Detailed history for obstetric cause of fecal incontinence
  - How many vaginal deliveries did you have?
  - H/o prolonged labor?
  - H/o forceps delivery? - Suggests traumatic vaginal delivery.
  - H/s/o a 3rd degree perineal tear during labor? (Disruption of anal sphincter in the tear = 3rd degree perineal tear.)
  - Mode of onset and progress? I.e. did incontinence develop soon after the problematic delivery, or did it occur later and progress gradually? Later occurrence and gradual progress ⇒ occult sphincter injury being unmasked by a lax pelvic floor and neuropathic sphincter failure owing to stretching of the pudendal nerves.

  Detailed history for iatrogenic cause of fecal incontinence
  - Which anorectal disorder did you have for which a surgery was performed?
  - Which operation was performed?
  - Did incontinence begin soon after the operation?

INTERPRETATION OF SYMPTOMS, AND DISCUSSION

Diet

Non-vegetarian diet predisposes to develop CA-colon, piles.

Vegetarian diet basically helps to prevent constipation due to high fiber-content and consequently leads to a lesser incidence of piles, fissure, CA-colon etc.

www.Ebook777.com

Pain

Basically, disorders below the dentate line (i.e. in the distal half of anal canal) are painful, and disorders above the dentate line are painless so long as they remain confined within the rectal wall.

- The presence of pain therefore suggests disorder below the dentate line, or disorder above the dentate line which has spread beyond the rectal wall.
- Examples of painless conditions: Uncomplicated internal piles, CA rectum confined within the rectal wall.
- Examples of painful conditions: Anal fissure, anorectal suppuration (fistula, abscess), acute perianal hematoma, complicated piles, CA rectum spread beyond the rectal wall, CA anal canal.

Continuous pain throughout the day ⇒ continuous tension on nerve endings. Examples: acute abscess, acute external piles (hematoma), CA anal canal.

Pain starting during/soon after defecation persisting for some time afterwards ⇒ anal fissure.

Pain caused by anal fistula *is intermittent*. Pain appears and gradually increases in intensity due to closure of the opening of the fistula and accumulation of pus within the track until it is forced open. The pain is relieved after discharge of the pus/blood-stained pus.

Site of pain: – Pain of a perianal abscess is localized to one or the other side of anus.
– Pain of pilonidal sinus is localized behind the anus.
– Bilateral sciatica-like pain may suggest spread of cancer of the rectum into pelvic cellular tissue/sacral plexus.

Intensity of pain: – Severe in acute anal fissure, acute abscess.
– Not so severe in chronic anal fissure, chronic abscess, cancer.

Nature of pain: – Sharp cutting/tearing/burning ⇒ anal fissure.
– Throbbing pain ⇒ abscess.

Acute inflammatory urological conditions of prostate/seminal vesicle/bladder neck *can* → *painful defecation*. Examples – acute prostatitis, prostatic abscess, (acute seminal vesiculitis).

Bleeding PR

- Bleeding occurring apart from defecation (i.e. occurring even without trying to pass stools), with staining of underclothes ⇒ the site of bleeding below (i.e. distal to) the anal sphincter, or the surrounding area. *Causes:* chronic anal fissure, prolapsed piles, prolapsed rectum, anal fistula, dermatitis, ulcer, anal warts, condylomata.
- Black/dark brown-colored stool which is tarry in consistency is called melena.
- Bright red blood in drops ⇒ bleeding from capillaries of internal piles.
- Bright red blood spurting with a jet spattering in the pan ⇒ bleeding from a small artery opened out due to ulceration.
- Purple-colored blood ⇒ bleeding from (dilated) veins mostly of internal piles.
- Dark red-colored blood ⇒ bleeding from rectum or colon.
- Streaks of blood smearing the sides of the stool OR blood apparent only on wiping basically ⇒ trivial bleeding OR small amount of bleeding.

Discussion

Intestinal contents remain liquid till they reach the descending colon. In the sigmoid colon, contents are stored, fluid absorbed and feces become solid. Normally, rectum remains empty except in constipation. Feces usually enter the rectum and anal canal only during defecation and immediately evacuated.

When the site of bleeding is anywhere from stomach to sigmoid colon, blood can get intimately mixed up with liquid intestinal contents and gets altered to black/dark brown color by enzymatic action → melena.

When bleeding originates from the ano-rectum, there is no chance for blood to get intimately mixed up with stools as the stool is solid, remains only for a short time in the rectum and anal canal, and also passage of formed stools is usually the cause of starting bleeding. This mechanism → hematochezia = passage of fresh blood PR – Purple/bright red colored.

Exceptions: 1. Proximal site of bleeding and yet leading to hematochezia instead of melena: This can happen if bleeding is massive e.g. massive bleeding DU, severely bleeding angiodysplastic lesion etc.

2. Rectal lesion leading to passage of altered blood instead of hematochezia: This can happen with a chronic ulcerative lesion, particularly CA in which continuous oozing of blood occurs in the rectum. As the blood is not passed immediately, it may get altered to dark color, and also gets mixed up with mucus. When certain amount of such discharge gets collected, it induces an urge to pass stools. This so-called stool consisting of only slime, actually does not contain fecal matter. Repeated passage of such discharge mimics dysentery, but without griping pain in abdomen. This especially occurs in the early morning as sufficient amount of discharge gets collected through night. – This is called 'spurious diarrhea of early morning'. (Spurious means false.)

Discharge of any kind occurring apart from defecation basically suggests the site of lesion usually beyond (i.e. distal to) the anal sphincter (anal verge), or the surrounding area.

Common causes: chronic anal fissure, prolapsed piles, prolapsed rectum, anal fistula, dermatitis, ulcer, cancer of the anus, anal warts, condylomata etc.

Itching is a non-specific symptom and may be associated with any anal/perianal disorder.

Free ebooks ==> www.Ebook777.com

Difficulties with defecation

A normal bowel habit is hard to define since it is influenced by social and dietary customs. Moreover, the definitions/criteria of constipation and diarrhea do not appear to be unanimously acceptable.

- *Constipation* may be defined as a decrease in the frequency of stools regardless of consistency and volume, to < 2 stools/week.
- *Diarrhea* may be defined as stools containing > 300 ml of fluid daily, OR may also be defined as frequency of > 3 semi-liquid stools/day.
- Normal consistency of stool is solid/semisolid.
- A hard consistency of stool does not necessarily signify any specific disease as such.
- Semi liquid consistency suggests colitis.
- Liquid (watery) consistency suggests enteritis.
- Tarry consistency = consistency similar to that of tar. Stool passed in melena has a tarry consistency.
- Greasy motion that floats on water ⇒ steatorrhea.
- Dark brown or black color suggests bleeding from GIT; the site of bleeding being anywhere from stomach to sigmoid colon.
- Pale and loose stool occurs due to intestinal hurry and steatorrhea.
- Clay-colored stool suggests obstructive jaundice.
- Passage of pure mucus ⇒ basically irritation of colonic mucosa. *Causes*: colitis, obstruction of colon, prolapse, pelvic abscess, IBS etc.
- Pink/red mucus ⇒ colitis. *Mechanism*: Inflamed mucosa bleeds as well as weeps mucus.
- Much darker red/purplish mucus = 'red current jelly' ⇒ advanced intussusception, mesenteric vascular occlusion. *Mechanism*: venous engorgement of ischemic mucosa.
- Passage of offensive dark red blood mixed with mucus and pus during defecation ⇒ ulcerative carcinoma of the rectum / anal canal.
- **Tenesmus** = painful and ineffective straining at stools ⇒ proctitis. It may also be a feature of IBS.
- Straining at stool does not signify any particular disease as such. However, it may often accompany constipation, and may also be a feature of IBS.
- **Dyschezia** suggests a growth in the ampulla of the rectum.
- **Going to the toilet several times a day but only actually defecating once or twice**, along with a h/o deep-seated perineal pain may suggest a solitary rectal ulcer.

Constipation

- Pencil-shaped (syn. pipe-stem-shaped) stools ⇒ stricture of the lower anal canal.
- Pellet-like stools ⇒ diverticular disease of colon.

Diarrhea/dysentery without griping pain in abdomen ⇒ chronic ulcerative condition of rectum, particularly carcinoma.
Griping pain in abdomen ⇒ enteritis/colitis.

Causes of **diarrhea alternating with constipation**

- A stenosing lesion of the left side of the colon.

Mechanism: In left side of the colon, bowel contents are solid and tend to obstruct the narrowed lumen, so for some days there is little or no stools. Scybalous mass proximal to the site of obstruction stimulates secretion of a large amount of mucus which is passed per rectum as mucoid diarrhea.

- It may also be a feature of IBS.

Alteration of bowel habit

Causes: Stricture, IBD (ulcerative colitis, Crohn's disease, indeterminate colitis, dysentery).

The nature of alteration of bowel habit caused by a stricture depends on the location of the stricture – whether occurring in the sigmoid colon/pelvi-rectal junction, in the (ampulla of) rectum or in the anal canal.

- *Increasing constipation* ⇒ stricture in the sigmoid colon or pelvi-rectal junction.
- *Sensation of fullness in the rectum and feeling that the bowel has not been completely emptied after defecation* ⇒ a growth in the ampulla of the rectum. This symptom is also a feature of intersphincteric abscess.
- *Sudden urgent call of stools particularly in the mornings with passage of liquid stools* ⇒ a growth in the ampulla of the rectum.

  Mechanism: As the growth ulcerates, an accumulation of mucus, pus, blood and feces occurs during the night and on rising from the bed the patient gets a sudden urgent call of stools – called the *spurious morning diarrhea.*
- *Pencil-shaped (Syn. pipe-stem-shaped) stools* ⇒ stricture of the lower anal canal.

Discussion:

This symptom viz. 'alteration of bowel habit' can be difficult to assess because some patients cannot be said to have any regular bowel habit. The range of normality is so wide—one person may normally have 3 bowel actions a day, another only one in 3 days!

www.Ebook777.com

Any deviation from a patient's norm, no matter how minor, must be considered significant if it is persistent and has been present for less than, say 2 years (practically a year). Serious disease is unlikely to have been present more than 2 years without some feature of the disease becoming manifest.

SCOPA

Causes: Piles, anal cushions, rectal polyp, prolapse of rectum.

Causes of a **swelling in the anal/perianal region**: prolapsed internal piles, skin-tag in association with piles, skin-tag in association with a fissure (sentinel pile), a perianal hematoma, venereal warts, condylomata.

Fever ⇒ sepsis e.g. anorectal abscess.

Most common causes of fecal **incontinence:** Obstetric injury, and a sequel/complication of an anorectal surgery.

In **GENERAL EXAMINATION,** specifically look for the following points

- **Posture**
  Patient preferring to stand, OR preferring to sit on the edge of a chair obviously seeming uncomfortable suggests strong possibility of (acutely) painful anorectal disorder.
- **General appearance**
  - Sick-looking appearance suggests (acutely) painful anorectal disorder.
  - A constipated patient often looks distressed, if not exactly sick-looking.

ABDOMINAL EXAMINATION

It may be better to examine the abdomen first, before carrying out a pelvic examination which is always an awkward proposition. It is better to have a patient who is in a relaxed state and willing to cooperate for a pelvic examination.

In abdominal examination, *specifically* look for evidence of intestinal obstruction.

PELVIC EXAMINATION should be carried out in the usual manner—refer to chapter 7.

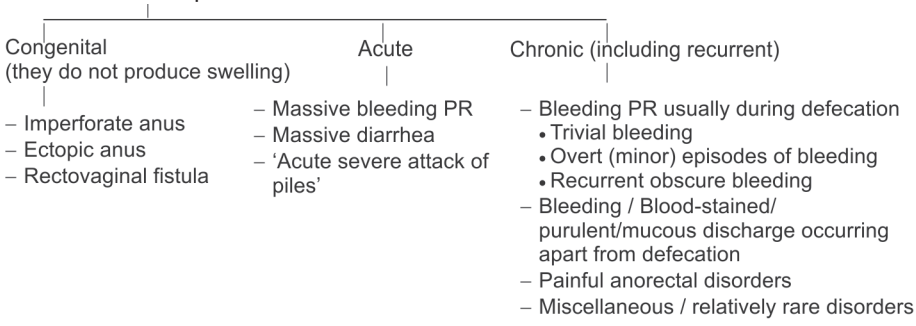
ARRIVING AT THE DIAGNOSIS

(Stepwise analysis of data)

PRINCIPLES AND PITFALLS

1. Although all symptoms are important in their own merit, the most important ones are pain, bleeding and discharge.
2. As many as 90% of rectal cancers can be felt digitally. It is unfortunate that this simple clinical examination is very commonly neglected.

Classification of presentation of anorectal disorders



Free ebooks ==> www.Ebook777.com

ACUTE MASSIVE BLEEDING PR

Commence resuscitation of shock (which may be in a state of compensation).
Arriving at the diagnosis, and principles of management in brief

– Severe bloody diarrhea i.e. severe dysentery
– Tenesmus (if there is proctitis)
± (Severe) Acute pain in abdomen
± Distension of abdomen
– Constitutional symptoms
± Anemia
– Dehydration
– Tenderness and guarding in abdomen (i.e. peritonism)
⇓
Acute severe colitis
OR Acute severe enterocolitis
OR Gangrenous ischemic colitis

Melena
↓
UGI endoscopy

Source of bleeding uncertain
↓
UGI endoscopy and/or emergency contrast meal study

Normal study

Source of bleeding disclosed
↓
Manage accordingly

Fresh blood

Source of bleeding definitely in the perineum/anorectum as concluded from patient's history and physical examination
↓
Manage accordingly

Bleeding must be considered to be arising from the lower GIT, though the possibility of a source from the 'middle GIT' should not be forgotten
↓
Conservative management. BTF often not necessary

Severe bleeding continues to threaten life
↓
Emergency laparotomy (perhaps after arteriography)

Most patients will settle down
↓
Commence specific investigation 1 or 2 days after bleeding has stopped. (Reason for waiting for 1 or 2 days is to allow time for the lower GIT to clear itself of blood.)
↓
Investigate in the sequence of:– Sigmoidoscopy – Ba enema – Ba meal (if it has not been done earlier) – Colonoscopy. All the investigations mentioned here may not be required in every case. The particular sequence for selection of these investigations has logic* though

Causative lesion disclosed
↓
The commonest lesions: diverticular disease of sigmoid, CA recto-sigmoid, polyp, angiodysplasia, acute exacerbation of IBD. Otherwise, the list of possible lesions is very long

Causative lesion not disclosed
↓
Hematological investigation

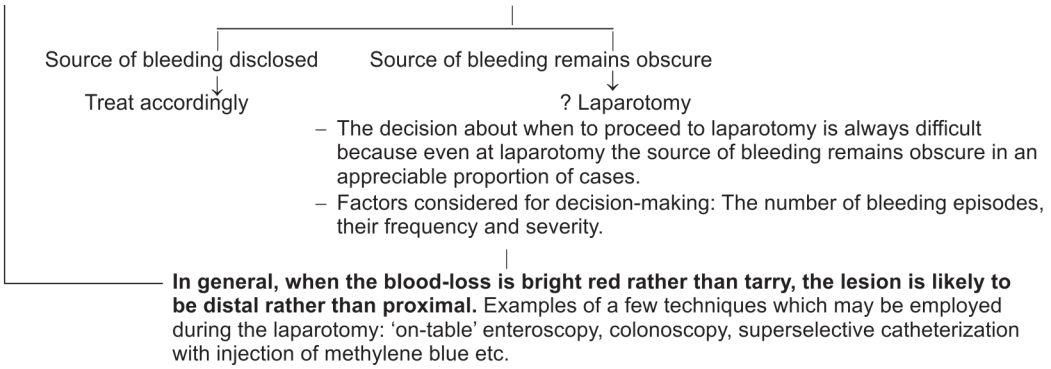
Hematological disorder disclosed
↓
Manage accordingly

Hematological disorder ruled out
↓
Wait and watch
If episode of bleeding recurs
↓
– Repeat the same protocol of management
+ ERCP to seek lesion in the biliary or pancreatic passages
± Arteriography (Urgent mesenteric arteriography. 'Urgent' as the bleeding point will be located precisely only if bleeding is brisk (at a rate of > 0.5 mL/min))
± Radionuclide-labelled red cell scanning

Contd...

www.Ebook777.com

Contd...



* – Sigmoidoscopy is more accurate than Ba enema.
– Ba enema should always precede Ba meal because the source of bleeding may be a lesion of the bowel that is partially obstructing the lumen, and inspissated barium from above may collect at the site and convert the partial obstruction into a complete obstruction.
– If sigmoidoscopy or Ba study demonstrate a lesion that is the likely source of bleeding, colonoscopy is unnecessary except that it may be considered for therapeutic purpose viz. excision of small and moderate sized (up to 3 cm) polyps by snare diathermy.

'ACUTE SEVERE ATTACK OF PILES' (= Acute painful anorectal disorders)

In a layman's vocabulary, 'piles' is a term describing anything that feels wrong in the region of the anal canal! *The chief causes*: acute anal fissure, acute anorectal abscess, acute perianal hematoma, prolapsed strangulated piles, prolapse of rectum, proctalgia fugax. They are easy to differentiate on the basis of history and a simple physical examination.

Clinical characteristics ↓	*Anal fissure (acute/ chronic)*	*Anorectal abscess**	*Perianal hematoma (Syn. Acute external pile)*	*Complicated piles*	*Anorectal fistula***	*Anal cancer*
Pain	– Severe in acute fissure Moderate in chronic fissure. – Sharp cutting / tearing / burning in nature	– Severe throbbing in c/o perianal / ischiorectal abscess – More of discomfort in c/o submucous / pelvirectal abscess	– Severe – Throbbing	– Moderate / Severe – Relieved, if the pile mass can be reduced	Recurrent attacks of slight-to-moderate pain relieved by discharge of pus / blood-stained pus	– Moderate – Dull
Relation of pain to defecation	Starts with defecation; lasts for 2–3 hours and disappears suddenly and completely only to recur at the next episode of defecation	None Pain is constant	None Pain is constant	None Pain is constant	None Pain is constant	None Pain is constant

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

Clinical characteristics ↓	*Anal fissure (acute/ chronic)*	*Anorectal abscess**	*Perianal hematoma (Syn. Acute external pile)*	*Complicated piles*	*Anorectal fistula***	*Anal cancer*
Bleeding PR / Discharge	– Drops of frank blood – in acute fissure – Mucoid discharge – in chronic fissure	–	–	Mucoid	Pus / blood-stained pus occurs apart from defecation, soiling the underclothes	Foul purulent, mixed with blood
SCOPA / External swelling	'Sentinel' pile at the lower end ⇒ chronic fissure	±	Present	Present	–	Present. May be absent initially
Fever	Absent	+. May be High-grade	Absent	–	Mild-moderate	–
Anemia	Absent	–	–	±	–	Present

*In a suspected case of **anorectal abscess** the diagnosis of which is thought on the basis of continuous anorectal pain (usually but not necessarily severe throbbing) or anorectal discomfort, associated with fever and malaise (almost invariable features), a preliminary judgment may be obtained as follows.

- Acute perianal inflammatory swelling at the anal margin ⇓ Perianal abscess
- Acute perianal cellulitis or frank diffuse swelling that is often larger and farther from that seen in a case of perianal abscess ⇓ Ischiorectal abscess
- No obvious e/o acute perianal inflammatory swelling, but pressure overlying the ischiorectal fossa → tenderness ± pitting edema ⇓ Ischiorectal abscess
- None of the previous 3 ⇓ Intersphincteric / submucosal / pelvirectal / very early (evolving) ischiorectal abscess / postanal abscess

Further differentiation may be done on pelvic examination and finally during examination under anesthesia.
DD of anorectal abscess: – Boils and gluteal abscess in either sex – Refer to page 177
– Bartholin's gland abscess in females – Refer to page 177
– Cowper's gland abscess and periurethral abscess in males—refer to page 176.

** Distance of the (single) external opening from the anal verge in a suspected c/o **anorectal fistula**

- < 4 cm ⇓ Low-level fistula → And, if the external opening is in relation to
  - the posterior half of the anus ⇓ the internal opening must be situated in the posterior midline
  - the anterior half of the anus ⇓ the internal opening must be situated exactly radially opposite to the external opening

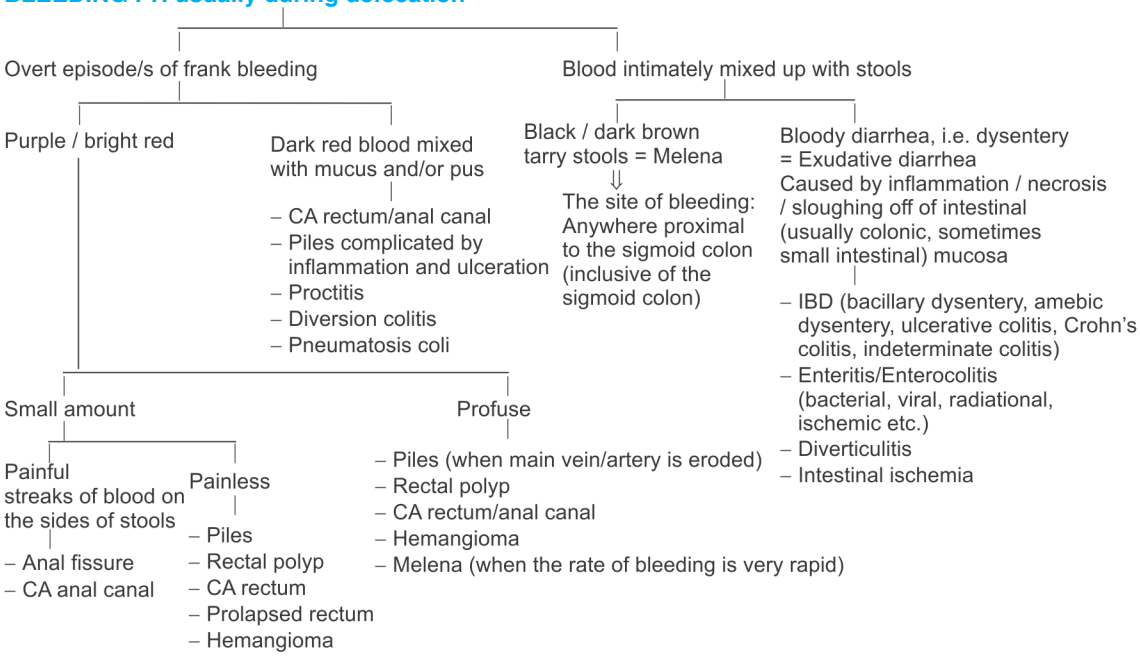
  according to Goodsall's law
- > 5 cm ⇓ High-level fistula: The track will horseshoe around the anal canal and open internally in the posterior midline according to Goodsall's law

www.Ebook777.com

TRIVIAL BLEEDING PR

= occasional streaks of blood on the sides of stools/toilet paper (blood apparent only on wiping).
Often there are no positive physical findings, although the patient may have 1st or 2nd degree piles.

BLEEDING PR usually during defecation



RECURRENT OBSCURE BLEEDING

= no identifiable cause can be established despite full routine, often repeat investigations.
Presentations: melena, fresh bleeding PR, anemia, and rarely hematemesis.
Causes

Foregut	Midgut	Hindgut
Dieulafoy lesion: rupture of a large tortuous submucosal artery normally found in the body of the stomach within 6 cm of the esophagogastric junction	– The commonest cause - small intestinal ulcer: drug-induced / associated with systemic disease like SLE, polyarteritis nodosa, RA, H-S purpura, etc. – Small intestinal tumor – Jejunal polyp/s – Jejunal divericulosis – Vascular anomalies – Meckel's diverticulum (ulceration of ectopic gastric mucosa) – one of the most common causes in children – Radiational enteritis – Aorto-enteric fistula (grafted patients) – Endometriosis in females – Hemobilia	The most common source: Proximal colon The most frequent lesion: Tiny angiodysplasia ~ 2 mm; easy to miss particularly in the cecum

Free ebooks ==> www.Ebook777.com

BLEEDING OR BLOOD-STAINED/PURULENT/MUCOUS DISCHARGE APART FROM DEFECATION

Refer to page 598.

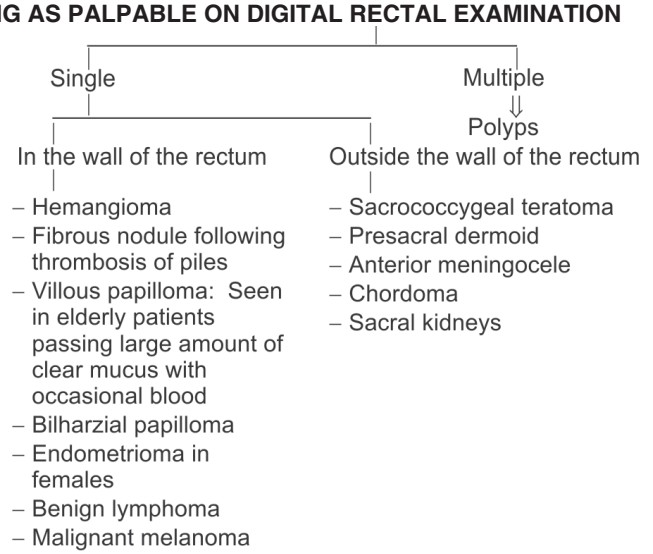
MISCELLANEOUS / RELATIVELY RARE DISORDERS

Venereal warts, condylomata, stricture, proctitis, proctalgia fugax, pruritus ani, solitary rectal ulcer, pneumatosis coli, and (chronic) swelling as palpable on DRE. Selected conditions are discussed below.

STRICTURE

– Concentric	– Eccentric
– Smooth surface	– Irregular surface
– (Usually) not ulcerated	– (Often) ulcerated
– Consistency—not as hard	– Consistency—typically stony hard
⇓	⇓
Benign stricture	Malignant stricture

(CHRONIC) **SWELLING AS PALPABLE ON DIGITAL RECTAL EXAMINATION**



SCHISTOSOMIASIS may → Bleeding PR, and large polypoidal lesions which may be misdiagnosed as cancer, usually in the descending/sigmoid colon.

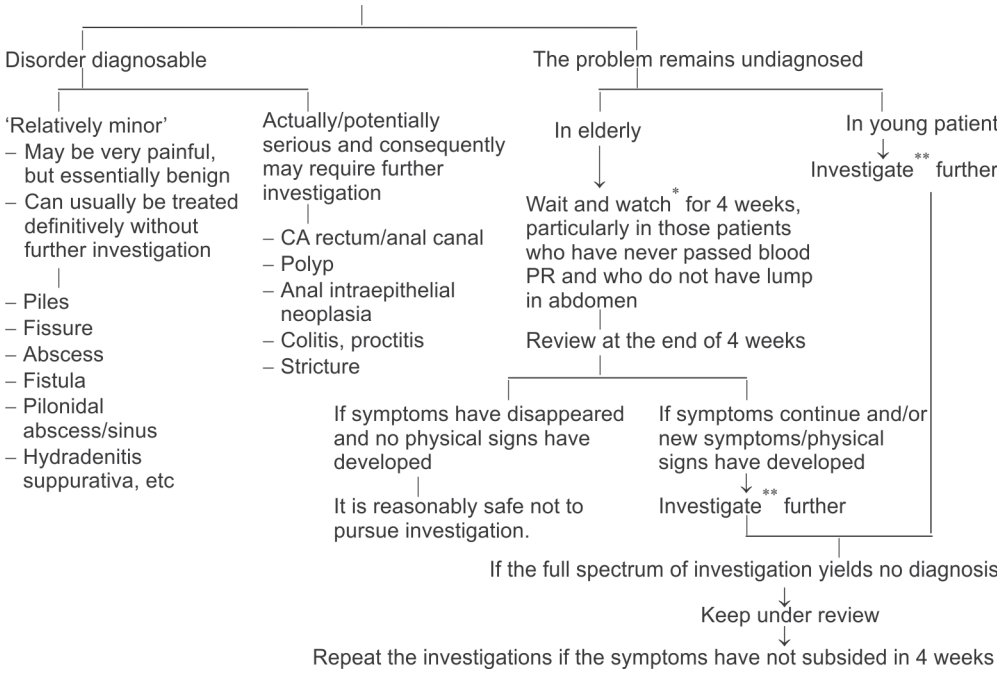
AN OVERVIEW OF DIAGNOSIS AND LINE OF INVESTIGATION IN BRIEF IN A CHRONIC ANORECTAL CASE

Look for intestinal obstruction.

Admit (as an emergency case) if patient has intestinal obstruction.

www.Ebook777.com

Conclusion on detailed history, physical examination and procto/sigmoidoscopy



* Reason for waiting: A full Ba-enema is a traumatic procedure and may well be difficult to tolerate by elderly. Moreover, experience shows that there is only very small return of +ve results of investigation.

** Apart form tumor markers, consider Ba enema – Ba meal – Colonoscopy – in this sequence if each previous investigation yields no +ve result.

DIFFERENTIAL DIAGNOSIS

Uncomplicated (internal) piles

Symptoms

– Painless bleeding PR during defecation is the earliest and the main symptom. Bleeding is separate from the stool and occurs in the form of drops or in jets spattering in the pan, or evident only on wiping.
  - Color of blood – purple or bright red.
  - Amount – variable.

  Once started, the episodes of bleeding occur for variable periods from a couple of days to a week. Recurrent episodes like this may occur for months/years.
– Prolapse — in grade 2 and 3 piles.
– Discharge: Discharge occurs only with prolapsing piles. Composed of mucus from engorged mucous membrane. It may get augmented by taking excess of liq. paraffin, which is almost invariably prescribed to these patients.
– Pruritus (itching) invariably follows discharge.
– Anemia, sometimes severe, if bleeding is persistent and profuse.
± Constipation.
± Dyspepsia.

Signs

– On inspection
  - In c/o 1st degree piles, there is no +ve finding.
  - In advanced cases, redundant folds of skin may be seen in the position of one/more of the 3 main piles.
  - In 3rd degree piles, the prolapsed piles are directly visible.

Free ebooks ==> www.Ebook777.com



Fig 41-1 4th degree piles

– On DRE: Uncomplicated piles are impalpable as they are compressible.
– On proctoscopic examination
  Purple or bright red colored mass of pile can be seen to be projecting in the lumen of proctoscope, on the background of normal rectal mucous membrane which is pale pink with thin longitudinal folds.

Notes on piles

Hemorrhoids (colloquially known as piles) are enlarged, prolapsed anal cushions.

Grades based on their progress:

– Piles presenting with bleeding alone = 1st degree.
– Piles which prolapse on defecation but reduce spontaneously = 2nd degree.
– Prolapsing piles which need manual reduction = 3rd degree.
– Irreducibly prolapsed piles = 4th degree.

Such grading does not necessarily relate to the amount of trouble.

Predisposing factors: one or more of the following.

1. Straining accompanying habitual constipation, enlarged prostate, urethral stricture.
2. Habit of sitting on the toilet for long periods.
3. Heredity.
4. Pregnancy.
5. CA rectum.
6. Portal congestion.

There are principal piles and subsidiary piles.

– The principal piles are arranged at 3 / 7 / 11 o'clock positions (– as seen with patient in lithotomy position).
– In between the 3 principal piles, additional (up to 4 or 5) small piles may occur – they are called subsidiary piles.

Pedicle of a pile is situated at the level of anorectal ring. The pile-mass is of variable size, and looks purple or bright red.

Complications of piles

1. Iron-deficiency anemia.
2. Profuse bleeding, if severe, may even lead to shock necessitating BTF.
3. Strangulation: Prolapsed pile gripped by sphincter → congestion → eventually arrest of venous return → strangulation.
   Strangulation → severe pain; patient often describes this event as an 'acute attack of piles'. Unless such piles are reduced within a couple of hours, thrombosis results.
4. Thrombosis.
5. (Superficial) Ulceration of thrombosed piles.
6. Incontinence of mucus and liquid feces leading to minor soiling and pruritus. Usually these symptoms are not particularly disabling. Disabling (extremely troublesome) incontinence suggests possibility of some cause other than piles.

www.Ebook777.com



Fig 41-2 Strangulated piles

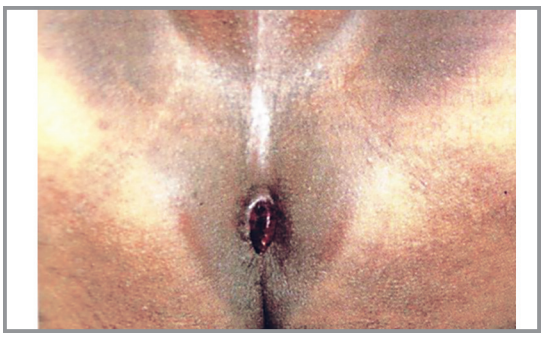


Fig 41-3 Thrombosed piles

(Chronic) External piles

Ideally, true external piles are those, which occur in association with advanced internal piles. In that case, they are called intero-external piles.

Symptom: The presence of something abnormal palpable at the anal verge.

DD: – 'Sentinel pile'.

– Dilatation of the veins of anal verge: A bluish cushion-like ring appearing when patient strains; essentially seen in those, who lead a sedentary life.

(Prolapsed) **strangulated piles**

– History is usually clear-cut: Sudden onset of severe disabling pain in a patient who has been having trouble with internal piles.
  Patient can hardly walk, and sitting is agony.
– O/E: One or more plum-colored shiny masses, weeping blood-stained mucus, and partially covered on their peripheral aspect with edematous anal skin.

(Prolapsed) **thrombosed piles**

– Pain experienced during strangulation passes off, but tenderness persists.
– Piles become purple/black. Considerable edema of the anal margin. Pile mass no longer remains soft; it becomes solid.

Acute perianal hematoma (S*yn*. **Acute external pile**)

– Acute painful and tender swelling at the anal margin following straining.
– Throbbing pain.
– Usually no h/o bleeding PR.
– No fever.
– Well-defined, rounded, purple/bluish dome-shaped perianal swelling.
– No regional lymphadenopathy.

The main DD: Prolapsed thrombosed pile.

It is essential to make an accurate diagnosis, as inappropriate incision of piles will result in considerable bleeding.

Cause of acute perianal hematoma: Rupture of hemorrhoidal vein due to straining, followed by the blood tracking subcutaneously in the space between the anoderm and the anal sphincter.

Predisposing factors: Bleeding diathesis, anticoagulation therapy.

Acute anal fissure

– Acute sharp, tearing or burning pain: starts during defecation, stays on for half an hour to few hours, and significantly diminishes or more often completely disappears only to recur with the next act of defecation.
– Bleeding PR: Thin streaks of bright red blood smearing the sides of stools, or blood dips into the pan. Amount of blood—usually small.
– H/o constipation ± (rather than aetiologically responsible, it is more likely a response to the pain.)

Free ebooks ==> www.Ebook777.com

On inspection
- Anus seems to be tightly contracted.
- By everting the anal canal using lateral traction on both buttocks close to the anal verge, the lower end of a longitudinal ulcer may be visible—situated at anal margin posteriorly in males, and posteriorly/anteriorly in females. Lower fibers of the internal sphincter also may be visible at the base of the fissure.
- Fissures may be multiple in syphilis.

On DRE
- Localized severe tenderness on the anal verge at the site of the fissure.
- Severe spasm of the anal sphincter; that precludes a complete DRE or a proctoscopy.

Chronic anal fissure
- Long history (at least 6 weeks), perhaps with acute exacerbations.
- Moderate pain.
- H/o constipation ±.

Examination findings are similar to those in acute anal fissure except for the following:
- The edge is inflamed, indurated and undermined.
- A skin tag called 'sentinel pile' is usually visible at the lower end of the fissure.
- A hypertrophied anal papilla may be visible at the proximal end of the fissure.
- Sphincter spasm may not be as severe as in acute fissure. Hence, a DRE or a proctoscopy may be possible if performed very gently.

Notes on anal fissure
- An anal fissure is a liner ulcer which occurs in the anal canal just distal to the dentate line.
- Most commonly occurs as a primary condition (exact cause is not known). Otherwise it may be caused by Crohn's disease or trauma.
- It affects both men and women of any age (including children). The highest incidence is between 30 and 40. It may occur soon after pregnancy and vaginal delivery.
- Criterion of a chronic fissure: A fissure that has been present for at least 6 weeks.

Prolapse of rectum

Basically, a h/o SCOPA during defecation and initially reducible, subsequently may become irreducible. Common in children and old age.

In children
- The parents notice a reddened area of mucosa at the anal verge.

± excess mucus and occasionally bleeding PR.

O/E
- It is possible that no abnormality is detected.
- If however prolapse has occurred at the time of examination: Carefully palpate between the finger and thumb to distinguish between two layers of mucosa and a complete prolapse.
- After reduction of the prolapse, digital examination reveals no detectable abnormality of the anal sphincter.

In adults
- Chief complaints: Prolapse on any act of straining like defecation, coughing, laughing or even walking etc; + soiling of underclothes from mucus, blood or feces.
- In addition, frank fecal incontinence OR paradoxically, constipation may occur.

O/E: Anus appears patulous, and sphincteric tone is deficient.

Partial prolapse = Protrusion of rectal mucosa **< 4 cm**.
- Color of protrusion – pale pink.
- Form of protrusion – either a tube or 2 lateral folds.

– *Prolapsed piles*	v/s	*Partial prolapse of rectum*
Look multiple and hence often segmental, pedunculated, rounded purple colored.		The protruded mass has an anteroposterior slit.

www.Ebook777.com

Complete prolapse appears like a truncated cone with circular folds over it, ≥ 7 cm in length.
There is an actual telescoping of the anterior anorectal wall down through the canal so that a groove can be felt posteriorly between the prolapsing wall and the posterior anal verge.
DD: Intussusception. Differentiating feature in favor of intussusception: A finger can be insinuated between the projection and the anus (particularly the anterior anal verge).
Notes on prolapse of rectum

- Rectal prolapse is defined as protrusion of rectum through the anal canal.
- Partial prolapse = protrusion of only the mucosa of lower rectum.
- Complete prolapse = protrusion of the entire thickness of the rectal wall.
- *Causes* of prolapse: weak anal sphincter, prolapsed internal piles, visceroptosis, following difficult labor in females, straining at micturition due to any cause, diarrhea, debilitating illness, various neurological conditions.
- *Complications*: 1. Fecal incontinence although it may be considered as a part and parcel of the whole pathological process of prolapse, 2. Ulceration, 3. Irreducibility, and 4. Strangulation leading to gangrene of the prolapsed segment.

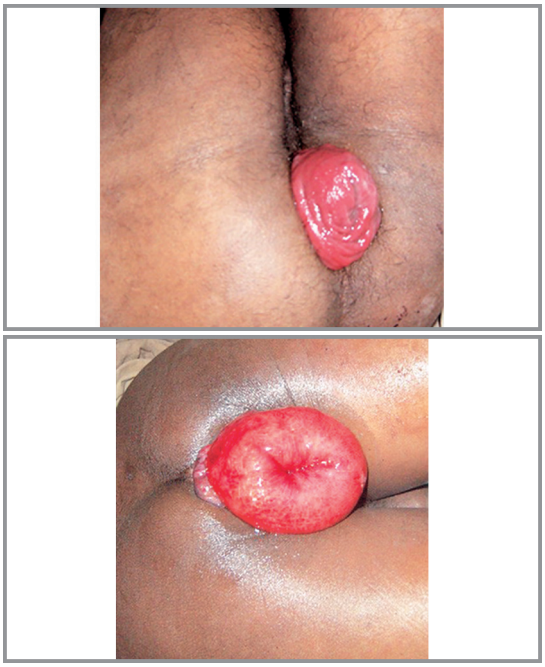


Fig 41-4 Examples of rectal prolapse

'Perianal abscess' is a generic term encompassing the collection of pus in the intersphincteric, perianal, ischiorectal, pelvirectal or postanal space.
The term 'submucosal' abscess is confusing. Probably, it may be considered basically as an intersphincteric abscess which has burst through the internal sphincter to lie in the submucosal plane.
Most patients have no predisposing factors. And most abscesses are cryptoglandular, initiated by blockage of the anal gland ducts.
Fever and malaise are common to all abscesses although these constitutional symptoms may be less marked in a perianal abscess, but more marked in rest of the abscesses which are deep-seated.

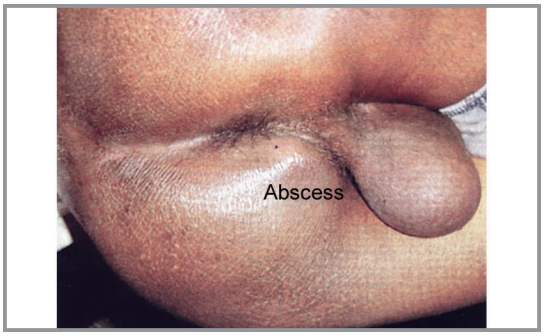


Fig 41-5 Acute perianal abscess

Acute perianal abscess

- Severe perianal pain, fever and malaise. Constitutional upset is relatively less marked.
- Acute inflammatory swelling at the anal margin between the internal and external sphincters.
- DRE is often not possible due to severe tenderness.

Ischiorectal abscess

- Severe perianal pain, fever and malaise.
- At an early stage: Acute inflammatory swelling may not be evident. Pressure on the ischiorectal fossa → tenderness ± pitting edema.
- In the advanced stage: A large area of tender induration overlying ischiorectal fossa (larger and further from perianal abscess).
- On DRE: Tender induration bulging into anal canal.

Free ebooks ==> www.Ebook777.com

Notes

– When a perianal or ischiorectal abscess discharges through the skin either spontaneously or as a result of surgical I & D, it may resolve completely. However, if the duct between the anal gland and the dentate line remains patent and becomes infected, the patient may then be left with a fistula.
– As the ischiorectal space is horseshoe-shaped, and there are no fascial barriers within it, infection can track posteriorly around the anus to affect the contralateral space.

Intersphincteric abscess

– Severe acute anal/perianal pain and a sensation of fullness in the rectum.
– Fever and malaise.
– Tender, indurated submucosal mass.
– May not be readily apparent on proctoscopic examination.

Note: Very difficult to detect clinically. Examination ↓ anesthesia is usually necessary.

Submucosal abscess

– Anal discomfort or pain, fever and malaise.
– On DRE: Mucous membrane is stretched and fixed over the swelling.

Pelvirectal abscess

– Rectal discomfort or pain, fever and malaise.
– On DRE: Mucous membrane is mobile over the swelling.

As the pelvirectal spaces encircle the anorectum above the levator muscles, abscesses can be bilateral and often present with a major constitutional upset.

Postanal abscess

Postanal space = space between external sphincter and levator ani.

Symptoms: Severe rectal discomfort, often radiating to sacrum, coccyx or buttocks; OR The pain will display sciatic distribution.

Differentiating features: Fever and exquisite posterior rectal tenderness.

Very often diagnosis is missed. A high index of suspicion is essential.

Notes

– *DD* of postanal abscess only on the basis of pain: Proctalgia fugax, coccydynia or lumbosacral strain.
– Almost inevitably, an internal opening in the posterior midline is identified at the time of drainage of the abscess.

Ano/rectal fistula

Ways of presentation

1. Following spontaneous discharge or I & D of an abscess previously, usually after all the inflammation and induration has settled down.
2. There may be no h/o abscess formation and the fistula may appear to arise *de novo*.

Symptoms

– Discharge of pus/blood-stained pus occurring apart from defecation, soiling the underclothes.
– Perianal discomfort.
– Recurrent attacks of slight-to-moderate pain with mild-moderate fever due to closure of the opening, relieved by discharge of pus/blood-stained pus.
– Occasionally, passage of flatus through the fistula.

On inspection

The external opening

- is usually close to the anal verge although sometimes it may be a few centimeters away, and rarely far away.
- may be single or multiple.
- may show hypertrophic granulation tissue.
- occasionally, raised with surrounding induration (except in TB).

On application of digital pressure over the fistula, pus may be seen exuding from the opening/s.

www.Ebook777.com

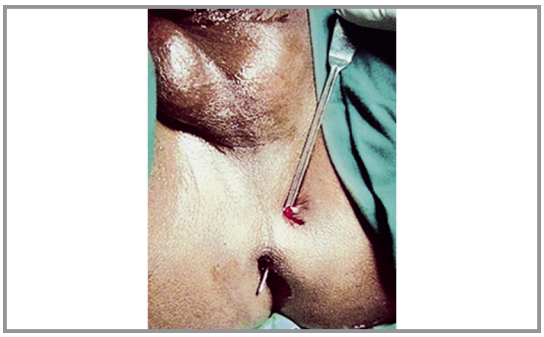


Fig 41-6 A c/o fistula being examined by probing (often ↓ anesthesia)

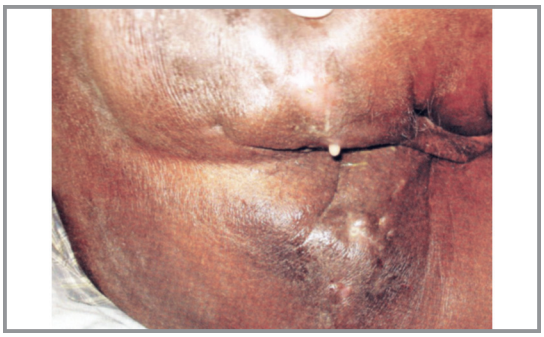
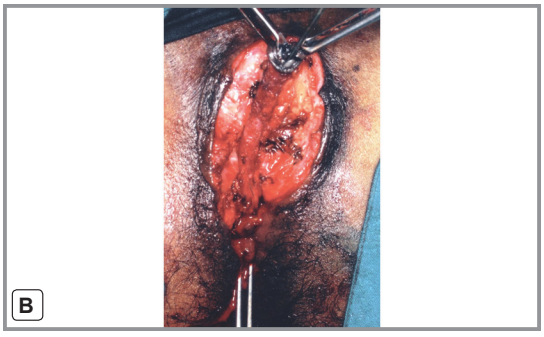


Fig 41-7 Multiple fistulae





Figs 41-8A and B Another case of a fistula. **(A)** External opening at 1 o'clock position, close to the base of the scrotum. **(B)** Fistulectomy. The tract has been 'cored out'.

On DRE

- Indurated elevation with a central hole suggests internal opening. Carefully assess the position of this vis-â-vis the anorectal sling
- On bidigital palpation (with one finger in the anal canal and thumb outside), fistulous track may be felt as a subcutaneous 'cord'.

Classification	*Criterion*
– Low-level	Internal opening lies below the anorectal ring
– High-level	Internal opening lies above the anorectal ring

Causes of fistula

- Most common—Idiopathic-nonspecific
- Other—TB, IBD, Bilharziasis, lymphogranuloma inguinale, stricture, colloid carcinoma of colon: any of these conditions may present as a case of anorectal fistula. These conditions → multiple fistulae.

Pilonidal sinus and abscess

Refer to chapter 43.

Perianal hydradenitis suppurativa

- Occurs in post-pubertal age only, as apocrine glands are not active until puberty
- The affected area becomes indurated; subsequently forms sinuses with discharge of small amounts of pus
- Other regions which can be similarly affected: The areas containing apocrine glands – axillae, breasts, inguinoscrotal region.

Free ebooks ==> www.Ebook777.com

Fibroepithelial anal polyp

Chief complaint: Prolapsing external anal swelling.

Usually associated with a previous history of perianal disease, including piles or anal fissure. Occasionally there may not be such preceding history.

Main DD: Prolapsing rectal adenomatous polyp on a long stalk. Differentiating point: Adenomatous polyp arises above the dentate line.

Note

Fibroepithelial polyp consists of hypertrophic epithelium arising on a stalk from the anal canal itself. Histology: Keratinized squamous epithelium supported by scarred fibrotic subcutaneous tissue.



Fig 41-9 Fibroepithelial polyp

Anal skin tags

Irregular bits of skin at the anal verge are known as skin tags.

May be associated with piles. The tags themselves are not piles.

Since the tags in themselves are otherwise asymptomatic, there is no particular indication to operate.

CA rectum

– Passage of dark red blood mixed with mucus and pus during defecation. (Mechanisms of bleeding PR: Directly from the growth; OR from the secondary piles developed as result of a growth higher up.)
– Alteration of bowel habit.
– No pain in the early stages. The presence of rectal pain is an ominous sign; it indicates a locally advanced cancer which is likely to be inoperable.
– O/E: - Ulcer with everted edge of hard consistency, hard base and surrounding induration.
  OR - Stricture with surrounding induration.
  OR - Polypoidal growth with surrounding induration.
  Classification based on mobility in relation to the pelvis: mobile / tethered / fixed. This classification has importance in determining the operability of the tumor.

Notes on colorectal cancer

– The usual cancer is adenocarcinoma.
– Macroscopic types of colorectal carcinoma: Ulcerative, stricturous (annular/tubular), polypoidal (fungating) and mucinous (colloid. Bulky tumor with a gelatinous appearance). In general, ulcerative and stricturous types are more common in the left side of the colon, and polypoidal type is more common in the right side.
– The majority of colorectal cancers occur in rectum and sigmoid colon.
– Modes of presentation of colorectal cancer in general:
  • Chronic symptoms.
  • Emergency: - Acute (often on chronic) intestinal obstruction.
    - (Fecal) peritonitis due to perforation either through the tumor itself or as a result of a closed loop obstruction when the ileocecal valve is competent.
– Rectal cancer very rarely presents as an emergency.

CA anal canal

– Passage of dark red blood mixed with mucus and pus during defecation.
– Discharge per rectum apart from defecation.
– Pencil-shaped (*syn*. pipe-stem-shaped) stools.
– Pain ±.
– O/E: Typical ulcer/ulcerated swelling at the anal margin or within the anal canal—with everted edge of hard consistency, hard base and surrounding induration.
– Inguinal metastatic lymphadenopathy (medial horizontal group) ±.
– Examination ↓ anesthesia is extremely important in evaluating the extent of the disease.

www.Ebook777.com



Fig 41-10 CA anal canal

Notes

– Almost always a squamous-celled carcinoma.
– Rarely adenocarcinoma may be seen but this is usually a very low rectal cancer which is involving the anal canal.
– Another very rare cancer—malignant melanoma.
– Local spread: Proximally and radially into the rectum and anal sphincters respectively. In advanced cases it may also spread to vagina.
– Lymphatic spread occurs to perirectal and inguinal nodes.

Colorectal polyps

– A polyp is defined as any pedunculated mass, without prejudice to its histological nature.
– Polyps arise from mucosa of the colon/rectum.
– Some polyps (adenomatous, familial hamartomatous and ?metaplastic) may contain a focus of invasive cancer. Any polyp greater than 2 cm in diameter must be assumed to be malignant unless proved otherwise on histological examination. When an adenoma is more than 2 cm in diameter, there is a 50% chance that it contains foci of invasive cancer.

Types of colorectal polyp: Inflammatory, hamartomatous, metaplastic and adenomatous.

Inflammatory polyps *(sometimes referred to as pseudopolyps)*

Occur in patients with colitis. Tend to be small, rarely exceeding 0.5 mm in diameter. Consist of inflamed congested mucosa with edematous changes in the submucosa.

Hamartomatous polyps are found in two forms: Juvenile and familial Peutz-Jeghers syndrome.

Juvenile *hamartomatous polyps*

– Painless bleeding PR, usually in children below the age of 10 years.
– SCOPA ±; the child's mother might have noticed it.
– O/E: Pedunculated bright red/cherry red smooth swelling which often tends to slip away from the examining finger. Very rarely, the polyp may be completely missed even during examination under anesthesia, if the polyp has a long stalk, and has slipped proximally high up.
– In 70% of cases, they are single; and 70% occur in the rectum and distal sigmoid colon.

Hamartomatous polyps occurring in ***Peutz-Jeghers syndrome***

Polyps are associated with pigmented lesions (bluish brown) on the face and on the lingual and buccal mucosa. Familial tendency is very strong. Almost always multiple. Found more commonly in the small intestine than in the colon/rectum. Unlike juvenile polyps, there is significant malignant potential.

Metaplastic polyps

Plaque-like growths 1–2 mm, rarely exceeding 5 mm in diameter. Rarely symptomatic.

Adenomatous polyps

– Mostly asymptomatic.
– May cause chronic occult blood-loss leading to anemia, or may present with overt bleeding PR.
– Diarrhea and tenesmus may occur due to mucoid discharge from the surface of the polyps, particularly in case of villous adenomas.

Free ebooks ==> www.Ebook777.com

– Retrograde propulsion of larger pedunculated polyps may produce abdominal pain, and in extreme cases lead to the development of colocolic intussusception.
– Highly variable in size. May be pedunculated or sessile.
– Diagnosis is made on either colonoscopy or barium enema. Colonoscopy is not 100% sensitive. Repeat colonoscopy can often demonstrate lesions which were missed on the previous colonoscopy, and miss lesions which had already been seen.

A villous papilloma/adenoma is a sessile adenoma made up of frond-like strands which grows as a carpet on the rectal mucosa.

– Spurious diarrhea from abundant mucus discharge → general breakdown of health, dehydration and hypokalemia.
– Lethargy, muscle weakness, mental confusion and in some extreme cases, renal failure can result due to hypokalemia and metabolic acidosis.
– On palpation: Feels typically like a soft velvety carpet. The base is soft and the tumor is mobile.

Familial adenomatous polyposis

A dominantly inherited condition characterized by the appearance of multiple adenomatous polyps throughout the large intestine—inevitably leads to colorectal cancer by the 4th decade of life. The severity of the polyposis is variable. Always consider this diagnosis when > 3 polyps are identified on clinical examination, on the background of a family history; and investigate.

Anal intraepithelial neoplasia (- a rather histological diagnosis of epithelial dysplasia)
Symptoms: Perianal discomfort, itching and bleeding.
On examination: The perianal epithelium may be thickened and may have a whitish appearance. This may be exaggerated by local application of acetic acid which is useful in targeting areas for biopsy.
In women it may be associated with similar intraepithelial neoplasia of the vulva/vagina/cervix.
Potential danger of intraepithelial neoplasia: Development of invasive squamous-celled CA of the anus.

Anorectal stricture

= stricture occurring anywhere from sigmoid colon to anal canal.
– Usual way of presentation: Alteration of bowel habit.
– Occasionally may present with acute or subacute intestinal obstruction.
– On DRE: a stricture or a growth may be felt.
Sometimes it is difficult to distinguish between a true stricture, and sphincter spasm. While examining under anesthesia, spasm will give way whereas true stricture will persist.
Causes: Congenital, post-traumatic (penetrating injury, impaction of fetal head during labor etc.), after injection of sclerosant, post-operative, post-radiation, lymphogranuloma inguinale, schistosomiasis, ulcerative colitis, diverticulitis, endometriosis in females, cancer, and in elderly patients as a result of habitual overpurgation.

Condyloma acuminatum (*Syn.* Venereal wart)
– A single or multiple cauliflower-like dry masses.
– Variable size.
– Usually follow gonorrhea.

Condylomata
– Fungating sessile growth / round or oval papules covered by a gray exudate with moist sodden surface and typical (offensive) smell.
– Usually due to syphilis.

Proctitis
– Discharge of dark blood, mucus and pus.
– Tenesmus.
– Fever.
– Rectal mucosa is tender, red, glossy (inflamed) with multiple small ulcers.
Causes: Non-specific infection, bacillary/amoebic dysentery, ulcerative proctitis/colitis, gonorrhea, lymphogranuloma inguinale, tuberculosis, Bilharziasis, strawberry proctitis, pelvic radiation, repeated trauma by finger manipulations in neurotic patients.

www.Ebook777.com



Fig 41-11 Venereal wart



Fig 41-12 Condylomata

Diversion colitis
= inflammation of the diverted (left behind) rectum usually along with distal portion of the sigmoid colon (as for example after a Hartmann's procedure).
Clinical features are similar to proctitis.
Histologically indistinguishable from ulcerative colitis.

Proctalgia fugax (*Fugax* = fleeting)
- Mode of onset of pain – sudden, often occurring in the middle of the night.
- Mode of disappearance of pain – spontaneous and fairly sudden.
- Very severe pain deep in the anorectal region, often with a lancinating series of exacerbations that shoot deep inside from the buttock region, lasting from a few seconds up to several minutes. The pain can be extremely severe and cause considerable alarm.
- The episodes may be separated by pain-free intervals of many days, weeks or months.
- Patient may be neurotic.
- The condition although recurring, is self-limiting and entirely harmless.

Probably caused by muscle spasm, but the real underlying cause is obscure.
DD: Syphilis (tabes dorsalis).

Pruritus ani (*Syn.* **Itching**)
On inspection: – a dermatological disease may be present.
– Itching → typically perianal excoriation and icthyosis (dryness, roughness and scaliness of the skin).
On DRE: anal tone might have been reduced.
On proctoscopy: an anorectal disorder like piles etc. may be disclosed.

The primary cause is probably minor anal leakage as a result of internal sphincter dysfunction leading to irritation
↓
Introduction of fungal infection will intensify itching ——————> ——————————> itching
↑ ↓
exacerbation of irritation ← scratching and brisk cleansing

In vast majority of patients the cause is not known (idiopathic).
Enterobial infestation *is particularly common in children*.
Otherwise, there is a large number of anorectal and dermatological conditions which may give rise to this symptom.

Solitary rectal ulcer
- Difficulty in defecation viz. going to the toilet several times a day but only actually defecating once or twice.
- Deep-seated perineal pain.

± passage of blood and mucus PR.

On DRE: an indurated area on the anterior aspect of the rectum.
Notes
- The term is a misnomer as the lesions may be multiple and ulcer may not be necessarily present.
- A red thickened area of rectal mucosa usually with a shallow ulcer in the centre, classically positioned on the anterior wall of the rectum opposite the puborectalis sling 5–8 cm above the anal verge.

Free ebooks ==> www.Ebook777.com

- Can occur at any age; most common in young adults.
- Patients spend an inordinate amount of time in the toilet trying to move their bowels.
- The diagnosis is established by endoscopy and biopsy.

Perianal Crohn's disease

- More common in patients with colonic and particularly rectal Crohn's disease but it may occur in isolation or in association with Crohn's disease elsewhere in the GIT.
- Potentially a premalignant condition.
- Examine ↓ anesthesia.
- *Morphological classification*:
  - Primary lesions: anal fissure, edematous piles, ulceration.
  - Secondary lesions: skin tags, anal stricture, perianal abscess and fistula, anovaginal/rectovaginal fistula, carcinoma.
  - Incidental lesions: skin tags, piles, perianal sepsis, hydradenitis suppurativa.

Ulcerative colitis

Severity of the disease is roughly classified into mild, moderate and severe.

Mild UC

- 'Small' amount of bleeding PR.
- < 4 loose motions a day.
- Absence of systemic symptoms and signs.
- Extracolonic manifestations ±: arthritis, uveitis, erythema nodosum. Sometimes, these manifestations may constitute the chief complaints and may pre-date the onset of diarrhea.

Moderate UC

- Bleeding PR invariably present.
- Frequency of diarrhea 4-6 a day.
- Mild colicky pain or discomfort in abdomen.
- (Low-grade) Fever.
- Loss of appetite, loss of weight, iron-deficiency anemia.
- Rapid deterioration with the development of severe disease or acute local complications (massive bleeding, toxic megacolon or colonic perforation) may occur at some stage during the course of the disease.

Severe UC

Modes of presentation: 1. Acute severe attack starting *de novo* – More common.
2. Progression of mild/moderate disease to severe form.

- Profuse bleeding PR.
- Severe diarrhea.
- Tenesmus.
- Fever.
- Severe pain in abdomen.
- Anemia, dehydration, acidosis. Hypovolemic shock ±.
- Abdominal distension of varying degree. Generalized abdominal tenderness and guarding.

Notes on ulcerative colitis

- An idiopathic condition of the rectum and colon. Rectum is nearly always involved and inflammation extends in continuity to involve varying lengths of the colon.
- The clinical course is variable. The severe intractable form is encountered in patients with total colitis.
- One of the biggest diagnostic challenges is to distinguish ulcerative colitis from Crohn's colitis. If it is impossible to reach a firm diagnosis using clinical, histological and radiological grounds, then the case may be labeled as indeterminate colitis.
- Local complications: Toxic megacolon, perforation, massive bleeding, stricture, cancer, perianal suppuration, giant inflammatory polyposis.

www.Ebook777.com

Crohn's colitis

– Can be difficult to distinguish from ulcerative colitis, but is usually possible to make a definitive diagnosis by employing a combination of clinical and histological features
– Crohn's colitis may be segmental, and if rectal sparing is observed, a diagnosis of ulcerative colitis can virtually be ruled out.

Clinical features	*Ulcerative colitis*	*Crohn's disease*
Bleeding PR	Very common	Unusual
Pain in abdomen	Infrequent	Common
Abdominal mass	Rare	Sometimes
Spontaneous fistula	Very rare/Never	Sometimes
Radiological features		
Distribution	Continuous with rectum	Often discontinuous
Mucosa	Granular. Shallow ulcers	Cobblestone appearance. Fissuring
	Pseudopolyps	Deep undermining ulcers
Strictures	Rare. Usually malignant	Often present
Prognosis	Surgery may be curative	Frequently relapsing course despite medical management or surgical intervention

Neutropenic colitis

A complication of chemotherapy, but it may also occur in other neutropenic patients.
Caused by superinfection by clostridial species of micro-organisms.
May lead to gas gangrene of the intestine.
Fulminating neutropenic colitis carries a mortality nearing 100%.

Pneumatosis coli

– Colicky pain in abdomen.
– Frequent bowel action. Mucus in stools. Bleeding PR.
– Large amounts of flatus.

Notes

– A rare condition of unknown aetiology affecting both the large and the small intestine.
– Association with COAD ±.
– Often asymptomatic. Usually discovered incidentally on abdominal X-ray.
  On plain X-ray: Gas-filled cysts in the submucosal and subserosal planes. On Ba study: Filling defects.

Angiodysplasia

Modes of presentation: – Chronic occult blood loss leading to iron-deficiency anemia.
– Recurrent obscure bleeding.
– Massive bleeding PR.

Characteristic age—over 60.

Notes

– They may occur anywhere in the GIT, but are commonest in the right side of the colon.
– Histologically—thin-walled vascular channels in the submucosa.

CONSTIPATION

Definition (of simple constipation): < 2 stools per week regardless of consistency and volume.
Constipation is a symptom rather than a disease, and there are multiple causes.

Free ebooks ==> www.Ebook777.com

The causes of constipation may be classified as follows

Locoregional

- Any stricturing lesion (benign or malignant) of the colon, rectum, anal canal or anus. Causes: following any inflammatory / ulcerative pathology like diverticular disease, Crohn's disease, etc; ischemia, postoperative, and cancer etc.
- Any painful anorectal disorder which inhibits the wish to defecate; e.g. fissure, abscess, strangulated piles, acute perianal hematoma, etc.
- Aganglionosis and/or lesions of the myenteric plexus: Congenital megacolon, idiopathic megacolon and megarectum
- Extrinsic compression by gravid uterus, any other big lump in the pelvis, etc.

Extracolonic

- Neurological disorders
  - Autonomic neuropathy (e.g. DM)
  - Parkinson's disease
  - Spinal cord injury*
  - Multiple sclerosis
- Endocrine disorders, e.g. hypothyroidism
- Connective tissue disorder, e.g. systemic sclerosis
- Any illness causing immobility
- Various drugs
- Psychological, e.g. depression, etc.

Unknown = Idiopathic
No structural abnormality

- Slow (generalized) colonic transit responsive to treatment
- Slow (generalized) colonic transit unresponsive to treatment
- Slow sigmoid colonic / rectosigmoid transit = 'obstructed defecation'

* Spinal cord transaction above L2 → constipation, but reflex emptying of the rectum can be brought about using enemas/suppositories. Injuries of sacral nerves and the cauda equina → lack of control and loss of tone of the external sphincter and this may lead to both constipation and incontinence.

ABSOLUTE Constipation

The patient "feels like passing flatus but is not able to do so, and feels that passage of flatus will relieve pain."

– This statement may be taken as absolute constipation ⇒ Complete intestinal obstruction. Refer to chapter 34.

Irritable bowel syndrome

Refer to chapter 33.

FECAL INCONTINENCE

Causes

(**A**) *Most common*

1. Obstetric injury: Following single or multiple vaginal deliveries, due to a combination of nerve damage and direct damage to the pelvic floor.
2. Iatrogenic injury during anorectal surgery:
   a. Laying open of an anal fistula which has transgressed the external sphincter.
   b. Overenthusiastic lateral internal sphincterotomy.
   c. Vigorous stretching of the anal canal for the treatment of fissure, piles etc.
   d. Vigorous retraction of the sphincter to facilitate endorectal surgery.

(**B**) *Other causes*

1. (Any) Anorectal disease.
2. Accidental trauma to the anorectum.
3. Fecal impaction.
4. Factors causing diarrhea: IBD, infectious colitis, malabsorption.
5. Patulous sphincter due to a neurological disorder: Pudendal nerve damage, peripheral neuropathy, stroke, multiple sclerosis, spinal cord damage.
6. Congenital disorder: Treated imperforate anus, megacolon, spina bifida.
7. Miscellaneous: Behavioral, encopresis.

Though a variety of specific causes may be responsible, the majority of cases are idiopathic, most commonly affecting elder parous women.

www.Ebook777.com

Severity of incontinence may be objectively assessed and documented using a grading scale (Refer to an authentic textbook on the subject).

Findings on DRE

– Reduced resting anal tone and squeeze pressure.
– A defect may be felt particularly in the anterior part of the external sphincter.

GUIDELINE STATEMENTS OF DIAGNOSIS OF COMMON ANORECTAL DISORDERS

Uncomplicated ….. degree pile/s at ….. o'clock position/s

± Subsidiary piles (details – number and positions)
± Associated with pruritus/anemia
± Most probable predisposing factor/s: …..

Acute perianal hematoma at ….. o'clock position

± Predisposing factor: bleeding diathesis/anticoagulation therapy.

Anal fissure

– Acute/Chronic (duration ~ …..)
– Posterior midline/Anterior midline/any other site.
– Idiopathic/Probably secondary to trauma/Crohn's disease/?malignant.
– Associated with constipation/passage of hard stools etc.

Partial / complete prolapse of rectum

– Most probably caused by ……
– with/without fecal incontinence.
± Complicated by ulceration/irreducibility/strangulation.

Anal fistula

– Low level / High level. (This judgment is based on two clinical points. 1. Distance of the external opening from the anal verge; and 2. The site of the internal opening (if palpable) in relation to the anorectal ring.)
– Level – uncertain clinically (– if the external opening lies at ~ 4–5 cm from the anal verge, and the internal opening is not palpable).
– Most probably nonspecific

OR

Probably secondary to ……

Anal skin tag/s

– Associated with difficulty in maintaining perianal hygiene/associated with pruritus.
– No other anorectal abnormality.

Painless/painful malignant growth of the rectum, most probably CA on the ….. wall, involving ~ …… of the circumference.

– Ulcerative/stricturous/polypoidal/mucinous.
– The lowest point of induration ….. cm from the anal verge.
– Mobile/tethered/fixed.
± with/without acute-on-chronic/chronic intestinal obstruction.
± Apparently without/with distant metastatic spread.

Free ebooks ==> www.Ebook777.com

42 ULCER

An ***ulcer*** is defined as a breach in the continuity of skin/mucous membrane due to molecular death of the tissue, cell by cell.
A ***wound*** is defined as a traumatic break in the continuity of any tissue.

GENERAL REMARKS

1. Legs are ulcer-prone and most of the varieties of ulcer can be seen in legs.
2. Floor, edge and base are the most important parts of any ulcer. Each of them has diagnostic and/or therapeutic significance.

HISTORY

- **Duration**?
- **M/o onset**?
  - Chronic ulcers practically present with insidious mode of onset.
  - Ulcers with apparently sudden mode of onset usually develop 'over and above some or the other preceding problem' such as an abscess/cellulitis/swelling/scar etc.

  Examples – An acute spreading ulcer may develop over an abscess/cellulitis.
  – A venous ulcer often develops over dermatitis.
  – A tuberculous ulcer usually develops following bursting of a tuberculous cold abscess.
  – A Marjolin's ulcer develops over a scar.
- **Progress**?
  - Over days ⇒ 'spreading ulcer'.
  - Over weeks ⇒ malignant ulcer.
  - Over weeks to months ⇒ chronic infection.
  - Stationary for months ⇒ delayed healing ulcer/callous ulcer.
- **Regress**?
  Regress over a period of days ⇒ healing ulcer.
- **Discharge** denotes the activity (and hence stage), and nature of the ulcer.
  - Does it foul smell? Foul smelling discharge ⇒ sepsis.
  - Type? - Blood (*syn*. sanguinous) ⇒ malignancy. An ulcer in the stage of healing also shows bloody discharge.
    - Pus ⇒ sepsis. Watery pus ⇒ TB. Blue-Green colored pus ⇒ infection with *P. pyocyneous*.
    - Serum—does not suggest anything specific really.
    - Chips/pieces of bone in the discharge ⇒ osteomyelitis.
  - Amount?: Profuse discharge ⇒ more activity ⇒ acute spreading ulcer.
    However, profuse discharge in a small chronic ulcer may be suggestive of a 'sinus' rather than the lesion being an ulcer.
    Discharge is usually absent in neuropathic ulcer, ischemic ulcer.

www.Ebook777.com

- **Pain**?
  - Pain is absent in 'neuropathic ulcer' (due to peripheral neuritis / nerve injury / leprosy / transverse myelitis etc.), early stage of a malignant ulcer.
  - The presence of pain ⇒ acute inflammation—usually experienced in acute spreading ulcers.

  The presence of pain or unusual sensations in case of a malignant ulcer may suggest perineural spread. This fact has a therapeutic implication: Involvement of a sensory nerve in the vicinity should be specifically looked for, and included in resection if found to be involved to prevent recurrence.
- **Fever**? ⇒ infection. If the ulcer is perforating, think about a possibility of osteomyelitis.
- **Disturbance of function / disability**?
- **Clicking sounds / crepitations**? ⇒ osteomyelitis.
- **Significant loss of weight**?
- H/s/o **regional lymphadenopathy**?
- H/s/o **factors responsible for chronicity**?
  - Arterial deficit, chronic venous insufficiency.
  - Anemia.
  - DM.
  - TB, leprosy.
  - Steroids, chemotherapy, (any other cause s/o) immunodeficiency.
  - Repeated trauma, persistent pressure, lack of rest, retained foreign body.
- **Bleeding**? A major bleed can be caused by erosion of a blood vessel under the base of an ulcer.
- **Treatment taken**?
- Have you been on any long-term medical treatment for any other disease?
- Is this the first episode of ulceration OR is this a **recurrent episode**? Details of previous episode/s, if any.
- **Past history**?
- **Smoking, alcohol, other** addiction? - Details.
- **Hypersensitivity**?

GENERAL EXAMINATION

A thorough G/E should be carried out.
Specifically look for clinical signs of wasting (i.e. loss of weight) and signs of undernutrition.

LOCAL EXAMINATION

INSPECTION

- **Number**?
  - Primary malignant ulcer is usually single.
  - Examples of multiple ulcers: TB, apthous etc.
- **Site**?

  Certain ulcers are more prone to occur at specific anatomical locations.
  - Squamous-celled CA: oral cavity, penis, vulva, and anal canal.
  - Basal-celled CA: on the face superior to an imaginary line joining the angle of the mouth to tragus of the ear.
  - TB: Neck > axilla > groin.
  - Ulcers associated with PAOD: Tips of fingers and toes, feet and legs (especially, shin).
  - Venous ulcers: Gaiter area (i.e. medial aspect of lower half of leg), lateral aspect of ankle and one-third of the way up the lateral aspect of the leg, and occasionally, extensive circumferential ulcer on the lower leg.
  - Neuropathic: Pressure points of the sole viz. heel, tips of the toes, and heads of the 1st and 5th metatarsals.
  - Bed sores: Sacrum, iliac crest, ischial tuberosities, greater trochanters, heels, scapulae and occipital region.
- **Extent**? Extent should be detailed in horizontal and vertical directions, or in relation to anatomical landmarks in the vicinity of the Ulcer.

Free ebooks ==> www.Ebook777.com

- **Shape**?
  - Generally, nonspecific ulcer in the stage of extension, and malignant ulcer has irregular shape.
  - Generally, perforating (neuropathic) ulcer has circular shape.
  - Square shape may suggest dermatitis artefacta.
- **Size**?
  - 'Traumatic ulcers', acute spreading ulcers in uncontrolled diabetics or immunocompromised patients, and bedsores can be very large.
  - Size of the rest of the ulcers usually varies between 0.5 and 5 cm.
- **Discharge** denotes the activity and pathological nature of the ulcer.
  - Profuse discharge of pus, especially foul-smelling ⇒ acute spreading ulcer.
  - In neuropathic and ischemic ulcers discharge is scanty or absent.
  - Pus: - Thick, yellow, creamy ⇒ Staphylococcal infection.
    - Thin, yellow ⇒ Streptococcal infection.
    - Thin, pale yellow/watery ⇒ TB.
    - Bluish/Greenish ⇒ infection with *P. pyocyneous*.
  - Serous discharge does not imply anything specific really.
  - Serosanguinous ⇒ malignant/'tropical' ulcer.
  - Sulfur granules ⇒ Actinomycosis.
  - Bone particles ⇒ osteomyelitis.
- **Floor**?

  Floor means the visible raw area of the ulcer.

  The floor consists of the tissues exposed by the deficiency in the integument.

  In ulcers, there is a loss of tissue which leads to depression of the floor in comparison to the surrounding skin, but in malignancy, gout and gumma there is proliferation of tissue leading to the floor which may be raised above the level of the surrounding tissue.

  The floor of an ulcer may be formed by granulation tissue/slough/eschar/crust (dried out discharge).

  The floor may have a cauliflower appearance.

  The floor may also exhibit pulsations, the presence of maggots, or movements of muscle/tendon underneath.

Granulation tissue = a sheet of delicate capillaries and fibroblasts covered by a thin layer of fibrin or plasma.

1. Pink, pin-point, flat, just below the level of the surrounding skin and easily bleeding on touch = 'healthy' granulation tissue ⇒ **healing** ulcer.

   Each pinpoint (granule) represents a single regenerating capillary.
2. Pale and smooth = 'unhealthy' granulations ⇒ **delayed healing** ulcer. Such an ulcer is often called an '**indolent**' ulcer. Indolent = Lazy. Basically, the presence of granulations suggests the body's attempt towards healing. However, unhealthy granulations suggest the presence of some factor/s creating hindrance in the healing process, which need to be identified and eliminated/controlled. If treatment for the same proceeds in correct direction, then granulations may start appearing rather healthier, and then healing can be expected to occur.
3. Large, fleshy and red = '**hypergranulation tissue**' ⇒ a foreign body or a pocket of pus (usually under the edge).



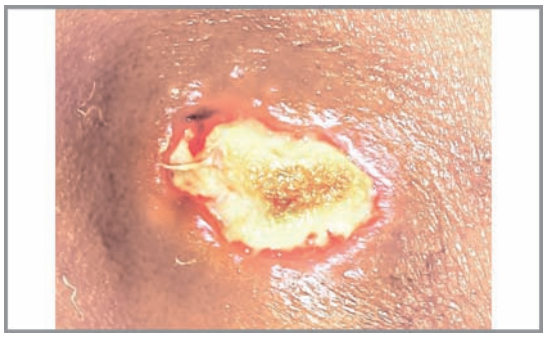
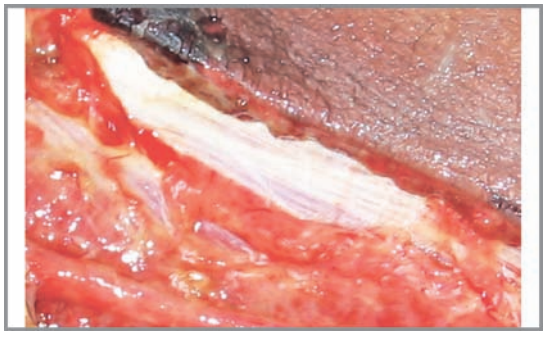
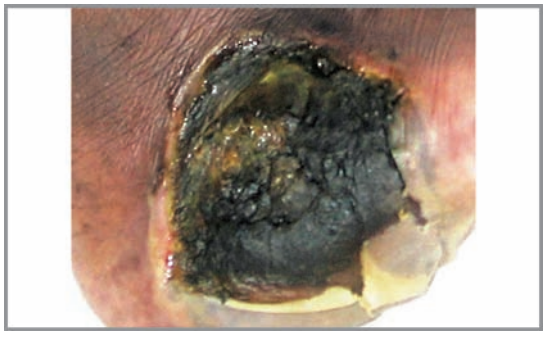
Fig 42-1 Healthy granulations

Slough = a piece of dead soft tissue (getting separated from the body).

Slough has structureless appearance. It is insensitive to touch.

The presence of slough ⇒ (a) essentially a deep ulcer, and/or (b) sepsis.

www.Ebook777.com

Color of the slough	*Composition/cause of the slough*	*Examples*
Pale yellow	Fat Fig 42-2	
Pale gray	Fat/muscle Fig 42-3	
Whitish	Deep fascia/tendon Fig 42-4	
Black	Seen in arterial/venous gangrene Fig 42-5	

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

Color of the slough	*Composition/cause of the slough*	*Examples*
Khakee	Seen in gas gangrene Fig 42-6	

'***Eschar***' = a slough produced by a burn, or by gangrene.

Cauliflower appearance ⇒ squamous-celled CA.

- **Edge**?

'Edge' = the junctional area between the margin and the floor.

'Margin' = the linear junction between the surrounding area and the edge.

Edge has a very special surgical importance because it is the most active part of an ulcer; 'activity' with reference to destruction.

Basically, an ulcer means destruction. Different pathological conditions cause ulcers by different modes of tissue destruction, which in turn result in different types of edge of various ulcers. Therefore, careful examination of the edge of an ulcer can give an idea about the pathological process responsible for ulceration in the given case, *wrt* the stage of the ulcer, and the nature of the disease.

Types of edge: sloping, inflamed/edematous, undermined, punched out, raised and everted, and raised and beaded.

Sloping edge is seen in healing ulcers.

Three zones of the sloping edge can be recognized: The innermost red—granulation tissue covered by a single layer of epithelium, the middle bluish—due to 2–3 layers of epithelium, and the outermost whitish—due to much thicker layer of epithelium. This centripetally gradually decreasing number of layers of epithelium gives the characteristic sloping appearance to the edge.

Inflamed (*reddened and edematous*) edge is seen in spreading ulcers.

Undermined edge suggests that destruction of deeper tissues is more than that of the skin (/mucosa).

Examples: 1. Tuberculous ulcer (thin and bluish edge). Mechanism: Most of the times a tuberculous ulcer results from bursting of a 'cold' collar-stud abscess. The opening in the skin is much smaller than the tissue destroyed by the abscess underneath.

2. Bed sores. Mechanism: Fat is more susceptible to pressure necrosis than skin, and therefore destroyed over a larger area.
3. Often, some portion of the edge of an infected ulcer (ulcer in a spreading stage) may be undermined due to destruction of fat in the respective portion.
4. During the initial stage of evolution of a neuropathic ulcer, the edge is often undermined.
5. Other examples of ulcers having undermined edge: tropical ulcer (undermined + slightly raised), chancroid (soft sore), typhoid ulcer, and amoebic ulcer.

Clinical methods to determine undermined edge:

a. Place a finger near the margin; press and pull the margin away firmly—the edge gets raised.
b. By probing.

Punched out edge is so called, because the ulcer looks as if it has been cut out by a punching machine.

Examples: established neuropathic ulcer, established bed sore, ulcer following sloughing off of gangrenous skin, dermatitis artefacta.

www.Ebook777.com

Raised and everted edge ⇒ squamous celled CA.

If there is a raw area projecting above the level of the surrounding skin surface, then there are 3 possibilities which need to be differentiated—a pyogenic granuloma, an ulcerated (benign) tumor or a cyst, or a squamous-celled CA.

Pyogenic granuloma	*Ulcerated (benign) tumor / cyst*	*Squamous-celled CA*
Essentially pedunculated. (The pedicle may be narrow or broad)	The side of the projection is covered by skin.	The entire surface of the projecting area, i.e. the top as well as the sides, is raw.
		

Raised and beaded edge ⇒ basal-celled CA.

The edge of a basal-celled CA can be raised and everted, if it has undergone squamous metaplasia.

Edge **may be *unremarkable*** particularly in chronic indolent ulcers.

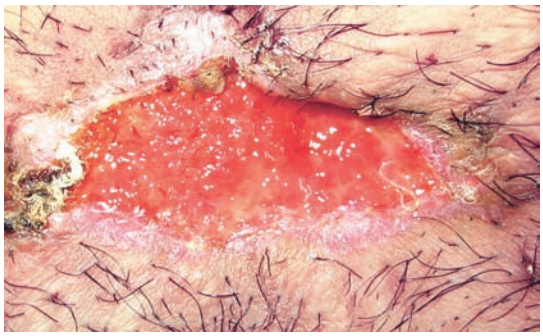


Fig 42-7 Sloping edge



Fig 42-8 Inflamed edge



Fig 42-9 Undermined edge (TB in this case)

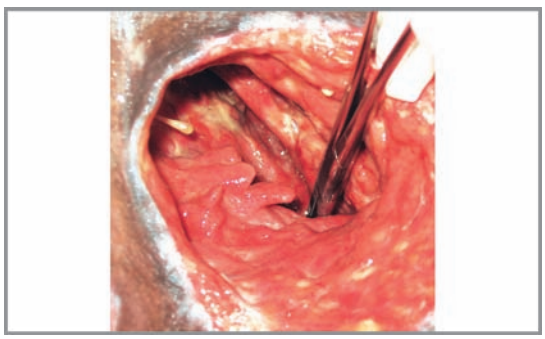


Fig 42-10 Punched out edge. The vertical edge makes the ulcer appear like a well. ~ half of a 6″ long artery forceps dipped in (- that gives an idea of the depth of the bed sore in this photograph)

Free ebooks ==> www.Ebook777.com



Fig 42-11 Raised and everted edge: SCC



Fig 42-12 Beaded edge: BCC

- **Color** (of the margin and the surrounding area close to the margin)?
  - Red—in acute inflammation.
  - Faint bluish—in TB.
  - Pale white—in a chronic ulcer in general.
- **Surrounding area**?
  - Edema. – edema may have many causes e.g. inflammation, CVI, causes responsible for generalized edema which may coexist with CVI, e.g. CHF, renal disease, hypoproteinemia, etc.
  - Cellulitis ⇒ acute inflammation.
  - Shiny, hairless and thin ⇒ ischemia.
  - Gangrenous (/ dark) skin ⇒ ischemia.
  - Ankle venous flare ⇒ grade 1 CVI.
  - Hyperpigmentation. Causes – TB, venous ulcer (grade 2 CVI).
  - Depigmentation ⇒ venous ulcer (grade 2 CVI).
  - Stasis dermatitis* ⇒ venous ulcer.
  - Lipodermatosclerosis† ⇒ very long-standing venous ulcer.
  - White ⇒ fibrosis ⇒ chronicity.
  - Scarred. Causes of scarring: TB, ulcer having undergone multiple cycles of breakdown and healing such as venous ulcer etc.
  - Signs of rheumatoid arthritis / vasculitis / DM.

Look for **any finding s/o etiology, complication, and causes of chronicity**.

PALPATION

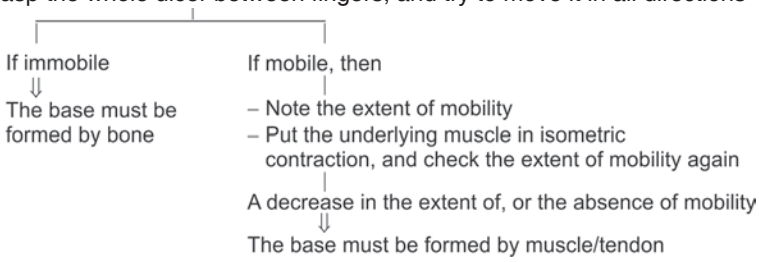
- **Temperature**?
  - Raised temperature ⇒ acute inflammation.
  - Decreased temperature ⇒ ischemia.
- **Tenderness**? ⇒ acute inflammation.
- **Mobility**?
  Grasp the whole ulcer between fingers, and try to move it.
  - Superficial and benign ulcers are usually mobile.
  - Fixity ⇒ a chronic ulcer, the base of which is formed by bone, OR infiltration of surrounding and deeper tissue in c/o a malignant ulcer.
- **Base**
  Base is the anatomical tissue on which the ulcer rests.
  Base is not visible except in perforating ulcers.
  In painful acute ulcers, it may be difficult to determine the base due to tenderness (which precludes grasping of the ulcer).

* Stasis dermatitis = chronic eczematous dermatitis, which initially involves medial aspect of leg just superior to the medial malleolus, and later, may involve the entire leg. Occurs in CVI.
Eczema = non-contagious inflammatory disease of the skin, characterized by erythema, edema, vesiculation, oozing, weeping and crusting.
† Lipodermatosclerosis = thick hyperpigmented skin with brawny edema of the skin and subcutaneous tissue.

www.Ebook777.com

Clinical methods to determine the base:

1. Grasp the whole ulcer between fingers, and try to move it in all directions



2. If the ulcer is big and cannot be grasped between fingers, then the base may be palpated with a sterile gloved finger across the floor, and then the related underlying muscle is put in isometric contraction as usual.

   Feeling of movement of the muscle and the contracting muscle becoming hard suggests that the base must be formed by muscle.

   Clinical significance of the 'base' :

   Basically, the base denotes the **depth** of the ulcer, **and activity** of the ulcer **in relation to the deeper tissues**.

   a. Depth of an ulcer is one of the loco-regional clinical points, which can give an idea about the **'healing potential'** of an ulcer. Deeper the ulcer, delayed the healing process.

   Classification of ulcers depending on the 'depth':

   (i) *Superficial* = *Shallow* = ulcer confined to the skin (/mucous membrane) only. Base is formed by subcutaneous (/submucous) tissue. Examples – apthous ulcer, herpetic ulcer, etc.

   (ii) *Deep* = involvement of skin and at least the subcutaneous tissue. Base is formed by at least the deep fascia (or equivalent, like Scarpa's fascia).

   (iii) *Perforating* = the base is formed by bone, which itself may be destroyed (i.e. destruction of tissue reaches right down to the bone). Classical example: neuropathic ulcers due to diabetic neuritis, alcoholic neuritis, leprosy, etc.

   b. **Base may extend** in the deeper tissues **beyond** the periphery of **visible** raw area as in a malignant ulcer. Therapeutic implication of this fact: While performing a 3-dimensional wide excision, adequacy of clearance should be judged on the basis of the *palpable* induration *rather than the visible* abnormality.

- **Consistency**—of the edge, the area immediately surrounding the ulcer, and the base; and inferences therefrom?
  - Induration of varying degree ⇒ chronicity.
  - Marked induration amounting to (stony) hardness ⇒ squamous-celled CA.
  - Bony hard ⇒ the ulcer is resting on bone, e.g. a chronic venous ulcer on the tibia, perforating ulcer on the calcaneus/head of a metatarsal.
  - A tuberculous ulcer lacks induration in spite of being chronic.
- (Easy) **Bleeding on touch** occurs in a healing ulcer or a malignant ulcer.
- **Surrounding area**?
- **Underlying bone/joint**?

REGIONAL EXAMINATION

- Examine for neurological deficit.
- Examine for arterial deficit.
- Examine for varicose veins, CVI.
- Examine the regional lymph nodes.

Free ebooks ==> www.Ebook777.com

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. In a small chronic ulcer, if discharge is profuse, then the lesion could be a 'sinus' rather than an ulcer.
2. Shape of a Marjolin's ulcer may be regular.
3. Floor of an ulcer may not be formed uniformly by the same type of tissue, especially when the ulcer is large. It is quite often possible for islands of slough as well as granulations to be present in the same ulcer. In such a situation whether to declare the ulcer as essentially infected or healing, will depend on the proportion of slough and granulations, coupled with other clinical criteria, and comparing the findings with findings on the previous days.
4. It is possible to have two different types of edges in different portions of the same ulcer, for example, edematous and sloping, edematous and undermined, sloping and undermined. A clinical conclusion should not be based only on one finding, without reviewing other physical findings.
5. The lack of induration suggests TB.
6. Ulcers in leprosy are often neuropathic rather than a true infection.

Causes of ulcer

A. Trauma: Mechanical (including self-inflicted (*dermatitis artefacta*)) / electrical / thermal / radiational.

B. Toxin: – Plant/animal poison (i.e. biological toxin).
– Domestic/industrial chemical.

C. Unrelieved pressure: – Tight band / ring / plaster, etc.
– Bed-ridden / chair-ridden position with unrelieved pressure on certain bony prominences.

D. An ulcer following I and D of an acute pyogenic abscess, or an ulcer resulting from infection of an operative wound is called **secondary infective ulcer**.

E. (Primary) **Infection / Infestation**

– Nonspecific infections often cause spreading ulcers in diabetic patients, immunocompromised patients. nonspecific infections may also cause chronic ulcers.
– Specific infections/infestations: TB, actinomycosis, blastomycosis, syphilis, fungal (sporotrichosis etc.), dermal leishmaniasis, guinea-worm.

F. Neuropathy: Peripheral neuritis (DM, leprosy, chronic alcoholism), peripheral nerve injury, spinal cord injury/tumor, syringomyelia, tabes dorsalis.

G. Vasculopathy: – Peripheral arterial occlusive disease.
– Vasculitis.
– Chronic venous insufficiency.

H. Gravitational ulcer: The exact cause is not known, although CVI plays an important role.

I. Tissue devitalization: – Sickle cell and other anemias.
– Edematous tissue (filariasis, hypoproteinemia, etc.).
– DM.
– Deficiency diseases (like nicotinic acid, folic acid → ulcers in mucous membrane).
– Nephritis.
– Iatrogenic extravasation of an irritant IV medicine/fluid.

J. Specific dermatological / systemic diseases.

K. Miscellaneous: Tropical ulcer, Bazin's ulcer, Martorell's ulcer, etc.

L. Primary/secondary cancers.

Regional ulcers

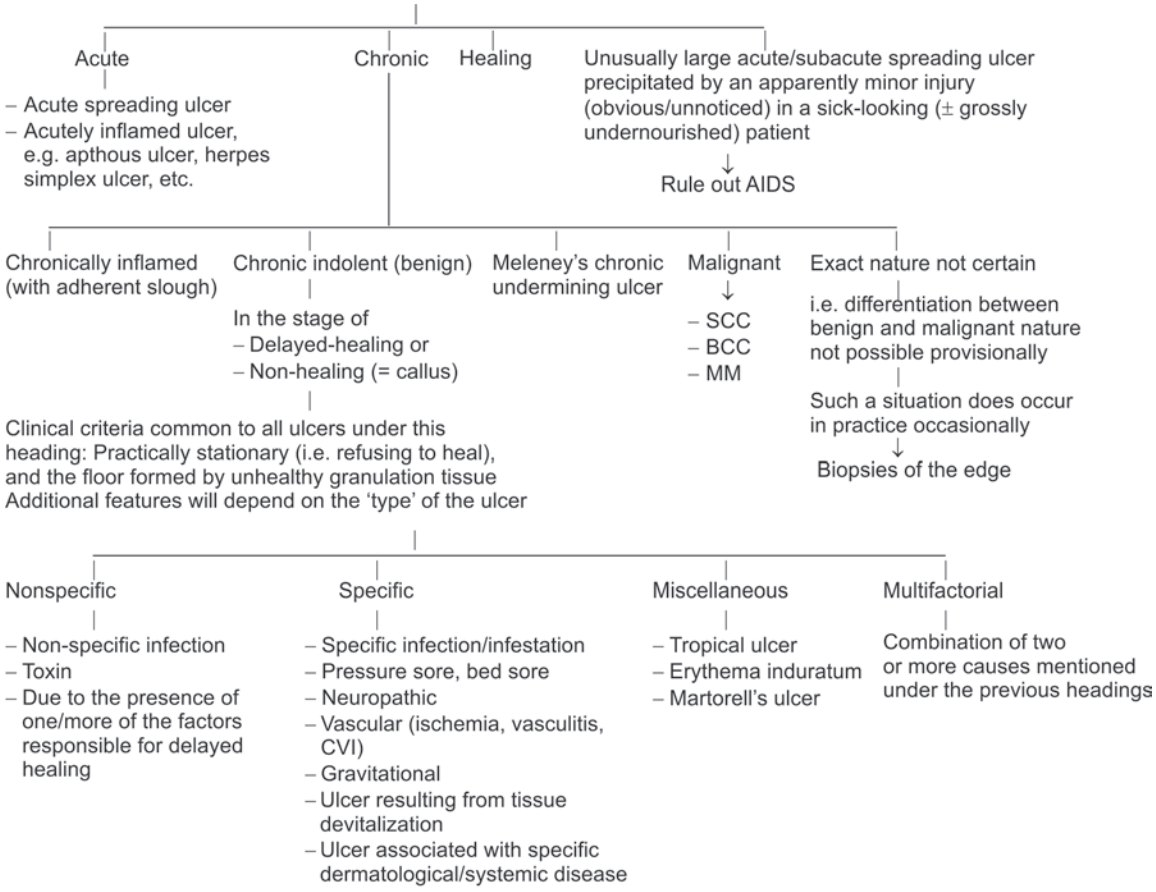
Certain types of ulcer are prone to occur in specific locations in the body.

Foot: – Traumatic, Nonspecific.
– Fungal.
– Neuro/Vascular.
– Irritable ulcer on the ankle, nerve terminal being exposed.
– Malignant melanoma.

www.Ebook777.com

Leg: – Traumatic, Nonspecific.
– Fungal.
– Vasculitic, venous, lymphatic.
– Gravitational.
– Tropical.
– Erythema nodosum, erythema induratum (Bazin's ulcer).
Oral cavity: Refer to chapter 51.
Penis: – Primary chancre, chancroid, balanoposthitis.
– Leukoplakia, malignant ulcer.
Nipple and areola: – Simple crack (fissure).
– Eczematous ulcer.
– Paget's disease.
– Ulcerated benign or malignant tumor.

CLINICAL STAGE AND POSSIBLE CAUSE OF ULCER



Factors responsible for delayed healing/non-healing of a chronic ulcer in general (ignoring the specific causes which in themselves are responsible for chronicity even otherwise)

A. GENERAL
– *Old age* (reduced protein turn-over, arteriosclerosis).
– *Malnutrition - including anemia, deficiency of vitamins and trace elements*.
- Vitamin 'A' is essential for epithelialization.
- Vitamin 'C' is essential for synthesis of ground substance and maturation of collagen.

Free ebooks ==> www.Ebook777.com

- Vitamin 'D' is essential for new-bone formation.
- Important trace elements: Zn, Ca, CO, etc.

– *Metabolic* disorders like DM, jaundice and gout.
– *Steroids, CT*.
– *Immunosuppression*.

B. LOCOREGIONAL

– (*Exceedingly*) *Long duration in itself may become a cause for non-healing*. *Mechanisms* : 1. Fibrosis of the edge. 2. The base too becomes fibrotic. And even if the ulcer sometime may appear to have healed, it is with an avascular scar tissue which is unstable and likely to break down again with minor trauma.
– *Repeated trauma,* e.g. due to sensory deficit (DM, nerve injury, etc.), scratching, rough handling during C and D.
– *Lack of rest* often due to unavoidable socio/economic pressures.
– *Persistent pressure* as in pressure sores.
– *Persistent (almost unavoidable) cause for infection,* e.g. smearing of a bed sore by urine/feces.
– *Site*: - Overlying a joint → delayed healing due to the lack of rest.
  - Base formed by a bone particularly devoid of periosteum.
– *Size*: Large size (> 5 cm in diameter). In large ulcers which are often chronic, the edge may become lined with skin i.e. the skin may get fixed to the base (e.g. bone), and then the edges cannot move towards each other, which in normal course happens by a physiological process called 'wound contraction'.
– *The presence of* a foreign body or a pocket of pus (usually under the edge) or sequestrum (in chronic osteomyelitis).
– *Deep or perforating ulcers* : Examples—neuropathic ulcer, bedsore etc.
– Surrounding *edema*.
– Vascular *insufficiency* (PAOD / CVI / lymphedema).
– *Improper technique of C and D*.
– *Irritation by drugs in the dressings / by discharge from the ulcer itself*.

Complications of an ulcer
Examples—osteomyelitis, telipes equinovarus, etc.

Any other associated disease—closely related/otherwise?

DIFFERENTIAL DIAGNOSIS

Healing ulcer

– Painless.
– Regressing in size.
– Minimal disturbance of function of the part of the body bearing the ulcer.
– Shape—regular.
– Discharge—thin watery film.
– Floor shows healthy granulations.
– Edge—sloping. The edge may bleed easily on touch (such as while changing the dressing) during the early stages of healing.
– Surrounding area—slightly edematous.
– No tenderness.

Acute spreading ulcer

– Painful.
– Rapidly progressing (over days).
– Marked disturbance of function of the part of the body bearing the ulcer.
– Purulent discharge (may be foul-smelling).
– Shape—irregular.
– Floor shows slough. Slough is black-colored (eschar) if the ulcer is caused by vasculitis.

www.Ebook777.com

– Edge—inflamed (reddened and edematous).
– Surrounding area shows cellulitis.
– Local temperature is raised.
– Tenderness is present.

Causes: acute nonspecific infection, DM (in practice, rule out diabetic ketoacidosis), vasculitis including pyoderma gangrenosum, etc.

Pyoderma gangrenosum

= destructive, non-infective ulceration of the skin which results from venulitis.

– The condition starts as tender erythematous nodules which undergo ulceration and is usually painful.
– Edge is bluish, usually raised and thickened, but sometimes may be ragged and undermined.
– Common site – lower limbs.
– May be associated with RA, ulcerative colitis, Crohn's disease, and myeloproliferative diseases.

Ulcerative form of cutaneous amoebiasis

Rapidly extending serpiginous phagedenic ulceration with a serrated margin and an undermined edge.
Pus oozing out from ulcers may contain parasites in large numbers.
Extensive ulceration may be mistaken for progressive bacterial cutaneous gangrene.

Chronically inflamed ulcer

Possible reasons responsible for chronicity: Inadequate debridement (including inadequate drainage of deep extensions or secondary tracts), improper or/and inadequate antibiotic treatment, inadequately controlled DM etc.

Basically starts as an acute spreading ulcer or infected wound, which attains chronicity (i.e. duration > 3 weeks).



Fig 42-13 Pyoderma gangrenosum



Fig 42-14 Close-up view of Fig. 42-13



Fig 42-15 Chronically inflamed ulcer

Free ebooks ==> www.Ebook777.com

Differentiating points:
- Painful.
- Purulent discharge.
- Adherent slough.

Chronic indolent ulcer
- Duration—in months or years.
- Stationary.
- Discharge—sanguinous or thin purulent.
- Disturbance of function—variable.
- Shape—variable, although regular.
- Floor—unhealthy (and often nodular) granulations.
- Edge—may be unremarkable especially if the ulcer is superficial.

A chronic indolent ulcer is said to be delayed healing because the floor is formed by unhealthy granulations. As chronic indolent ulcers are known to break down due to even minor trauma after healing has been achieved, care of the part (often the foot) and preventive measure are of immense importance to prevent recurrence.
A non-healing ulcer is also called callus ulcer as it practically completely refuses to heal, e.g. a gravitational ulcer.
Criteria of a callus ulcer: – Duration in years.
– Marked induration in the base, edge, and the surrounding area of the ulcer. After a while, fibrosis itself becomes one of the additional factors responsible for non-healing.

Chronic nonspecific ulcer
Basically, a chronic indolent ulcer.
Differentiating points
- Progress—initially rapid, then stationary. Regression ±.
- Shape—irregular.
- Discharge—thin pus.
- Surrounding tissue—slightly edematous.
- Base—slightly indurated.
- Regional lymphadenitis—non-tender and discrete.

Tuberculous ulcer/s
Basically, chronic indolent ulcer/s.
Differentiating points
- Shape—irregularly oval.
- Size—~ 2 cm in diameter.
- Floor consists of thin pale granulations simulating 'apple jelly'.
- Edge—faint bluish and undermined.
- Surrounding area—hyperpigmented / scarred.
- Base—not indurated.
- Depth—'deep'.

Supporting points
- Multiple.
- Situated in the typical site prone for tuberculous lymphadenitis viz. neck > axilla > groin in the descending order of occurrence; and the ulcer/s seem to have occurred in the course of a natural history of tuberculous lymphadenitis on bursting (or I and D) of cold abscess.

Lupus vulgaris
Scrofuloderma } Refer to chapter 15.

www.Ebook777.com

Leprous ulcers
In fact these ulcers are ulcerated swellings.
– Sites—dorsum of fingers and toes and sometimes over other non-weight-bearing areas.
– Floor is covered with pale granulations.
– Smears may show plenty of AFBs.
Notes
Ulcers occurring in a leprosy patient are called 'leprotic' ulcers.
Leprotic ulcers are of two types:
1. Neuropathic—Nonspecific ulcers occurring under pressure points commonly on the soles.
2. Leprous—Specific ulcers resulting from breakdown of cutaneous nodules in patients with uncontrolled lepromatous leprosy. Discharge is infective. Complications: Secondary amyloidosis, CA.

Etiology, clinical features and management is different in both these types of leprotic ulcers.

Actinomycotic ulcer/s
Basically, chronic indolent ulcer/s.
Differentiating point: The floor shows sulfur granules.

An ulcer following **guinea worm infestation**
Basically, (often) a known c/o guinea worm infestation.
– Initially copious discharge of sero-sanguinous fluid; subsequently discharge becomes scanty.
– Edge – punched-out.
– Base – soft.

Dermal leishmaniasis
– No constitutional symptoms.
– Situated on exposed parts of the body.
– Single / multiple.
– 4–8 cm in diameter.
– Crater in the centre and granulation tissue at the periphery covered by scab.
– Surrounding skin shows red indurated rim.

Primary syphilitic sore
Develops at the site of inoculation of the treponemes – penis, vulva, lip, tongue, nipple, rectum, etc.
– Sexually active adult of either gender.
– Incubation period—3–4 weeks.
– No constitutional symptoms.
– Starts as an indurated papule, subsequently erodes to form a painless ulcer.
– Usually single, round/oval, 1–2 cm in diameter.
– Thin colorless serous discharge.
– Floor – often covered with crust; pink granulations are apparent on removing the crust.
– Raised hyperemic edge sloping towards the center where there is a shallow ulcer.
– Surrounding area shows a dusky red edema.
– Base is indurated simulating the feel of a button.
– May simulate other penile/vulval lesions – traumatic like fissures/tears/ burns, chancroid, herpes genitalis, furuncle, carcinoma, balanoposthitis and lymphogranuloma venereum.
– Lymphadenopathy.

While clinically examining the patient, the prepuce must always be fully retracted as otherwise tiny sores in the coronal sulcus may be missed.
T. pallida can be demonstrated in the discharge by dark-field microscopy.



Fig 42-16 Primary syphilitic sore on the penis

Free ebooks ==> www.Ebook777.com

Ulcer may resolve at any stage of its development and thus be quite atypical.
The serum tests do not become +ve for up to a month after the appearance of the sore, hence initial negative results must not be interpreted as excluding primary syphilis. The tests should be repeated for up to 3 months where doubt persists.

Gumma (Tertiary Syphilitic ulcer)
Time of development – from 6 months up to many years after inoculation of the infection.
- Age—40–60, of either gender.
- Duration—in months.
- Progress—slow. Regression ±.
- Discharge—dry, thick, and foul-smelling.
- Painless.
- Single / multiple.
- Site—overlying subcutaneous/submucous bones (skull, palate, sternum, clavicle, scapula, ulna, tibia), SCM muscle, tongue, breast, testis.
- Shape—circular or serpentine.
- Size—1–2 cm in diameter.
- Floor consists of wash leather (yellow-gray colored) slough. Floor is moderately depressed.
- Edge—punched-out.
- Surrounding area shows coppery red pigmentation, and is slightly edematous.
- Base—indurated.
- No regional lymphadenitis.

Healing of the ulcers leave behind a 'tissue-paper' scar.

Pseudomalignant form of cutaneous amoebiasis
Slowly progressive papillary lesions with ulceration: – Firm consistency.
– Irregular heaped up border of hard consistency.
Clinically, may be mistaken as a malignant lesion.

Bed sore (Pressure ulcer)
- Sites: Bony prominences subject to unrelieved pressure—sacrum, iliac crest, ischial tuberosities, greater trochanters, scapulae and occipital region.
- Fever suggests infection, osteomyelitis.

Assessment should include overall physical and mental health, social support and risk of recurrence, as follows:
- Co-morbid conditions:
  - Old age (reduced protein turn-over, arteriosclerosis).
  - Malnutrition—including anemia, deficiency of vitamins and trace elements.
  - Metabolic disorders like DM, jaundice and gout.
  - Immunosuppression.
  - Collagen vascular disease.
  - Cancer.
- Psychosis, depression, learning ability.
- Social support, life-style.
- Risk of recurrence with respect to curability of the primary condition which predisposed to the development of bed sore, and one/more of the above-mentioned factors.

Early warning signs before the development of an established (open) bed sore:
- Itching or pain in the skin (if sensation is preserved).
- Color-change: - Red, blue or purple on darker skin.
  - Red on lighter skin. Erythema blanches with pressure, especially in patients with risk factors.
- Temperature: ↑/↓.

www.Ebook777.com



Fig 42-17 Neuropathic ulcer (in evolution)

Risk factors:
- Decreased mobility / Immobility.
- Decreased sensation.
- Fecal / urinary incontinence.
- Poor nutritional status.

Neuropathic ulcer (*Syn*. Neurotrophic ulcer)
Basically, a chronic indolent ulcer situated under an abnormal pressure point in patient suffering from peripheral neuritis (DM / leprosy / chronic heavy alcoholic) / peripheral nerve injury / spinal cord injury/tumor / syringomyelia / tabes dorsalis.
Differentiating points:
- Pain—absent / mild.
- Discharge—scanty / almost absent. Purulent foul-smelling discharge ⇒ gross secondary infection.
- Shape—often circular. Size—1–2 cm in diameter.
- Floor consists of grayish white slough or unhealthy granulations.
- Edge—undermined (during evolution) or punched-out (in established case).
- Base—initially formed by the flexor tendon; subsequently by the bone.
- A judgment of the extent of proximal spread of infection can be obtained by gently probing the ulcer with a (sterile) probe.
- Surrounding area: - The area immediately surrounding the edge – thickened (keratotic).
  - Rest of all the surrounding area beyond the perimeter of callosity is essentially normal.

Notes:
The skin over the abnormal pressure points on the sole becomes particularly thick and prone to flaking (hyperkeratosis). A minor abrasion may become infected, and track deep and silently over a wide surrounding area without this being obvious through the thickened overlying skin. When grossly infected, (surgical) drainage must be complete to be effective, and the whole abscess cavity must be deroofed. The extent of spread can be accurately determined by probing, and the overlying skin cut away.
There are 2 mechanisms of development of neuropathic ulcer—extrinsic and intrinsic—and they work either in isolation or in combination. It is beyond the scope of this book to discuss the pathogenesis of neuropathic ulceration by extrinsic and intrinsic mechanisms (Please refer to the textbook 'Essential Surgical Practice: Higher Surgical Training in General Surgery, 4th edition, 2002).

Ischemic indolent ulcer primarily due to a PAOD ± additional factor/s responsible for chronicity
Basically, a chronic indolent ulcer situated on the tip of finger/toe, or situated on the leg; may have been preceded by severe burning pain.
Differentiating points:
- Discharge—scanty / almost absent.
- History and physical findings suggestive of a chronic PAOD (Refer to chapter 46).

Free ebooks ==> www.Ebook777.com



Fig 42-18 'Claw foot' deformity resulting from intrinsic neuropathy (The 2nd toe was surgically amputated for microvascular gangrene)



Fig 42-19 Vasculitis ulcer

Notes:

- An ulcer is qualified for an ischemic ulcer when there is insufficient tissue perfusion due to arterial insufficiency, irrespective of the original cause of the ulcer.
- In an individual with an ischemic lower limb, an ulcer at a pressure point or at a site of recent trauma may reasonably be assumed to be ischemic in nature, although logically there are other possibilities.
- Ulceration and gangrene are the end result of many disease processes other than ischemia, so that their role as evidence of ischemia must not be accepted uncritically.
- An ulcer in an ischemic limb may be initiated by one of the two mechanisms: 1. Trauma, which can naturally occur at any site. 2. Simple (persistent) pressure over an area such as the ankle malleoli where the bone is subcutaneous. Such pressure commonly arises in bed-bound patients.

  Secondary infection originating at the site of ulcer can worsen the ischemia: Tissue edema (due to infection-inflammation) $\rightarrow \uparrow$ in interstitial pressure, which may rise higher than the low capillary perfusion pressure of a severely ischemic limb leading to thrombosis of blood in microcirculation, and the effect can spread proximally to the small arteries.

Vasculitis

Vasculitis is characterized by inflammation within the blood vessel wall.

This inflammatory process may involve many blood vessels and consequently organ systems.

The clinical features result from ischemia to the tissues supplied by the damaged vessels and are often accompanied by the constitutional systemic features of anorexia, malaise, fever, and weight loss which result from widespread inflammation. These conditions produce a range of symptoms from a mild obliterative disorder to necrotizing vasculitis.

While cutaneous vasculitis, RA vasculitis and vasculitis associated with CTDs affect mainly the microvasculature, vasculitis affecting the larger vessels must be considered in cases of unexplained vascular pathology.

The vasculitides represent a clinical challenge for many disciplines.

Venous ulcer, primarily due to a CVI (mostly **post-thrombotic**)

Basically, a chronic indolent ulcer situated in the medial malleolar region, that is presumed to be wholly or partly due to venous disease.

Edge may be unremarkable.

Differentiating points:

- Ulcer situated in the medial malleolar region.
- Margin may be irregular. Size—usually 2–3 cm in diameter, but may be larger.
- Surrounding area may show one/more of the following features:
  - Edema. Causes responsible for generalized edema may coexist.
  - Ankle venous flare.

www.Ebook777.com

- Areas of hyperpigmentation or depigmentation.
- (Stasis) dermatitis.
- Lipodermatosclerosis.

– Floor—sloughy, or consists of (unhealthy) granulations. A necrotic floor suggests an alternative diagnosis or co-existing pathology like arterial disease.
– Depth—Usually deeper especially if long-standing.

Notes

Chronic PAOD, DM, and RA often co-exist in patients with CVU.

Ulcers that are resistant to treatment, are multi-factorial in origin, and sustained healing will only be achieved once all the aetiological factors have been addressed.

(True) Varicose ulcer

Basically, a chronic indolent ulcer situated on the gaiter area (i.e. medial aspect of lower half of the leg) / lateral aspect of the ankle, that is presumed to be wholly or partly due to venous disease. Varicose ulcers above the gaiter area are unusual.

Edge may be unremarkable.

Differentiating points

– Vertically generally oval (margin may be irregular).
– Size—variable.
– Floor—sloughy, or consists of (unhealthy) granulations. A necrotic floor suggests an alternative diagnosis or co-existing pathology like arterial disease.
– Depth—'shallow', however may become deep if the ulcer is very log-standing.

Gravitational ulcer

– Duration—very long-standing. Nevertheless, in the initial period, h/o multiple cycles of healing and breakdown ±.
– Site—gaiter area, or one-third of the way up from the lateral malleolus on the later aspect of the leg.
– Shape—vertically oval / irregular. However, in severe cases the medial and lateral ulcers may merge to form a circumferential ulcer.
– Floor—covered by slough, although may be interspersed with islands of pale granulations.
– Base—usually indurated due to chronicity.

Etiopathology:

Actually the cause is not known. However, venous stasis may play a significant role.

Ulcer/s associated with sickle cell anemia

– Situated on the legs above the malleolus.
– Closely resemble venous ulcers.

Iatrogenic ulcer, due to extravasation of …….

– Definite h/o extravasation of an irritant (painful) IV fluid / medicine f/b development of an acute spreading ulcer, which may attain chronicity.
– The ulcer may be small/large, superficial/deep.



Fig 42-20 Post-thrombotic venous ulcer



Fig 42-21 Gravitational ulcer

Free ebooks ==> www.Ebook777.com

Radiational ulcer
Basically, a chronic indolent ulcer with a h/o RT.
The ulcer is surrounded by an area of pigmentation and dermatitis.

Tropical ulcer
– To begin with, acute, nonspecific localized necrosis of skin and subcutaneous tissue, which is endemic in tropical countries.
– *Natural history* : 1. May rapidly extend to involve deeper tissues (muscle, bone). OR 2. Heals after a long period leaving a parchment-like pigmented scar.
– Occurs in young men. Almost always occurs on the lower extremity.
– Symptoms: Pain, and constitutional symptoms.
– Usually solitary.
– Foul-smelling purulent discharge / Copious sero-sanguinous discharge.
– Floor consists of pale dirty slough.
– Edge—raised and undermined.
– Surrounding skin—edematous.

Acute stage may be followed by chronic stage. The ulcer may refuse to heal and retain practically the same size for months/years.
– Floor consists of unhealthy granulations.
– Surrounding skin—atrophic (thin) and depigmented.
– Edge—firm and sclerotic.
– Base—indurated.

Notes:
Etiology: Not clear. Usually follows trauma, often trivial.
Organisms found fairly constantly: *Fusiform bacilli* and *Borrelia vincenti*.
It is possible that some unidentified organisms may be involved.

Erythema induratum—due to localized areas of fat necrosis.
– Adolescent girls.
– Symmetrical purplish nodules occur, especially in the calves, and gradually break down to form indolent ulcers; heal by leaving behind pigmented scars.

Meleney's chronic undermining ulcer
Results from infection by a microaerophilic streptococcus.
– Usually develops after surgery on the intestinal and urogenital tract.
– The infection and necrosis start in the subcutaneous tissues and progressively deepens to involve the pelvis.

A malignant ulcer/ulcerated swelling, most probably **squamous-celled carcinoma**, clinical stage …...
Basically there are two patterns of growth, although these can overlap – protuberant type and infiltrative type.
Differentiating points common to both:
– Offensive discharge.
– Dirty necrotic floor.
– Raised everted edge.
– Stony hard induration of the edge and at the base.
– May bleed easily on touch.
– Regional metastatic lymphadenopathy after about three months.

1. *Protuberant type* : Raised cauliflower-like ulcerated swelling.
2. *Infiltrative type* : Begins as a warty nodule; breaks down to form a progressively enlarging ulcer.

Supporting points
– Middle age.
– Site: - Lip, tongue, vulva, penis, anus.
  - Old scar, area of radiation burns, sun-exposed areas in fare-skinned patients (including sites of actinic keratosis), site of the bite by an elapid snake may be years earlier.

www.Ebook777.com

Marjolin's ulcer
= a squamous-celled carcinoma developing in an old scar that is often *unstable* (i.e. the one which repeatedly breaks down and heals).
Differentiating points
– An ulcer occurring in an unstable chronic scar.
– Hardening and cracking of skin are usually the first indications of malignant change.
– Shape – irregular **or regular**. Size – variable.
– Edge – unremarkable (as the ulcer is shallow). The absence of typical raised and everted edge does not exclude malignant nature.
– Base – indurated.

Regional metastatic lymphadenopathy does not occur as long as the ulcer is confined to the scar. Reason – the lymphatics in the scar have been destroyed. Regional metastatic lymphadenopathy may occur only if and after the ulcer has spread to the normal tissue.

Basal-celled carcinoma
Malignant melanoma
} Refer to chapter 15.

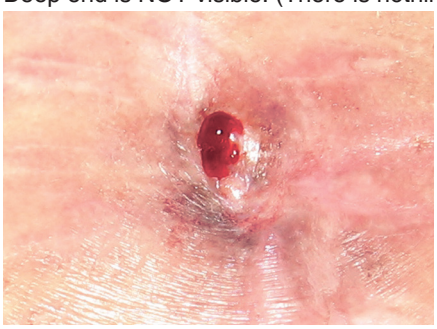
Free ebooks ==> www.Ebook777.com

43 SINUS / FISTULA

Sinus means a blind tract leading from a surface down into a tissue, and lined by granulation tissue, fibrous tissue or epithelium. This definition belongs to a pathological sinus, and the same is used in clinical practice too.
In Anatomy, the word 'sinus' is used to denote a space e.g. cavernous sinus, sagittal sinus, paranasal sinuses, laryngeal sinus etc.
Fistula means a communicating tract between a hollow viscus and exterior, or between two hollow viscera, and lined by granulation tissue or epithelium. A fistula is always pathological. Unlike anatomical sinuses, there are no anatomical fistulae in postnatal life. However, fistulae do occur normally in fetal life, fulfilling developmental or physiological needs; e.g. V-I duct, urachus, ductus venosus (transferring placental blood to IVC, bypassing the liver), ductus arteriosus (transferring fetal blood to aorta, bypassing the lungs).

GENERAL REMARKS

1. A sinus cannot always be easily differentiated from a fistula clinically, as the internal opening of a fistula may be difficult to demonstrate.
2. **Sinus *v/s* ulcer**

Sinus	*Ulcer*
Deep end is NOT visible. (There is nothing like a floor.)  Amount of discharge is out of proportion to the size of the visible raw area (the opening), and is often profuse. Pressure on surrounding tissue often brings out some discharge.	Floor is readily visible. 

3. The mouth of a sinus / fistula can be a very small feature which may lead to a false sense of underestimation of the underlying pathology.
4. A sinus **often leads to** an abscess cavity.
5. The **key to the correct treatment** of a sinus is detection of any associated deep abscess cavity or complex deep extensions. Failure to do this will result in a recurrence at the same or adjacent site.

www.Ebook777.com

6. **Classification** of fistulae in general: (**A**)

External	Internal
Involve the skin *Usual cause*: as a complication of surgery viz. unrecognized injury to a viscus, leakage of anastomoses *Common cause other than the above*: primary abscess formation from intestinal pathology, subsequently discharging through the skin, e.g. anorectal suppuration f/b anorectal fistula *Common complications*: (a) Excoriation of skin and digestion of abdominal wall—particularly serious feature of pancreatic, duodenal and proximal small intestinal fistulae (b) Fluid and electrolyte imbalance—particularly, with proximal small intestinal fistula	Affect adjacent organs contiguously OR more commonly through an intervening abscess cavity *Usual cause*: complication of a GIT disease, e.g. intestinal TB, cholecystitis, radiation enteritis, CA-colon, etc. *Complications* depend on the exact site and pathology. Examples: (a) Entero-enteric fistula → malabsorption and steatorrhea due to bacterial overgrowth and bile salt pool (b) Bilio-enteric fistula → cholangitis (c) Vesicocolic fistula → severe cystitis

(**B**) Classification of fistulae based on the number of tracts:
- Simple
- Complex: Complex fistulae have many tracts.

7. (Specific) Sinuses/fistulae occur in specific anatomical areas. However, some sinuses/fistulae may occur in general in any region of the body.

   Examples of general sinuses / fistulae:
   - Sinus following inadequate drainage of an abscess
   - Osteomyelitic sinus
   - Tuberculous sinus
   - Neoplastic sinus / fistula (i.e. overlying a (deeply situated) malignant tumor).

8. **Parts**
   - Of a sinus:
     - Mouth = opening(s)
     - Tract/s
     - Blind end
   - Of a fistula:
     - Mouth = external opening(s)
     - Primary tract. Sometimes secondary tract(s) may be present
     - Internal opening.

9. **Causes of persistence** of a sinus / a fistula

   (Physical) – Lack of rest. Common examples—location being near a joint, anal canal (frequent contraction of sphincter leading to not only a lack of rest, but also forcing a fecal matter into the fistula).
   – Inadequate drainage due to non-dependency / small opening / tortuosity of the tract.
   – Deep soft tissue cavity with rigid walls.
   – Persistent obstruction distal to the level of the fistula.

   (Biological) – Persistent source of infection; common in—dental sinus, anal fistula.
   – Other causes of persistent source of infection in general: a foreign body (thorn, hair, stitch etc.) / necrotic tissue / fecolith / worm, etc.
   – Recurrent infection; common in—sinuses on the foot, bronchopleural fistula.
   – Specific chronic infection—TB, actinomycosis, amebiasis, syphilis.
   – Osteomyelitis with/without sequestrum/non-collapsible cavity.
   – Dense fibrosis around the tract preventing adequate supply of blood to the granulation tissue which is a prerequisite for healing.
   – Epithelialization of the tract.
   – Malignancy, e.g. orocutaneous fistula following oral malignancy.

Free ebooks ==> www.Ebook777.com

HISTORY

The most common chief complaint: H/o persistent or recurrent episodes of discharge of ------- from --------, since -----.

- **Duration**?
  Examples of congenital sinus/fistula: Congenital periauricular sinus, congenital branchial sinus/fistula, congenital TEF, umbilical sinus/fistula due to partially/completely patent V-I duct or urachus, congenital RVF etc.
- **M/o onset**?
  - Following a significant trauma: Examples—bronchopleural fistula, A-V fistula
  - Following an obstructed labor: An example—VVF
  - Following I & D of an acute pyogenic abscess suggests sinus formation following inadequate drainage of the abscess. Similar history may be available in a thyroglossal fistula
  - As a failure of an operation: An example—thyroglossal fistula following incomplete excision of a thyroglossal cyst.
  - As a complication of an operation: Examples—fecal fistula, biliary fistula, salivary fistula.
  - Fistulae created with therapeutic intention: Examples—cysto-gastrostomy for internal drainage of a pancreatic pseudocyst, various gastrointestinal anastomoses, various ostomies, A-V shunt for hemodialysis, etc.
  - Following osteomyelitis
  - Over and above a pre-existing swelling: Examples—tuberculous chronic lymphadenitis / cold abscess; malignant ulcerated swelling as in oral malignancy, etc.
  - Following anorectal suppuration: Anal fistula, and rarely anal sinus.
- **Discharge**
  - Persistent OR Intermittent?
  - Amount? Is it increasing or decreasing?
  - Foul-smell or any other peculiar smell such as urinary/fecal etc.?
  - Bone chips?

  INTERPRETATION
  - Profuse discharge ⇒ more activity (of a disease and consequently inflammatory pathology).
  - Foul smelling discharge of pus ⇒ sepsis
    In general,
    - Persistent, profuse purulent discharge ⇒ acute/active sinus / fistula
    - Intermittent, seropurulent discharge is seen in chronic sinuses
    - In a c/o fistula, progressively decreasing amount of discharge may suggests healing of the fistula
  - Urinary smell ⇒ fistula involving the urinary tract
  - Fecal smell ⇒ a fecal fistula OR an internal abscess
  - The presence of bone chips ⇒ osteomyelitis.
- **Pain**?
  The presence of pain ⇒ (acute) inflammation.
- **Fever**? ⇒ infection of soft tissue / possibility of osteomyelitis.
- **Disturbance of function** / **disability**?
- **Clicking sounds** / **crepitations**? ⇒ osteomyelitis.
- **Significant loss of weight**? — may suggest TB, malignancy.
- H/s/o **retained foreign body** such as a thorn, stitch (postoperative) etc?
- H/s/o **sequelae** / **complications**
  - Recurrent attacks of formation of acute pyogenic abscess with throbbing pain f/b spontaneous bursting with discharge of pus mixed with blood? (- s/o recurrent episodes of secondary infection)
  - Soiling and excoriation of skin surrounding the lesion?
  - Social embarrassment? (- applicable usually to urinary / fecal fistulae)
  - Symptoms due to fluid-and-electrolyte imbalance? (- applicable to a high-output fecal fistula)

www.Ebook777.com

- **Treatment taken**?
- Have you been on any long-term medical treatment for any other disease?
- Is this the first episode or is this a **recurrent episode**? Details of previous episode(s), if any?
  Some sinuses are known to recur in spite of adequate excision e.g. pilonidal sinus.
  Otherwise, causes of recurrence could be: inadequate excision, malignant nature etc.

LOCAL EXAMINATION

INSPECTION

- The **mouth,** i.e. (external) opening/s: Single or multiple?

  Multiple openings may represent either multiple sinuses or fistulae, or they may represent secondary tracts belonging to a sinus/fistula basically with only single tract/internal opening
  - Single opening (obviously of single sinus / fistula): Examples—sinus following inadequate drainage of an abscess, branchial sinus, thyroglossal fistula, etc.
  - Single / multiple openings, due to single / multiple sinuses / fistulae respectively: Examples—osteomyelitic sinus, TB sinus, preauricular sinus, urethral fistula.
  - Always multiple openings due to multiple sinuses: An example—actinomycosis
  - Multiple secondary external openings of essentially single basic tract: Commonly found in anal fistula and pilonidal sinus
  - Pertaining pilonidal sinus:
    - Multiple openings strictly in the midline suggest multiple secondary openings de novo.
    - Multiple openings away from the midline suggest that the multiple secondary openings have followed spontaneous bursting or I and D of abscesses.
- **Site** gives a definite clue to the diagnosis.
  - Osteomyelitic: Commonly occurs near the end of long bones
  - TB sinus: In the neck, overlying ribs
  - Actinomycotic: Lower jaw, feet. The openings are situated on the summit (top) of subcutaneous pustules.
    A single sinus over the lower jaw is likely to be due to osteomyelitis, but multiple indurated sinuses overlying the same region or upper part of the neck suggest actinomycosis
  - Preauricular sinus(es): At the root of the helix or on the tragus
  - Branchial sinus / fistula: Inferior 1/3rd of neck near anterior border of the SCM muscle
  - Thyroglossal fistula: In the midline in the neck
  - Pilonidal sinus: In the midline in the natal cleft at the level of coccyx
  - Urethral fistula: Ventral surface of penis / scrotum
  - A guinea-worm sinus occurs classically in the lower leg. Reason: This is an anatomical part which gets submerged in water more frequently and hence suitable for an adult female guinea worm to discharge her embryos through a sinus in the water where they are expected to be eaten by Cyclops—the vectors through which life-cycle of guinea worm will continue.
- **Size of the external opening**?
  Wide opening resembling an ulcer, with undermined edge suggests TB.
- **Edge of the external opening**?
  - Raised and everted ⇒ malignant sinus
  - Red and edematous ⇒ (acute) inflammation
  - Heaped up edge of sodden white epithelium suggests chronicity
- **Granulation tissue at the external opening**?
  - Sprouting granulation (hypertrophic) tissue ⇒ foreign body in the depth / pocket of pus / sequestrum / malignancy
  - Pale flabby granulation tissue ⇒ TB

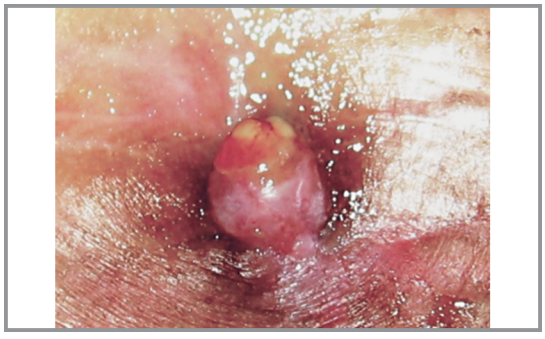


Fig 43-1 Heaped-up sodden epithelium

Free ebooks ==> www.Ebook777.com

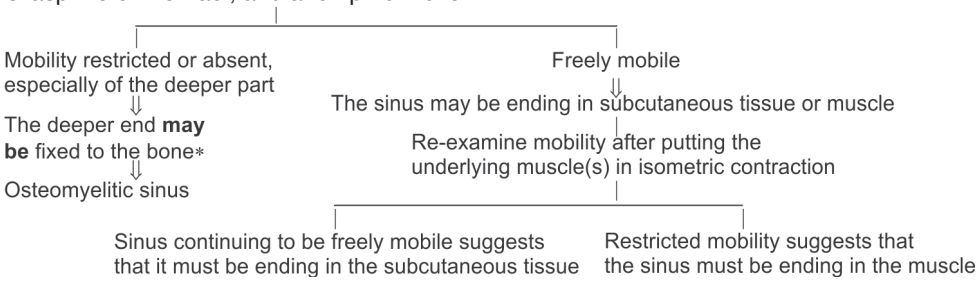
- **Discharge—smell, nature and amount**?
  - Foul smelling discharge of pus ⇒ sepsis
  - Urinary smell ⇒ fistula involving the urinary tract
  - Fecal smell ⇒ a fecal fistula
  - Discharge of saliva ⇒ a salivary fistula
  - Serosanguinous / watery ⇒ TB
  - Mucous discharge suggests that the tract must be lined by epithelium e.g. branchial, thyroglossal, etc.
  - Foul smelling sebum, often blood stained, ± hair: Typically suggets pilonidal sinus
  - Sulfur granules ⇒ Actinomycosis. Size of the granules is very small—pinhead—and cannot be seen with naked eyes. Pus has to be collected in a test tube and seen in a powerful light
  - Bone particles ⇒ osteomyelitis
  - Amount of discharge in c/o a fistula depends upon the viscus at the deep end and the disease responsible.
- **Any foreign body**?
  The presence of **hair** ⇒ pilonidal sinus.
- **Surrounding area**?
  In c/o a sinus:
  - Hyperpigmentation, thickening and brawny edema suggests chronicity
  - Cellulitis ⇒ active infection
  - Scarred, in osteomyelitis
  - Hyperpigmentation may suggest TB
  - Bluish pigmentation ⇒ actinomycosis.

  In c/o a fistula, the state of the surrounding skin depends on nature (and amount) of the discharge.
  - Sodden skin ⇒ serous / seropurulant discharge
  - Excoriated skin ⇒ fecal fistula.
- **Underlying / related swelling, if any**?
  For example, a tuberculous sinus may be related to an underlying swelling due to lymphadenitis / epididymitis / arthritis / osteomyelitis.
- **Any other finding** (s/o Etiology, complication), if any?

PALPATION

Palpation of the sinus / fistula *per se* = palpation of the external opening and the tract wall.

- **Temperature**?
  Raised temperature ⇒ (acute) inflammation.
- **Tenderness**? ⇒ (acute) inflammation.
- **Thickening**? ⇒ fibrosis ⇒ chronicity.
  A tuberculous sinus is characterized by the absence of induration.
- **Mobility**?
  The sinus may end in subcutaneous tissue / muscle / bone.
  Grasp the entire tract, and attempt to move it.



www.Ebook777.com

- **Surrounding area**?
  - Cellulitis ⇒ (acute) inflammation
  - Indurated in actinomycosis. In actinomycosis the whole affected area is indurated involving the subcutaneous tissue with multiple sinuses; it is not that multiple sinuses have their own separate indurated areas with intervening tissue being normal.
- **Palpate the underlying / related swelling, if any, in its own respect.**
- **Underlying bone / joint**?
- **Gently probe** with a sterile malleable probe, maintaining aseptic precautions.
  - Detect the direction, depth and multiplicity of tracts
  - Probing may help to reveal whether the lesion is a sinus or a fistula
  - Try to detect (by 'sounding') any foreign body or sequestrum
  - Sequestrum gives a metallic **note** on tapping while living bone gives a dull note
  - Fresh discharge on withdrawal of probe?
  - Note the characteristics.

* Immobility of a sinus over a bone, or touching a bone on probe examination is **not necessarily a proof** of bone involvement.

The criteria of bone involvement

On palpation of the bone: 1. Thickening enlargement. 2. Irregularity of the surface. 3. Rounding of sharp margin, if any, on comparing with the opposite normal side.

And *on probing*, the area touched should be rough.

REGIONAL EXAMINATION is essential to find out a causative feeding lesion, if any.

In TB, it may be present

OR

Just underneath the sinus, e.g. lymphadenitis, epididymitis, arthritis, osteomyelitis, renal TB, etc.

Far away
i.e. coming from a distant tuberculous lesion of bone, joint, bursa, serous cavities like pleural, etc.

Incidentally, the feeding lesions of a cold abscess also fall in the same categories.

- Examine the **lymph nodes** of the respective drainage area
- In c/o a sinus / fistula **in the loin**: Examine the spine, ribs, and urinary system
- In c/o a sinus / fistula **around the anus**: Perform DRE and proctoscopic examination.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. In the field of general surgery, any postoperative fistula encountered is likely to originate in the gastrointestinal or urinary tract. The first suggestion of a fistula is the appearance at the abdominal wall, either through a wound, through a drain-track, or at some other point near the wound, of fluid that looks like urine or feces.

Characteristic appearance and smell of urine
⇓
Urinary fistula. Usually no doubt. However, if doubtful, then confirmation can be obtained by IV injection of indigocarmine
↓
The dye is excreted by kidneys within a few minutes

Feculent appearance and smell
⇓
– A fecal fistula
OR
– Fluid from an internal collection infected with *Escherichia coli*—simulates a fecal leak
↓
The simplest bedside investigation for confirming the diagnosis of fistula
↓
Give the patient an oral dose of methylene blue. The dye is readily recognized in the effluent, and the length of time that elapses between ingestion and appearance is a rough measure of the distance along the alimentary tract of the origin of the leak

Free ebooks ==> www.Ebook777.com

2. A sinus cannot always be easily differentiated from a fistula, as the internal opening of a fistula may be difficult to demonstrate.
3. A sinus **often** leads to an abscess cavity.
4. **Causes of persistence** of a sinus / a fistula: Refer to page 629.

STEPWISE ANALYSIS OF DATA

Step 1
Acute OR Chronic?
Persistence of the lesion for > 3 weeks, or recurrent episodes qualify the case to be chronic.

Step 2
– The exact site?
– The possible organ/tissue involved?

Step 3
Determine various pathological possibilities at the involved site; these are quite specific.

Step 4
Determine the possible etiology / factor(s) responsible for persistence/recurrence.

Step 5
Determine if any complication is present.

Step 6
Any associated disease—closely related / otherwise?

Diagnosis can be attempted by **COMBINING STEPS 2 AND 3**

Preauricular
1. Congenital sinus.
2. Sinus associated with mastoiditis (infection tracking along the zygomatic arch).
3. Tuberculous sinus—from preauricular or parotid lymphadenitis.
4. Parotid salivary fistula

(Acquired) **Periauricular sinus**

Submandibular / mandibular
1. Dental sinus associated with a root abscess
2. Osteomyelitic sinus
3. Tuberculous sinus
4. Actinomycotic sinus
5. Salivary fistula
6. Branchial sinus / fistula

Submental
1. Dental sinus associated with a root abscess
2. Sinus following bursting of an infected dermoid cyst
3. Osteomyelitic sinus
4. Tuberculous sinus
5. Thyroglossal fistula

Midline cervical
Thyroglossal fistula

Lateral cervical
1. Tuberculous sinus—commonly following lymphadenitis; rarely following tuberculous osteomyelitis of cervical spine.

www.Ebook777.com

2. Branchial sinus / fistula
3. Pharyngeal fistula
4. Thoracic duct fistula

Breast

Mammary duct fistula

Causes of persistence of a fistula after drainage of empyema thoracis

1. Too late drainage (leading to severe thickening and fibrosis of pleura).
2. Improper drainage due to a too anterior, a too high, or a too low opening.
   a. Too anterior or high an opening becomes non-dependent.
   b. Too low an opening = opening in the costo-phrenic recess. Movements of the diaphragm may block the tube and consequently the drainage.
3. Too early removal of drainage tube (before full expansion of the lung has occurred).
4. Too late removal of drainage tube (leading to fibrosis around the tract).
5. Specific chronic infection e.g. tuberculous empyema.
6. The presence of a foreign body (Piece of gauze or rubber tubing).
7. Osteomyelitis of the cut rib ends.
8. Broncho-pleural fistula (recurrent infection).
9. Unsuspected esophageal perforation.
10. Unsuspected cancer or other chronic disease of the lung.
11. Non-closure of abscess cavity due to rigidity of walls; particularly thickening of visceral pleura prevents expansion of the lung.
12. Epithelialization of the tract.

Umbilical—*in newborn and infants*

1. Fecal fistula.
   Conditions, which may be mistaken for fecal fistula: (a) Adenoma (*Syns*. bud, polyp, raspberry tumor, enteroteratoma), (b) Granuloma (due to infection of the umbilical stump).
2. Urinary fistula.

Umbilical—*in adults*

1. Fecal fistula.
   Causes:
   a. Patent V-I duct becoming manifested later in life.
   b. Rupture of abscess involving the intestine—TB / nonspecific.
   c. Bursting of CA-transverse colon.
   d. Sloughing off of a strangulated umbilical / paraumbilical hernia (leading to spontaneous cure).
2. Urinary fistula.
   Causes:
   a. Completely patent urachus, with bladder outflow obstruction.
   b. Postoperative following operation upon GU tract (including hysterectomy).
3. Biliary fistula—following rupture of acute empyema of gallbladder.
4. Sinus following inadequately drained abscess in abdominal wall or incompletely patent urachus
5. (Infected) Pilonidal sinus
6. Ruptured infected sebaceous cyst

   → Purulent discharge.

7. Endometrioma (in women).

Abdomen

A. *Spontaneous external fistula*: Pseudomyxoma peritonei arising from a mucinous carcinoma of appendix.
B. *Postoperative intestinal* fistula—following operation upon any segment of the GI tract.

Free ebooks ==> www.Ebook777.com

C. *Causes of fistula following appendicectomy*
1. Unsuspected ileocecal TB.
2. Unsuspected actinomycosis of cecum.
3. Unsuspected CA-cecum.
4. Obstructive lesion distal to the cecum.
5. Chronic abscess cavity with rigid walls.
6. Incomplete removal, with open lumen of the stump.

On the **Back in the midline**
A congenital sinus associated with spinal dysraphism.

Renal angle
1. Osteomyelitic sinus due to chronic osteomyelitis—tuberculous or non-specific—of the 12th rib or thoracic spine.
2. Fistula due to empyema thoracis.
3. Renal fistula. Causes: Pyonephrosis, perinephric abscess, postoperative.
4. Ureteric fistula. Causes:
   a. Following ureterotomy: improper reconstitution (eversion of mucosa), distal obstruction.
   b. Improper closure of ureteral stump during nephrectomy (results in vesicoureteral reflux).
5. Colonic diseases.
6. Sloughing off of the colonic wall due to (a) inadvertent crushing or mass ligation or electrocoagulation for the bleeding points. (b) Pressure from the drainage tube.
7. Suppurative diseases in the liver or subdiaphragmatic spaces.

Groin
1. Osteomyelitic sinus due to chronic osteomyelitis—tuberculous or nonspecific—of superior end of femur / pelvic bone / S-I joint / lumbar spine (along the iliopsoas muscle in c/o the latter two).
2. Tuberculous sinus following inguinal or external iliac lymphadenitis.
3. Rupture of a cold abscess traveling along the processus vaginalis in spermatic cord.
4. Rupture of an infected psoas bursa.
5. Fistula following sloughing off of strangulated Richter's type of femoral hernia.
6. Fistula following TO abscess draining along the round ligament of uterus (may be bilateral), sometimes may follow infected unabsorbable suture material used during TL.
7. Fistula following perinephric abscess draining along the psoas sheath.
8. Retroperitoneal abscess from colonic disease.
9. Rupture of an abscess of the pouch of Douglas (just above the inguinal ligament).

Sacrococcygeal region
1. A coccygeal dimple/pit.
2. A congenital sinus in the midline of the back associated with spinal dysraphism.
3. Pilonidal sinus.
4. Osteomyelitic sinus due to chronic osteomyelitis of the sacrum / coccyx.
5. A (congenital) sinus in connection with post-anal dermoid.

Perineal region
1. Anal fistula.
2. Urethral fistula.
3. Pilonidal sinus.
4. Chronic osteomyelitic sinus arising from ischial tuberosity, sacrum or coccyx.
5. Infected post-anal dermoid.
6. Rupture of a prostatic abscess. Rare. Lies anterior to the anus. H/o passing small prostatic stones ±.
7. Sinus from a Bartholin's gland abscess.
8. Lymphogranuloma involving the perineum.

www.Ebook777.com

Foot

1. Sinus following inadequate drainage of an abscess.
2. Sinus due to a retained foreign body.
3. Osteomyelitic sinus.
4. Mycetoma.
5. Guinea-worm sinus.

DIFFERENTIAL DIAGNOSIS

Tuberculous sinus / fistula

- Nonspecific manifestations of TB (Loss of appetite, loss of weight, low-grade evening fever with night sweats)
- Often multiple sinuses
- Wide opening resembling an ulcer with undermined edge, and the opening lies flush with the surface
- Pale flabby granulation tissue
- Discharge—serosanguinous / watery
- Surrounding area—hyperpigmented
- NO induration around the tract
- A causative feeding lesion nearby or far away ±. Most common—tuberculous lymphadenitis f/b cold abscess which bursts.

Chronic (nonspecific) osteomyelitic sinus

- Preceding h/s/o acute osteomyelitis
- Bone chips in the discharge
- Situated on a specific site (which is prone for osteomyelitis)
- Sprouting granulations at the opening
- Surrounding area—scarred
- (Deeper part of the sinus is) fixed to the bone
- Underlying bone on palpation and on probing of the sinus, shows signs of chronic osteomyelitis.

Cervicofacial actinomycosis*

- Long duration—in months / years
- Predisposing conditions—carious teeth / trauma
- Multiple sinuses situated in the lower jaw + upper part of the neck
- The openings of the sinuses are situated onto the top of the subcutaneous pustules
- Bluish pigmentation
- Induration of the whole affected area
- Sulfur granules in the discharge.

Mycetoma—foot** with 'mild'/'moderate'/'severe' disability. Refer to Fig 13-12.

- Long duration—in months/years
- Multiple sinuses
- (Bluish) Pigmentation
- Induration of the whole affected area.

Congenital preauricular sinus

- H/o it being present for a long time / since childhood / since birth
- Situated on the root of the helix or on the tragus

* For relevant details on parasitology, refer to chapter 13.
** For relevant details on bacteriology and parasitology, refer to chapter 13.

Free ebooks ==> www.Ebook777.com

- Normally remains asymptomatic as it is lined by epithelium
- Becomes symptomatic when it gets infected: → pain / purulent discharge / abscess. Center of the abscess usually lies anterior to the punctum (opening of the sinus), because the sinus track runs forwards away from the ear.

Pathology: Imperfect fusion of anterior aural tubercles forming the ear.

(Acquired) **Periauricular sinus**

- Results from I and D of an infected peri-auricular cyst.

Apparently simple periauricular cysts and sinuses may have extensive and deep branching ramifications which pass in close proximity to the facial nerve.

Parotid salivary fistula following I and D of a **parotid abscess** OR spontaneous bursting of a parotid abscess; and **the cause of persistence being stricture / stone** in the parotid duct

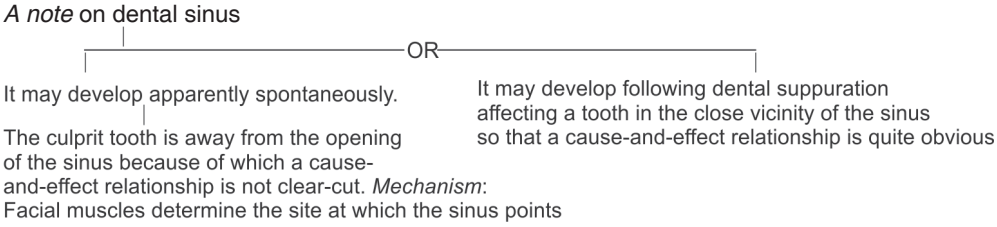
- Preceding h/s/o acute pyogenic parotid abscess
- Discharge of saliva from an opening in the region of parotid gland
- Sogging of the surrounding skin.

Parotid salivary fistula may occur following **superficial parotidectomy**

Dental sinus

- Preceding h/s/o root abscess
- Caries of tooth—usually in the close vicinity, but can be away
- Sinus track can be felt as a fibrous cord leading to the bone underneath the carried tooth.

A note on dental sinus



Curiously, the source of the lesion is often not recognized, and then the sinus is mistaken as an infected sebaceous cyst / even a SCC or BCC. The possibility of a SCC/BCC is heightened as excision is always f/b recurrence.

Thyroglossal fistula (Always acquired. Never congenital)

- *Modes of onset*
  1. Following spontaneous bursting or I & D of an infected thyroglossal cyst.
  2. Following incomplete excision of thyroglossal tract during the operation of removal of a thyroglossal cyst.
- Intermittent discharge of mucus
- Recurrent attacks of inflammation
- Situated (practically) in the midline, between the level of thyroid isthmus and hyoid bone. Long-standing fistulae are inclined to be situated low in the neck
- Transversely disposed elliptical opening. The superior lip of the ellipse has a hood-like fold of skin. During swallowing and during protrusion of tongue, especially the lower lip of the elliptical opening moves up accentuating the upper lip's hood-like effect.

The internal end of the fistula opens at the foramen cecum of the tongue.

Congenital branchial sinus / fistula

- Present since birth
- Situated in lower 1/3rd of the neck, just anterior to SCM (This specific location has embryological basis)
- Occasionally, bilateral
- Normally remains asymptomatic as it is lined by epithelium
- Mucoid / mucopurulent discharge
- Otherwise, may suffer recurrent attacks of infection (due to the presence of lymphoid tissue in the wall).

www.Ebook777.com



Fig 43-2 Thyroglossal fistula

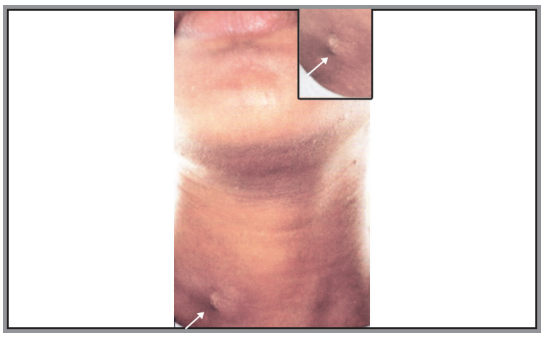


Fig 43-3 Branchial sinus/fistula



Fig 43-4 Mammary duct fistula

If it is a fistula, then the tract opens internally on the posterior pillar of the tonsillar fossa (just behind the tonsil). However, mostly it is a sinus track ending blindly on the lateral pharyngeal wall. Clinically it is not possible to say for sure as to whether it is a sinus or a fistula because of the following reasons. 1. The internal opening is not visible even if it is a fistula. 2. The entire tract is not amenable to palpation, i.e. the part of the tract deeper to the deep fascia cannot be palpated.

Acquired branchial sinus—usually it is one of the differentials and not a definite diagnosis.

Supporting points

– Mode of onset: Spontaneous bursting, or I & D of an infected branchial cyst
– Situated in the carotid triangle.

Mammary duct fistula

= a fistula between (the epithelium of) a lactiferous duct and the skin
– A preceding h/s/o recurrent attacks of subacute abscess (at intervals of a few months) or a chronic subareolar abscess
– Opening of the fistula often situated on the areolar margin
– Nipple inverted or retracted.

Causes

1. Inversion of nipple
2. Mammary duct ectasia/periductal mastitis

} → Attacks of subacute abscess or a chronic subareolar abscess.

Umbilical fecal fistula in the newborn and infants

– Occurs due to completely patent V-I duct
– 'Small' amount of discharge ⇒ a narrow fistula—most likely to close spontaneously
– 'Larger' amount of discharge ⇒ a wide fistula / a fistula which may be associated with atresia of the intestine distally

Free ebooks ==> www.Ebook777.com

Umbilical urinary fistula in the newborn and infants

Discharge of urine from the umbilicus ⇒ patent urachus associated with bladder outflow obstruction.

A patent urachus without distal obstruction (bladder outflow obstruction) does not give rise to an evident fistula as contraction of bladder starts at the dome and passes towards the base, thereby temporarily closing the vesical opening of the fistula.

Other causes of umbilical discharge in children: umbilical bud, umbilical granuloma: Refer to chapter 81.

Patent urachus leading to umbilical urinary fistula

Seldom reveals itself until maturity, or even old age / unless bladder outflow obstruction develops.

Contraction of bladder starts at the dome and passes towards the base, thereby temporarily closing the vesical opening of the fistula. Hence, the fistula remains unobtrusive until a time when the bladder is overfull, usually due to outflow obstruction.

Postoperative digestive tract (esophageal/gastric/intestinal) fistula

This is usually a serious condition except for the most distal (low-output) colonic fistula.

Classification

1. Based on the mode of development
- Congenital. An example – completely patent Meckel's diverticulum
- Spontaneous, due to a disease
  Examples—(a) pseudomyxoma peritonei, (b) complication of diverticulitis (e.g. colovesical fistula developing due to proximity between the sigmoid colon and dome of the bladder), (c) cancer of the GIT.
- Postoperative.
  Causes/Examples: (a) Anastomotic leak leading to an external fistula a few days after surgery.
  (b) Cecal fistula following an appendicectomy on a gangrenous appendicitis, or following opening of an appendix abscess.
  (c) From blow-out of a gangrenous patch of intestine after the relief of a strangulated hernia, the gangrenous patch not being obvious 'on-table' and hence having been left behind inadvertently.
  (d) Following opening of an abscess connected with chronic diverticulitis or cancer of a viscus e.g. CA-colon.
  (e) Following operation on the intestinal tract affected by TB / actinomycosis / amebiasis / regional enteritis.
- Post-RT several months/years after the treatment, owing to the late development of endarteritis obliterans and chronic microvascular ischemia.

2. Morphological classification: External / Internal (GIT ↔ UT, GIT ↔ vagina, GIT ↔ GIT i.e. between 2 segments of the GIT itself).
- Bile-stained discharge, causing severe excoriation of the skin ⇒ fistula involving the duodenum / jejunum
- Discharge looking like fluid fecal matter ⇒ ileal / cecal fistula
- Semi-solid or solid fecal matter ⇒ fistula from the (distal) colon.

3. Based on number / branching pattern
- Single = directly between intestine and skin
- Branched = Single internal (i.e. intestinal) opening, and > 1 external opening
- Multiple = multiple internal (i.e. intestinal) openings at different sites, especially seen with infected pancreatic necrosis.

4. Based on fluid output: – High output = > 1 L/day.
– Medium output = 500 mL–1 l/day.
– Low output = < 500 mL/day.

Causes of persistence of a fecal fistula :
- Obstruction distal to the point of origin of fistula
- The presence of a foreign body
- The presence of a chronic disease in the involved segment of gut e.g. TB, actinomycosis, regional enteritis, cancer, etc.
- A very long duration itself, by virtue of growing of epithelium from the skin along the track to meet epithelium from the body cavity.

www.Ebook777.com

The fistula may be lined by :

1. Mucous membrane; it may protrude above the skin surface; such a fistula will never close spontaneously.
2. Granulation tissue.
3. Fibrous tissue and partly epithelialized.

Pathology / effects of an external fistula:

1. The fluid coming out of the fistula may not constitute the total volume of leak; the remainder may be spreading in the abdomen causing peritonitis. This disaster may present by (a) distended silent abdomen. (b) the development of shock or (c) symptoms and signs of peritonitis. An urgent laparotomy in order to seal or exteriorize the leak is mandatory.

2. Losses of fluids and electrolytes can be considerable. The more proximal the fistula in the digestive tract the greater the volume of the intestinal secretions and proteins lost, and more digestive their properties. These deleterious effects are worst with a duodenal fistula. The composition of the electrolyte-loss can vary depending on the site of the fistula.

3. Digestion of the skin is maximum with a duodenal fistula.

Duodenal fistula

The most usual causes:

1. A complication of gastrectomy: A serious complication of Billroth-II reconstruction with a maximum incidence about 4th day.
2. An abscess connected with a perforated duodenal ulcer.
3. Traumatic rupture of duodenum.
4. A complication of transduodenal sphincterotomy / right nephrectomy / right colectomy.
   - The fistula discharges bile and pancreatic juice leading to excoriation of skin.
   - Excessive discharge may quickly lead to dehydration, electrolyte imbalance, and hypoproteinemia.

External biliary fistula

Spontaneous	Postoperative
Example—following rupture of acute empyema of gall bladder. Usually develops through umbilicus	More common than the spontaneous variety. Causes: – Damage to the main ducts – Leakage of biliary anastomosis

Pancreatic fistula—usually follows operative trauma to the gland which in most cases was not suspected at the time.

- In the presence of fistula, the pancreas tends to hypersecrete, and up to 2 liters may be lost in a day. This along with loss of electrolytes may constitute a menace to life
- Autodigestion of the skin: A 'pure' pancreatic juice fistula is much less corrosive than a mixture of duodenal and pancreatic juice, because in the former the enzymes are not activated by the duodenal contents
- 'Pooling' of the discharge may occur deeply in the wound essentially due to non-dependency.

Spontaneous closure occurs in most of the cases. Failure is usually due to inadequate dependant drainage.

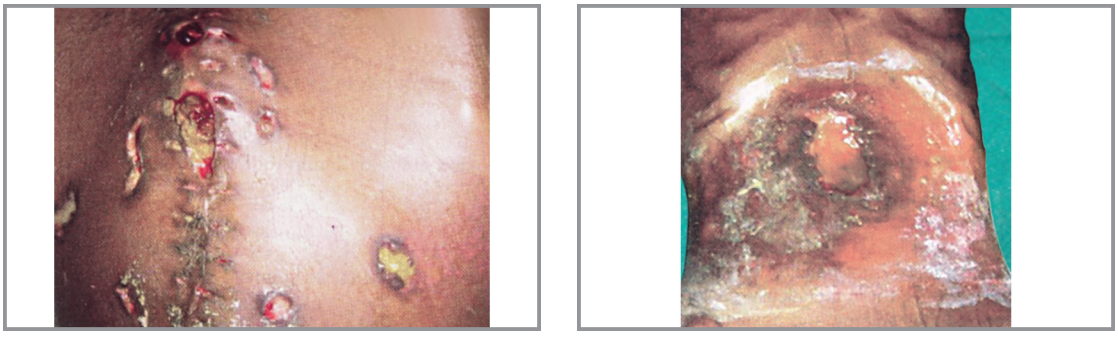


Fig 43-5 Examples of postoperative fecal fistulae

Free ebooks ==> www.Ebook777.com



Fig 43-6 A chronic fecal fistula (colonic)

Coccygeal dimple
- A shallow groove / hole in the skin over the coccyx
- No significance. Disappears during growth.

Coccygeal pit—up to 1 cm long.
During infancy feces may be pressed into it by the napkin, causing it to be misinterpreted as a small fecal fistula.

A congenital sinus in the midline of the back associated with spinal dysraphism
- A tiny sinus in the lumbosacral region
- The depth cannot be seen and a probe indicates that there is small track, not always strictly in the midline
- Dermoid cysts arise at any point along the sinus tract and often lie adjacent to the conus
- Clinical presentation varies from repeated attacks of unexplained meningitis to neurological deficits arising from the presence of an intraspinal mass.

Sacrococcygeal pilonidal sinus
- More common in young (20–30) men than in women. Patients presenting later in life may give a history dating back to this period
- Many patients are exceptionally hairy, and are usually obese
- *Clinical presentation*: A chronic (persistent) or a recurring sinus
- H/s/o acute recurrent pyogenic abscess ±
- Site: in the natal cleft at the level of coccyx, several centimeters posterior to the anal verge.
  Multiple openings may be present: Multiple openings actually mean multiple secondary openings of essentially single basic tract.
  These secondary openings may develop far out on the buttocks or in the perineum.
  - Multiple openings strictly in the midline suggest multiple secondary openings *de novo*
  - Multiple openings away from the midline suggest that these openings have followed spontaneous bursting or I and D of abscesses
- Discharge—foul-smelling blood-stained sebum ± hair
- Tuft of hair may project from the opening/s. Shafts of hair may lie loose in the sinus, or embedded in granulation tissue, or embedded deep in mature scar tissue.

On probing:
- The blind end stops short of bone
- The probe passes upwards and forwards towards the sacrum (that is how differentiating itself from an anal fistula).

- *It is possible however, that a pilonidal sinus may be mistaken for an anal fistula and vice versa*
- *During the episodes of secondary infection*:
  - Even if a pyogenic abscess forms, constitutional symptoms are often slight. Abscess is often located just to one side of the midline in the natal cleft

www.Ebook777.com

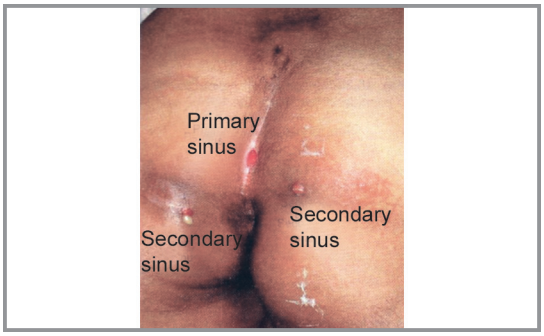


Fig 43-7 Pilnodal sinus

- The discharge becomes mucopurulent
- Variable number of pits in the upper natal cleft may be seen to exude pus.

– Pilonidal sinus may be asymptomatic, and detected incidentally on examination.
– *Other sites in the body where pilonidal sinus may be found* : Umbilicus, web spaces of hands of barbers.

Urethral fistula: Refer to chapter 54.

Ano/rectal fistula: Refer to chapter 41.

Free ebooks ==> www.Ebook777.com

44 (RECURRENT ATTACKS OF) DISCHARGE FROM THE UMBILICUS

GENERAL REMARKS

Basically, the discharge may originate from acute-on-chronic sepsis, a sinus, or a fistula.

1. **Acute-on-chronic sepsis** may occur in the umbilicus itself (omphalitis) or in the anterior abdominal wall.
   a. Omphalitis occurs essentially due to poor hygiene; with or without umbilical stone.
   b. Sepsis in the anterior abdominal wall may occur *de novo*, or it may occur as a complication in an existing sinus e.g. an incompletely patent urachus or VI duct.
2. **Sinus**
   A sinus may form following inadequate drainage of anterior abdominal wall abscess (including umbilical), or it may be pre-existing due to incompletely patent urachus/VI duct, or as a pilonidal sinus.
3. **Fistula**
   a. Fecal: Patent VI duct manifesting later in life, rupture of abscess involving intestine (TB/non-TB), bursting of CA transverse colon, sloughing off of a strangulated paraumbilical hernia.
   b. Urinary:
      – Completely patent urachus with bladder outflow obstruction
      – Postoperative following operation upon GUT.
   c. Biliary: Following rupture of acute empyema of gall bladder.

HISTORY

Mode of onset

- Sudden OR insidious?
  In c/o sudden m/o onset: H/s/o acute pyogenic abscess preceding the discharge?
  Causes of acute pyogenic abscess
  – Umbilical abscess (omphalitis) *per se*
  – Anterior abdominal wall abscess *de novo*
  – Secondary infection in an existing sinus such as an incompletely patent urachus/VI duct. (A deep tender swelling in the midline inferior to umbilicus ⇒ abscess in the extraperitoneal fat—usually due to an infected urachal remnant.)
- **Progress, regress**?
- **Smell of the discharge**?
  – General foul-smelling discharge ⇒ sepsis
  – Fecal smell ⇒ fecal fistula
  – Urinary smell ⇒ urinary fistula.
- **(Additional) symptoms s/o etiology**
  – The presence of hair in the discharge? ⇒ pilonidal sinus
  – Preceding symptoms of an intra-abdominal disease of a few months duration e.g. intra-abdominal abscess, CA colon, etc.?
  – H/s/o a paraumbilical hernia?
  – H/s/o bladder outflow obstruction?
  – Recent past h/o any operation upon the GUT?

www.Ebook777.com

- Recent past h/s/o empyema of gallbladder?
- In a woman: Cyclical discharge of blood each time during menstrual period, associated with pain in the umbilical nodule? ⇒ endometriosis.

- **Treatment taken** (medications / any other), and response to it?
- Is this the first episode or is this a recurrent episode? Details of previous episode/s, if any.

LOCOREGIONAL EXAMINATION

INSPECTION

- **Of skin surrounding the umbilicus**
  - The skin may be sodden due to serous / seropurulant discharge
  - Excoriated skin suggests fecal fistula
- **Of the abdomen in general**—in the usual manner.

PALPATION

- Temperature?
- Tenderness?
- **Press on the surrounding area to reproduce discharge**.
  a. Smell of the discharge?
  b. Nature of the discharge?
    - Mucous discharge could be due to a patent VI duct
    - Biliary discharge suggests biliary fistula
    - Serous discharge occurs in umbilical granuloma
    - Serosanguinous discharge occurs in umbilical granuloma, umbilical stone
    - Frank sanguinous discharge in a woman—could be due to endometriosis
- **Part the edges of the umbilicus to look for** hair/stone/s (often black colored)
- Palpate the **tract**
- Palpate the **rest of the abdomen**
- Subumbilical (depth wise) lump may be due to an enterocyst or urachal cyst
- **Gently probe the tract** (with a sterile probe, observing aseptic precautions).

DIFFERENTIAL DIAGNOSIS of selected disorders causing discharge from the umbilicus.

For causes of 'discharge from the umbilicus' in general, refer under the heading 'General remarks' at the outset.

Umbilical stone

- Often black in color. Composed of desquamated epithelium which becomes inspissated and collects in the deep recess of umbilicus
- Eventually → infection-inflammation, and a blood-stained discharge.

Dermatitis of and around the umbilicus

- Fungal and parasitic infestations are more difficult to eradicate from the umbilicus than from the skin of the abdomen
- Sometimes, the dermatitis is consequent upon a discharge from the umbilicus
- In fat patients, intertrigo is seen.

Patent VI duct

- *Suspect* this when there is mucous discharge from the umbilicus; a subumbilical / infraumbilical lump may be associated
- Frank fecal discharge is very rare.

Urachal fistula

- *Suspect* this when there is urinary discharge from the umbilicus in a patient with e/o bladder outflow obstruction.
- Refer to chapter 43.

Free ebooks ==> www.Ebook777.com

Urachal sinus

– *Suspect* this when there is purulent discharge from the umbilicus. A subumbilical / infraumbilical acute or acute-on-chronic cellulitis / abscess may be associated.

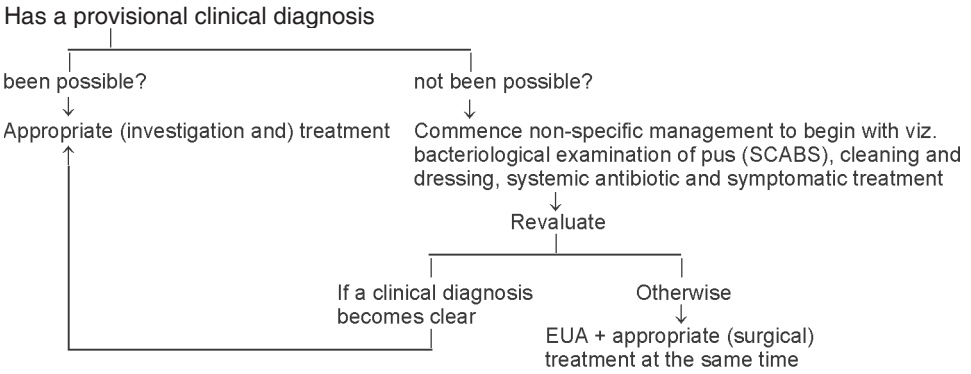
Pilonidal sinus

– Many patients are exceptionally hairy, and are usually obese
– *Clinical presentation*: A chronic (persistent) or a recurring sinus
– H/s/o acute recurrent pyogenic abscess ±
– Discharge—foul-smelling blood-stained sebum ± hair.

Umbilical bud (*Syns.* Umbilical adenoma / polyp / raspberry tumor / enteroteratoma)
Umbilical granuloma

} Refer to chapter 81.

PLANNING OF MANAGEMENT



www.Ebook777.com

45 PAIN IN THE LIMB

Cause of pain in the limb can be obvious or obscure.

The 'obvious causes' include: Trauma (including sudden jerky movement, overuse / exercise), acute cellulitis, and swelling.

Well localized pain suggests that the tissue underneath the site of pain could be diseased.

↓

- Obvious local pathology in the skin/subcutaneous tissue
- Fascia / bursa / tendon-sheath / ligament in 'relatively superficial plane'
- Muscle—strain / abscess
- Bone (An exception—osteoporosis)
- Joint (An exception—proximal joints like shoulder, hip, sacroiliac and spine).

Tissue-wise causes of segmental or diffuse pain

- Arteries—peripheral arterial occlusive disease
- Veins—superficial thrombophlebitis, DVT
- Lymphatics—lymphangitis
- Nerves. Usually lesion in the proximal portion of nerve (root / plexus / trunk) leads to diffuse pain. However, lesion in the distal portion of the nerve also may give rise to (diffuse) pain in the whole limb
- (Inflammatory) Disorders of fascia / bursa / tendon-sheath / ligament in 'deep plane'
- Muscle—acute infectious fevers, deficiencies of vitamin B complex, calcium; infiltration with *Trichinella spiralis*
- Bone—osteoporosis
- Proximal joint disorders.

HISTORY

- **Exact site**?
  Is it localized OR segmental OR diffuse involving the whole limb?
  ↓
  - If in the arm: Is it confined to the arm, OR Does it extend distally beyond the elbow?
  - If in the thigh: Is it confined to the thigh, OR Does it extend distally beyond the knee?
- **Duration**?
- **Mode of onset**?
- **Progress** / **Regress** / **Fluctuation**?
- **Intensity**
  - Does it distract you from your usual activity?
  - Does it compel discontinuation of usual activity, and to take rest ± analgesic?
    - Mild = no distraction of usual activity
    - Moderate pain compels discontinuation of usual activity, and to take rest ± analgesic. May compel to consult a doctor, if pain does not subside by above measures
    - Severe pain compels to seek urgent medical help.

Free ebooks ==> www.Ebook777.com

- **Nature**?
  Throbbing / cramping / burning / shooting / aching / vague?
- **Precipitating/Aggravating factor(s)**?
  - Trauma / sudden jerky movement / overuse / exercise?
  - History suggestive of acute infectious fever?
    OR
  - Did the pain begin spontaneously?
- Have you been suffering from **a cardiac/aortic disease**?
- **Relieving factor(s)**?

Interpretation in case of severe agonizing acute/subacute pain

- Severe agonizing continuous bursting pain, radiating down the course of an artery
- In a patient with a debilitating and dehydrating illness, e.g. typhoid, etc.
  OR
- In a patient known to have a cardiac / aortic disease
- Sudden mode of onset. Rapid / dramatic progress.

} ⇒ ***Acute arterial occlusion***

Further history to be asked

- **Burning / tingling / numbness**?
  Cause of paresthesia: Ischemia of peripheral nerve endings.
- **Paresis / paralysis**?
- **Coldness and deadness** of the limb?

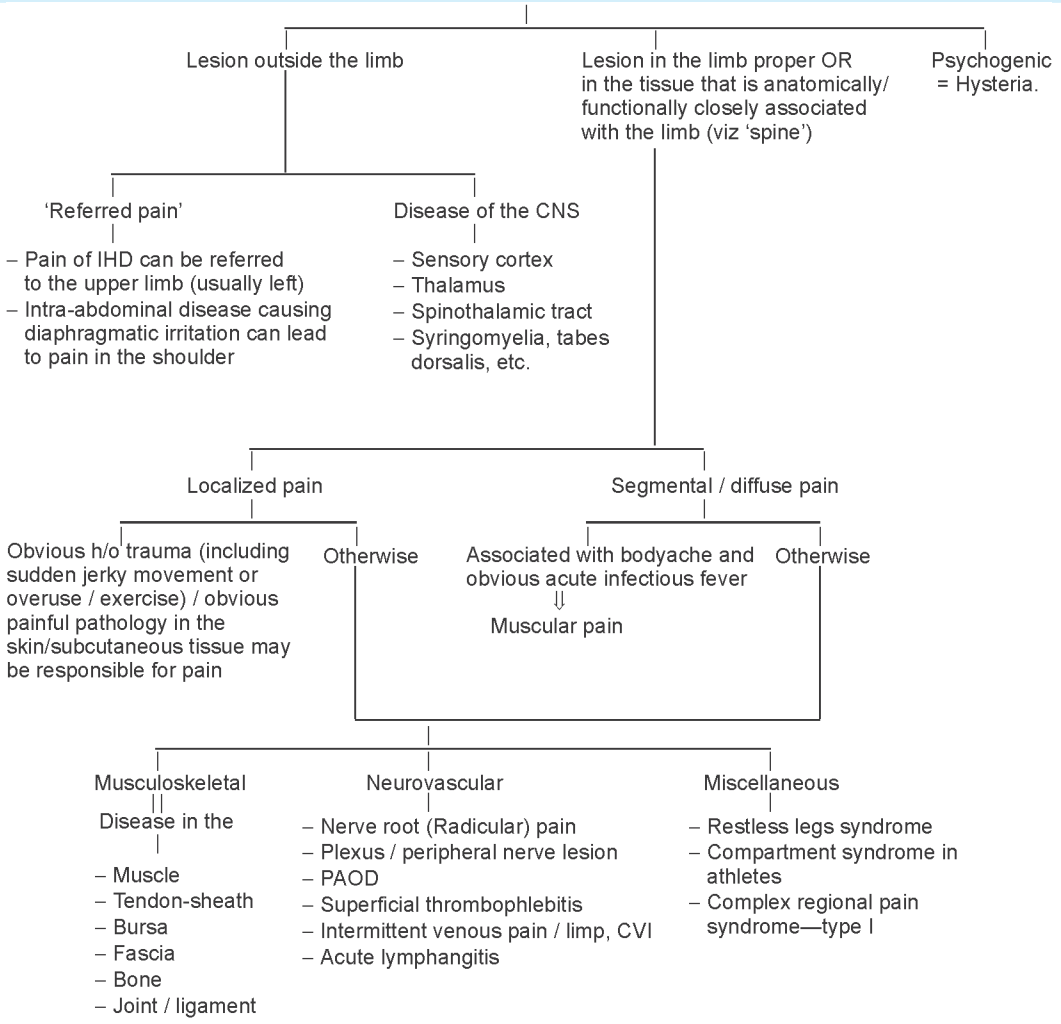
EXPOSE both the limbs completely for comparison.

LOCOREGIONAL EXAMINATION

- **Asymmetry**?
  - May be due to wasting or edema.
- **Attitude** of the limb? A fixed attitude suggests disorder of a joint or spasm of a muscle.
- Skin **rash**?
- **Swelling**?
- **Tenderness**?: Refer to chapter 5.
- **Weakness**?
- **Neurological deficit**?
- **Pulsations**?

www.Ebook777.com

ARRIVING AT A PROVISIONAL CLINICAL IMPRESSION



DIFFERENTIAL DIAGNOSIS

Cervical degenerative disease (e.g. spondylosis)

– Pain in the neck itself, and referred to the cervical paraspinal muscles, shoulder and arm—typically as far as but not beyond the elbow
– Unilateral or bilateral, or it can move from one side to the other
– Aching in nature.

Cervical degenerative disease is extremely common and few people escape some degree of neck pain at sometime in their lives.

Degenerative spinal pain mimicking the disorders of hip and thigh

– Low back pain, and pain referred to the paraspinal muscles, hip, groin, buttock, thigh—typically as far as but not below the knee. Pain in the thigh is almost always on the anterior, lateral or posterior aspect and virtually never on its medial aspect
– Beware of pain on the medial aspect of the thigh—it could be due to a pelvic pathology instead, (particularly malignancy) irritating the obturator nerve

Free ebooks ==> www.Ebook777.com

- The referred pain can be more severe than the low back pain itself, so that an alternative diagnosis such as osteoarthrosis of the hip may be thought of (wrongly)
- Referred pain can extend from the groin into the scrotum and testis, mimicking inguinal and scrotal disorder
- Aching in nature
- Brought on by 'using' the lower back such as on bending, lifting heavy objects, standing or sitting
  Pain is relieved by lying down.

Pain in the fascia / bursa / tendon-sheath / ligament may be perceived 'relatively superficially' or 'relatively deep'.

- When perceived 'relatively superficially', the pain is well localized. A therapeutic test (if required): Infiltration of local analgesic in the area of pain relieves the pain
- When perceived 'relatively deep', the pain is diffuse, continuous and steady.

Muscle pain

- Pain increases after exercise and decreases after rest
- Muscle may become more tense and firm than usual.

Causes of muscle pain

- Strain = an overstretching or overexertion of some part of the musculature; → localized pain and tenderness (both direct and indirect (indirect means when the muscle is put in its action)).
- *Intramuscular hematoma*
- *Myositis ossificans*
- *Intramuscular abscess*
- *Muscle tumor*

  → localized pain and swelling.
- *DVT* → diffuse pain
- *Vitamin B-complex deficiency, Ca deficiency* → diffuse pain
- *Trichinella spiralis infiltration* → diffuse pain
- *Fibromyalgia* → generalized pain at rest with localized tender areas
  - No objective clinical / laboratory abnormalities
  - Psychiatric symptoms commonly coexist
- *Polymyalgia rheumatica* → generalized pain particularly in proximal muscles
  - Elderly patient
  - Often associated with giant cell arteritis
- *Benign myalgic encephalomyelitis* → generalized pain induced by exercise
  - Fatigue
  - A puzzling disorder often occurring after viral illness
  - May be merging with depressive symptoms
  - No clear diagnostic criteria
- *Metabolic myopathy* → generalized pain induced by exercise.
  - Family history ±
  - Weakness / wasting
  - Swelling
- *Inflammatory myopathy* (*Dermatomyositis*) → generalized pain.
  - Weakness / wasting
  - Skin rash
- *Parkinson's disease* → generalized pain at rest
- *Collagen vascular disease* → pain at rest
  - Skin rash
- *Drug-induced myopathy*:
  - H/o exposure to toxin
  - Weakness / wasting

www.Ebook777.com

- *Alcoholic myopathy*
- *Physiological* pain—is induced by exercise
- *Tetanus*
- *Tetany*
- *Spasticity*
- *Neuroleptic malignant syndrome*

  } → Stiffness / spasm

- *Malignant hyperthermia* → Stiffness / spasm
  - The condition is characterized by a sudden rise in body temperature while undergoing GA.
  - Certain hereditary myopathic disorders, e.g. myotonic dystrophy, central core disease are unduly prone to this condition.

Causes of **tendon pain**: Tenosynovitis—simple / suppurative / stenosing.

Simple tenosynovitis

- H/o excessive / unaccustomed use commonly of the hand / foot
- Commonly seen in connection with the extensor tendons of the hands, and the tendoachillis
- Local edema
- Soft crepitus (like rustling of tissue paper) may be palpable on moving the fingers.

Suppurative tenosynovitis: Refer to chapter 15.

Stenosing tenosynovitis

- A condition of unknown etiology in which the sheath of a tendon thickens, apparently spontaneously, so as to entrap the tendon. → pain and limitation of movements
- The most common examples: **1**. The common sheath of the abductor pollicis longus and extensor pollicis brevis tendons of the wrist ('de Quervain's disease), **2**. Retinaculae of the flexor tendons of the fingers and thumb in the palm ('Trigger finger').

Supraspinatous tendonitis ('Painful arc syndrome', 'Frozen shoulder')

- Pain felt in the shoulder especially on abduction and rotation (as the inflamed portion of the supraspinatus tendon passes between the head of the humerus and the acromion: this occurs in the middle 3rd of the abduction arc so as to give a typical clinical picture—*the painful arc syndrome*).
- Natural tendency to protect the shoulder by holding it still → formation of adhesions → rapid onset of shoulder stiffness
- Localized tenderness over the insertion of supraspinatus tendon.

Natural history: Pain and stiffness increase × 3 months but thereafter remain static usually × a further period of 3 months. Subsequently, pain gradually settles; the range of movement gradually increases until by about the end of 9 months and the joint returns essentially to normal.

'Frozen shoulder' is not a specific clinical or pathological event; it is merely a response of the shoulder to any locally painful pathological process.

Tennis elbow

- Pain in the elbow at rest, and in particular while moving the hand in such a way as to put the hand and finger extensors in contraction
- Localized tenderness on the lateral epicondyle at the site of origin of the extensors of the forearm.
- Active contraction against resistance of the extensors of the hand → tenderness.

Golfer's elbow

- A precisely similar condition to tennis elbow, save that it affects the origin of the flexor muscles of the forearm to the medial epicondyle
- Localized tenderness on the medial epicondyle, however, tenderness is often less well localized than in tennis elbow
- Active contraction against resistance of the flexors of the hand → tenderness.

Free ebooks ==> www.Ebook777.com

TOS (Thoracic Outlet Syndrome)

= symptoms in the upper limb (including the hand) due to compression of nerves or/and blood vessels within the thoracic outlet.

Actual compression occurs between two normally, and occasionally with one abnormally present anatomical components of the thoracic outlet, and accordingly the patient may be said to have a cervical rib syndrome, scalenus anticus syndrome, costoclavicular syndrome, hyperabduction syndrome, etc. However, all ultimately belong to the common heading of TOS.

Clinical types according to the primary structure subjected to compression: 1. Arterial TOS (subclavian artery), 2. Venous TOS (subclavian vein), 3. Neurogenic TOS (brachial plexus nerve roots).

The diagnosis of neurogenic TOS, as compared to vascular TOS, often remains more difficult, confusive, and elusive.

Obstruction of the subclavian vessel(s) is less frequent and often present in conjunction with nerve compression.

Accurate diagnosis and correct patient selection for treatment remains a core problem due to the wide range of symptoms. This is compounded by the fact that there is no one diagnostic investigation specific to the syndrome.

Coexistence of neurogenic and vascular types of TOS affects the decisions and plans for surgical treatment.

Neurogenic TOS

Clinical pattern recognition of symptoms is *the basis of diagnosis*. Physical examination, including some specific clinical tests may be supportive. Reports of specific investigations are negative or equivocal in most cases; diagnosis remains essentially clinical. The principal value of investigation is to exclude other conditions, and strengthen the diagnosis of neurogenic TOS.

Differentiating points

– Pain extending up to shoulder, neck, and upper back
– The lack of distribution of symptoms into patterns referable to a single peripheral nerve.

Supporting point: Localization of the 'trigger points' over the scalene triangle.

Predisposing factors: 1. Occupational or recreational activities involving repeated use of the arm/s in elevated position.

2. A certain spectrum of injuries to the head/neck/upper limb.

Patients with no apparent predisposition may also suffer from neurogenic TOS.

Age and gender: Common in women between 20 and 40.

Symptoms

- *Pain and paresthesia* throughout the hand and arm without any localization to a specific peripheral nerve distribution; may involve different areas of the upper extremity.
  Pain may extend up to shoulder, neck, and upper back. Symptoms in the neck and proximal back may be functionally more disabling.
  May be unilateral or bilateral.
- *Positional effects*: The symptoms are reproducibly exacerbated by activity that requires elevation or sustained use of the arm/hand, e.g. simply reaching for objects overhead, lifting, prolonged typing, combing, etc.
- *Headache*: Typically occipital; most likely referred from spasm of trapezius and paraspinous muscles.
- *Weakness and muscle atrophy* may indicate severe and long-standing problem.
- *Disability* of mild and tolerable degree is associated with positional complaints in the majority of individuals.

To some extent, such symptoms are common in the normal population.

Progressively disabling symptoms are associated with clinically significant neurogenic TOS. This is typically associated with a long h/o consultations and partial/ineffective treatment.

- *Vascular symptoms* may be associated.

Examination

- Assess both the passive and active range of movement of the upper extremity and the neck.
- Examine for tenderness over the shoulder joint for rotator cuff tendonitis.
- Examine for tenderness over the trapezius. The presence of tenderness may indicate fibromyalgia.
- Identify the extent of any local muscle spasm over the scalene triangle itself as well as over the pectoralis, trapezius, and parascapular muscles.
- Localize the specific areas that reproduce symptom pattern upon focal digital compression. Such localized areas are called 'trigger points'.
- Examine the peripheral nerves to exclude ulnar nerve entrapment or carpal tunnel syndrome.

www.Ebook777.com

Functional assessment

– The extent of disability?
– The patient's expectations for the potential to continuing working?

Differential diagnosis *of neurogenic TOS*

Condition	*Differentiating features*
– Cervical disk disease	Neck pain and stiffness, arm weakness, and paresthesia involving thumb and index finger (C_5-C_6 disk). Symptoms improve with arm elevation. Positive findings on CT/MRI
– Cervical arthritis	Neck pain and stiffness. Arm and hand paresthesia infrequent. Positive findings on X-ray spine
– Cervical spine strain / sprain	(Post-traumatic) Neck pain and stiffness localized posteriorly along the cervical spine. Paraspinal tenderness. Relief with conservative measures over weeks to months
– Brachial plexus injury	Caused by direct injury or stretch. Arm pain and weakness. Hand paresthesias. Symptoms—constant; not intermittent or positional. Positive findings on neurophysiologic studies
– Rotator cuff tendonitis	Localized pain and tenderness over biceps tendon, and shoulder pain on abduction. Positive findings on MRI. Relief from NSAID, local steroid injection, or arthroscopic surgery
– Fibromyositis	(Post-traumatic) Inflammation of trapezius and parascapular muscles. Tenderness, spasm and palpable nodules over the affected muscles. May coexist with TOS and persist after surgery
– Ulnar nerve compression/ entrapment	Pain and paresthesia in the hand in ulnar nerve distribution, positive findings on nerve conduction studies
– CTS	Pain and paresthesia in the hand in median nerve distribution, positive findings on nerve conduction studies

Persistent neurogenic TOS

Criterion: The absence of relief of symptoms even for a short time following the initial surgery.
The cause of persistent symptoms in most cases is another condition. The diagnosis should be completely re-evaluated. Consider the possibility of persistent TOS if the symptoms do not respond to conservative treatment for at least several months.

Recurrent neurogenic TOS

Criteria: Patient having got a good symptomatic improvement from operation, later experiences symptoms. In majority of cases recurrences are observed within the first 2 years of the primary operation.
The symptoms are often the same as those present before the initial operation, and the diagnosis is made in the same manner essentially on the basis of clinical criteria.

Arterial TOS: Refer to chapter 46.

Venous TOS: Refer to chapter 50.

Patellar tendonitis

Pain and tenderness in the ligamentum patellae either at its attachment to the patella or at its distal attachment to the tibia.
Tenderness is felt also on active extension of the knee.

Achilles' tendonitis

Pain due to inflammation and thickening of the loose connective tissue in which the Achilles' tendon slides, OR
Pain may originate at the site of insertion of the tendon.
Causes: – A period of greatly increased unaccustomed walking.
– Wearing of shoes with ill-fitting heel.
– Occasionally, could be spontaneous.

Free ebooks ==> www.Ebook777.com

Plantar fasciitis
Pain and localized tenderness under the heel.
Pain is felt especially at heel-strike, and interferes with walking.
Intensity of pain—often severe.
Etiology—uncertain.

Carpal tunnel syndrome
- Pain classically localized to the distribution of the median nerve in the hand, but may be felt anywhere in the arm
- The poor localization of the pain may make it easy to confuse the condition with other causes of pain in the arm
- Paresthesia
- Incoordination
- Fingers may feel stiff, especially first thing in the morning
- Frank weakness of the muscles of the hand innervated by the median nerve (the easiest to examine – abductor pollicis brevis, wasting of which is obvious).

O/E: Often no +ve findings.
- Slight tenderness overlying the carpal tunnel ±
- Holding the fingers and the hand in fully flexed position × a few minutes may → pain / ↑ in pain
- Abnormal neurological signs in the distribution of the median nerve in the hand ±.

Differentiating point: Pain worse at night; may be relieved by hanging the hand over the edge of the bed.
A good description from the patient of the distribution of the sensory disturbance in the hand is usually diagnostic.
Causes of carpal tunnel syndrome:
- Unknown in an otherwise normal person.
- Pregnancy
- Post-Colles' fracture, RA, myxoedema.

Bursa is a fluid-filled cavity lined by flattened endothelium similar to synovium which develops between tendons / bones / skin to allow easier movement between them.
Types of bursae
1. Natural = the normal anatomical bursae, e.g. subscapular, prepatellar, olecranon, etc.
2. Adventitious—develop secondarily as a result of prolonged pressure over bony prominences, e.g. bunion of hallux valgus, Billingsgate hump.

Bursitis = inflammation of a bursa.
Types of bursitis
1. Simple bursitis: – Follows injury or unaccustomed exercise, e.g. inflammation of the bursa anterior to the tendoachillis following a cross-country run
2. Chronic bursitis: – Follows repeated pressure or slight injuries to the natural bursae, e.g. olecranon bursitis (student's elbow), prepatellar bursitis (housemaid's knee), infrapatellar bursitis (clergyman's knee), ischial tuberosity bursitis (weaver's bottom).
3. Acute suppurative bursitis results from direct infection by penetrating wounds or spread of local cellulitis.

Bone pain
Usual features
- Well localized
- Constant dull aching
- Swelling ±
- Localized tenderness.

Pain due to osteoporosis:
- Diffuse
- No local signs
- Difficult to diagnose without X-rays.

www.Ebook777.com

Common causes of bone pain:
– *In both upper/lower limbs*: Osteomalacia, tumor, osteomyelitis
– *Specific to lower limb*: Paget's disease.

Osteoid osteoma → – Persistent severe pain unrelieved by rest, more at night; relieved significantly by aspirin
– The only +ve physical sign may be bone tenderness, however even this is often absent; so the patient may be regarded as hysterical.

Joint pain
– Pain in distal (= distal to shoulder/hip) joints: Fairly localized
– Pain in proximal joints (Shoulder, hip, S-I and spine): Diffuse; felt in the whole limb
– Painful restriction of movements
– Pain starting immediately on walking—practically on taking the first step ⇒ osteoarthrosis
– 'Fixed attitude'
– Swelling is evident in case of superficial joints
– Wasting of surrounding muscles ±
– Tenderness
– Muscle spasm.

Common causes of joint pain:

In both upper/lower limb	*Specific to upper limb*
Osteoarthrosis	Calcific tendonitis
Arthritis: RA, infective, etc.	Rotator cuff tear
Bursitis.	Tennis elbow (periarticular), Golfer's elbow.

Sprain = a joint injury in which some of the fibers of a supporting ligament are ruptured but the continuity of the ligament remains intact.

Neurogenic pain
– The most common cause of segmental/diffuse pain.

Usually, lesion in the proximal portion of nerve (root/plexus/trunk) leads to diffuse pain. However, lesion in the distal portion of the nerve also may give rise to (diffuse) pain in the whole limb.

Pain due to *nerve root lesion or pressure in the intervertebral foramen* = ***Radiculopathy***
– Pain ↑ed by coughing or by movement
– Paresthesias—almost as common as pain
– Motor weakness
– Areflexia.

} The neurological findings are 'pure', i.e. not only appropriate, but exclusive to the root compressed

The presence of diffuse arm/hand pain and sensory disturbance that is not confined to a single dermatome ⇒ either the possibility of radiculopathy is ruled out, OR suspect a multilevel compressive radiculopathy if the reasons are compelling.

Objective neurological signs are not needed to make the diagnosis, but the pattern and distribution of pain and sensory disturbance must be as close to that expected anatomically.

Pain due to ***plexus*** / ***peripheral nerve*** *lesion*
– Burning in nature
± Sweating, cyanosis, edema of extremity
± associated neurological deficit.

Common causes of neurogenic pain	*Upper limb*	*Lower limb*
Pressure on the **nerve root**	– Spinal cord tumor – Localized pachymeningitis – PID (Prolapsed Intervertebral Disc)	– Spinal cord tumor – Localized pachymeningitis – PID (Prolapsed Intervertebral Disc) – Spondylolisthesis – Ankylosing spondylitis – Osteoarthrosis

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

Common causes of neurogenic pain	*Upper limb*	*Lower limb*
Pressure in the **intervertebral foramen**	– Deformities of the spine – Collapse of the vertebra – Spondylosis – Hyperabduction arm syndrome – Subclavian artery aneurysm – Infiltration by Pancoast's tumor or other cancer in the lower part of the neck.	– Deformities of the spine – Collapse of the vertebra – Spondylitis – Sacralization of the 5th lumbar vertebra – Osteoarthrosis
Pressure on the **nerve plexus or trunk**	– Thoracic outlet syndrome – Brachial neuritis / neuralgia – Postirradiation damage – Neurofibroma	– Pregnancy – Psoas abscess – Intrapelvic tumor
Other causes	– Neuritis, Herpes zoster – Injury—causalgia (complex regional pain—type II) – Elbow tunnel syndrome – Carpal tunnel syndrome – Radial neuropathy after humeral fractures – Traction on nerves due to occupation / deformities	– Neuritis – Injury—causalgia (complex regional pain—type II) – Sciatic nerve tumor

Spinal degenerative disease	***v/s***	***Radiculopathy***
– Pertaining cervical degenerative disease: If there is referred pain in the arm, it is confined to the arm, i.e. does not extend beyond the elbow		Radiating arm pain in c/o cervical radiculopathy—almost always present going beyond the elbow as far as the wrist or hand
– Pertaining lumbar degenerative disease: If there is referred pain in (the anterior, lateral or posterior aspect of the) thigh, it is confined to the thigh, i.e. does not extend beyond the knee		Radiating pain in c/o lumbosacral radiculopathy—almost always present all the way down as far as the foot Exception—L4 radiculopathy in which pain radiates only as far down as the knee
– No neurological deficit		Neurological findings if present are 'pure', i.e. exclusive to the root compressed

Radiculopathy *v/s peripheral nerve syndromes* (e.g. carpal tunnel syndrome, elbow tunnel syndrome, etc.)
Peripheral nerve syndromes can 'split' sensation in fingers, but nerve root syndromes never do so.
For example—the sensory disturbance from median nerve compression affects the thumb, index, middle and half of the ring finger. In ulnar neuropathy it involves the little finger and half of the ring finger.

PID
The factor which precipitates pain—prolonged standing, e.g. standing in a queue. Pain is relieved only by sitting or lying down; just standing still to take rest is not sufficient (*cf* intermittent arterial pain (Boyd's type III)). And pain takes longer time to get relieved, often > 20 minutes.

Spinal canal stenosis causing cauda equina compression
Pain associated with change in posture.
Associated neurological deficit ±.

Meralgia paresthetica
Burning, tingling pain over the outer aspect of the thigh, increased when standing or by walking.
A patch of sensory impairment may be evident over the outer aspect of the thigh.
Caused by neuritis of the lateral cutaneous nerve of the thigh.

www.Ebook777.com

Pain due to acute arterial insufficiency
Pain due to chronic peripheral arterial occlusive disease
} Refer to chapter 46.

Superficial thrombophlebitis
A painful inflamed cord extending along the course of a superficial vein.
Occasionally, thrombosis may be extensive—with severe pain and systemic toxicity.
Predisposing factors
– Cannulation for infusion
– A complication of varicose vein
– TAO (Smoker's arteritis)
– Blood dyscrasias, including polycythemia, thrombocythemia, leukemia
– CA—pancreas / bronchus / kidney.
} In these situations, thrombophlebitis occurs spontaneously.
Except when it arises in septic puncture sites, superficial thrombophlebitis is usually nonbacterial.

Acute lymphangitis
– Prodromal symptoms followed by high-grade fever with rigors
– Red blushes / streaks in the skin, corresponding to the inflamed lymphatics
– Patches of acute cellulitis along the course of lymphatics suggests more severe infection
– In most severe forms, abscesses develop at the site of lymphangitis
– Toxemia is often severe and is greater the more the infection has extended proximally.
Notes
– The condition is commonly caused by *Streptococcus pyogenes*, when infection spreads beyond a point of infection to the group of draining lymph nodes, where an abscess may form. Occasionally, the infection bypasses the 1st station of lymph nodes to affect another group at a higher level
– Recurrent attacks due primarily to filariasis, tend to develop in endemic areas.

Intermittent venous pain / limp
– A bursting pain or discomfort in the whole limb following use of the limb or exercise, relieved only by resting and elevation of the limb (as venous return is assisted by gravity)
– The pain is not relieved rapidly by simply standing still.
Caused by partial occlusion of usually the common or external iliac vein.

Pain associated with CVI (Chronic venous insufficiency)
May be associated with one or more of the following symptoms:
– Aching on dependency / before periods
– 'Heaviness' in the legs
– Restlessness in the legs
– Edema of foot
– Typical skin changes suggestive of CVI: (a) 'Stasis dermatitis', (b) Discoloration of skin in the gaiter area or lateral malleolar area: Ankle flare, hyper- or depigmentation, lipodermatosclerosis
– Active / healed / repeatedly recurring ulcer.

Restless legs syndrome
– Intolerable burning, tingling sensation or pain in both legs, occurring only when sitting or lying down, and relieved by walking.
– No associated neurological abnormality.

Compartment syndrome (shin splints) in athletes (*syn.* **anterior tibial syndrome**) after prolonged / unaccustomed exercise such as jogging.
– Severe aching in the anterior compartment of the leg
– 'Dropped big toe', i.e. inability to extend the big toe (as the extensor hallucis is the one that is affected first).
– O/E: The anterior compartment may be tense and tender.

Free ebooks ==> www.Ebook777.com

Complex regional pain syndrome (CRPS)

Clinical criteria are variable.

- Spontaneous/evoked pain/hyperalgesia beyond the territory of a single peripheral nerve and disproportionate to the initiating event
- Nature—any
- May be intermittent or may be constant
- May force the patient to adopt a protective posture
- Sympathetic dysfunction—reported as vasomotor instability: Variable symptoms: extremity may be warm and red or cold and blue, purple or mottled. Sweating particularly in palms/soles may be increased, decreased or unchanged
- Typically peripherally situated intermittent or permanent swelling may be reported at some stage
- Edema: Pitting / brawny
- Trophic changes in the skin: May be reported later in the course of the disease
- Motor dysfunction—dystonia / tremor / loss of strength
- Joint swelling and stiffness ±.

The condition excludes:

- Pain and other findings that are anatomically, physiological, and temporally appropriate to some form of injury
- Myofascial pain syndrome.

Types

- CRPS type I: Follows an inciting noxious event
- CRPS type II: Results specifically from a nerve injury.

Causes of CRPS

1. Traumatic: #, dislocation, sprain, crush injury, burns, etc.
2. Nontraumatic: Prolonged bedrest, thrombophlebitis, metabolic bone disease, MI, tumors, etc.
3. Idiopathic.

Causes of exercise-induced leg pain and / or weakness

- PAOD—very common
- Partial occlusion of usually the common or external iliac vein
- Neurogenic: Neurogenic pain is found only with severe lumbar degenerative disease, especially spinal stenosis.

Differentiating points in favor of intermittent arterial pain / limp:

- Pain distance is generally stable in the medium term; a patient with leg pain after walking 20 m on one day, after walking 200 m on another day and occasionally pain at rest is unlikely to be suffering from intermittent vascular pain
- In case of Boyd's type III: Pain disappears within 1 minute on standing still, leaving at most only a heavy feeling.

Differentiating points in favor of neurogenic pain:

- The commonest level involved is $L_{4/5}$, where the stenosis compresses the L_5 nerve root → pain radiating down the lateral aspect of the leg to the dorsum of the foot, and weakness of the dorsiflexors of the foot
- Usually both limbs are affected, but only one limb may be affected
- Classically a patient with major spinal canal stenosis walks or stands with the spine held flexed by 30°—the position which maximizes the space available for the central and lateral nerve roots. This is a good pointer to a spinal cause for the pain.

Differentiating points in favor of intermittent venous pain / limp:

- A bursting pain or discomfort in the whole limb, relieved only by resting + elevation of the limb
- The pain is not relieved rapidly by simply standing still.

ON THE BASIS OF PROVISIONAL CLINICAL IMPRESSION MADE, AND WITH THE HELP OF 'DIFFERENTIAL DIAGNOSIS', REVIEW THE CASE TO ARRIVE AT THE DEFINITE DIAGNOSIS.

www.Ebook777.com

46 ACUTE ARTERIAL INSUFFICIENCY AND CHRONIC PERIPHERAL ARTERIAL OCCLUSIVE DISEASE (PAOD)

DEFINITIONS OF TERMS

By convention, occlusion means a complete obstruction, and stenosis means partial obstruction (narrowing). 'Occlusive diseases' however, include stenotic conditions.

Ischemia is defined as a lacking in blood flow.
In any vessel, the lumen must reduce by 75% of the cross-sectional area, before there is a significant reduction in flow.

Acute arterial insufficiency = ischemia of < 2 weeks duration.

Chronic arterial insufficiency = ischemia stable for > 2 weeks.

Acute-on-chronic arterial insufficiency = worsening symptoms and signs (within a span of < 2 weeks) in a patient known to have a chronic arterial occlusive disease.

Anatomical classification of blood vessels: Arteries, microcirculation, and veins.

- *Major arteries* (*Syn*. *Large* arteries) = arteries with transporting function – 'Elastic' arteries—aorta and its major branches. They expand rapidly during systole and then recoil – the recoil maintains BP during diastole
- *Medium and small* arteries = arteries with distributive function – 'Muscular' arteries. They contract and dilate as appropriate depending upon the need of direction in which to shunt the flow of blood
- *Microcirculation* involves arterioles (0.1 mm), capillaries and venules.

Applied classification of limb vessels

- *Proximal*: - In the lower limb: vessels proximal to the popliteal, including the popliteal
  - In the upper limb: vessels proximal to the wrist, including radial and ulnar at the wrist
- *Distal*: - In the lower limb: crural vessels, and vessels in the foot
  - In the upper limb: vessels in the hand.

This classification has a therapeutic implication. A proximal vessel disease is more amenable to interventional corrective procedures, particularly surgical. A distal vessel disease is less amenable or not amenable to the same.

Arteriosclerosis

'Sclerosis' = hardening (*syn.* induration), especially from inflammation.
'Arteriosclerosis' = thickening (due to hardening) and inelasticity of arteries. – This generic term is applied for 3 patterns of arterial disease, all of which cause thickening and inelasticity: Atherosclerosis, medial calcific sclerosis, and arteriolosclerosis. Atherosclerosis is the dominant pattern.
Atherosclerosis → narrowing of the vascular lumen, and is also associated with degenerative changes in media and adventitia.
The consequences of complications of atherosclerosis can be serious in smaller arteries such as those of the heart and brain, because they may cause total blockage. Atherosclerosis has a predilection for arterial supply of the heart, brain and aorta.

- Cardiac ischemia → CAD (Coronary Artery Disease) (*Syns*. CHD (Coronary Heart Disease), IHD (Ischemic Heart Disease)) → various clinical manifestations like angina, MI and sudden death
- Cerebral ischemia and infarction → 'stroke'
- Aortic atherosclerosis predisposes to development of aneurysm, which may rupture and cause massive fatal bleeding
- Atherosclerotic narrowing of the ostia of renal arteries → renal ischemia and HT
- Involvement of the lower limb arteries viz iliac / femoral / popliteal → a chronic PAOD
- Rupture of a weakened artery may → intracranial bleeding.

Free ebooks ==> www.Ebook777.com

Since all the above-mentioned consequences of atherosclerosis form a group of 'closely related conditions', with a patient presenting with one of them, careful search must be made for the remaining.

GENERAL REMARKS

Risk factors especially for the development of occlusive disease

- Unmodifiable → Male gender, and strong f/h/o IHD
- Modifiable
  - Major risk factors
    1. Hypercholesterolemia. Causes / associations: Familial, diet rich in cholesterol, DM, hypothyroidism, etc. Familial cause is the most outstanding. There is no single level that is specifically risky. The higher the level, greater the risk. The risk is ↑ with ↑ LDL, and ↓ HDL levels
    2. Hypertension. Elevation of both systolic and diastolic pressures
    3. Cigarette smoking
    4. DM. → early development and rapid progression of atherosclerosis
  - Other risk factors
    1. Dietary factors: (a) Diet rich in cholesterol, CHOs. (b) Low levels of antioxidant vitamins viz. A, C, E and β carotene. (c) Softness of drinking water. "The softer the water, the harder the arteries"
    2. Obesity
    3. A lack of regular physical exercise
    4. A stressful competitive lifestyle
    5. Type 1A personality: Competitive nature, and less ability to control anger
    6. A prolonged use of OC pills

When ≥ 2 of the above-mentioned major risk factors are present, they multiply the risk.

Major (positive) **risk factors specifically** for the development of PAOD

- Men over 45
- Women over 55 or with premature menopause (no HRT)
- Family h/o premature atherosclerosis
- HDL < 35 mg%
- HT
- Smoking
- DM.

Medial calcific sclerosis

= calcifications in the media of muscular arteries (medium sized muscular arteries in individuals > 50 years of age).
→ nodularity on palpation, and readily visualized on X-ray.
There is no encroachment on the lumen; the condition is largely of anatomical interest alone.

Arteriolosclerosis

= sclerosis of small arteries and arterioles.
Most often associated with HT and DM.
Often prominent in kidneys.
Thickening of the walls → luminal narrowing.

Vasculitis

Definition: Inflammatory and often necrotizing changes in the walls of the vessels themselves.
The clinical features result from – 1. ischemia to the tissues supplied by the damaged vessel, and 2. widespread inflammation leading to constitutional symptoms viz. anorexia, malaise, fever, loss of weight.
The severity of symptoms may vary from a mild obliterative disorder to necrotizing vasculitis.
Five main categories of vasculitis syndromes: Polyarteritis nodosa group, hypersensitivity vasculitis, granulomatous (giant cell) arteritis, temporal (cranial) arteritis, Takayasu arteritis, specific disorders (smoker's arteritis (TAO), rheumatoid arthritis, SLE, etc.), and idiopathic.

www.Ebook777.com

Vasculitis syndrome	*Vessels involved*	*Organs/tissues affected*	*Remarks*
Polyarteritis nodosa	Medium and small arteries	GIT, liver, kidney, pancreas, muscles, other sites	The disease often becomes chronic and relapsing
Hypersensitivity vasculitis	Arterioles, capillaries, venules	Widespread, but particularly skin, serosal membranes, glomeruli	– Type III hypersensitivity reaction – Examples of various causal antigens: endogenous antigens, sulfonamide, penicillin, β-hemolytic streptococci, hepatitis B, etc. – Usually self-limiting, but can be recurrent / become chronic
Temporal (cranial) arteritis	Large, medium, small arteries	Widespread, predilection for temporal (cranial) arteries	-
Takayasu arteritis	Large and medium arteries	Aorta, pulmonary artery, origins of aortic branches	Typical victim: young women 20–30
TAO (*Smoker's arteritis*)	Small arteries, microcirculation, veins and nerves	Upper and lower extremities	– Remitting, relapsing inflammatory thrombosis with extension through vessel wall to involve veins and nerves. Cessation of smoking may bring relief from further attacks – Men between 25 and 50 who are heavy tobacco smokers – Extremely rarely reported in non-smokers or in women

Causes of acute limb ischemia

1. ***Mechanical injury***: Accidental OR iatrogenic.
   ↓
   Open or closed; with fracture or without fracture.

   Mechanisms of accidental traumatic ischemia:
   - Pressure from displaced fragments of fractured bone—the commonest
   - Pressure from an expanding hematoma
   - Occlusion due to hematoma in the wall of the artery itself
   - Intimal tear → elevation of a flap → occlusion
   - Minute intimal injury can induce thrombosis in turn leading to occlusion
   - Arterial spasm. This is an arteriographic / operative diagnosis of exclusion. **"Spelling of spasm = C L O T!"**. That means spasm as a possible cause of acute traumatic ischemia should never be made as a clinical diagnosis. Spasm may be considered as a retrospective diagnosis if all organic possibilities are excluded on surgical exploration.
2. ***Cold injury***—frostbite, immersion foot / hand.
3. ***Embolism***

   Causes / sources:
   - Left side of the heart: MS, MI, SBE, aneurysm (left ventricular), atrial myxoma, arrhythmia
   - Aorta: Aneurysm, atheromatous plaque
   - Large proximal artery (brachiocephalic / subclavian / iliac / femoral / popliteal)—stenosis, atheromatous plaque, aneurysm, intra-arterial drug abuse
   - In some cases the source of emboli is not found.
   - Peripheral vein thrombosis with atrial septal defect—called paradoxical embolism.

Free ebooks ==> www.Ebook777.com

Emboli usually lodge at one or more of the following sites.

- Aortic bifurcation
- Common femoral artery

} In lower limb

- The proximal brachial artery at the level of the profunda
- The brachial artery in the mid-arm level at the origin of the superior ulnar collateral
- The brachial bifurcation
- The radial or/and ulnar arteries

} In upper limb.

4. ***Acute thrombosis***—precipitated due to slowing of blood stream in debilitating and dehydrating illnesses, e.g. typhoid, etc.
   - Transient arrhythmias may cause transient hypotension sufficient to occlude critical peripheral arterial stenosis whether it is in native vessels or within peripheral arterial bypass graft
   - Thrombosis may develop *in situ* on an underlying arterial disease such as atherosclerotic stenosis, aneurysm or extrinsic compression (e.g. compression within the thoracic outlet)
   - Leriche's syndrome.

Note that cardiac arrhythmia (even if transient) can lead to acute limb ischemia by 2 mechanisms: (a) Embolism, (b) Arrhythmia → hypotension → thrombosis of peripheral arteries or bypass grafts with pre-existing critical stenosis. Nonetheless, prolonged hypotension from any cause may lead to slowing of blood stream → thrombosis and occlusion of even otherwise normal arteries.

Patients presenting with ALI can present a challenge to the most seasoned vascular surgeon. The therapeutic window to allow successful restoration of arterial blood supply without tissue loss can be quite small, and highly dependant on any preexisting arterial disease.

Skeletal muscle can withstand total ischemia for up to 6 hours before irreversible muscle damage will occur.

Causes of chronic PAOD

Macrovascular disease
- Atherosclerosis
- Vasculitis—TAO
(- Medial calcific sclerosis)
- Aneurysm
- Compression of the main artery
  Examples: An SOL, fibromuscular band, cervical rib, costoclavicular compression, hyperabduction syndrome, malposition syndrome, post-RT extensive fibrosis, etc.
- Ligation of the main artery

Microvascular disease / Tissue devitalization

Obstructive
1. Atherosclerosis: DM
2. Vasculitis
3. Microemboli arising from the heart / aorta / proximal vessels
4. Hyperviscosity syndrome
   - Myeloma
   - Polycythemia rubra vera

Vasospastic
1. Primary Raynaud's disease
2. Secondary Raynaud's syndrome
   - Thoracic outlet syndrome
   - Hand-arm vibration syndrome
   - Hammer-hand syndrome (syn hypothenar hammer syndrome)
   - Hypothyroidism
   - CTD, most commonly systemic sclerosis
   - Complex regional pain syndrome II (Reflex sympathetic dystrophy)
   - Drugs

There are ***2 major spectra of chronic PAOD***—1. Intermittent pain, and 2. Critical limb ischemia (CLI). Management strategies and outcome of these two are different.

The patient with a preceding h/o intermittent pains or even early rest pain will be seemingly able to tolerate a longer period of ischemia due to the existence of collaterals compared to the patient with previously normal vessels who having suddenly occluded their major inflow has no effective pre-established collaterals.

Ischemia of the upper limb occurs considerably less frequently.

The incidence and spectrum of vascular disease are widely different between the upper and lower extremities.

Lower extremity	*Upper extremity*
Atherosclerosis of arteries and DVT are most common	Atherosclerosis is rare in the upper extremity, however, and most commonly affects the left subclavian artery
DVT is associated with hypercoagulable conditions and stasis	DVT is occasionally associated with hypercoagulable conditions, but most commonly associated with trauma (thoracic outlet compression or central venous catheterization)

www.Ebook777.com

In other instances, the explanation for the differences in the distribution of the diseases in unknown. The distribution of diseases affecting the upper limb is unique.

Disease	*Subclavian*	*Axillary*	*Brachial*	*Forearm*	*Hand (digital)*
Atherosclerosis	√				
Giant cell arteritis	√				
Takayasu disease	√	√			
Fibromuscular dysplasia		√			√
Embolic		√	√		√
CTD				√	√
DM				√	√
Repeatitive trauma					√
Hypercoagulation					√
Cryoglobulins					√
Pressors / PVC					√

- For unexplained reasons, the upper limb contains more precapillary vasoconstrictors than the lower limb. As a result, vasomotor problems are more common in the hand
- The structures responsible for extrinsic arterial compression are bones, muscles, tendons, and ligaments. In upper limb, compression is most likely to occur at the thoracic outlet
- Digital gangrene implies significant multilevel disease or extensive palmar/digital arterial disease
- Although the clinical presentation of large arterial emboli is usually dramatic, symptoms often improve rapidly as the emboli break up and travel distally. The degree of ischemia seen on physical examination also improves, but rarely returns to normal
- Many patients with stenotic / occlusive disease in the brachio-cephalic or subclavian arteries will remain asymptomatic
- Major collateral arteries exist around all major joints in the upper limb. This accounts for the low incidence of symptomatic upper limb arterial insufficiency despite the presence of occlusive lesions.
- Occasionally, in the upper limb, it may be quite difficult to differentiate between acute and chronic arterial insufficiency as the extensive nature of collateral vessels can leave an arm asymptomatic despite quite severe disease
- Vasospasm does occur even normally in the hand in response to cold or strong emotional stimuli. Vasospasm severe enough to lead to complete cessation of blood flow is called **abnormal vasospasm**
- Patterns typical of lower limb ischemia are rarely seen in the upper limbs where coldness and discoloration of the digits are most common presenting symptoms, and ulceration and gangrene less frequent
- If left untreated, ischemia of the upper limb often results in severe functional impairment even in the absence of overt tissue loss
- Establishing the underlying etiology of upper limb ischemia is often more complex, particularly in chronic disease when it may be a manifestation of a systemic disorder
- Upper limb acute ischemia is generally a result of trauma or embolic disease. Acute thrombosis is usually secondary to an atherosclerotic stenosis, subclavian aneurysm or extrinsic compression within the thoracic outlet

In the case-taking that follows, some points are applicable to upper limb ischemia, some to lower limb ischemia and the remaining are applicable to both. Reader is expected to use discretion.

HISTORY

- **Occupation**
  - Does it involve the use of vibrating tools like (pneumatic) drilling machine, hammers, chain saws, grinders, wrenches, etc?
  - Does it involve a lot of typing?
  - Carpentry, plumbing, farming, mechanic?
  - Exposure to ergot or PVC (Polyvinyl chloride)?

Free ebooks ==> www.Ebook777.com

Do you pursue specific **sports** activity regularly?
- Baseball, handball, volleyball, tennis, golf
- Swimming, weight lifting.

Symptoms of arterial insufficiency basically depend upon 1. whether the insufficiency is acute or chronic, and 2. the severity of the degree of ischemia.

Chief complaint: Often, **segmental** / **diffuse pain** in the limb

↓

- **Duration**?
- **Mode of onset**?
- **Progress**, **regress**, **fluctuation**?
- **Intensity**?
- **Nature**?—throbbing / cramping / burning / bursting / shooting / aching / vague.
- **Precipitating/aggravating factor/s**?
  - Trauma / sudden jerky movement / overuse / exercise?
  - H/s/o acute infectious fever?

  OR
  - Did the pain begin spontaneously?
- Have you been suffering from **a cardiac** / **aortic disease**?

Interpretation at this juncture

- Severe agonizing continuous bursting pain, radiating down the course of an artery
- in a patient with a debilitating and dehydrating illness, e.g. typhoid, etc.
  OR in a patient known to have a cardiac/aortic disease
- Sudden mode of onset. Rapid/dramatic progress.

⇒ ***Acute arterial occlusion***

|

Further history to be asked

↓

- **Burning** / **tingling** / **numbness**?
  Cause of paresthesia: ischemia of peripheral nerve endings.
- **Paresis** / **paralysis**?
- **Coldness and deadness** of the limb?

History to be continued in a chronic case

- **Intermittent pain** in the limb?
  - Exact site?
  - Duration?
  - Mode of onset?
  - Nature?—cramping / aching / burning / bursting?
  - *In c/o lower limb:*
    - Is the pain brought on walking / exercise?
    - Does the pain come on walking more or less the same distance every time you walk?
    - If you continue to walk after the pain begins, does the pain disappear or remain steady, or does it worsen compelling you take rest?
    - Is standing still (to take rest) enough to relieve pain or do you have to lie down to get a relief from pain?
    - Have you noticed any change in the distance that you could walk comfortably? – Is the distance getting shorter?
    - Have you noticed any change in the rest time? – Is it getting prolonged? (This point is applicable to only Boyd's type III).
  - *In c/o upper limb:*
    - Is the pain brought on by writing / manual labor / exercise?

Instead of pain, sometimes there is feeling of only tiredness/weakness/sensation of cold or pins-and-needles.

www.Ebook777.com

- **Pain in the** (lower) **limb even at res**t?
  - Exact site?
  - Duration?
  - Mode of onset? Was it preceded by the stage of intermittent pains for months / years?
  - Nature?—cramping / aching / burning / bursting?
  - Progress?
  - Relieving factors?
- **Positional pain** and **paresthesia** in the **upper limb**?—may be suggestive of *thoracic outlet syndrome*.

  Do they seem to be occurring in the distribution of a specific nerve? } Neurological symptoms.
  - Which position of the arm precipitates/aggravates the symptoms? } Neurological symptoms.
    – Is it the elevated position?
  - Muscular weakness, particularly in the hand?
  - Intermittent cold discoloration of hands—often of a positional nature and frequently worse at night? } Arterial symptoms.
  - Easy fatiguability? } Arterial symptoms.
  - H/o hyperextension neck injury such as whiplash or occupational repetitive strain injury?
- **Chronic indolent ulcer** situated on tips of fingers / toes / in the legs?
  - may have been preceded by severe burning pain in the limb.
  - Discharge? Amount? – Discharge from a chronic indolent ischemic ulcer is usually scanty or almost absent.
- **Blackening of one or more digits** or **more extensive area** (s/o gangrene)?
- Do you get spasmodic (intermittent) attacks of a series of color changes in the skin of hands/feet (may be following exposure to cold or emotional stimuli, or may be even spontaneously)? (—**Raynaud's phenomenon**)
  - Sudden development of pallor and a sensation of cold and numbness
  - The color next changes to blue or deep purple, and finally to bright red. Pain and tingling are prominent during these later stages.
- Frequency of attacks?
- Within how much time do the symptoms settle?
- In between the attacks, are you completely normal, OR do you do experience some symptoms?
- Are the attacks followed by complete normality, OR do the attacks leave behind some degree of irreversible color change?
- **Acrocyanosis**?

  = The skin color changes described above (under the heading of Raynaud's phenomenon) are permanent rather than spasmodic.

  The areas affected are tips of the extremities, e.g. fingers, toes, tip of the nose.
- In c/o upper limb ischemia: Vertigo / dizziness / bilateral visual disturbances / ataxia? (⇒ **vertebrobasilar insufficiency** ⇒ subclavian artery occlusive disease)
- **Review of systems**
  - SOB, diminished exercise tolerance?
  - Pain in the chest, palpitations, syncope, HT, IHD, RHD?
  - Headache, giddiness, visual disturbance (blurring, diplopia), blackouts, stroke, convulsions?
  - Hypothyroidism?
  - Pain in abdomen after meals?
  - Increased or decreased frequency of micturition, color of urine, hematuria?
  - Chronic skin disorder, joint pains, bodyaches, RA, any other CTD?
- Contraceptive history? – Ask particularly with reference to OC pills.
- Have you been suffering from any other disease such as, e.g. **hepatitis-C**, **a viral infection**, **cancer,** etc?
- **Treatment taken**?

  Response to the treatment?

Free ebooks ==> www.Ebook777.com

- Have you been consuming **any medicines** / receiving any other treatment for any other disorder?
  - β-blockers for HT?
  - CT drugs?
  - Accidental intra-arterial injection of drugs meant for IV use?
- **Past history**
  - Similar illness in the past?
  - Injury, accident, operation especially in the recent past? (—for pseudoaneurysm)
  - H/o rheumatic disease?
- **Smoking** / consumption of **tobacco** in any other form?
- **Family history**
  - Of similar illness?
  - Of DM, HT, and IHD?
- **Personal history**: Appetite, sleep?

INTERPRETATION OF SYMPTOMS, AND DISCUSSION

Age

- Infants: Nonspecific symmetrical digital gangrene
- Children: Hereditary cold fingers
- 22–55: Smoker's arteritis
- 55 onwards: Atherosclerosis. However, diabetics can suffer from atherosclerotic occlusive disease at a younger age.

Gender

- Smoker's arteritis is almost exclusively seen in men
- Raynaud's disease is more common in women.

Occupation and sports

- Occupations involving **vibrations or small but repetitive trauma to fingertips** as in typists can lead to persistent vasospasm
- Incidence with respect to duration of the job involving the use of vibratory tools: About 50 percent of workers are affected by 3 years. The condition may not improve after discontinuing the use of vibratory tool; in some it may even worsen
- Vibrations lead to a fixed digital ischemia and finger-tip necrosis by various mechanisms causing structural arterial damage
- Early symptom which may suggest the development of vasospasm: Paresthesia
- Hammer-hand syndrome (*syn.* hypothenar hammer syndrome; basically it is a Raynaud's) occurs with repetitive pounding to the palm in those people who use their hand as hammer as in (a) baseball, handball, volleyball, tennis players, and golfers, and (b) carpenters, plumbers, farmers, etc. who all use hand-held tools such as hammers, wrenches. Repetitive trauma to the palm from using the hand as a hammer leads to damage (an aneurysm formation, thrombosis, and subsequent embolization to the fingers) to the palmar branch of the ulnar artery
- Thoracic outlet syndrome occurs in swimmers, weight lifters
- Exposure to ergot, vinyl chloride may → vasospasm
- Low socio-economic status predisposes to smoker's arteritis.

'Intermittent arterial pain / limp' (*syn.* 'intermittent claudication')

= pain brought on by activity like walking / exercise, etc. of reproducible degree. Pain or other symptoms may give a sort of limp; actual limping itself is quite unusual.

– The level of arterial block is usually one joint proximal to *the site of pain*.

Site of pain	*The most probable level of block*
Sole	Tibials
Calf	Femoropopliteal. Exceptionally, it may be due to aortoiliac disease in many cases
Calf and thigh	Iliofemoral
Calf, thigh and buttock	Aortoiliac

- *Mode of onset:* Insidious.
- *Stages in the progress* of the symptom (judged from Boyd's classification)
  - Type I: Walks—pain—continues walking—pain disappears
  - Type II: Walks—pain—continues walking—intensity of pain remains steady—can continue to walk
  - Type III: Walks—pain—continues walking—intensity of pain increases—forced to take rest.

www.Ebook777.com



Fig 46-1 The typical posture adopted by a patient with rest pain in the foot.

– *Progress* is judged from 'pain/limp distance' and 'rest time'
  - Pain distance = the distance at which pain/discomfort starts in the muscles on walking
  - Progressive decrease in this distance ⇒ progress of the disease. However, it is a subjective distance which can vary from day-to-day
  - Various variables to be kept in mind before drawing a conclusion: the speed of walking, walking uphill against walking on the plain road, walking against wind, changes in general condition like anemia, etc.
  - Rest time—applicable to Boyd's type III = the time for which patient is compelled to take rest before walking can be resumed. Progressive increase in rest time ⇒ progress of the disease
– *Other causes* of intermittent pain from which intermittent arterial pain / limp needs to be differentiated:
  - Venous pain / limp
  - Osteoarthrosis
  - PID
  - Sciatica

  } For differential diagnosis, refer to chapter 45.

Diagnostic implications of intermittent arterial pain / limp

1. It is a very important symptom s/o chronic arterial insufficiency.
2. It gives a fairly accurate idea about the level and the severity of block.

'Rest pain'

= severe unbearable pain not readily controlled by analgesics, felt in the foot (occasionally may extend upwards to the thigh) even when the patient is at rest.

– Continuous
– Can be throbbing
– Usually follows the stage of intermittent pain for a few months / years
– Progress—rapid
– Often worse at night, due to reduction in sleeping cardiac output
– Lying down or elevation of foot worsens the pain, due to the absence of gravity-induced hydrostatic pressure
– May be relieved somewhat, if at all, by sitting with the affected limb hanging over side of the bed or drawn close to the body.

Pathogenesis of rest pain: Ischemia of somatic nerves. "It is the cry of dying nerves".

Rest pain ⇒ 'Critical limb ischemia'

Other causes of deep-felt and similarly intractable pain from which rest pain needs to be differentiated: Pain of nerve or nerve root irritation.

Differentiating point	*Arterial rest pain*	*Pain due to nerve / nerve root irritation*
– The distribution of pain and paresthesia	Stocking distribution	Characteristic of a nerve / nerve root
– Abnormalities of motor function and reflexes	Absent	±

Raynaud's phenomenon

– Raynaud's phenomenon may be encountered as an early manifestation of true occlusive disorder.
– Symptoms usually settle within 15–20 minutes.
– Associated h/o skin rash / joint pains / bodyaches (muscle pains) / dysphagia / RA, etc. ⇒ secondary Raynaud's due to a CTD
– True vasospasm is invariably intermittent. **An exception** to this is chemical-induced vasospasm such as in the case of ergotism, toxins or various substances used by intravenous drug abusers. In these situations, prolonged vasospasm may result in irreversible damage.

Free ebooks ==> www.Ebook777.com

Nonhemispherical neurological manifestations (i.e. vertigo / dizziness / bilateral visual disturbances / ataxia) and Intermittent arterial pains in the upper limb = '**Subclavian steal syndrome**'—most frequently encountered on the left side.

Review of systems
The review of systems symptoms will serve important purposes: 1. Detect etiology. 2. Detect other systems which might have been affected by virtue of certain vascular disorders being generalized, e.g. atherosclerosis.
EXAMPLES
- Pain in the chest may suggest IHD
- RHD may be responsible for an (acute) embolic gangrene
- Hematological disorder particularly causing increased blood viscosity, e.g. polycythemia, myeloma, cryoglobulinemia, lymphoma, etc. → microvascular occlusive disease of the digits
- Headache may be migrainous, for which an ergot alkaloid might have been consumed
- Giddiness / syncope / stroke suggest CVD
- Hypothyroidism, Grave's disease, Addison's disease, Cushing's may be responsible for microvascular spasm in the digits
- Chronic pain in abdomen typical of 'intestinal angina' may suggest chronic mesenteric ischemia
- RA / other CTDs may be responsible for vasculitis.

OC pills may predispose to increased tendency to VTE.

Disorders which may lead to microvascular occlusive disease of the digits:
- Purpura fulminans, parvovirus, etc.
- Digital ischemia can be a paraneoplastic syndrome
  - Mode of onset of digital ischemia—sudden
  - Asymmetrical involvement
  - Severe symptoms, in many cases progressing to digital gangrene
  - Some patients who present with unexplained digital occlusive disease later in follow-up are found to have had an occult cancer
  - Treatment of cancer may result in remission of Raynaud's.

Drugs
β-blockers used for HT may lead to Raynaud's. However, the drug is not contraindicated in every patient with Raynaud's.
Many other drugs and CT drugs may be responsible for secondary Raynaud's.
Accidental intra-arterial injection of drugs meant for IV use can cause severe vasospasm leading to digital ischemia with gangrene and digital loss.

Smoker's arteritis (TAO)
Medium-sized arteries onwards (i.e 'distalwards') are affected.
Modes of presentation: 1. Chronic ischemia with very painful digital tip ulcerations and necrosis. 2. Distal extremity vasospasm.
"No tobacco, No TAO".

Family h/o HT, IHD ⇒ atherosclerosis.
Atherosclerosis, DM are important factors which predispose to a PAOD.

Appetite and sleep of a patient suffering from rest pain may be severely affected.

POSITION of the patient: Supine, with both limbs exposed adequately for comparison.

LOCOREGIONAL EXAMINATION IN ACUTE LIMB ISCHEMIA

INSPECTION

- **Edema**, **generalized blue discoloration**, and **large collateral veins**? ⇒ venous ischemia.

Further clinical points refer to arterial insufficiency

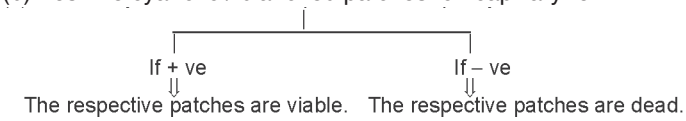
- **Discoloration**?—serial color changes occur with successively advancing degree of ischemia. **Extent**?
  1. Marked pallor.
  2. Mottled (= marbled) appearance = mixture of areas of pallor, cyanosis and redness. Pallor is due to the lack of blood in microcirculation. Cyanosis is due to extremely sluggish flow of blood, which → maximum deoxygenation of Hb. Redness is due to reactive hyperemia.

www.Ebook777.com

Buerger's test is very useful in these patients:

Step I: Elevate the limb and — (a) Inspect in general. Occurrence of blanching suggests that capillary integrity is still maintained.

(b) Test the cyanotic / blanched patches for 'capillary refill'



Step II: Ask the patient to sit up and hang the limb down. If most of the skin becomes bright red, then it is viable.

Mechanism: reactive hyperemia. The duration it takes to become bright red depends on the severity of ischemia. The longer this takes, more critical is the ischemia.

Some of the above-mentioned signs, which are mainly dependent on changes in color of the skin, can be appreciated only in fair-skinned patients.

3. Congested and purple-blue cyanosed appearance ⇒ pregangrenous stage.
4. Black discoloration ⇒ frank gangrene.

- **Trophic changes** s/o (a pre-existing) chronic arterial insufficiency?
  - Wasting, thin and shiny skin, sparse hair, shrunken digits due to loss of pulp, deformed nails with transverse ridges, and chronic indolent ulcer
  - The presence of trophic changes basically suggests ALI occurring in the already diseased arterial tree. The most probable cause of ALI in this setting could be acute thrombosis
  - *A word of caution with respect to therapy*: Injudicious use of balloon thromboembolectomy can cause intimal disruption and put the limb at further risk rather than saving it.
- '**Venous guttering**'?
  = venous channels are depressed beneath the skin surface, and look like pale blue gutters.
- **Extrinsic abnormality along the course of main artery**?

PALPATION

- (Skin) **Temperature**?
  Clinical method
  - Feel with back of the fingers
  - Compare with the opposite side
  - Check from distal to proximal to determine the possible level of arterial block.

  Usual finding: Cold. *Fallacy*: **The limb may feel warm if it was covered with a blanket just prior to examination**.
- **Tenderness**?
  - Tenderness of the (apparently) affected muscles ⇒ necrosis (bad prognosis).
  - Tenderness along the artery ⇒ recent thrombosis
  - Excruciating tenderness on passive flexion / extension of the digits / foot / hand causing stretching of tendons suggests possibility of acute compartment syndrome.
- **Consistency**?
  - Suppleness of the muscles ⇒ advanced ischemia, although salvageable—indicates urgency of the situation
  - Stiffness due to rigor mortis ⇒ grade 3 (i.e. nonsalvageable) ischemia
  - Does the limb feel unusually tense?
  - Could be due to acute compartment syndrome
- **Pitting on pressure** ⇒ thrombophlebitis
- **Sensation**?
  - 'Partial' (or 'inconstant') loss of sensation qualifies for grade 2 ischemia
  - Profound anesthesia qualifies for grade 3 ischemia.
- **Pulsations**?

  The level of block lies proximal to the highest impalpable pulse and distal to the lowest palpable pulse.

Free ebooks ==> www.Ebook777.com

- **Function**?
  - The absence of muscle weakness ⇒ grade 1 ischemia. *A fallacy:* Intact tendon attachment may produce movement of the dead digit
  - Paresis ⇒ grade 2 ischemia
  - Paralysis ⇒ grade 3 ischemia.
- **Any palpable abnormality along the course of the main artery**?
  In the case of upper limb, do not forget to palpate for a cervical rib in the supraclavicular fossa. In the absence of an obvious mass, tenderness may be suggestive of a cervical rib.

AUSCULTATION

- Bruit ⇒ stenosis. Bruit is present beyond the site of stenosis
- Systolic murmur ⇒ aneurysm
- Continuous machinery murmur ⇒ AV fistula

SYSTEMIC EXAMINATION

In cardiovascular system examination, specifically search for the source / cause of acute embolism.
In nervous system examination, specifically search for simultaneously present or past e/o cerebrovascular insufficiency.

Clinical grading of the severity of acute ischemia

Grade	*Sensory loss*	*Muscle weakness*	*Doppler signals*		*Inference / comments*
			Arterial	*Venous*	
1	None	None	Audible BP > 30 mm Hg	Audible	Viable. Salvageable
2	+	±	Inaudible	Audible	Threatened; but salvageable if promptly treated
3	Anesthesia	Paralysis. In the end, rigor mortis (i.e. stiffening)	Inaudible	Inaudible	Major tissue loss Nonsalvageable. Permanent nerve damage inevitable

Always revaluate the case at the end of ~ 1 hour.

LOCOREGIONAL EXAMINATION IN CHRONIC LIMB ISCHEMIA

INSPECTION

- **Edema**? Dependent edema is usually present in a patient having rest pain in the foot. **Extent**?
- **Asymmetry** between the 2 limbs?, *unless both are affected.*
  Causes:
  - Wasting of the tissues. Wasting is one of the trophic changes
    OR
  - Dependent edema usually in a patient having rest pain in the foot.
- **Discoloration**?
  - Pallor / cyanosis / red / purple—indicate pregangrene
  - Black discoloration of a part ⇒ gangrene.

  Extent of discoloration?
- **Trophic changes**?
  (Other than wasting of the part.)
  - Skin in general looks thin and shiny (due to edema)
  - Hairs—sparse
  - Digits—shrunken due to the loss of pulp
  - Nails—lusterless, deformed, and show transverse ridges
  - A chronic indolent ulcer ±.

www.Ebook777.com



Fig 46-2 Wasting of the right leg, and gangrene of the forefoot

- **Venous guttering**?
  = venous channels are depressed beneath the skin surface, and look like pale blue gutters.
- **Extrinsic abnormality along the course of main artery**?

PALPATION

- (Skin) **Temperature**?
  Clinical method
  – Feel with back of the fingers
  – Compare with the opposite side
  – Check from distal to proximal to determine the possible level of block.
  Finding
  – Cold temperature suggests ischemia
  – Normal temperature suggests normality; however, in early (noncritical) stages of ischemia, temperature may be normal. So, normal temperature does not necessarily rule out arterial insufficiency.
  A fallacy: **The limb may feel warm if it was covered with a blanket just prior to examination.**
- **Tenderness**?
  – Tenderness of the (apparently) affected muscles ⇒ necrosis
  – Tenderness along the artery ⇒ recent thrombosis.
- **Pitting on pressure**? ⇒ thrombophlebitis
- **Sensation**? Sensation is preserved unless gangrene has supervened
- **Pulsations**?
  – Weak / absent ⇒ ischemia
  – Normal pulsations ⇒ normality / asymptomatic disease / mild intermittent arterial pain / limp.

↓
Carry out the 'disappearing pulse' test
↓
Ask the patient to exercise to the point of pain, and immediately palpate the pulses again

Pulsations disappear, and reappear after a period equivalent to 'rest time' in the given case	Normal pulsations continue to remain palpable
⇓	⇓
The presence of occlusive disease	Occlusive disease may be clinically ruled out for the time

Explanation: Exercise → vasodilatation as a normal physiological response. The arterial inflow reduced by occlusion, cannot keep pace with increasing vascular space caused by vasodilatation. → a fall in arterial pressure leading to disappearance of pulse.
Basically, whether the pulses distal to the level of block will be absent or present, and when present, feeble or normal, will be determined by the combination of 2 factors viz. the level of block, and how well the collateral circulation is developed. In a patient giving h/o intermittent pains, even if various peripheral pulses in the lower limb are palpable and normal, the possibility of PAOD should not be ruled out without doing the 'disappearing pulse' test.

Free ebooks ==> www.Ebook777.com

Inference when pulsations are weak/absent: The level of block lies proximal to the highest impalpable (or weak) pulse and distal to the lowest palpable (normal) pulse.

- **Function**?
  The absence of muscle weakness does not rule out gangrene, as intact tendon attachment may produce movement of the dead digit.
- **Any palpable abnormality along the course of the main artery**?
  In the case of upper limb, do not forget to palpate for a cervical rib in the supraclavicular fossa. In the absence of an obvious mass, tenderness may be suggestive of a cervical rib.
- **Measurement**
  For objective evidence and documentation of wasting which may not be grossly apparent, measure the circumference of both limbs (for comparison) at a fixed distance from a fixed anatomical landmark.

AUSCULTATION

– Bruit ⇒ stenosis. Bruit is present beyond the site of stenosis
– Systolic murmur ⇒ aneurysm
– Continuous machinery murmur ⇒ AV fistula.

VARIOUS TESTS with specific purpose

Capillary refilling time
= the time taken by a blanched area to return to normal color.
Press down the tip of a nail / pulp of a digit / a cyanotic patch for a couple of seconds to make it blanched.
Release the pressure, and observe the time taken for return of normal color.
Normal time—just a couple of seconds.
Longer the refilling time, poorer the state of circulation.

Buerger's test and capillary filling time
– Elevate the (lower) limb slowly, and observe for the development of pallor
– The angle at which pallor develops is called '*the Buerger's angle of vascular sufficiency*'
– Lesser the angle, greater the insufficiency. The angle of ≤ 20° ⇒ critical ischemia
  (In a normal person, pallor does not develop even when the limb is elevated to 90°.)
– Now ask the patient to sit up hanging the limbs down over the edge of the bed
– Observe the time taken for the return of normal color (due to filling up of the dilated capillaries)
– The filling time of ≥ 20 seconds ⇒ critical ischemia
– In dark-skinned patients, the color changes cannot be appreciated and consequently this clinical test is not helpful.

Venous refilling time
– Elevate the limb and stroke gently to empty the veins. Then ask the patient to sit up hanging the limbs down over the edge of the bed
– Observe the time taken by the veins on the dorsum of the foot to fill up
– Normally the dorsal veins fill up within 5–10 seconds
– A delay of ≥ 60 seconds ⇒ marked impairment of arterial flow through the main channels or through the collaterals.

A fallacy
The veins should not fill up from above due to incompetent valves.
In such situation: Empty the veins by elevating the limb. Apply a BP cuff proximally and inflate it rapidly to 60 mm of Hg, and then perform the test.

Disappearing pulse
Applicable only for intermittent pains with normally felt pulses. Refer to page 671.

Tests for thoracic outlet syndrome: Roos' test, Tinel's test, Adson's test, costoclavicular compression test, and hyperabduction maneuver test.

www.Ebook777.com

Roos' test
Reproduction of symptoms on repeated hand clenching with both shoulders abducted to 90° and elbows flexed to 90° ⇒ the presence of thoracic outlet syndrome.

Tinel's test
Reproduction of symptoms by 90° abduction and external rotation of the arm may suggest the diagnosis of thoracic outlet syndrome.

Adson's test
Purpose: Detection of compression of subclavian artery due to cervical rib/scalenus anticus syndrome.
Principle: Anatomical relations between the artery, the rib and the scalenus anterior muscle.
Procedure: While monitoring the radial pulse, ask the patient to take a deep breath and then extend the head and turn the face towards the side being tested.
Inference: Pulse getting feeble / disappearing ⇒ +ve test; ± the hand may become cool and pale, and bruit may become audible in the supraclavicular fossa.
- Alteration of pulse even before turning the face suggests cervical rib
- Alteration of pulse after turning the face suggests scalenus anticus syndrome.

Costoclavicular compression test
Purpose: to detect compression of subclavian artery between the 1st rib and the clavicle.
Principle: Anatomical relations between the artery, the rib, and the clavicle.
Procedure: While monitoring the radial pulse, ask the patient to achieve an exaggerated military position (i.e. shoulders back and **downwards**).
Inference: Pulse getting feeble / disappearing ± development of bruit in the supraclavicular fossa ⇒ +ve test.

Hyperabduction maneuver test
Purpose: to detect compression of subclavian artery caused by pectoralis minor tendon.
Principle: Anatomical relations.
Procedure: While monitoring the radial pulse, abduct the arm passively.
Inference: Pulse getting feeble / disappearing ± development of axillary bruit ⇒ +ve test.

Test for arterial occlusive disease in the hand: Allen's test.

Allen's test
Purpose: 1. Detection of arterial occlusive disease in the hand. 2. Clinical study of palmar arch in the hand.
Procedure
1. Ask the patient to clinch the fist tightly. Apply tight pressure on both the radial and ulnar arteries. Ask the patient to open out the clinched fist. Release the pressure from over the radial artery alone.
   Observe the time taken for the blanched palm to regain normal pink color.
2. Carry out the same procedure but with release of pressure from over ulnar artery this time.
   Inference:
   - Normal filling time—up to 5 seconds.
   - > 5 seconds ⇒ an 'organic' occlusive disease.

   Fallacy: The filling time does not become abnormal in vasospastic disease.
   Clinical application: Selection of the side for doing an AV shunt for hemodialysis.

SYSTEMIC EXAMINATION

In nervous system examination, specifically search for simultaneously present or past e/o cerebrovascular insufficiency.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. In localized atherosclerosis with partial stenosis, distal pulse will still be palpable but systolic bruit may be heard distally.

Free ebooks ==> www.Ebook777.com

2. The primary diagnostic modality in the diagnosis of both lower and upper limb ischemia is the physical examination. Palpation (of pulses) is a reliable sign. Localization of the level of occlusion is based on the presence or absence of pulses at each arterial level.
   Pulse status is not entirely reliable for the evaluation of chronic arterial ischemia of the upper limb because of the rich collateral network.
3. If proximal pulse(s) are absent, the distal pulses will also be absent.
4. In normal individuals, one pedal pulse may be absent. In a patient giving h/o intermittent pains in the lower limb, the absence of a pedal pulse does not necessarily prove the diagnosis of PAOD.
   On the other hand, the presence of pulses does not rule out the presence of a PAOD.
   Up to 15 percent of normal subjects experience obliteration of the (radial) pulse with a thoracic outlet maneuver. A +ve thoracic outlet maneuver on its own does not make the diagnosis of a thoracic outlet syndrome.

ACUTE ISCHEMIA

5. **The diagnosis must be made clinically, without recourse to complex methods of investigation such as arteriography that might delay** treatment.
   Skeletal muscle can withstand total ischemia for up to 6 hours before irreversible muscle damage will occur. The time commences from the point of start of pathology, **and not from the time of admission** of the patient to hospital.
6. Edema, generalized blue discoloration, and large collateral veins ⇒ venous ischemia. However, if venous obstruction is sudden and severe, signs of ischemia precede the development of limb edema. It is only when edema occurs later that the diagnosis of venous ischemia becomes clear.
7. The site of block is usually higher than the upper limit of subjective and visible disturbance.
8. Regarding arrhythmia: An attack, e.g. paroxysmal atrial fibrillation, can be transient, and the pulse rate and rhythm can be quite normal when the patient is evaluated for acute limb ischemia.
9. The absence of Doppler-detectable venous flow at the ankle level is a particularly poor prognostic sign.

PHYSICAL CARE OF THE ACUTELY ISCHEMIC LIMB AWAITING DEFINITIVE TREATMENT

(a) Ischemic tissues are sensitive to pressure. ∴ Carefully protect the limb from external pressure. – The simplest method is to keep it in nests of cotton wool.

(b) O_2 requirements of ischemic tissues increase with temperature. ∴ Keep the limb cool by leaving it exposed and, if necessary, in the path of the draught from a fan. Do not cover the limb with bedclothes.

CHRONIC PAOD

10. In a case of intermittent pains Boyd's type III, noting down the current pain distance and rest time is crucial to the management by comparing the values over weeks or months. The values may remain static, or suggest deterioration or improvement on the basis of which further strategy of management can be planned.
11. Sometimes, a patient may have a clear h/o intermittent arterial pains, and a femoral pulse that is strong enough to be acceptable as within normal limits, but a bruit is heard on auscultation over the vessel in the groin. These facts ⇒ some irregularity of the arterial tree proximal to the groin, even though it has not been sufficient to obliterate the femoral pulse.
12. Patients with critical limb ischemia (due to a PAOD) develop symptoms for 3 weeks or more before presentation.

STEPWISE ANALYSIS OF DATA

Step 1: Clinical setting

Acute (/Acute-on-chronic) case	Chronic case usually with +ve signs	Intemittent pains proximal to the foot, with all peripheral pulses palpable even after exercise

www.Ebook777.com

Step 2

a. The **limb/s** affected?
   – Affection of only one lower limb is usually due to embolus lodged in common femoral artery
   – Affection of both lower limbs ⇒ a saddle embolus lodged at the aortic bifurcation.

b. The **level of block**?: **The most important** subjective and objective **clinical points** to determine this are: The site of pain, temperature difference, pulsations, and arterial bruit.

Step 3: **Etiopathology**, including the severity of ischemia?

Look at the *clinical background*, *the 'degree of acuteness'*, and *whether a pre-existing chronic arterial insufficiency has been absent or present.*

a. – A clear-cut case of trauma ⇒ traumatic ischemia
   – Debilitating and dehydrating illness, e.g. typhoid, etc. ⇒ acute thrombosis
   – Cardiac arrhythmia (even if transient) may lead to embolism.

b. Within the framework of acuteness, there are degrees of suddenness. An embolus impacts and immediately transforms a limb with normal circulation into an ischemic limb. Hence, a dramatic transformation of a previously normal limb into an ischemic one suggests embolism.
   Relatively lesser degree of suddenness suggests thrombosis.

c. E/o a pre-existing chronic arterial insufficiency ⇒ acute-on-chronic ischemia – and is usually due to thrombosis developing *in situ*.
   The necessity to differentiate acute ischemia *de novo* from an acute-on-chronic ischemia has a therapeutic implication. Acute ischemia, usually caused by embolism is more amenable to a successful invasive intervention. Patients with acute-on-chronic ischemia tend to do badly if operated upon. Injudicious use of balloon embolectomy in these patients can cause intimal disruption and put the limb at further risk.

If embolism is the cause, what is the source of embolus?

Severity of ischemia?



Step 4

Any associated disease/s?:
– Closely related, e.g. HT, IHD, CVD, abdominal angina, bronchitis, etc.
– Other.

Step 5 Inference on **revaluation at the end of 1 hour**?

If in terms of history and examination, there is evidence that the circulation in the limb is improving (this usually occurs due to opening up of collateral circulation), it may be meddlesome to operate unless it later becomes apparent that the improvement had ceased and the viability of the limb was still in doubt.

The possible collateral circulation is anatomically and functionally more effective in the upper than in the lower limb. In the upper limb it may be safe to await spontaneous improvement provided that there is no actual gangrene of the limb and that observation for an hour reveals no deterioration.

Even in the lower limb, after a definite decision to operate has been made, it is worth reassessing the patient at the end of the usual hour of premedication before actually proceeding with the operation.

Differential diagnosis of cold injury

Frostbite

= frank freezing of tissues, which may eventually culminate in gangrene; commonly superficial and limited to digits, occasionally affecting even a larger part of the limb.

Symptom: Numbness.

Signs: The part looks wrinkled and white, and is cool to touch.

Immersion foot / hand (*Syn.* Trench foot)

This results from continuous moist exposure to conditions of near 0° C temperature, as may occur in those who are campaigning over wet ground.

Free ebooks ==> www.Ebook777.com

Mild cases

– Burning, paresthesia/anesthesia, subjective sensation of cold
– Blisters form
– Skin takes on a characteristic blue/purple-red appearance

In ***severe cases*** the extremity passes through 2 stages:

Stage 1: Lasts from a few hours to several days.
– The affected part becomes edematous, cyanotic and cold
– Pulsations in the neighboring arteries—weak.

Stage 2: – Continues for 6–10 weeks
– Pain—usually worse at night
– Malaise, mild fever ±
– ↑ in edema
– Part becomes dark red
– Blebs containing serous / hemorrhagic fluid
– Albuminuria ±.

Note: Even if the entire hand/foot and all the digits are discolored, it is unusual for a digit to be lost, particularly in the hand. If gangrene develops it is usually superficial and, with general systemic care, will separate naturally.

CHRONIC CASE USUALLY WITH +ve SIGNS

Discomfort (during the earliest stage of the disease) OR pain in the foot/ leg / thigh / buttock / hand / forearm /arm, with or without trophic changes	Discomfort and / or pain confined to the digits
⇓	Usually spasmodic (intermittent); rarely permanent
Macrovascular disease	All peripheral pulses—normal
	⇓
	Microvascular disease

MACROVASCULAR DISEASE

↓

Step 2

a. The **limb/s** affected?
b. Does the disease appear to be—most probably **monolevel OR** most probably **multilevel**?

Multilevel disease = block in both proximal and distal vessels. Clinically it may not be possible to say with certainty if a block is multilevel for the following reasons: (i) In proximal vessel disease of sufficient severity to cause impalpable proximal pulses, even the distal pulses become impalpable. (ii) There is no specific clinical test to definitely prove or rule out distal vessel disease in the presence of impalpable proximal pulses.

The presence of multilevel disease can be guessed on the basis of the following facts: (i) Cause of the disease, e.g. smoker's arteritis (TAO), atherosclerosis in a diabetic patient; (ii) Relatively rapid progress of the disease, and (iii) Critical ischemia. Patients with critical ischemia tend to have complex multilevel disease (blocks in multiple proximal and distal vessels as well as in some of the collateral vessels).

c. – If the disease is thought to be monolevel: The **level of block**?
– If the disease is thought to be multilevel: The **most proximal level of block**?
The most important subjective and objective **clinical points** to determine the level (or the most proximal level) of block are: the site of pain, temperature difference, pulsations, and arterial bruit.

Step 3

Etiopathology

a. The **cause** of block, risk factors?
b. **Severity** of the degree of ischemia?
   i. **Nondisabling intermittent pain**. *Note down the current pain distance*.
   ii. **Disabling intermittent pains**. *Note down the current pain distance and rest time*.
   (Disability is relative, being governed by – age, occupation, and leisure interests. Objective assessment of functional impairment with QOL measurements is a desirable and good practice.)

www.Ebook777.com

iii. – Rest pain requiring regular analgesics × at least 2 weeks
– +ve Buerger's test with the angle of vascular sufficiency ≤ 20°
– Chronic indolent ulcer or/and gangrene.
} ⇒ **Critical ischemia** (Any single criterion is accepted)

c. **Possibility of collaterals having developed**?—Judge this from the venous refilling time.

Step 4

Complication?
An example—depression, irritability sometimes progressing to even suicidal tendency may be seen in a patient suffering from rest pain.

Step 5

Any **associated disease**(s)?:
– Closely related, e.g. HT, IHD, CVD, abdominal angina, bronchitis, etc.
– Other.

Unusual / rare arterial causes of intermittent pains in the lower limbs

1. *Popliteal artery entrapment syndrome*
   – Results from the artery coursing around the medial head of gastrocnemius muscle rather than between its 2 heads
   – Suspect in a young individual, especially if athletic
   – Can be bilateral
   – Forced active plantar flexion / dorsiflexion of the foot against resistance may → weakening / disappearance of the pedal pulsations
   – Popliteal vein also may be involved in ~ 10% of patients.
2. *Persistent sciatic artery*
   A congenital disorder, resulting from the persistence of the sciatic artery (the embryonic axial artery of the lower limb), replacing the iliofemoral system.
   – Often bilateral
   – Intermittent pains affect the buttocks and thigh.
3. *Fibromuscular dysplasia*: Common sites of affection—renal, carotid, external iliac.
4. *Cystic adventitial disease*: There is cystic abnormality of the adventitia of the popliteal artery.
   – Pains may come on suddenly
   – Flexion at the knee may → disappearance of the pedal pulsations.

PAOD affecting the proximal vessels of upper limb

Common causes
– Atherosclerosis of brachiocephalic or subclavian artery can present with both occlusive and atheroembolic symptoms
– TOS
– Iatrogenic trauma resulting in arterial dissection and acute thrombosis: This cause is becoming more frequent with the increasing use of brachial access for coronary angiography.

Uncommon causes: Vasculitides, e.g. smoker's arteritis, giant cell arteritis, Takayasu arteritis, radiation-induced arteritis. They usually present with occlusive symptoms; seldom present with embolic episodes.
Rare causes: Vasospasm due to ergot poisoning, vasospasm due to fungal contamination of some strains of grain.

Thoracic outlet syndrome

= symptoms in the upper limb (including the hand) due to compression of nerves or/and blood vessels within the thoracic outlet.
Obstruction of the subclavian vessel(s) is less frequent and often present in conjunction with nerve compression.

Arterial TOS
Distinct features
– The frequent presence of a bony abnormality, usually a complete cervical rib
– Need to address a primary arterial injury and secondary emboli

Free ebooks ==> www.Ebook777.com

– Frequent indication for operative treatment
– Complete resolution of presenting symptoms on treatment.

Pathology

The most common site of arterial compressive injury is the costoclavicular space. Bony abnormalities other than complete cervical rib which may be responsible are: incomplete cervical rib, long cervical transverse process, first rib abnormalities that produce protuberances, pronounced widening or arching that narrows the costoclavicular space, and (usually post-traumatic) abnormalities of the clavicle such as hypertrophic callus, deformity.

Repetitive motion (overuse) is a significant, although not essential, contributor for arterial injury.

The most common presenting arterial pathology is a focal aneurysm with secondary embolization. Earlier in the pathogenesis, stenosis may be noted with mild poststenotic dilatalion. Progression may result in complete occlusion and retrograde thrombosis with embolization into the cerebral circulation.

Clinical features of arterial TOS

– Occupational or recreational history of arm overuse ±, such as in painter, mechanics, weight-lifters, etc.
– Cold intolerance
– Raynaud's phenomenon
– Easy fatiguability
– Forearm exertional pain, rest pain, ulceration, gangrene
– Although uncommon, stroke may be the presenting complaint
– Intrapleural rupture of an aneurysm may occur rarely but can be fatal.

On examination of the neck

– A mass ±
– A bruit ±
– Pulse and BP may be diminished on the affected side. A drop of > 20 mm Hg in brachial systolic blood pressure compared with the normal side is suggestive.

Examine the nail-beds and fingers for ischemic signs.

Positional signs may be supporting. *A pitfall:* The positional signs may be present in normal individuals.

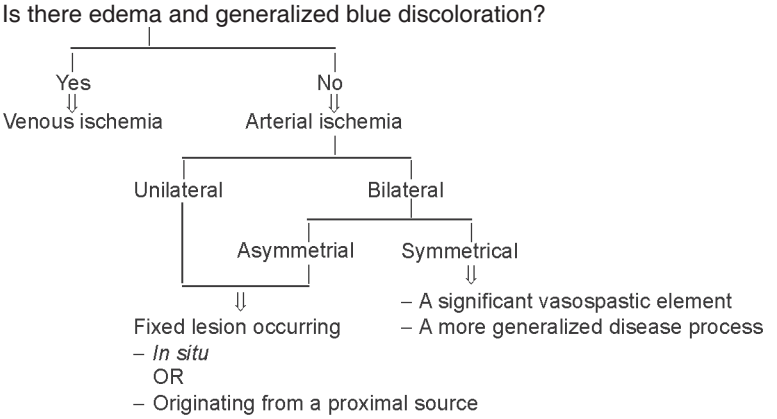
Arterial compression is a frequent finding in normal asymptomatic individuals and may be noted incidentally during imaging of a neck mass or contralateral symptomatic lesion.

Accurate diagnosis and correct patient selection for treatment remains a core problem due to the wide range of symptoms. This is compounded by the fact that there is no one diagnostic investigation specific to the syndrome.

MICROVASCULAR DISEASE

Step 2

Is there edema and generalized blue discoloration?



www.Ebook777.com

Step 3

Is the patient known to have a systemic or regional disorder which may predispose to microvascular disease?

Yes	No
A clue to careful history and investigation will be available comparatively easily	It may be difficult to arrive at the diagnosis Patient may have a primary Raynaud's

The microvasculature can be affected by many disease processes. The end result is common to all viz. vascular damage and ischemia. The clinical challenge is not in making the diagnosis of ischemia but in determining which pathological processes are ongoing, thus allowing speedy and effective management of the underlying disease. With the help of a careful clinical history, examination, and a selection of specific blood tests, it may be possible to arrive at the correct diagnosis.

Although microvascular disorders are divided into two broad categories (obstructive and vasospastic) some overlap does occur between these two groups, e.g. in atherosclerosis, CTDs, etc.

Differential diagnosis of selected microvascular disorders

- Small areas of palpable purpura
- Tissue around the lesions—healthy and vascularized with no decrease in capillary return } ⇒ **Microembolism**.
- Constitutional symptoms and widespread rash ⇒ septicemia-induced infected microembolism, e.g. meningococcal septicemia.

Examples of **CTDs** *which may* cause *microvascular disease*: SLE (Systemic lupus erythematosus), APS (Antiphospholipid syndrome), SS (Sjögren's syndrome), SSc (Systemic sclerosis), PMR (Polymyalgia rheumatica), GCA (Giant-cell arteritis), polyarteritis nodosum.

Raynaud's phenomenon—a cold-related disorder affecting young women; more prevalent than in men.

Primary Raynaud's disease	(Secondary) Raynaud's syndrome
There is no associated disorder Exaggeration of normal central and local vasomotor response to cold/ emotions/other stimuli	– There is associated underlying disorder – The prognosis is determined by the underlying disorder which must first be detected, monitored and treated *Pathology*: Diffuse obstructive small vessel disease within the palmar and/or digital vessels

- Found in up to 25% of young adults living in countries with a cool climate
- Young women < 45 are predominantly affected
- The full triphasic color change is not essential for diagnosis. A number of patients have blanching alone
- Apart from digits, the tip of the nose, tongue and earlobes may also be involved
- Stimuli other than cold and emotion can provoke an attack, e.g. trauma, hormones, various chemicals including those in tobacco smoke
- A wide spectrum of disease may be associated with Raynaud's phenomenon

Diagnosis of 'Raynaud's'

Criteria: Episodic attacks of well-demarcated blanching or cyanosis of one or more digits brought on by exposure to cold or emotion and rarely spontaneously. Well-demarcated change occurs with local vasospasm, and this avoids the problem of overdiagnosis in normal individuals who simply have pale fingers. It is surprisingly difficult to provoke a typical vasospastic episode in the OPD or vascular laboratory.

Free ebooks ==> www.Ebook777.com

Type of Raynaud's	*Gender*	*Features / comments*
Primary	Usually female	< 45 The absence of pain and lack of tissue damage (during attacks) may suggest primary Raynaud's. A degree of intermittent vasodilatation occurring during the attack allows just enough blood flow to protect the fingers from severe ischemia. Discomfort, but not pain, may occur during the hyperemic recovery phase. Investigations—normal
Secondary	Male / female	Any age. The presence of pain and/or residual damage during attacks suggests secondary Raynaud's. *Abnormal findings on physical examination suggesting secondary Raynaud's*: – Skin abnormalities: telangiectasias, digital ulcerations, pits, mottling, cyanosis, nail fold infarcts, tight shiny skin suggestive of a CTD – Pulses may be abnormal – +ve thoracic outlet maneuvers – +ve Allen's test – Vascular laboratory abnormalities ± – +ve autoantibodies.

INTERMITTENT PAINS PROXIMAL TO FOOT, WITH ALL PERIPHERAL PULSES PALPABLE EVEN AFTER EXERCISE

Carefully re-examine and investigate for intermittent pains of the cauda equina or McArdle's syndrome. If these conditions are ruled out, then consider the possibility of functional pain.

Intermittent pains of the cauda equina

→ sciatic pain (sometimes in association with neurological signs) produced by interference with the blood supply to the cauda equina by narrowing of the spinal canal, with or without protrusion of an intervertebral disk. (It is a province of a neurologist/neurosurgeon.)

McArdle's syndrome

– Young age
– The diagnosis is confirmed by special histochemical staining techniques of material obtained by muscle biopsy.

www.Ebook777.com

47 LIMB GANGRENE

DEFINITIONS

Gangrene is defined as death with putrefaction of macroscopic portions of tissue. (Putrefactive element even though present, may not be clinically obvious in dry gangrene.)

The feature which distinguishes gangrene from necrosis and infarction is putrefaction. In every case of gangrene there is not only death of a part, but also infection with saprophytic putrefying bacteria which adds rottenness to death.

From pathological perspective, gangrene = cell death + coagulative necrosis + liquefactive necrosis.

In dry gangrene, coagulative pattern of necrosis is dominant.

In moist gangrene, liquefactive pattern of necrosis is dominant.

Ischemia = a lacking in blood flow.

Necrosis = the sum of morphological changes that occur following cell death in a living tissue.

Examples of morphological changes occurring in the nucleus—pyknosis, karyolysis, karyorrhexis.

Necrotic cells are dead cells, but dead cells are not necessarily necrotic. For example, formalin-fixed 'normal' cells studied in histology are dead but not necrotic. Fixation preserves the cells in their original natural state in which they existed in the body.

Infarct = a localized area of ischemic necrosis in an organ or a tissue resulting from occlusion of either its arterial supply or venous drainage (and the process is called infarction).

GENERAL REMARKS

1. Gangrene may involve only a part of the limb (more common) or the entire limb. Gangrene may involve all the tissues of the affected part, or gangrene may be confined to only skin or fascia or muscle. When gangrene commences in the subcutaneous tissue plane, initially the skin may look normal; skin involvement is secondary to thrombosis of the perforating vessels coursing through the infected necrotic area.
2. Patterns typical of lower limb ischemia are rarely seen in the upper limbs where coldness and discoloration of the digits are most common presenting symptoms, and ulceration and gangrene less frequent.
3. Establishing the underlying aetiology of upper limb ischemia is often more complex, particularly in *chronic* disease when it may be a manifestation of a systemic disorder.

   In contrast, upper limb *acute* ischemia is generally a result of trauma or embolic disease. Acute thrombosis is usually secondary to an atherosclerotic stenosis, subclavian aneurysm or extrinsic compression within the thoracic outlet.

Free ebooks ==> www.Ebook777.com

4. Clinical presentation of a case of limb gangrene can be one of the following.

Marked constitutional symptoms due to obvious **gross trauma** (exception: Radiation injury) **OR necrotizing infection**. **Gangrene per se does not constitute the chief complaint**	(Acute) **gangrene with only 'mild' / 'moderate' constitutional upset** OR Gangrene occurring **on the background of a predisposing condition** OR **obvious precipitating cause**	**Spontaneous chronic gangrene without constitutional** symptoms	Acute constitutional upset due to secondary infection developing in a patient having a spontaneous chronic gangrene
↓ Commence resuscitation Shock must be assumed	– Acute thrombosis – Embolism – Venous gangrene – Diabetic gangrene (Necrotizing infection in a patient with uncontrolled DM) – Cold injury: Frost bite, immersion foot / hand	– Macrovascular disease. Pulsations distal to the level of block absent – Microvascular disease / tissue devitalization. Pulsations—usually normal	

5. Causes of gangrene: Trauma, necrotizing infection, vascular disorders, tissue devitalization, other less common causes.

Trauma
- Crush injury
- Injury to the main vessel, including iatrogenic injury (e.g. during arteriography)
- Thermal: Deep burns, frost bite, immersion foot / hand
- Electrical burn
- Chemical burn: Carbolic acid, etc.

Necrotizing infection. Refer to chapter 13.

Acute thrombosis
- Precipitated due to slowing of blood stream in debilitating and dehydrating illnesses
- Transient arrhythmias may cause transient hypotension sufficient to occlude critical peripheral arterial stenosis whether it is in native vessel or within peripheral arterial bypass graft
- Thrombosis may develop *in situ* on an underlying arterial disease.

Embolism *from one of the sources mentioned below* :
- Left side of the heart: MS, MI, SBE, aneurysm (left ventricular), atrial myxoma, arrhythmia
- Aorta: aneurysm, atheromatous plaque
- Iliac / femoral / popliteal artery—stenosis, plaque, intra-arterial drug abuse
- Peripheral vein thrombosis with atrial septal defect—called paradoxical embolism.

Venous gangrene: Massive venous thrombosis of main and collateral veins → arterial spasm and later on edema, infiltration and swelling of the arteries.

Diabetes mellitus

When uncontrolled	Otherwise
↓ Predisposes to acute/ subacute necrotizing infection	↓ Spontaneous chronic gangrene *Mechanisms*: – PAOD due to macrovascular atherosclerosis – Microvascular disease / tissue devitalization

Atherosclerosis in diabetic patients v/s atherosclerosis in non-diabetic patients
- Diabetics are ~ 5 times more prone to atherosclerosis than non-diabetics
- The process is more accelerated in diabetic patients; diabetic patients requiring vascular surgery are therefore likely to be younger than non-diabetics
- Atherosclerosis tends to affect the distal arteries (tibial and peroneal) more in diabetics, in contrast to the proximal arteries (above knee) in non-diabetics
- The media of arteries in diabetes tends to become calcified often in excess of the degree of atherosclerosis that is present

www.Ebook777.com

Macrovascular disease. Refer to chapter 46.
Atherosclerosis is a degenerative disease affecting the arteries of conduction viz. aorta, iliac, femoral, popliteal and proximal crural arteries.
Gangrene may be due to ischemia following stagnation rather than terminal occlusion.

Vasculitis. Refer to chapter 46.

Microvascular disease /tissue devitalization. Refer to chapter 46.

Less common causes *of gangrene*: Acrocyanosis, scleroderma, periarteritis nodosa, erythromelalgia, venous gangrene, 'ainhum', cystic myxomatous degeneration, Takayasu's arteriopathy (pulseless disease of females), radiation injury.

HISTORY

- **Chief complaint(s) / mode of presentation**
  1. Basically obvious gross trauma f/b coldness of the limb?
     OR
  2. A small wound which got complicated by rapidly spreading acute cellulitis and toxemia?
     OR
  3. Sudden mode of onset of symptoms s/o acute arterial insufficiency in a patient
     – with debilitating and dehydrating illness e.g. typhoid etc? (⇒ acute thrombosis).
     OR
     – known to have a cardiac / aortic disease? (⇒ embolism)
     OR
     – known to have a chronic peripheral arterial occlusive disease? (⇒ acute thrombosis)
     OR
     – with acute venous thrombosis also having an atrial septal defect? (⇒ paradoxical embolism)
     OR
  4. Essentially spontaneous development of blackening of digit(s) / more extensive area of the limb? **Mode of onset** is insidious **and progress** gradual in this situation.

Mode of onset is sudden **and progress** rapid / dramatic in these situations.

- **Symptoms s/o acute arterial insufficiency**
  – Severe agonizing continuous bursting pain, radiating down the course of the artery?
  – Burning, tingling, numbness? Cause of paresthesia: Ischemia of peripheral nerve endings.
  – Paresis / paralysis?
  – Coldness and deadness of the limb?
- **Acute spreading ulcer with cellulitis preceding the development of blackening**? (Usually, but not always necessarily, in a diabetic patient).
- **Symptoms s/o chronic arterial insufficiency**

In the lower limb ↓

– Fatigue?
– Do you experience severe unbearable pain in the foot even at rest ?
  - Is the pain worse at night / on lying down / on elevating the foot?
  - Any relief by hanging the limb over side of the bed ?
– Earlier history for a few months / years of intermittent pains-on-walking?
– Chronic indolent ulcer?

In the upper limb ↓

– Fatigue?
– Episodes of coldness and discoloration (a series of pallor f/b blue f/b crimson red f/b returning to normal over a period of a few minutes / hours) of the digits? ⇒ Raynaud's phenomenon ↓

Have you noticed any particular precipitating factor like exposure of the digits to cold environment such as immersion in water?

Free ebooks ==> www.Ebook777.com

- **Review of systems**
  - SOB, diminished exercise tolerance?
  - Pain in the chest, palpitations, syncope, HT, IHD, RHD?
  - Headache, giddiness, visual disturbance (blurring, diplopia), blackouts, stroke, convulsions?
  - Hypothyroidism?
  - Pain in abdomen after meals?
  - Increased or decreased frequency of micturition, color of urine, hematuria?
  - Chronic skin disorder, joint pains, bodyaches, RA, any other CTD?
- **Contraceptive history**? - Ask particularly with reference to OC pills.
- **Treatment taken**?
  Any response to the treatment?
- Have you been consuming any other medicines / receiving any other treatment for any other disorder?
- **Past history**
  - Similar illness in the past?
  - Injury, accident, operation especially in the recent past? (- for pseudoaneurysm)
  - Rheumatic disease?
- **Smoking** / consumption of **tobacco** in any other form?
  "No tobacco, No TAO".
- **Family history**
  - Of similar illness?
  - DM, HT, and IHD?
- **Personal history**: Appetite, sleep?

INTERPRETATION OF SYMPTOMS, AND DISCUSSION: Refer to chapter 46.

GENERAL EXAMINATION

In a suspected arterial insufficiency, BP should be taken in all 4 limbs using cuff of appropriate size.

- **Marked toxemia ± drowsiness ± shock** ⇒ Necrotizing infection, except for non-gas-forming clostridial cellulitis
- **'Mild' to 'moderate' constitutional upset** is seen in acute gangrene caused by acute vascular insufficiency
- A patient with a spontaneous chronic gangrene caused by a chronic PAOD may look sick due to **distress caused by pain**.

POSITION for locoregional examination: Supine, with both limbs exposed adequately for comparison.

LOCOREGIONAL EXAMINATION IN ACUTE GANGRENE CAUSED BY NECROTIZING INFECTION

– i.e. when a wound (**noticed** / **unnoticed**) got complicated by a rapidly spreading acute cellulitis and toxemia, progress being rapid / dramatic.

INSPECTION

- **Edema**? } Extent?
- **Discoloration**?
- **Demarcation**? Indistinct demarcation ⇒ possibly advancing gangrene.
- **Discharge**?
- **Other findings** which may differentiate the cause of gangrene?

PALPATION

- **Temperature**?
- **Tenderness**?

www.Ebook777.com

Direct tenderness
Examine for tenderness by direct pressure all round the circumference of the entire limb, as well as up the abdominal/thoracic wall.
Indirect tenderness—on stretching of the muscles by passive (hyper)flexion / (hyper)extension at the distal joints.
By both the methods, determine **the highest point (**i.e. **the most proximal level) of tenderness**.

- **Consistency** (of the muscles)?
- **Crepitus**?
  Determine **the highest point (**i.e. **the most proximal level) of crepitus**, when present.
- **Pitting on pressure**?
  Determine **the highest point (**i.e. **the most proximal level) of pitting on pressure**.
- **Sensation**?
- **Pulsations**?
- **Function**?

SPECIFIC FINDINGS IN VARIOUS NECROTIZING INFECTIONS: Refer to chapter 13.

LOCOREGIONAL EXAMINATION IN ACUTE GANGRENE CAUSED BY ACUTE VASCULAR INSUFFICIENCY

INSPECTION

- **Edema**—often present. – Extent?
- Black **Discoloration** ⇒ frank gangrene
  Proximal extent of the discolored area?
- **Demarcation**? – Usually indistinct ⇒ possibly advancing gangrene
- '**Skip areas**' = patches of gangrene in the proximal apparently normal segment of the limb.
  ⇓
  Impending proximal spread of gangrene.
- **Trophic changes** s/o a pre-existing chronic arterial insufficiency? (Refer to p. 669 chapter 46)
  The presence of trophic changes basically suggests ALI occurring in the already diseased arterial tree.
  The most probable cause of ALI in this setting could be acute thrombosis.
- **Venous guttering**?
- **Any visible abnormality along the course of the main artery**?

PALPATION

- (Skin) **Temperature**?
- **Tenderness**?
- **Consistency** (of the muscles)?
- **Sensation**?
- **Pulsations**?
- **Function**?
  Usually paralysis is present in the affected part in which gangrene is established.
  However, when gangrene is confined to only the foot/hand, movements of digits may be possible due to action of the long tendons taking origin proximally in the leg or forearm respectively. Hence, preservation of active movements of digits does not rule out the presence of gangrene.
- **Any palpable abnormality along the course of the main artery**?

AUSCULTATION

Bruit / systolic murmur / continuous machinery murmur?

Free ebooks ==> www.Ebook777.com

SYSTEMIC EXAMINATION

Cardiovascular system: Specifically search for the source / cause of acute embolism.
Nervous system: Specifically search for simultaneously present or past e/o cerebrovascular insufficiency.

LOCOREGIONAL EXAMINATION IN SPONTANEOUS GANGRENE (Usually chronic dry gangrene)

INSPECTION

- The gangrenous area looks **Shriveled**
- Black **Discoloration** ⇒ frank gangrene
  Proximal extent of the discolored area?
  Fallacy: Dark/Black-colored ointment, scabs may be mistaken for black discoloration of skin.
- The line/zone, and depth of **Demarcation**?
  - Demarcation in a dry gangrene is usually clear-cut
  - Demarcation may be skin deep / muscle deep / bone deep
- '**Skip areas**' = patches of gangrene in the proximal apparently normal segment of the limb
  Indistinct demarcation or/and the presence of skip areas ⇒ impending proximal spread of gangrene.
- **Trophic changes**?
- **Venous guttering**?
- **Any visible abnormality along the course of the main artery**?

PALPATION

- (Skin) **Temperature**?
  Usual finding: Cold. *Fallacy*: **the limb may feel warm if it was covered with a blanket just prior to examination**.
- **Tenderness**?
  along the artery ⇒ recent thrombosis.
- **Consistency** (of the gangrenous part)?
  Often hard, if the part has become mummified.
- **Pitting on pressure** ⇒ thrombophlebitis.
- **Sensation** – absent.
  Fallacy : during testing, the adjacent normal part may be stimulated with a false +ve presence of sensation.
- **Pulsations**?
  The level of block lies proximal to the highest impalpable pulse and distal to the lowest palpable pulse.
- **Function**?
  Refer to p. 685.



Fig 47-1 Note black discoloration and clear-cut line of demarcation

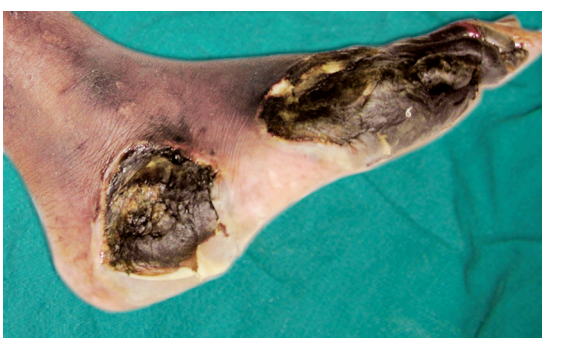


Fig 47-2 'Skip area'

www.Ebook777.com

- **Any palpable abnormality along the course of main artery**?
- Palpate an ulcer if present, in the usual way.
- **Measurements**—for objective assessment of wasting.

AUSCULTATION

Bruit / systolic murmur / continuous machinery murmur?

SPECIFIC TESTS to determine the level of adequacy of skin circulation

Buerger's test (of vascular sufficiency)

– Elevate the (lower) limb slowly, and observe for development of pallor.
– The angle at which pallor develops is called 'the Buerger's angle of vascular sufficiency'.
– Normally, pallor does not develop even when limb is elevated to 90°. Lesser the angle, greater the insufficiency. The angle of < 20° ⇒ critical ischemia.
– The extent of pallor gives an idea about the level of adequacy of skin circulation.

In dark-skinned patients, the color changes cannot be appreciated and consequently this clinical test is not helpful.

Scratch test

– Scratch the skin from thigh to ankle with a fork. → a red wheal if circulation is sufficient
– The lowest level where the red line is seen ⇒ the level of adequate skin circulation.

Venous refilling time

Refer to p. 672 chapter 46.

Check O_2 saturation using a pulse-oxymeter skin probe.

Multiple puncture test

– Make multiple deep punctures in the skin observing aseptic precautions at various levels in the limb one after the other
– Note the bleeding
– A free bleeding ⇒ good skin circulation.

SYSTEMIC EXAMINATION

Specifically search for simultaneously present or past e/o cerebrovascular insufficiency.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. Ulceration and gangrene are the end result of many disease processes other than ischemia, so that their role as evidence of ischemia must not be accepted uncritically.
2. When acute arterial insufficiency is responsible for gangrene, the acute ischemia initially gives rise to symptoms and signs other than blackening e.g. pain, paraesthesia, paresis/paralysis, prostration, discoloration (pallor f/b cyanosis f/b mottled appearance with blisters), pulselessness, coldness, and venous engorgement.
   Blackening suggestive of established gangrene occurs in a day or two without marked constitutional symptoms.
3. A patient in a cardiac care unit may be having an acute MI/arrhythmia which may predispose to acute embolism. As far as arrhythmia is concerned, an attack e.g. paroxysmal atrial fibrillation can be transient, and pulse rate and rhythm can be quite normal when the patient is evaluated for ALI.
4. The appearance of the highest (i.e. the most proximal) margin of the gangrenous area depends upon the level of adequacy of blood supply there.
   – A clear-cut zone of inflammation demarcating the dead from living tissue ⇒ sufficient perfusion to support an acute inflammatory response
   – If perfusion is inadequate, the zone of inflammation is weak and indistinct, and the area of gangrene spreads.
5. The spread of infection may travel proximally along various tissue planes and long tendon sheaths.

Free ebooks ==> www.Ebook777.com

STEPWISE ANALYSIS OF DATA

1. The **limb/s** and **the (apparent) extent of area** in the limb affected?
   Pertaining to gangrene caused by acute vascular insufficiency:
   Affection of only one lower limb is usually due to embolus lodged in common femoral artery.
   Affection of both lower limbs ⇒ a saddle embolus lodged at the aortic bifurcation.
2. Decide—whether **gangrene is present or not**?
   Clinical signs of established gangrene:
   - Discoloration—mottled purple/brownish/greenish OR black.
   - Demarcation.
   - Loss of temperature, sensation, pulsations, and function.

   Pitfalls
   - Line / zone of demarcation is apparent clearly only in dry gangrene and that too only after gangrene is completely established.
   - In gangrene caused by microvascular diseases/tissue devitalization, all the pulses (down to pedal/wrist) are palpable and normal.
   - In reference to 'loss of function': Clinically, function of only muscles/tendons can be tested. (The loss of) Function of a cutaneous patch of gangrene cannot be tested clinically.
   - In case of gangrene caused by microvascular disease/tissue devitalization, the actual visible area of established gangrene may be fairly tiny situated at the tip of a digit. The practical difficulties in this situation which need to be addressed are (a) What is the state of affair of the deeper tissue? – Is it gangrenous or not, and if so, how much is the exact extent; and consequently (b) How much should be the magnitude of surgical debridement? In practice it is often found that the deeper tissue might not have undergone gangrene but it is destroyed by necrotizing cellulitis from secondary infection. Although a surgical procedure may begin at the least for debridement, it should be scheduled as a formal amputation at an appropriate level if deemed necessary; an informed consent for the same having been obtained in advance.
3. Is the gangrene—**dry or moist**?

Clinical feature	*Dry gangrene*	*Moist gangrene*
– Smell	±	Offensive
– The part	Shrunken (mummified)	Swollen
– Color	Black	Mottled purple, brownish, or greenish to black
– Blisters	Absent	May be present
– Line / zone of demarcation	Present	Absent
– Consistency	Hard	Soft



Fig 47-3 Dry gangrene



Fig 47-4 Moist gangrene

www.Ebook777.com

4. Is the gangrene—**static or advancing**?
   - Clear-cut line/zone of demarcation } ⇒ Static gangrene.
   - The absence of 'skip areas'
   - Indistinct zone of demarcation } ⇒ Advancing gangrene.
   - The presence of 'skip areas'
5. **Cause of gangrene**?

A. Basically, is it **acute*** or **chronic** (including previous recurrent attacks of self-limiting acute episodes)?

Look at the clinical background / presentation

(a) Obvious **gross mechanical trauma**: Possible causes of gangrene: Vascular injury, clostridial gas gangrene

(b) **Marked constitutional upset**, gangrene *per se* probably not constituting the presenting chief complaint:
–Think of necrotizing infection

(c) **E/o acute arterial insufficiency with no e/o pre-existing chronic arterial insufficiency. Constitutional upset may be 'mild' or 'moderate'. Predisposing/precipitating cause may or may not be obvious**: – Think of embolism or acute thrombosis

(d) **E/o acute arterial insufficiency with e/o pre-existing chronic arterial insufficiency. Constitutional upset may be 'mild' or 'moderate' predisposing/precipitating cause may or may not be obvious**: – Think of (acute) thrombosis

(e) **DVT**: Think of venous gangrene

(f) Obvious **cold injury**: Think of frost bite/ immersion foot / hand

Usually not associated with constitutional symptoms

Chronic spontaneous gangrene

Peripheral pulses —weak or absent ⇓ Macrovascular disease

All peripheral pulses including (pedal / wrist)—palpable and normal ⇓ Microvascular disease / tissue devitatalization

* Occasionally a patient, particularly diabetic, may present apparently with acute gangrene of toe(s) with 'moderate' constitutional upset.

Actually it might have been a neuroischemic ulcer progressing to chronic spontaneous gangrene. As this pathology usually begins to develop at the tip of a toe (the area which is not routinely casually self-inspected) the patient may not be aware of the problem during the early period. It is when secondary infection flares up causing extension of cellulitis with discharge of foul smelling pus that the patient presents as 'an acute case'. By this time, constitutional upset may have also worsened from mild to moderate.



Fig 47-5 Chronic spontaneous gangrene due to tissue devitalization in a diabetic patient.

B. Think of **less common causes** if gangrene does not seem to have been caused by any of the more common causes.

6. Determine the proximal extent of pathology / level of occlusion

In c/o gangrene caused by necrotizing infection

The proximal extent of the spread of infection?

This is judged from the highest point (the most proximal level) of edema, tenderness and crepitus.

In c/o gangrene caused by an acute or chronic macrovascular occlusion

The level of occlusion?

(a) The site of occlusion lies proximal to the highest impalpable pulse and distal to the lowest palpable pulse.

(b) Bruit may be heard beyond the site of stenosis.

Free ebooks ==> www.Ebook777.com

7. Decide the **adequacy of skin circulation**. This can be done by one / more of the following methods.
   - Level of coldness: Though inaccurate, is a good clinical guide
   - Extent of pallor in Buerger's test
   - Scratch test
   - Venous refilling time
   - O_2 saturation checked using a pulse-oxymeter skin probe
   - Multiple puncture test.
8. **Any associated diseases**?
   - Closely related e.g. HT, IHD, CVD, abdominal angina, bronchitis, etc.
   - Other.

DIFFERENTIAL DIAGNOSIS

Necrotizing fascitis / myositis *v/s* infected vascular gangrene: Refer to chapter 13.

Clostridial myositis *v/s* non-clostridial (streptococcal) myositis: Refer to chapter 13.

Venous gangrene

Usually follows massive venous thrombosis.

- Predisposing causes—intra-abdominal or occult malignancy
- The digits or even the whole foot become edematous, cyanotic and eventually necrotic
- Zone of demarcation—unclear i.e. transition of abnormal area to normal looking area is gradual
- Temperature at the outset—often warm rather than cool.

Note

Providing the cause of venous occlusion is removed, tissues which initially appeared irreversibly affected often recover, with the loss of only the most seriously damaged superficial areas. For this reason it is better to defer amputation for several days to discover the full and final extent of the improvement that can be achieved. So, although the appearance of the limb may be alarming, it is a mistake to embark on early amputation (unless there is compartment syndrome with impending muscle necrosis) since tissue loss can often be minimized by aggressive medical treatment.



Fig 47-6 Ainhum. Necrosis yet to develop in this case.

Cold injury—refer to chapter 46.

Ainhum

It is a disease of unknown Aetiology affecting a toe, usually the little toe. A fissure appears at interphalangeal joint of a toe. The fissure is followed by a fibrous band that encircles the digit and causes necrosis.

Clinical points to be considered (in general) **for selecting the level of amputation**

- The site of (rest) pain
- The line / zone of demarcation
- Temperature
- Crepitus (when present)
- Pulsations
- The level of adequacy of skin circulation.

www.Ebook777.com

48 AMPUTATION STUMP

A patient with an amputation stump may not have any complaint as such, except for being an amputee. However, such a patient may be brought in to the practical viva examination hall, as 'a c/o amputation stump.' However, one or more of the following complaints may be associated with an amputation stump during early or later postoperative period.

– Pain
– 'Phantom limb'
– Breakdown of the wound
– Discharge (from a sinus / ulcer / eczema)
– Deformity at the proximal joint
– Inability to manage with a prosthesis: Difficulty in fitting / difficulty in using an otherwise well-fitting prosthesis.

HISTORY

Narrative History

The patient may present with one or more of the chief complaints mentioned above.

Interrogative History

- Ask the **details of each complaint** as usual viz. mode of onset, progress, regress, fluctuation, quantity, quality, precipitating/aggravating factors, relieving factors etc.
- **What was the indication for amputation**? – Details.
- H/s/o **postoperative complication**?
- About the **prosthesis**:
  - Type of the prosthesis? Cost of the prosthesis?
  - How long have you been using the prosthesis?
  - Any problem in managing with the prosthesis?
- **Recurrence of symptoms** in the same limb / contralateral limb, **which had necessitated the amputation**?
- **Treatment taken**?
- **Addictions**?

LOCAL EXAMINATION of the amputation stump

INSPECTION

- Length?
- Shape: Conical / cylindrical / irregular?
- Does the stump appear to be bulky?
- Skin:
  - Any redundant skin / Infolding of the skin / Areas of blackening?
  - Surrounding skin?

Free ebooks ==> www.Ebook777.com

- Scar:
  - Site, disposition, color, shape and size?
  - Does the scar seem to be bearing the weight?
- Any sinus (on the scar)? – Look for the usual details
- Any ulcer (on the scar)? – Look for the usual details
- Eczema?

PALPATION

- Temperature?
- Tenderness?
- Any bony projection?
- Is the skin freely mobile over the underlying bone?
- Is the scar freely mobile over the underlying bone?
- Palpate a sinus / ulcer, if present, in the usual manner.

MOVEMENTS

- Passive:
  - Range?
  - Contracture?
- Active:
  - Grade?
  - Range?
  - Voluntary control?

REGIONAL EXAMINATION

- Neurovascular deficit?
- Regional lymphadenopathy?

EXAMINATION OF THE CONTRALATERAL LIMB AND OTHER LIMBS

Examine carefully to detect signs of the disease which had necessitated amputation, so that appropriate therapeutic measures can be commenced at once.

EXAMINATION OF THE PROSTHESIS

- Socket: Lining, areas of wear and tear?
- Sole?
- Artificial joint?

ARRIVE AT THE CONCLUSION: IS THE STUMP IDEAL OR NOT?

CRITERIA OF AN IDEAL AMPUTATION STUMP

1. The patient should be a**s**ymptomatic.
2. Length of the **s**tump should be **s**tandard.
   - This fact is applicable to lower limbs. A standard length is essential for functionally effective and also aesthetically acceptable prosthetic limb. Prosthesis may easily slip out of a short stump. Too long a stump, even if 'vascularly' viable, also may not be acceptable for various reasons.

www.Ebook777.com

Level of amputation	*Standard length*	*Remarks*
a. Above knee (AK)	25 cm from tip of greater trochanter	A stump longer than that does not allow a prosthesis with a knee joint mechanism to be fitted functionally. It may be technically possible to fit such a prosthesis, but that would result in lowering the level of artificial knee joint compared to the level of the contralateral normal knee joint. Walking would not be feasible with the two knee joints at different levels
b. Below knee (BK)	12–18 cm from tibial tuberosity. (Fibula should be sectioned 2 cm higher than tibia.)	A prosthesis would tend to slip out of the stump that is shorter than 12 cm A stump longer than 18 cm would level below the muscle bulk of the calf, where blood supply is poor, thereby risking the chances of stump healing
c. Through the foot	Metatarsals should be preserved as much as possible	If preservation of metatarsals is not possible, then it may be appropriate to select Syme's or even more proximal level of amputation but amputation through the tarsus should be avoided as the stump tends to tilt (due to the loss of attachment of dorsiflexors) leading to plantar-flexion deformity of the stump
d. Through the great toe	-	The great toe should be preserved as much as possible. Reason: Great toe has a considerable functional value, particularly in 'take off' of normal walking. The most important part of great toe is head of the 1st metatarsal. Hence, at least head of the 1st metatarsal should be preserved if it is not possible to preserve the entire great toe; the distal phalanx matters much less
e. Amputation of the 2nd toe	-	→ Severe hallux valgus deformity. Hence, this amputation should be avoided as far as possible
f. Amputation through the 3rd/4th/5th toe(s)	-	→ little disability

There is nothing like 'standard' length in upper limb amputation. Every possible centimeter should be preserved. Upper limb prosthesis has more of cosmetic value. Prosthesis does not have to bear weight of the body, neither it is useful for locomotion.

3. **S**hape of the stump should be conical, so that prosthesis can fit easily.
4. **S**car should not be exposed to pressure. The site of scar is unimportant in modern prostheses with total contact socket.
   In forearm and leg amputations however, the scar should be disposed transversely. Reason: Anteroposteriorly disposed scar in these amputations tends to get drawn up between the 2 bones.
5. **S**kin should not be infolded, and it should slide easily over the bone.
6. There should be no redundant (bulky) **s**oft tissue.
7. There should be no protruding **s**pur of bone.

Free ebooks ==> www.Ebook777.com

49 VARICOSE VEINS AND CHRONIC VENOUS INSUFFICIENCY

DEFINITIONS

Varicosity
Dilatation and tortuosity of a superficial vein of the lower limb, due to permanent loss of its valvular function with resultant ambulatory venous HT.

CVI (Chronic Venous Insufficiecny)
There is no universally accepted definition of CVI. However, it is applied to the patients, who have developed irreversible skin damage as the result of sustained ambulatory venous HT.

CVU (Chronic Venous Ulcer/Ulceration)
A chronic ulcer, situated between the level of malleoli and tibial tuberosity, that is presumed to be wholly or partly due to venous disease.

Post-thrombotic syndrome (PTS) = venous claudication, edema, and skin changes of CVI leading to ulceration.

GENERAL REMARKS

1. LSV has a number of reasonably constant tributaries in the thigh and calf. It is these tributaries, not the main trunk (which is invested in supporting fascia), which often become varicose.
2. The direct medial calf perforators communicate with the posterior arch vein, and not the main LSV trunk.
3. AV malformation or extensive cavernous (= venous) malformation may be mistaken for varicose veins at first sight, if not seen carefully. Congenital AVM usually occurs below the age of 20 (varicose veins do not occur before the age of 20). Veins will be pulsating in AVM.
4. The great majority of individuals with (primary) varicose veins are otherwise asymptomatic.
5. Varicose veins do not by their mere presence constitute an indication for (invasive) treatment, because complications of untreated varicose veins such as superficial thrombophlebitis are quite rare.
6. Chronic PAOD, DM, and RA often co-exist in patients with CVU.

   Many ulcers, particularly those that are resistant to treatment, are multi-factorial in origin, and sustained healing will only be achieved once all the aetiological factors have been addressed.
7. *Discussion on* ***obesity as related to varicose vein disease***
   a. Obesity is one of the predisposing factors in the development of primary varicose veins.
   b. Obesity is one of the risk factors for the development of venous thromboembolism. DVT → PTS, which in turn → secondary varicose veins.
   c. CW Doppler study is less accurate in obese patients.
8. The mere presence of uncomplicated (primary) varicose veins does not qualify for CVI. It is only when the (primary) varicose veins progress to develop irreversible skin damage that they qualify for the aetiology of CVI in the given case.

www.Ebook777.com

HISTORY

- **Occupation**
  Does your occupation involve long hours of standing?

The chief complaint: Basically dilated, tortuous (± elongated) swelling(s) in the lower limb(s) *with or without one or more of the following additional symptom(s)*:

- Looking unsightly. Does the patient seem to have been concerned about cosmetic appearance of the limb?
- Itching, scaling and weeping? – suggest dermatitis (eczema).
- Discoloration of skin in the gaiter area / lateral malleolar area?
  Causes of discoloration: ankle flare, hyper- or depigmentation, lipodermatosclerosis.
- Active / healed / repeatedly recurring ulcer in the lower leg (gaiter area / lateral malleolar area)?
- Bleeding (due to trauma)?
- Pain? – Details.
- Cramps? – May be vague; they may not necessarily be suggestive of intermittent arterial pains.
- Aching on dependency / before periods?
- 'Heaviness' in the legs?
- Restlessness in the legs?
- Paresthesia?
- Edema?
- Tenderness?

Interrogative history

- Are these recurrent varices (especially after a surgery) OR otherwise?
- Ask for the duration, mode of onset, progress, regress, fluctuation, quantity, and quality etc. of each complaint as applicable
- Pain? – Details.
  - H/s/o venous 'limp'?
  - H/s/o arterial pain—intermittent / at rest?
- Review of systems
- Obstetric history
- Gynecologic history
- Contraceptive history
- Treatment taken and response to it?
- Have you been on any long-term medications?
- Past history of
  - Calf pain and edema in the leg of sudden onset, (usually unilateral)?
  - Prolonged immobility due to any cause like an injury / accident / prolonged surgery / stroke / sepsis, etc?
  - Any major surgery, in particular pelvic surgery / hip surgery / lower limb surgery?
  - Any accident particularly involving the lower limb?
  - Venous thromboembolism?
  - Any major (medical) illness?
  - Estrogen therapy?
- Addictions—smoking, alcohol, other?
- Hypersensitivity?
- Family history?

INTERPRETATION OF SYMPTOMS, AND DISCUSSION

Age

- Varicose veins usually occur above the age of 20 years. They are more common as age advances
- Occurrence of (severe) varicose veins in young age indicates a significant role played by hereditary factor
- Varicose veins occurring below the age of 20 should be considered to be 'fistula varicosities' (i.e. due to congenital AVM), unless proved otherwise
- Old age predisposes to develop CVI.

Free ebooks ==> www.Ebook777.com

Occupation
Occupations involving long hours of standing predispose to (primary) varicose veins. Examples: bus conductors, policemen, salesmen, surgeons, etc.

Obstetric history
Incidence of varicose veins increases with parity. Mechanisms: 1. Gravid uterus → increased intra-abdominal pressure → increased hydrostatic pressure. 2. Hormonal influences during pregnancy → smooth muscle hypotonia.

Cosmetic issues
Many individuals, especially young women, seek treatment for varicose veins for cosmetic consideration. Possibly because they are embarrassed to admit that cosmesis is the main issue, they frequently complain of various lower limb symptoms as well.

Pain
- Dull aching pain / fatigue / undue tiredness in the legs that occurs towards the end of the day, which is relieved by lying down for ~ 15–30 minutes is characteristic of varicose veins
- Pain and tenderness in a cordlike inflamed area – can be due to superficial phlebitis
- A bursting discomfort in the leg following use/exercise, relieved only by resting + elevation = 'venous limp'. - It is one of the manifestations of PTS (Other manifestations of PTS being edema and skin changes of CVI culminating into CVU)
- Particular reason to exclude PAOD in a patient suffering from varicose veins: Patient with ABPI of 0.6 due to uncorrectable PAOD is a contraindication for both compression treatment as well as surgical treatment for CVI.

Past history
All the points in the past history are meant to explore the possibility of post-thrombotic syndrome (PTS) as a cause of CVI/ secondary varicose veins.
A clear h/o DVT is found in only ~ 20% of patients with chronic venous insufficiency. In addition, an unknown, probably unquantifiable number of people develop DVT without ever knowing it.

Family history
- Varicose vein disease, CVI and CVU are known to have a familial predilection
- Occurrence of (severe) varicose veins in young age indicates a significant role played by hereditary factor.

H/o Hypersensitivity
This is important, as one of the complications of injection sclerotherapy is hypersensitivity, and therefore one has to be careful.

Parts Exposed: The whole lower limbs + the lower abdomen.

POSITION of the patient
Initially standing on a platform (A platform is for the benefit of the clinician). Later, lying down.
And finally lying down alternating with standing often many times, while specific clinical tests are being performed.

LOCOREGIONAL EXAMINATION

INSPECTION

Dilated, tortuous (± elongated) veins.

- **Territories involved** (i.e. site)?
  - Predominantly medial aspect of leg, proceeding up towards the saphenous opening ⇒ LSV territory
  - Posterior aspect of leg, proceeding up towards the popliteal fossa ⇒ SSV territory
  - Determination of territory may not be as easy as it may appear
  - Tributaries leading to long or short saphenous vein, wander over a wide area of the limb and below the knee there may be a real difficulty in deciding whether a given varix belongs to the long saphenous or to the short saphenous territory
  - Varicosity of the vein of Giacomini—present or absent?
    Vein of Giacomini = a communicating vein between LSV and SSV
    Comes off LSV in the thigh, passes posteroinferiorly around the popliteal fossa, and joins SSV a little inferior to SPJ. It can transmit the incompetence of LSV to the SSV even though SP valve is competent
  - If iliac system is occluded, then collaterals develop to bypass the occluded system. The collaterals are seen as dilated veins running across the lower abdomen between the groin/s and the umbilicus.

www.Ebook777.com



Fig 49-1 Truncal varices



Fig 49-2 Reticular varices

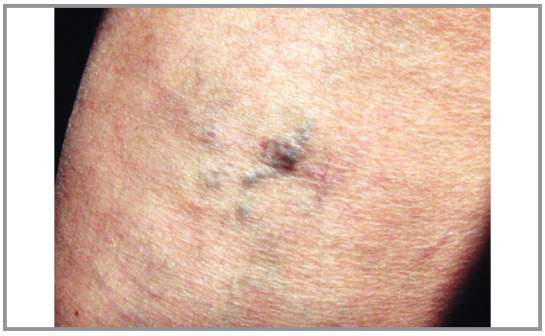


Fig 49-3 Spider veins



Fig 49-4 Fistula varicosities (AVF for dialysis)

- **Form**?
  1. ***Truncal*** varices= varicosity of the main stem (LSV / SSV), or its 'major' tributaries.
     – > 4 mm in diameter (may be much larger)
     – No discoloration of the overlying skin.
     As far as LSV is concerned, the tributaries which may be considered as 'major' in the thigh and leg: Anterolateral vein of the thigh, posteromedial vein of the thigh, anterolateral vein**s** of the leg, and the posterior arch vein.
  2. ***Reticular*** varices = varicosity of 'minor' tributaries of the main stem.
     – < 4 mm in diameter
     – The overlying skin—blue.
  3. ***Spider*** veins = venous telangiectasia. (By definition, vascular lesions characterized by dilatation of a group of small blood vessels.)
     – Usually $\leq$ 1 mm in diameter
     – The overlying skin—purple / bright red.
- **Pulsations**?
  The presence of pulsations $\Rightarrow$ varicose veins developed due to local effects of an AVM. Such varicose veins are called 'fistula varicosities'.
- **Saphena varix**?
  = saccular dilatation of the terminal portion of LSV.
- '**Blow outs**'?
  Indicate the sites of incompetent perforators.
- Scaling $\pm$ weeping $\Rightarrow$ **Dermatitis (Eczema)**
  Dermatitis is characterized by itching, scaling $\pm$ weeping.
  Types:
  – Endogenous—due to varicose/venous 'stasis' dermatitis—mechanism not understood
  – Exogenous—due to application of external substances—called 'contact' dermatitis.

Free ebooks ==> www.Ebook777.com



Fig 49-5 Saphena varix



Fig 49-6 Ankle flare

- **Ankle flare**?
  The presence of ankle flare qualifies for grade-1 CVI.
- **Hyper- or depigmented areas**?
  Their presence qualifies for grade-2 CVI.
- **Lipodermatosclerosis**?
- **An active ulcer**?
  Situated in the gaiter area / lateral aspect of the ankle / circumferentially on the lower leg. Vertically generally oval. Note the usual attributes of the ulcer 'on inspection'.
- Reddening along the varicosed vein ⇒ **phlebitis**.
- **Scar**—site, disposition, color, shape and size?
  Various causes of scarring could be: following a healed chronic venous ulcer / following a venous surgery, an arterial surgery, hip/knee joint surgery particularly their replacement surgery.
- **Edema**?
  Note its extent.
  There are several causes of generalized edema (e.g. right heart failure, renal disease, hypoproteinemia etc.) that may co-exist in a patient with CVI.
- **Trophic changes suggestive of a peripheral arterial occlusive disease**?
- **Any extrinsic abnormality along the course of a main artery**?
  If present, it may be responsible for chronic arterial insufficiency, by compressing the artery.

PALPATION AND PERCUSSION

- **Temperature** along the varicosed vein?
  Increased temperature along the varicosed vein may be due to phlebitis.
- **Temperature** of the entire limb in general?
  This should be felt for to rule out a peripheral arterial occlusive disease.
- **Tenderness**?
  Tenderness along the varicose vein, may be due to phlebitis.
- **Confirmation of the territory**
  A technique that is useful even in fat patients in whom the veins may be difficult to see: Map out a vein by tapping at one spot with the index finger of one hand and perceiving the vibrations transmitted along its channel proximally to a point several centimeters distant, with fingers of the other hand placed lightly against the vein. Follow the vein along its course in this manner segment by segment until it definitely enters the long or the short saphenous system.
- **Thickening**?
  - Cord-like thickening of the varicose vein ⇒ phlebitis.
  - Brawny edema of skin and thickening of subcutaneous tissue (due to fibrosis) in the area of hyper- or depigmentation ⇒ lipodermatosclerosis, which in turn ⇒ grade-2 CVI.

www.Ebook777.com

- **Palpable Thrill**?
  ⇒ 'fistula varicosity'.
- Palpate an ulcer, if present, in the usual manner.
- **Palpate the pulses**—to rule out a peripheral arterial occlusive disease.
  If pedal pulses are not easily palpable, **measure ABPI**.
  An ABPI of < 0.8 (at rest) ⇒ hemodynamically significant arterial disease.
  Is there any palpable **extrinsic abnormality along the course of a main artery**?
  If present, it may be responsible for chronic arterial insufficiency, by compressing the artery.
- **Mark the 'blow outs'** with a skin pencil (or any other suitable means).
  During the course of physical examination, subsequently, after the patient is made to lie down, search for defects in the deep fascia (by palpation), specifically at these marked sites.

Schwartz's test
Purpose: Determination of the 'severity' of varicosity *wrt* the extent of valvular failure.
The test is performed in standing position.
Principle
Basically, valvular failure leads to reflux, which in turn, leads to varicosity. Due to valvular failure, the varicosed segment of vein is no longer divided by competent valves into a series of low-pressure segments; instead, there is a continuous unbroken column of blood in it.
Procedure
Place 2 or 3 fingers upon the fossa ovalis. Tap the most prominent portion of the varicose vein with fingers of the other hand and try to feel for thrill-like impulse at the fossa ovalis. Reverse the role of both hands, tap upon the fossa ovalis, and try to feel for thrill-like impulse upon the vein lower down. Repeat the same procedure again and again, to test longer and longer segment of vein each time.

Conclusion: Longer the abnormal segment, severe the varicosity.

Examine for **deformities**, **weakness of muscles**, and the **joints for a reduced range of mobility**.

AUSCULTATION

Machinery murmur upon the varicosed vein ⇒ 'fistula varicosity'.

SPECIFIC CLINICAL TESTS

These tests are designed to determine the following:

1. The points of leak – at SF junction / SP junction / perforators. Obliteration (ligation) of the point/s of leak (incompetent valves) is of crucial importance in the treatment.
2. Whether the deep veins are occluded or patent. Occlusion of deep veins is a contraindication for any kind of invasive treatment upon the superficial veins.

Clinical tests for determination of **SF incompetence**

1. **Visible impulse-on-coughing**
   Ask the patient to cough, and observe at the saphena varix if present, or at the fossa ovalis, and the varicose vein itself for a visible impulse.
   The presence of an impulse ⇒ SF incompetence.
2. **Palpable impulse-on-coughing**
   Ask the patient to cough, and palpate upon the saphena varix when present, or upon the fossa ovalis, and then upon the LSV **in the thigh** (and not merely just inferior to the fossa ovalis).
   The presence of thrill-like impulse ⇒ SF incompetence.
   Mechanism of this test: Impulse-on-coughing → increase in intra-abdominal pressure → increase in pressure in the external iliac and femoral veins – that is transmitted across the incompetent SF valve to the varicosed LSV due to actual reflux of blood (this can be seen in duplex scanning).
3. **Trendelenberg test**
   Principle: Reflux of blood across incompetent SF valve leads to filling up of varicose veins, which have been initially emptied.
   Procedure
   With the patient in standing position, mark the sites of blowouts and/or the prominent areas of varicosity. Then, ask the patient to lie down. Elevate the limb till the veins are emptied.

Free ebooks ==> www.Ebook777.com

Occlude the terminal portion of the LSV either by applying a tourniquet just inferior to the fossa ovalis, or by applying pressure with a thumb upon the fossa ovalis (saphenous opening).

Ask the patient to stand up quickly.

While observing the sites (already marked for advantage) of blowouts or/and the prominent areas of varicosity, remove the occlusion of the LSV.

Inference

Immediate filling up of the blowouts/the prominent areas of the varicosity ⇒ SF incompetence. Sometimes, 'blood' is seen rushing from above downwards.

Clinical test for determination of **SP incompetence**: **Trendelenberg test**

The principle, procedure and inference are similar as while testing for SF incompetence except that in this test, the terminal portion of SSV needs to be occluded by applying a tourniquet just below the knee joint, and the inference is applicable to SP junction.

Clinical test for determination of **perforator incompetence**: **Multiple tourniquet test**

Principle

Blowouts (= additionally distended segments of varicose veins) indicate the sites of incompetent perforators. The blowouts receive blood from 3 sources—1. Normal source. 2. Refluxed blood from incompetent SF/SP junction; and 3. Incompetent perforators. To prove that a perforator is incompetent, blood reaching from the remaining 2 sources needs to be cut down; hence, one tourniquet each is tied just above and just below the blowout.

Procedure

Position of the patient—lying-down.

Elevate the limb till the veins are emptied. Run down a finger along the surface marking of the main stem as well as major tributaries of the LSV (/ SSV), with particular attention on the sites already marked out at the blowouts. Palpable gaps (defects) in the deep fascia indicate the sites of perforators, which are considered to be incompetent.

Tie one tourniquet each just above and just below the level of a perforator so that, in case of 2 adjacent perforators, 3 tourniquets will be required; in case of 3 adjacent perforators, 4 tourniquets will be required.

Ask the patient to stand up, and observe the sites of blowout.

Inference

Reappearance of the blowouts ⇒ incompetent perforators. Otherwise, incompetence of the perforators is ruled out.

Remove the tourniquets.

Clinical test to determine if the **deep veins are occluded or patent**: **Perthe's test**

Principle

Sudden stoppage of return of venous blood while arteries still continue to supply blood, will lead to stagnation and increased tension in the tissues, and consequently pain in about 5 minutes. If the whole of the superficial system of veins is kept emptied by bandaging the limb, pain will occur only if the deep veins are occluded. Pain will not occur if deep veins are patent.

Procedure

Initially the patient should lie down. Elevate the limb till the veins are emptied.

Apply (an elastic) bandage from the base of the toes up to groin with the pressure just enough to keep the superficial veins empty.

Then, ask the patient to walk about briskly for 10 minutes.

Inference:

– Experiencing pain suggests that the deep veins are occluded
– The absence of pain suggests that the deep veins are patent.

EXAMINE **THE OPPOSITE SIDE**

SYSTEMIC EXAMINATION

Examine particularly for neurological and locomotor disease

Assess the patient's mobility, and also watch how the patient walks. Flat foot, fixed ankle, reduced mobility at knee/hip suggests marked impairment of foot and calf muscle-pump leading to exacerbation of venous HT.

(It may be unrealistic to advice that every patient with varicose veins should undergo a DRE to exclude a pelvic mass.)

IT IS WORTH TAKING PHOTOGRAPHS DURING EACH REVIEW, FOR COMPARISON at a later date.

www.Ebook777.com

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITALLS

1. There is a weak relationship between various lower limb symptoms and venous disease.
2. Acute lipodermatosclerosis may be difficult to distinguish from acute cellulitis although the latter is unusual in venous ulcers unless there is associated arterial disease. Unlike lipodermatosclerosis, cellulitis is associated with marked pain and tenderness, constitutional symptoms, leucocytosis, and sometimes lymphadenitis.
3. **Try to answer** the following questions; that will help in making a correct diagnosis and plan the management strategy for an individual patient.
   - Are the symptoms truly present?
   - Can the symptoms and signs be attributed to venous disease?
   - Are there other pathological processes contributing to the clinical presentation?
   - What are the anatomical and functional venous abnormalities responsible for this patient's symptoms and signs?
   - Will this patient benefit from treatment (surgery in particular)? – Use your own judgment and experience to answer this question.

STEPWISE ANALYSIS OF DATA

Step 1

Anatomical aspect of the diagnosis

- **Limb/s** affected: Right / left / both?
- **Territories** involved: LSV / SSV / both?
- Physical **form**: Truncal varices / reticular varices / spider veins / combinations?
- In case of isolated reticular varices (without truncal disease): The exact location: Above-knee OR below-knee?
- In case of truncal varicosity: **Extent,** and the exact **point/s of** '**leak**' (i.e. the point/s of incompetence)?

Step 2

Etiology: Primary / secondary?

Primary *varicose veins*	***Secondary*** *varicose veins*
Predisposing factors: Pregnancy, occupations involving prolonged standing, familial tendency, a big lump in abdomen	Causes: Actual physical or functional occlusion of deep veins, 'fistula' varicosities (congenital / acquired)
Incompetence of direct perforators may be associated	Incompetent direct perforators almost always associated
Rarely cause ulcer, **even when long-standing**	Ulcer—common
Ulcers, if at all occur,: variable in site; vertically generally oval, and they easily heal. – Called '**true varicose ulcers**'	Ulcer—constant in position viz. most commonly medial malleolar region; larger in size, deeper, surrounded by hyperpigmentation and induration. Circumferential ulcer may occur. – Called '**post-thrombotic**' ulcer

Pathology?

- In case of primary varicose veins: The mechanism of loss of valvular function is not yet fully understood
- In case of secondary varicose veins occurring as a part of PTS: (i) Valves in the deep veins and perforating veins are damaged. (ii) Diameter and compliance of the deep veins are reduced, thus leaving the veins functionally occluded. Due to these pathophysiological aftermaths of DVT, blood from the deep compartment

Free ebooks ==> www.Ebook777.com

is forced via incompetent perforators, to the skin of gaiter area, and through distended so-called secondary varicose veins, proximally. Thus, the secondary varicose veins virtually develop to form collateral channel for deep venous blood.

Step 3

Complication/s?: Dermatitis (eczema), CVI, lipodermatosclerosis, (history of) bleeding from trauma, phlebitis.

***Widmer's classification* for gradation of CVI**—based on outward clinical appearance of the lower limb:

- Grade 1 = Venous flare
- Grade 2 = Hyper- / depigmented areas
- Grade 3 = Open / healed ulceration.

Step 4

Any associated ailments

Closely related OR—otherwise?

↓

a. Deep venous incompetence.

   In case of primary varicose veins, deep venous incompetence (which usually follows a past episode of thrombosis) is not necessarily considered to have a 'cause and effect' relationship. It may co-exist by a chance. However, the coexistence has an important therapeutic implication. Surgical option, which might have been appropriate otherwise, will have to be selected very carefully if deep venous incompetence co-exists.

   As far as secondary varicose veins are concerned, deep venous incompetence will be virtually associated with it, albeit, deep venous incompetence is a cause of secondary varicose veins.

b. Deep venous occlusion.
c. PAOD.
d. DM.
e. RA etc.

www.Ebook777.com

50 LIMB EDEMA

Truly, edema is **defined** as a collection of fluid in the interstitial spaces. It usually leads to a circumferential swelling of the limb.
Edema-like chronic diffuse swelling also may result in a circumferential swelling of the limb, and hence a need to differentiate it from true edema.

Causes of edema-like chronic diffuse swelling

1. Lipoderma. In fatty persons excess of subcutaneous fat may allow abnormal diffusion of lymph into the tissue spaces.
2. Diffuse lipomatosis.
3. Diffuse neurofibromatosis—plexiform neurofibromatosis, elephantiasis neuromatosa.
4. Pretibial myxedema associated with Grave's disease.

GENERAL REMARKS

Clinical settings

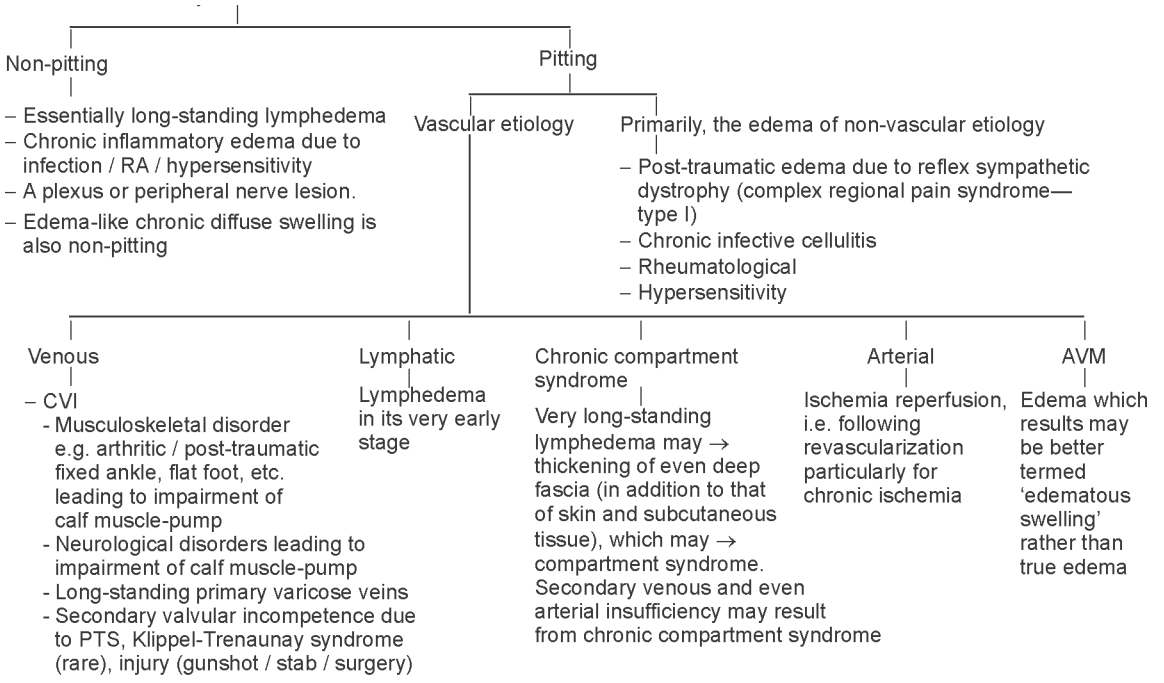
Acute	Chronic
– Painful	– Usually painless
– Mode of onset—sudden	Exceptions: -Post-traumatic edema due to reflex sympathetic dystrophy -Edema due to AVM
	– Mode of onset—insidious

Causes of acute edema:

– Trauma: Soft tissue trauma like contusion / hematoma in a bulky muscle (e.g. calf muscle hematoma); fracture of bone, 'immersion hand / foot' (a cold injury), burn injury
– (Vasculotoxic) Snake bite, bites by other land / sea animals / insects
– Acute venous thrombosis (DVT)
– Compartment syndrome
– Infective cellulitis
– Rheumatological
– Hypersensitivity
– Superficial thrombophlebitis } – May or may not be associated with edema
– Acute lymphangitis – Refer to chapter 45.

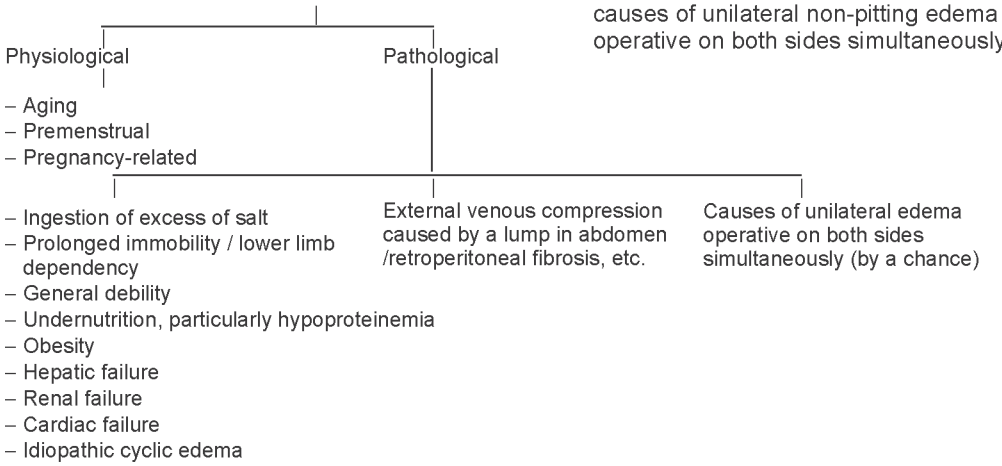
Free ebooks ==> www.Ebook777.com

Causes of essentially unilateral chronic edema



– External venous compression caused by a swelling, retroperitoneal fibrosis, etc.
– Prolonged dependency due to any reason e.g. limb with rest pain
– Residual symptoms (pain, early fatigue and limb edema) which may occur in some patients, after recovery from the acute event (thrombosis) of venous TOS
– Persistent edema due to residual venous obstruction following the acute event (thrombosis) of venous TOS.

Causes of (simultaneously) **bilateral chronic edema**. Such edema is usually pitting-type unless causes of unilateral non-pitting edema are operative on both sides simultaneously.



Examples of **mechanisms** of development of edema

1. Increased capillary pressure as in DVT, cardiac failure.
2. Increased capillary permeability as in acute inflammation.
3. Impaired lymphatic drainage as in filariasis.
4. Decreased osmotic pressure of blood as in hypoproteinemia.

www.Ebook777.com

HISTORY

All points mentioned below may not be applicable in every case of edema; the clinician is expected to use discretion.

- **The limb/s** involved—**unilateral** OR (Simultaneously) **bilateral**?
- Duration?
- Mode of onset?
- Where did it commence (distally in the foot, or proximally in the thigh)?
- Progress / regress / fluctuation?
- Painful / painless?
- Paresthesia?
- Can you move the digits?
- Is this the first such episode, OR is this a recurrent episode? – Details of the previous episodes if any?

Further history to be asked in **acute edema**

↓

- **Trauma**? – Details.
- Severe unaccustomed physical activity of the limb? (– Severe unaccustomed physical activity is also a type of trauma.)
- Wherever applicable: Does the POP cast appear to be very tight?
- Snake-**bite** / insect-bite?
- How long have you been **immobile / bed-ridden**? – Details
- H/s/o **thoracic outlet syndrome**?
- Have you been suffering from **a cancer**?
- Did you recently undergo an intervention (surgical/percutaneous) for an acute limb ischemia?
  - Details of the ischemic episode?
  - How many hours after the onset of symptoms of acute arterial insufficiency did you undergo the intervention? Was it > 6 hours?
- **SOB** / (**pleuritic**) **chest pain** / **hemoptysis**?
- **Fever**?
- H/s/o **RA**?
- H/s/o **hypersensitivity**?

Further history to be asked in **chronic edema**

↓

- Do your shoes get tight?
- Are the rings tight on your fingers?

} These points confirm edema.

- Have you had to let out your belt?
- Have your clothes gotten too tight around the middle?

Further history to be asked essentially in **unilateral** chronic edema

↓

- H/s/o any chronic infection—either spontaneous or following an injury, with a purulent discharge?
- H/o RA?
- H/s/o hypersensitivity?
- Difficulty in walking due to any musculoskeletal disorder like arthritic / post-traumatic fixed ankle, flat foot etc?
- Neurological disorder?
- H/o very long-standing varicose veins?
- Past h/s/o DVT?
- Recent or long past h/o injury/surgery upon the limb?
- H/s/o AVM?

Free ebooks ==> www.Ebook777.com

Further history to be asked essentially in **bilateral** chronic edema (All points may not be applicable to every case. Use discretion.)

- Do you routinely develop it in premenstrual period?
- Are you pregnant?
- Did you consume excess of salt?
- How long have you been immobile / bed-ridden?
- Known to have a chronic liver disease?
- Fatigue / malaise / myalgia / arthralgia / headache?
- Forgetfulness / confusion / poor concentration / personality changes?
- Known to have a kidney disorder?
- Hematuria / dysuria / nocturia / polyuria?
- Is the edema worse in the morning, or is it worse at night?
- Do your eyelids get puffy in the morning?
- Known to have a chronic heart disease?
- SOB (orthopnea / paroxysmal nocturnal dyspnea) / diminished exercise tolerance?
- Pain in the chest / palpitations / syncope / HT / IHD?
- H/s/o Grave's disease?

INTERPRETATION OF SYMPTOMS, AND DISCUSSION

AGE

– In children, congenital AVF may have to be considered
– Age > 40 in itself is one of the predisposing factors for the development of edema of lower limbs
– Young women may suffer from premenstrual edema
– 30–40 year old women may suffer from idiopathic cyclic edema.

GENDER

Premenstrual edema, idiopathic cyclic edema occur in women.

DIET

Ingestion of excess of salt may → edema.

OCCUPATION

An occupation involving long hours of standing may lead to edema of the lower limbs.

THE SITE OF COMMENCEMENT

In c/o DVT

– Pain often originates in the leg as DVT often originates in the soleal plexus of veins
– However, pain originating in the groin / iliac fossa, and edema originating in the thigh ⇒ origin of DVT in the iliofemoral segment.

Lymphedema almost always commences on the foot, and extends proximally upwards.

Lymphedema commencing proximally rather than distally should raise a suspicion of malignancy.

FLUCTUATION IN THE APPEARANCE

Non-lymphatic edema usually fluctuates i.e. appears on dependency, worse at the end of the day, and absent in the morning.

Established lymphedema does not fluctuate with changes in position of the limb.

The presence of **PARESTHESIA**
Inability to move the digits i.e. **PARESIS** / **PARALYSIS** } May suggest compartment syndrome.

RECURRENT EPISODES particularly of acute edema may be due to VTE (DVT).

INJURY

– Direct injury to a vein ⇒ TE
– Injury to a soft structure or bone in the vicinity of a vein → compression, which in turn may → TE
– Injury to the leg / forearm with / without fracture, but leading to concealed bleeding may → compartment syndrome

www.Ebook777.com

– Severe unaccustomed physical activity of a limb may → VTE—especially seen in upper limb. –The phenomenon is called 'effort syndrome'
– A tight POP cast may → compartment syndrome.

POISONOUS BITE
A vasculotoxic snake-bite, e.g. a viper → compartment syndrome.

PROLONGED IMMOBILITY FROM ANY CAUSE predisposes to DVT.

THORACIC OUTLET SYNDROME predisposes to VTE.

A MALIGNANCY predisposes to VTE.

REPERFUSION of a limb for grade (late) 2 acute ischemia, treated after 6 hours may → compartment syndrome.

SOB / PLEURITIC CHEST PAIN / HEMOPTYSIS in a suspected case of, or in a case known to have DVT ⇒ PE.
– Usually these symptoms develop with a sudden m/o onset
– Insidious mode of onset of these symptoms ⇒ comparatively less severe degree of PE
– A sudden cardiovascular collapse in a patient, who otherwise appeared to be alright previously ⇒ massive PE.

GENERAL EXAMINATION

Look specifically for the following findings:

- **Posture**, gait and general activity?
  Patient with acute edema usually lies down, with the affected limb flexed to a variable degree depending upon the 'tenseness'.
- Eyes and **facial expression**?
  Anxious face may suggest PE.
- **Vital parameters**, **JVP**?
  – Low-grade fever may be present in VTE
  – Tachycardia, tachypnea, hypotension and raised JVP ⇒ PE
  – **Pulses may be normal in compartment syndrome**
- Height, **weight**, BMI, body proportion?
  Obesity predisposes to DVT.
- **Mouth**
  Cyanosis of tongue may suggest PE.
- **Nails**
  – Cyanosis ⇒ PE
  – **Capillary refilling time may be normal in compartment syndrome**
- **Skin**
  Skin rash (erythema nodosum) may suggest rheumatological disease as a cause of edema.
- Lymphadenopathy?

PARTS EXPOSED AND POSITION

– In c/o lower limb edema: Both lower limbs + the lower trunk
– In c/o upper limb edema: Both upper limbs + the chest.

LOCOREGIONAL EXAMINATION

INSPECTION

- **Compare** the two limbs, particularly at the feet and legs.
  Note their relative size and the prominence of veins, tendons, and bones. Edema may obscure the veins, tendons, and bony prominences.

Free ebooks ==> www.Ebook777.com

- **Site**?
  - Venous edema tends to occur in dependent parts, e.g. lower limbs in upright position, overlying sacral region in recumbent position
  - Lymphatic edema may occur in either limbs / overlying scrotum depending upon the site of pathology.
- **Extent**?

  How far up the limb does the edema extend?

  In DVT, – Edema of the ankle / edema of the ankle extending up the lower leg ⇒ occlusion of the crural veins

  – Edema of the entire lower limb ⇒ iliofemoral thrombosis.
- **Surface**?

  Smooth / nodular?

  (Purple-red) nodules in a c/o lymphedema should raise the doubt about lymphangiosarcoma.
- **Skin**?
  - Normal / shiny?—shiny appearance ⇒ severe edema
  - Color—normal / red (i.e. erythematous) / white (pale) / blue (cyanotic) / dark (blackish / hyperpigmented) / depigmented?
  - Dermatitis, dermatophytosis, onychomycosis?
  - Verrucae (wart-like epidermal proliferations)?
  - Papillae (small nipple-shaped projections)?
  - Ulcer?
  - Lymphorrhea / chylorrhea? (= discharge of lymph/chyle from skin vesicles) – Due to lymphangiectasis.
- **Veins**?
  - Distended veins ⇒ venous occlusion as a possible cause
  - Dilated and tortuous veins ⇒ development of collateral superficial venous channels. For example—dilated veins in the subcutaneous tissue plane of antero-lateral abdominal wall (i.e. the trunk) occurs in c/o extensive DVT.
- **Joints**?

  Look for effusion.

PALPATION

- **Temperature**?
  - ↓ed temperature may be suggestive of compartment syndrome / venous ischemia / gangrene
  - ↑ed temperature may be suggestive of DVT / cellulitis.
- **Tenderness**?

  1. *Direct method of eliciting tenderness*
  - Flex the limb at the knee (to relax the muscles) and palpate the calf. With finger-pads, gently compress the calf muscles against the tibia, and search for tenderness and / or cords
  - Search for tenderness over the femoral vein just medial to the pulsation of the femoral artery at the groin.

  2. *Indirect method*

In a case suspected of having DVT

With the knee in extension, **passively** and yet forcibly dorsiflex the foot

Tenderness ⇒ DVT

In a case suspected of having compartment syndrome

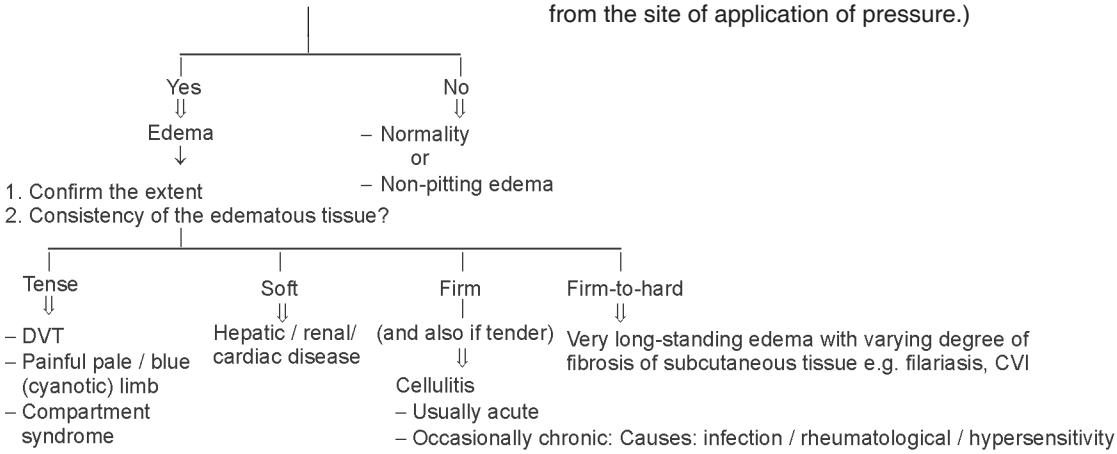
Passive flexion / extension of the foot and toes or hand and fingers → stretching of the muscles of the respective compartment, which in turn → tenderness, if the muscles have become ischemic

www.Ebook777.com

- **Press firmly but gently** with the thumb for at least 5–15 seconds—1. over the dorsum of each foot, 2. just behind each medial mallolus (on the bone), and 3. over the shins.

  Points to be noted

  Is a pit-depression caused by the thumb? (Pitting occurs due to forceful expulsion of edema-fluid from the site of application of pressure.)



- **Texture of the skin**: Normal / thickened?
- **Pulses**?

  The presence of normal pulses does not rule out the possibility of compartment syndrome.

  It may be practically difficult to palpate pulses if edema is tense. Inability to palpate pulses does not necessarily mean that the pulses are absent.
- **Measurements**
- **Musculoskeletal disorder**?
  - Joint effusion, ruptured Baker's cyst may suggest rheumatological disease as a cause of edema
  - Arthritic / post-traumatic fixed ankle, flat foot, reduced mobility at the knee / hip from any cause, etc.
- **Neurological disorder** leading to reduced mobility?

EXAMINE **THE OPPOSITE LIMB**

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. Acute edema may be pitting or non-pitting; that depends on the tension of fluid causing the edema. And anyhow, arriving at the diagnosis in a c/o acute edema does not depend on whether it pits or not.
2. DVT may not have demonstrable signs, and diagnosis often depends on high clinical suspicion (and investigation).

   About half of the patients with DVT in the calf have tenderness and cords deep in the calf.

   As the absence of calf tenderness does not rule out DVT, even the presence of calf tenderness is not necessarily always due to DVT. Calf tenderness may be nonspecific, and may be present without thrombosis.
3. Venous edema *v/s* lymphatic edema

Clinical feature	*Venous edema*	*Lymphatic edema*
– Duration	'Short'	'Long'
– M/o onset	Sudden or insidious	Insidious
– Progress	Fluctuating	Steady. May fluctuate only in the earliest stages
– Toes	Unaffected	Involved
– Lymphorrhea	Absent	Present
– Skin	– Normal – Abnormal in c/o CVI	Thickened

Free ebooks ==> www.Ebook777.com

4. If a patient in a forearm cast complains of pain, believe him. Split and spread the cast, and elevate the limb. The patient may be suffering from compartment syndrome.
5. Edema-like chronic diffuse swelling may initially be mistaken for (true) edema.

STEPWISE ANALYSIS OF DATA

CLINICAL SETTING

Acute

Is the limb cold?

If yes,

Consider one of the following possibilities

- Acute arterial insufficiency
- Painful blue limb ± impending venous gangrene
- Advanced stage of compartment syndrome

Otherwise

Consider one from the remaining causes of acute edema

Chronic

True edema

Essentially unilateral

Refer to page. 704

Bilateral

Refer to page. 704

DIFFERENTIAL DIAGNOSIS

Acute venous thrombosis (DVT)

- This condition may affect the deep veins / superficial veins / both systems simultaneously
- Isolated involvement of superficial system is rare
- The deep venous system is so commonly involved that acute venous thrombosis is taken for granted as deep vein thrombosis (DVT) unless specified otherwise
- Determination of the etiology of DVT is often not difficult
- In the absence of recognizable precipitating event, thrombophilia should be suspected and patient should be investigated accordingly.

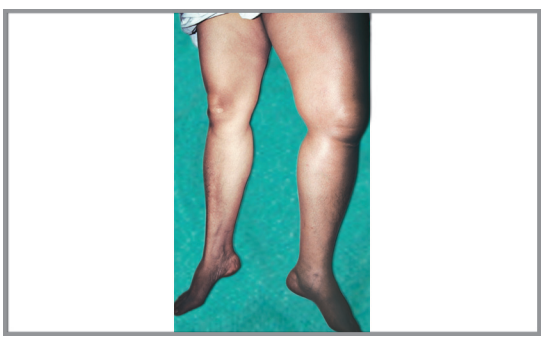


Fig 50-1 Acute DVT

www.Ebook777.com

Causes of DVT (Risk factors for venous thrombosis)

Primary

Due to a primary hypercoagulable state = Thrombophilia
DVT may occur without any obvious precipitating cause

1. Antithrombin III deficiency: Increased risk of DVT, mesenteric vein thrombosis, arterial thrombosis. Rare before the age of 20 years. Usually thrombosis occurs in association with other risk factors
2. Protein C and S deficiencies: < 40 years of age
   VT also in mesenteric, renal and cerebral circulation
3. Antiphospholipid syndrome: Risk increases with age
   The syndrome occurs in patients with lupus anticoagulants and anticardiolipin antibodies.
   A high incidence of venous and arterial thrombotic events including recurrent DVT, PE, IVC thrombosis, MI, stroke, failure of vascular reconstruction
4. Hyperchromocysteinemia: Inherited or secondary to deficiencies of vitamins B_{12}, B_6 and folate
   Increased risk of recurrent venous and arterial thrombo-embolic events
5. Hypofibrinolytic state

Secondary

DVT is usually of this variety
An obvious precipitating cause almost invariably recognized. *Risk factors*:

- Age > 40
- Previous VTE
- Prolonged immobility due to any cause like an injury / accident / prolonged surgery / stroke / sepsis, etc.
- Any major surgery, in particular pelvic surgery / hip surgery / lower limb surgery
- Any accident particularly involving the lower limb.
- Malignancy, CT
- Any major (medical) illness e.g. IBD, etc.
- Estrogen therapy

Risk factors for postoperative VTE

- Those listed above
  +
- Major joint replacement
- Peripheral vascular reconstruction
- Surgery for cancer / IBD
- Cardiovascular complications
- Thrombophilia

Causes of secondary DVT specifically applicable to upper limb

- The end result of chronic thoracic outlet compression
- May arise out of the blue often in a young person, precipitated by unaccustomed physical activity or jerky movements of the limb—called 'effort syndrome'
- Cancer
- Pressure on the veins (axillary / subclavian) from crutches or abducted position of the limb
- Indwelling catheters in any of the great veins of the upper limb
- May be truly spontaneous

Criteria for diagnosis of DVT

There is no clinical differentiating point for the diagnosis of DVT. However, diagnosis of DVT can still be made fairly confidently on the basis of the basic and supporting points.

Basically, acute painful edema / uncomfortable heavy limb, ± fever.

In c/o lower limb: Acute pain and edema in the calf, usually unilateral. Acute pain in the groin/iliac fossa, edema starting in the thigh, and tenderness in the groin over femoral vein ⇒ iliofemoral thrombosis.

Supporting points

- Erythema / pallor / cyanotic tinge
- Distended collateral superficial veins
- ↑ed local temperature
- Tenderness on (gently) squeezing the affected segment of the limb e.g. calf / thigh. (Tenderness in the calf on forceful dorsiflexion of the foot (Homan's sign) is not accurate.)
- Pitting or non-pitting edema. The limb is often tense
- The presence of predisposing (risk) factor(s) ±.

A circumscribed although elongated swelling, redness, warmth and a subcutaneous cord ⇒ superficial thrombophlebitis.

Complications of severe extensive DVT:

- Painful pale limb—due to arterial spasm
- Painful blue (cyanotic) limb
- Venous gangrene.

Free ebooks ==> www.Ebook777.com

Venous TOS—leads to subclavian-axillary vein thrombosis.
The intensity of symptoms and signs is directly related to the length of the occlusion, the existence of prethrombotic venous collateral pathways, and the amount of the activity of the involved limb.

Clinical features of the acute event :
Edema, cyanotic discoloration of the limb and distended subcutaneous collaterals.
M/o onset—sudden.
The edema usually involves the entire limb (including hand), and is **characteristically non-pitting.**
In time, venous engorgement develops of the superficial collateral veins over the arm, base of the neck, and anterior chest as collateral drainage is established.
Pain in the arm or axilla, usually worsens with exertion.

Causes of delay in making the diagnosis: 1. The lack of a clear precipitating event 2. The presence of only few symptoms due to ample collaterals having been developed. 3. On Doppler study: the area of acute thrombosis may be hidden under the clavicle, however, and the axillary and basilic veins may appear normal.

Residual symptoms which may occur in some patients, after recovery from the acute event: Pain, early fatigue and limb edema.

Persistent symptoms: These are related to residual venous obstruction rather than reflux as against in the lower limb. They are likely to reflect an above-average activity level often with an ongoing need for active use of the involved limb. Pain and edema are made worse by dependency and active use of the involved limb.

Painful blue (cyanotic) limb
– A painful pale limb may progress to a painful blue limb within 24–48 hours
– Usually found in patients with advanced malignant disease
– Severe bursting pain
– Extreme edema of the entire limb
– Cyanosis
– In ~ 50% of patients venous gangrene develops affecting the toes and foot initially within 24–48 hours of onset. ⇒ extension of thrombosis from the main venous trunk and collaterals, into the venules and capillaries resulting in secondary arterial insufficiency.
Causes of considerable morbidity and mortality: 1. The development of gangrene necessitating amputation, and 2. PE.

Compartment syndrome
This condition may affect leg or forearm, as the fascial compartments in these segments of the limbs are quite tight, i.e. not 'accommodative', and consequently they may get tightly compressed by blood, exudate or swollen muscle. Conversely, the fascial compartments of the thigh and arm are not so tight, i.e. they are 'accommodative'. Hence, compartment syndrome does not occur in the thigh/arm.
Compartment syndrome may complicate certain specific clinical conditions; hence, making an early diagnosis should not be difficult. However, unfortunately, diagnosis is delayed due to a low index of suspicion or/and a negligent attitude towards genuine symptoms (particularly severe pain) complained by the patient.

Conditions/situations that are prone to develop compartment syndrome :
– A supracondylar fracture in a child
– Dislocation of elbow
– An unsplit cast or a tight bandage on a forearm fracture
– A fractured tibia, dislocation of knee
– A badly treated fracture / a crush injury / a bullet wound
– Occasionally, soft tissue injury (only, without fracture) causing hematoma, e.g. injury to the thenar muscles
– Vasculotoxic snake-bite.

Symptoms
– Severe pain
  In case if a fracture has been reduced, then the pain should become less. Severe postoperative pain is the critical early symptom
– Inability to move the digits
– Numb digits.

www.Ebook777.com

Physical findings

– Temperature: Initially may be ↑ed; subsequently ↓ed
– Excruciating tenderness.
  Stretching of the tendons coursing through the affected compartment, by passive flexion / extension of the digits / foot / hand → excruciating tenderness.
  Examples:
  - Tenderness on (passive) plantar-flexion ⇒ affection of the anterior compartment of the leg
  - Tenderness on (passive) foot extension ⇒ affection of the posterior compartment(s) of the leg
  - Tenderness on (passive) extension of fingers / hand ⇒ affection of the anterior (flexor) compartment of the forearm
  - Tenderness on (passive) flexion of fingers / hand ⇒ affection of the posterior (extensor) compartment of the forearm (rare)
– Unusually tense limb
– Pulses—absent / weak / normal
– Capillary refilling time—reduced / normal.

A pitfall

The presence of normal pulses and capillary refilling time do not necessarily exclude compartment syndrome.

Vasculotoxic snake bite

Examples of snakes with vasculotoxic venom: Viper, adder, rattlesnake.

Symptoms

– Painful local edematous swelling within 1–2 hours of bite
– SOB ± —due to pulmonary edema or/and pleural effusion due to generalized ↑ in capillary permeability
– Syncope ±.

Physical findings on G/E

– Conjunctival and facial edema ± —due to generalized ↑ in capillary permeability
– Hypotension and shock may be caused by hypovolemia resulting from leakage of blood / plasma into the bitten limb.
  A massively swollen limb can accommodate half the total blood volume
– Angioedema ±.

Physical findings on locoregional examination

– 2 discrete fang-marks may be visible
– The local edema originally at the bite site may spread to involve the whole limb and even adjacent trunk, reaching its peak in 24–72 hours
– Bruising—at the site of bite ± along the track of lymphatics ± overlying the painful enlarged regional lymph nodes
– Blistering after 12–24 hours at the site of bite ± extending up the limb
– Bite on digits may be complicated by necrosis
– Bites near fascial compartments may → compartment syndrome.

Systemic complications which may occur

– Spontaneous bleeding from various sites may → oral bleeding from gums, epistaxis, GI bleeding, hemoptysis, hematuria, subconjunctival bleeding, bleeding in the skin.
  Intracranial or/and massive GI bleeding may prove fatal
– Arrhythmia ± —due to bleeding and necrosis of cardiac muscle and coronary vasoconstriction
– Neurotoxic signs are unusual. Russell's viper found in south India and Sri Lanka may → generalized rhabdomyolysis.

Acute (infective) cellulitis

– Painful and tender edematous swelling
– M/o onset—sudden
– Progress—rapid
– Precipitated by an injury which may be trivial or even unnoticed.

Free ebooks ==> www.Ebook777.com

- Constitutional symptoms ±
- Edematous swelling:
  - Red glossy
  - Pits on pressure
  - Consistency—firm (brawny edema)
- Regional acute lymphadenitis ±.

Differentiation of erysipelas from cellulitis

- Typical rosy-red rash; disappears on pressure
- Sharply defined irregular edge, which is raised from the level of the skin
- Vesicles contain serum (not pus).

(- In c/o face: Erysipelas can spread to the pinna (being cuticular affection.)

Rheumatological disease, e.g. RA.

Supporting points

- Joint effusion
- Ruptured Baker's cyst
- Skin rash (erythema nodosum).

Acute angioedema

= a vascular reaction of deep dermis / subcutaneous tissue / submucosal tissue leading to localized edema caused by dilatation and increased permeability of capillaries. Basically it is a type of urticaria characterized by development of wheals (localized areas of edema) on the skin on any site of the body. Whenever there is involvement of deeper dermis / subcutaneous tissue / submucosa, it is termed angioedema instead of urticaria.

Types (Classification) of angioedema :

A. Clinical
   1. Acute: Pink colored wheals with central pallor. Occur asymmetrically anywhere in the body. Itching.
   2. Chronic: May last for months/years. In many cases, it is not possible to pinpoint the allergen.

B. Aetiological
   1. Allergic.
   2. Non-allergic (Non-immunogenic): Dermatographic / cholinergic / urticarial vasculitis.

Induced by injury/pressure. A slight scratch may produce marked response	Induced by emotion / exercise / variations in atmospheric temperature	Paresthesia rather than itching Duration: > 24 hours Minimal response to antihistaminic

Acute inflammatory edema

- Associated with erysipeloid inflammation of skin of the affected part. Erysipeloid inflammation always precisely affects the same area; but the associated edema may spread considerably beyond it
- Parts which may be affected other than limbs—eyelid, lip, tongue
- Recurrent attacks of painful and tender edematous swelling. Red and hot
- M/o onset—sudden
- Progress—dramatic / rapid
- Regress—slow. Regress is slower and slower with succeeding attacks. And eventually, edema may become permanent.

Pretibial myxedema associated with Grave's disease

- Legs and dorsum of feet
- M/o onset—insidious
- Itching ±
- Orange-peel appearance
- Coarse hair
- Hyperpigmentation
- Thickening of skin.

Cause: Infiltrative dermopathy—deposition of mucin-like substance in the skin at this specific site.

www.Ebook777.com

A plexus or peripheral nerve lesion
– Burning pain in the limb
– Sweating
– Cyanosis
± associated neurological deficit.

CVI (Chronic Venous Insufficiency)
Symptoms (apart from edema)
– Cramps—may be vague; not necessarily suggestive of intermittent arterial pains
– Aching in the legs on dependency / before periods
– 'Heaviness' / restlessness in the legs
– Discoloration of skin in the gaiter area / lateral malleolar area
– Tenderness
– Clear-cut past h/o DVT, OR Past history suggestive of factors which predispose to develop DVT and subsequently post-thrombotic syndrome:
  - Calf pain and edema of sudden onset in the leg, usually unilateral
  - Prolonged immobility due to any cause like an injury / accident / prolonged surgery / stroke / sepsis, etc.
  - Any major surgery, in particular pelvic surgery / hip surgery / lower limb surgery
  - Any accident particularly involving the lower limb
  - Venous thromboembolism
  - Any major (medical) illness
  - Estrogen therapy.

Physical findings (apart from edema)
– Discoloration of skin in the gaiter area / lateral malleolar area—due to venous flare, or areas of hyperpigmentation / depigmentation
– (stasis) Dermatitis
– Lipodermatosclerosis
– A chronic indolent venous ulcer ±.

Widmer's classification for gradation of CVI—based on outward clinical appearance of the lower limb:
- Grade 1 = Venous flare
- Grade 2 = Hyper- / depigmented areas
- Grade 3 = Open / healed ulceration.

Lymphedema
Symptoms
– Painless
– Mode of onset—insidious
– Almost always commences distally on the foot, and extends upwards
– Two situations in which the case should be investigated aggressively to rule out malignancy: **1**. Age at onset—beginning from middle age onwards. **2**. Lymphedema commencing proximally rather than distally
– Initially fluctuating like any non-lymphatic edema i.e. appears on dependency, worse at the end of the day, and absent in the morning. As the condition establishes, it is less and less affected by changes in position, and is present all the time
– Recognized precipitating events in c/o (primary) lymphedema: Infection in the form of cellulitis, injury, surgery, venous disease, etc.

Physical findings
– 'Buffalo-hump' on the dorsum of foot, infilling the submalleolar depressions
– Skin changes in advanced cases: Dermatitis, dermatophytosis, onychomycosis, verrucae, papillae, ulcer – usually due to associated CVI
– Lymphorrhea / chylorrhea due to lymphangiectasis
– Purple-red nodules, non-healing bruises, non-healing ulcer(s) should raise the doubt about lymphangiosarcoma
– The edema pits-on-pressure during the initial period; but becomes non-pitting as the condition advances.

Free ebooks ==> www.Ebook777.com

***Clinical staging** of lymphedema*

Stage 1: Fluctuating, and pitting-on-pressure.

Stage 2: Persistently present, i.e. does not significantly reduce on elevation of the limb; and non-pitting.

Stage 3: Irreducible skin changes develop—fibrosis, papillae.

Causes of lymphedema (Etiological classification of lymphedema)

Secondary

The most common form of lymphedema

Causes

- Trauma—especially degloving injuries of extremities
- Postcellulitis Acute bacterial lymphangitis is a frequent triggering event
- Filariasis
- Malignancy and / or its treatment (radical excision of / RT to the regional lymph nodes)
- Severe external compression
  - Pressure from bands / rings / garters
  - Extensive scars after injury / burns
  - Amniotic bands → congenital lymphedema
- Venous disease. Many patients with CVI have a degree of lymphatic impairment that contributes to edema

Primary

Lymphedema congenita

Within a year of birth

- More likely to be bilateral
- More likely to involve the whole (lower) limb
- M > F
- Sporadic / familial

Lymphedema precox

1–35 years

- The most common form of primary lymphedema
- Peak incidence—shortly after menarche
- F > M
- Unilateral > bilateral
- BK > involvement of the whole (lower) limb

Lymphedema tarda

After 35 years of age

- Often associated with obesity
- Lymph nodes are replaced with fatty and fibrous tissue

Hyperplasia or lymphangiectasis due to reflux from thoracic duct due to failure of valves. Milky vesicles may be present in the skin which may burst and discharge chyle

Diffuse lymphangioma of the whole limb

- Elephantiasis lymphangiectatica

Elephantiasis and lymphedema are two distinct entities.

Elephantiasis follows lymphedema.

Elephantiasis = a state of diffuse thickening of skin and subcutaneous tissue due to fibroplasia. Elephantiasis is confined to skin and subcutaneous tissue. It does not extend beyond the deep fascia.

When elephantiasis involves the lower limb, in majority of cases, it is restricted to below the knee.

AVM

Present from birth, but may become symptomatic in later life.

Presenting symptoms

- In young children: Obvious swelling or limb enlargement, disfigurement, discoloration (birthmark)
- In adults: Obvious swelling or limb enlargement, disfigurement, discoloration (birthmark), limb length discrepancy, aching, sensation of heaviness, varicose veins (fistula varicosity)



Fig 50-2 (Filarial) Elephantiasis



Fig 50-3 Post-axillary dissection lymphedema

www.Ebook777.com

– In women, exacerbation of symptoms occurs during menarche / pregnancy
– Pain.

Signs

– Skin discoloration
– Characteristics of the edematous swelling:
  - Soft / firm and 'spongy' consistency
  - Non-compressible. Compressible only if there is predominantly venous component
  - Pulsatile
  - Palpable thrill
  - Continuous machinery murmur. Bruit ±
– Fistula varicose veins.

Edema due to aging / prolonged immobility or lower limb dependency / general debility results from muscle pump dysfunction.

Premenstrual edema

– Young age
– Edema particularly of the whole body: Ring becomes tight. Edema of breasts → feeling of tightness / discomfort / pain in the chest.

Hepatic failure

– Jaundice ±
– General failure of health. Easy fatigability
– Edema, ascites, pleural effusion—due to fluid and salt retention
– Episodes of variceal bleeding from portal HT
– Bleeding tendency
– Mental impairment—due to encephalopathy
– Muscle wasting
– Palmar erythema
– Tachycardia
– 'Water-hammer' pulse (= collapsing type of pulse)

} Due to hyperkinetic circulation—increased stroke volume, wide pulse pressure and decreased peripheral resistance.

– Spider nevi (> 3)
– Gynecomastia (applicable to male patients)
– Splenomegaly
– Ejection systolic murmur
– Thrombocytopenia.

Renal failure

– Puffiness of face more in the morning
– Oliguria
– Hiccoughs
+ Absolutely any nonspecific symptom and specific symptom from all body systems.

Cardiac failure

– Exertional dyspnea / orthopnea / paroxysmal nocturnal dyspnea
– Attacks of pulmonary edema: Awakening from sleep with extreme suffocation; gasping for breath; profuse sweating; skin getting cold, pale and cyanosed; wheezing; expectoration of frothy and pinkish sputum. Attacks gradually subside over 15–20 minutes
– Fatigue
– Insomnia
– Daytime oliguria combined with nocturia
– Pulsus alternans.

Free ebooks ==> www.Ebook777.com

- Cheyne-Stokes breathing
- Raised JVP
- Basal rales with expiratory wheezing
- Bubbling rales all over the lungs ⇒ acute pulmonary edema
- Pleural effusion ±, particularly on the left side
- Left ventricular hypertrophy. Gallop rhythm
- Symptoms and signs due to etiology: Valvular heart disease / constrictive pericarditis / HT / IHD / arrhythmia / chronic obstructive lung disease.

Idiopathic cyclic edema

- 30–40 years of women ('unfortunately' often who are emotional)
- Long history in years
- Remissions and relapses off and on. Attacks may coincide with stress and strain
- Tighter fitting of ring, bangles and clothes
- Puffiness of face and hands on waking up in the morning. Edema of the lower limbs occurs as the day progresses
- Dyspnea on exertion ±.

Commonly confusing DD: Edema associated with hypothyroidism.

Retroperitoneal fibrosis

Refer to chapter 33.

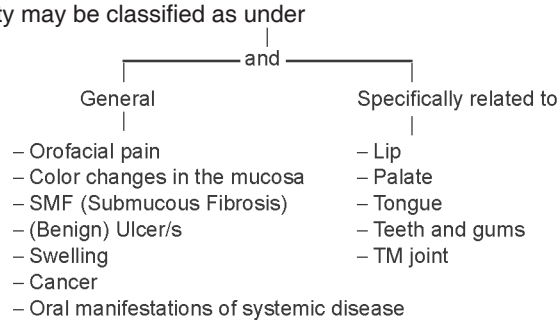
Reflex sympathetic dystrophy (- Complex regional pain syndrome): Refer to chapter 45.

www.Ebook777.com

51 ORAL CAVITY OR OROFACIAL PAIN

GENERAL REMARKS

1. Many systemic conditions can manifest initially within the mouth. Indeed, features of oral disease may lead to the diagnosis of previously unrecognized systemic disorders.
2. Noting any color change is probably the single most important early finding suggestive of oral cancer.
3. Any lesion in the oral cavity that has failed to resolve or improve within 2–3 weeks should be biopsied to exclude malignancy.
4. A single oral ulcer is more likely to be malignant than multiple ulcers, particularly if asymptomatic.
5. High index of suspicion is necessary for the diagnosis of early oral cancers, which are frequently asymptomatic.
6. The prognosis deteriorates the more posterior the malignant lesion is in the oral cavity.
7. Apart from defining the site of lesion in anatomical term, analyze as to whether the area is keratinized (dorsal surface of the tongue, hard palate) or nonkeratinized (ventral surface of the tongue, buccal mucosa).
8. Complaints of the disorders of oral cavity may be classified as under



LOCOREGIONAL EXAMINATION of the mouth

Examine the external aspect of the face, and then examine the cavity.

Examination of the oral cavity

INSPECTION

- **Lips**
  - Any deformity—congenital / otherwise?
  - Evert the lips and examine the mucosal aspect
  - Retract the cheek for further intraoral examination.
- **Inner** (i.e. mucosal) **aspect of the cheek**
- **Teeth**
  - Number?
  - Unerupted / partially erupted teeth?
  - Condition of the teeth?

Free ebooks ==> www.Ebook777.com

- **Gums**
  - Hyperplasia?
  - Gingivitis?
- **Tongue**
  - Ask the patient to put the tongue out. Inspect the dorsal surface
  - Wrap a piece of gauze around the tip of the tongue. Hold the tongue from over the gauze and pull it gently to either side and examine the lateral borders
  - Ask the patient to place the tip of the tongue against the roof of the mouth
- Inspect the undersurface of the tongue and **floor of the mouth**
- Depress the tongue with a depressor and inspect the **tonsils and oropharynx**
- Ask the patient to tip the head backwards and open the mouth widely. Inspect the **palate and uvula**.

PALPATION

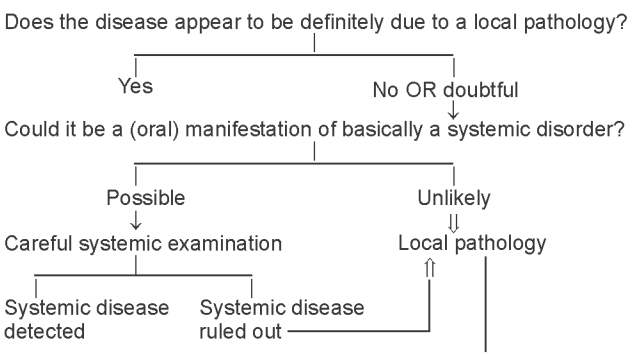
- **Lips and cheeks**: Palpate bidigitally
- **Tongue**

  Ask the patient to keep the tongue within the mouth at rest in relaxed state. Palpate with a gloved (index) finger.

  Reason for keeping the tongue within the mouth relaxed: It is extremely important to feel for induration to determine the size of any lesion. If the tongue is protruded, the contracted muscle (tongue is essentially a muscular organ) may give a false impression of induration and consequently an error in clinical judgment.
- **Floor** of the mouth
  - Palpate **bimanually**: Insert a gloved finger into the mouth along the groove between the alveolus and the tongue as far back as possible and then press gently on the floor of the mouth
  - Place fingers of the other hand under the jaw
  - The actual process of palpation involves a gentle attempt to make fingers of both the hands meet. Fingers will not meet but in the attempt, details of the tissues/structures in the floor of the mouth can be evaluated. Start palpating from behind forwards up to the midline
  - Normal and abnormal structures which can be thus palpated—submandibular salivary gland, duct of the gland, the presence of any stone, etc.

  Bimanual palpation enables to differentiate between the swellings of (/ arising from) the structures deep to the mylohyoid muscle and superficial to the mylohyoid muscle. Example: A submandibular salivary gland is felt bimanually whereas an enlarged submandibular lymph node can be felt only by the finger outside but not by the intraoral finger.

GENERAL GUIDELINE for arriving at the diagnosis of a lesion in the oral cavity



– A single lesion that is painless: Make every attempt to rule out cancer
– Advanced cancer is painful
– Benign lesions are often painful irrespective of size and etiology

www.Ebook777.com

HISTORY

Pain

- Exact site?
- Is it extending? Extension may suggest spreading inflammation
- Does it radiate anywhere, for example to the ear?
- Duration?
- Mode of onset?
- Progress / regress / fluctuation?
- Intensity?
- Nature: Throbbing / burning / electric shock-type / dull aching, etc.
- Precipitating (triggering) / aggravating / relieving factor? Such as—chewing / fine touch / a blast of cold wave
- Continuous or intermittent?

Associated symptoms

- H/o injury to the jaw?
- Any clicking sound in the TM joint?
- Inability to open the mouth (– due to trismus)?
- Any swelling, ulcer?

EXAMINE the head, face, neck and oral cavity.

ARRIVING AT THE DIAGNOSIS of orofacial pain

GENERAL OUTLINE

Look for an organic cause first

↓

If no obvious organic cause could be found

Could it be due to adenoid cystic carcinoma of salivary gland?	Atypical pain	Psychogenic
↓	– Common in women > 30	– Symptoms often ill-defined
Keep an index of suspicion	– Usually in the maxillary region	– Sleep not disturbed
↓	– Dull continuous ache	– Other psychological complaints ±
Investigate accordingly if necessary.	– Appetite and sleep not disturbed	– Patient otherwise appears quite well
	– Objective signs absent	– Objective signs absent
	– Antidepressants sometime help	– Investigation reports negative

If organic cause is thought to be responsible for pain, evaluate further according to the following steps

Step 1

Tissue/organ involved?

- Loco-regional
  - Bony / articular
    - – Jaw (maxilla / mandible)
    - – TM joint
  - Dental
  - Soft tissue
    - – Oral mucosa (including mucoperiosteum) Usually → burning pain. The 'burning mouth syndrome'.
    - – Nerve (Trigeminal neuralgia)
    - – Blood vessels (Temporal arteritis)
    - – Salivary glands (Adenoid cystic carcinoma, other tumors).
    - – Glossodynia
- Referred
  - – Ischemic heart disease
  - – Glossopharyngeal neuralgia
  - – Nasal disease

Free ebooks ==> www.Ebook777.com

Step 2 – Etiopathology?

Jaw
Fracture, maxillary sinusitis, alveolar abscess, tumor, occult cavities in the jaw bone.

TM joint
Osteoarthritis, loose meniscus, trismus, locked jaw, atypical pain.
Refer to chapter 52.

Dental
- Malocclusion, unilateral mastication
- Poorly fitting dental prosthesis
- Pulpitis, root abscess, etc.

'Burning mouth syndrome'
- Ill-fitting dentures
- Xerostomia, i.e. dryness of mouth
- Tongue thrusting
- 'Geographical tongue'
- Hematological disorders
- Diabetes mellitus
- Chilli and/or spicy diet. Nutritional deficiency states
- Food allergy
- Smoking, alcoholism
- Drugs
- Candidiasis
- Psychogenic, e.g. cancer phobia.

Nerves
Trigeminal neuralgia, glossopharyngeal neuralgia.

Vascular
Giant-cell arteritis.

Salivary glands
Adenoid cystic carcinoma.

DIFFERENTIAL DIAGNOSIS of selected causes of orofacial pain

Trigeminal neuralgia
- Middle-aged / elderly
- Unilateral pain confined to one or more divisions of trigeminal nerve, most often mandibular and/or maxillary
- Severe. Electric shock like or shooting in nature
- Pain either begins spontaneously, or may be triggered by even fine touch (apart from chewing or brushing teeth) or a blast of cold wave
- Typically periodic lasting from seconds to minutes. Subsequent attacks may be longer, and incapacitate the patient.

Possible causes
- An arterial or a venous loop in contact with the trigeminal sensory root where it enters the pons at the root entry zone (REZ). Vascular contact with the trigeminal REZ is more likely to occur as vessels elongate and become ecstatic with aging, or as systemic HT develops
- A (benign) tumor compressing the nerve
- Multiple sclerosis.

Glossopharyngeal neuralgia
- The site of pain—posterior third of the tongue, or in the tonsil, and sometimes in the ear.
  The pain is virtually always unilateral.

www.Ebook777.com

– Swallowing precipitates the pain
– Rest of all the features are similar to trigeminal neuralgia.

Almost all cases are idiopathic, with no specific cause identified.

Neuralgia = pain due to inflammation or partial injury or neoplastic involvement of nerves / nerve roots. It is always severe, often intractable, and requires special measures to treat.

Adenoid cystic carcinoma of salivary gland

Tumor may be present for **some years** before diagnosis. Before the mass becomes palpable or evident, the patient may have spent some years visiting different specialists with the complaint of facial pain.

COLOR CHANGES in the oral mucosa

White lesions:
– Developmental: Fordyce spots, white sponge nevi
– Candidiasis
– Frictional keratosis due to excessive tooth brushing or ill-fitting prosthesis
– Leukoplakia, cancer.

Red lesions:
– Thinning and atrophy of oral mucosa
– Vascular lesions, e.g. hemangioma, purpura
– 'Geographic tongue' (erythema migrans)
– Candidiasis
– Erythroplakia.

Red/white lesions:
– Speckled leukoplakia
– Stomatitis nicotina
– Lichen planus
– Cancer.

Purple, violet lesions.

Pigmentation:
– Amalgam tattoo
– Racial (natural)
– Nevi
– Drug-induced (contraceptive pills, antimalarial drugs, etc.)
– Heavy metal poisoning
– Endocrine disorders (e.g. Addison's disease)
– Certain syndromes (e.g. Peutz-Jeghers)
– Malignant melanoma.

WHITE LESION

HISTORY

- Duration? Acute lesion may be due to pseudomembranous candidiasis (thrush)
- Mode of onset?
- Progress?
- Associated disorder already existing? e.g immunocompromised status from any cause like AIDS, leukemia, etc.
- Have you been taking (broad spectrum) antibiotic(s) or steroid inhalation?
- H/o excessive tooth brushing?
- Any jagged tooth or ill-fitting prosthesis (crown / denture, etc.)?
- Tobacco consumption in any form?

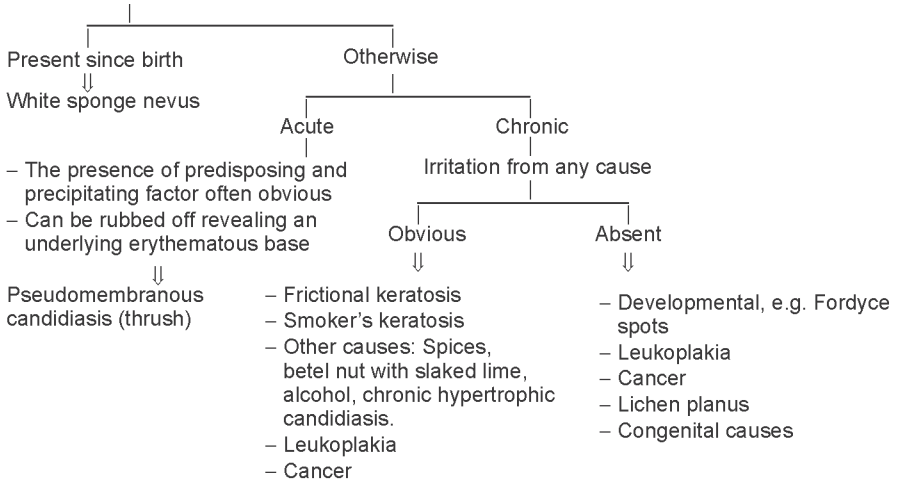
LOCAL EXTRAORAL EXAMINATION (Examine the external aspect of the face, and then proceed with intraoral examination.)

Free ebooks ==> www.Ebook777.com

LOCAL INTRAORAL EXAMINATION

- Number?
- Site and extent. Does the site appear to be prone to irritation from any cause such as jagged tooth or ill-fitting prosthesis (crown/denture, etc.)?
- Margin, shape, size, surface?
- 'Satellite lesions'?
- **Try to rub it off**—Is it possible? If so, does it reveal an underlying erythematous base?

ARRIVING AT THE DIAGNOSIS



Notes

– *'Keratosis'* (= hyperkeratosis)
Etiological factor is readily recognized and withdrawal of the cause may lead to regression of the white patch.

– *'Leukoplakia'*
(Various) Criteria: A hyperkeratotic lesion the etiological agent of which is not recognized / known, OR the lesion has been established to a point where it will not regress after withdrawal of the causative agent. The presence of dyskeratosis is essential, according to one school of thought.

DIFFERENTIAL DIAGNOSIS

Fordyce spots (These are intraoral sebaceous glands)
– Yellow / white.
– Usually on the buccal mucosa.

White sponge nevus
Rough white-colored plaque present from birth.

Acute pseudomembranous candidiasis (thrush)
– White plaques that can be rubbed off revealing an underlying erythematous base
– Predisposing condition: Basically immunocompromised status from any cause like AIDS, leukemia, etc.
– Precipitating factor: (Broad spectrum) antibiotic(s), steroid inhalation.

Frictional keratosis of gum due to **excessive tooth brushing**
– Obviously, h/o excessive tooth brushing and the white-colored lesions situated on the gums
– Associated erosion at cervical margins of the teeth ±.

Frictional keratosis due to **ill-fitting prosthesis**
H/o chronic irritation due to prosthesis like a crown / denture, etc.

www.Ebook777.com

Leukoplakia

Definition: Any white lesion in the oral cavity (or external genitalia) which cannot be rubbed off, and which cannot be characterized clinically or pathologically as any other disease.

The affected mucosa looks as if painted by white paint which has dried and cracked, and may be homogenous or speckled on an erythematous base.

- Size—variable—ranges from a small, circumscribed white plaque to an extensive lesion involving wide areas of oral mucosa
- 'Milky blush' on mucosal surface is the early stage appearance. Otherwise, it can be yellowish or gray
- Surface—smooth / wrinkled / verrucous (typical warty surface) / nodular, and many lesions are traversed by cracks
- Consistency—soft or firm. **Induration suggests malignant change and is an indication for immediate biopsy**. Even otherwise, biopsy (preferably excisional) of leukoplakia is indicated.

'Hairy' leukoplakia

- Usually affects lateral border of the tongue appearing characteristically as white striae
- Caused by EB virus
- Strongly associated with HIV infection.

Malignant melanoma

- Usual site—palate
- Usually ill-defined margins
- Satellite lesions ±.

Congenital causes of white lesion/s:

- **A symmetrical developmental hyperkeratotic patch on the undersurface of the tongue**—A premalignant condition
- **Leukokeratosis mucosae oris**: All the oral mucous membranes have a filmy white appearance.

RED LESION

HISTORY

- Duration?
- Mode of onset?
- Progress?
- Associated disorder already existing?, e.g. immunocompromised status from any cause like AIDS, leukemia, etc.
- Have you been using a denture?

LOCAL EXTRAORAL EXAMINATION (Examine the external aspect of the face, and then proceed with intraoral examination.)

LOCAL INTRAORAL EXAMINATION

- Number?
- Site and extent?
- Margin, shape, size, surface?
- **Try to rub it off**—is it possible?

DIFFERENTIAL DIAGNOSIS

Median rhomboid glossitis

- The abnormal area lies in the midline immediately anterior to the foramen cecum
- Smooth / lobulated oval / triangular patch
- Color—a rather deeper than the rest of the tongue.

DD : CA tongue. However, CA tongue rarely occurs in the midline.

Geographic tongue

- The normal rough and coated areas together with scattered smooth red areas that are denuded of papillae → a map-like pattern that changes over time

Free ebooks ==> www.Ebook777.com

– The patches spread and recede in an irregular fashion and fresh patches appear.

The condition is benign. Etiology is not known.

Known predisposing conditions: Acute GI problems, congenital heart defect.

Denture-induced candidiasis

A red patch confined to the area of hard palate covered by the denture.

Erythroplakia

Definition: Any bright red velvety plaque, which cannot be characterized clinically or pathologically as any other lesion.

– Well demarcated from adjacent normal epithelium. Usually irregular
– Surface—smooth or may be nodular
– All erythroplakic areas must be completely excised and subjected to histopathological examination.

RED / WHITE LESION

HISTORY

- Duration?
- Mode of onset?
- Progress?
- Have you been on antihypertensive, hypoglycaemic or any other drug (inducing lichen planus)?
- Do you have any skin lesions?

LOCAL EXTRAORAL EXAMINATION (Examine the external aspect of the face, and then proceed with intraoral examination.)

LOCAL INTRAORAL EXAMINATION

- Number?
- Site and extent?
- Margin, shape, size?
- Surface. Does it appear to be erosive, atrophic?
- **Try to rub it off.** Possible?

DIFFERENTIAL DIAGNOSIS

Speckled leukoplakia

– Red patch with white foci
– Commonly affect the nonkeratinized regions.

High-risk of malignant change, especially in smokers and when affecting the floor of the mouth.

Biopsy (preferably excisional) is indicated.

Stomatitis nicotina

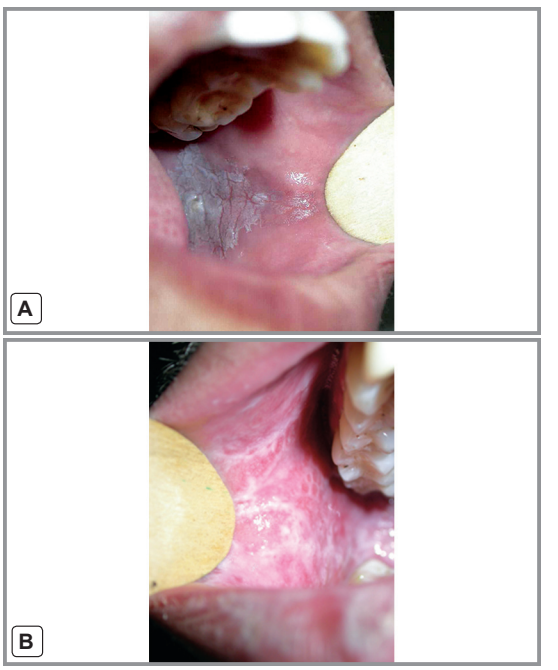
– White appearance of the hard palate with numerous red dots (secondary to blocked minor salivary glands)
– Induced by pipe, cigar or reverse smoking.

Note: It is not considered to be potentially malignant, but since these patients smoke, there is an increased risk of cancer any way.

Lichen planus

– Classical site—nonkeratinizing mucosa bilaterally. However, can occur on the lateral border of the tongue.
– May be associated with certain drugs (antihypertensive, hypoglycemic, etc.)
– Skin lesions may be present, affecting the flexor surface of the arms.

Note: Erosive and atrophic variants (out of many) are sometimes associated with cancer.



Figs 51-1A and B Lichen planus

www.Ebook777.com

PURPLE, VIOLET LESION

Kaposi's sarcoma

– Usually affects the hard palate
– Sessile or only slightly raised or pedunculated swelling.

AMALGAM TATTOO

– Previous dental treatment with amalgam filling material
– Otherwise asymptomatic
– Do not blanch or change shape on pressure
– If large enough, may show up on a plain radiograph.
Usually persist for life.

MALIGNANT MELANOMA

– Usually affects the palate
– Color—variable—from brown to black, gray or even white
– Margins—usually ill-defined
– Satellite lesions ±.

SUBMUCOUS FIBROSIS

A progressive disease in which fibrous bands form beneath the oral mucosa. These bands progressively contract so that ultimately mouth opening is severely limited. Tongue movements may also be limited.
Lips and cheeks also become stiff; loose their elasticity.
Mottled or marbled pallor of the mucosa of cheeks, palate, tongue and gums.
Etiology: Betel nut chew, chilli, tobacco, vitamin deficiencies; or etiology may be obscure.
It is a premalignant condition.

BENIGN ORAL ULCERS

Causes

– Injury
– Infection: TB, syphilis
– Recurrent aphthous stomatitis—minor, major and herpetiform aphthous ulcers
– Systemic diseases may present initially with oral ulceration
Examples:
  - Drug-induced
  - GI disorders, e.g. celiac disease, Crohn's disease
  - Blood disorders
  - Vesicobullous lesions: Skin disorders like epidermolysis bullosa, pemphigus, erythema multiforme, lichen planus.

HISTORY

- Single or multiple?
- Site?
- Duration?
- Mode of onset?
- Progress?
- Pain?
- Injury from dentition, e.g. sharp tooth / poorly fitting denture, etc.
- Constitutional / systemic symptoms?
- Have you been taking any medicines?

LOCAL EXTRAORAL EXAMINATION (Examine the external aspect of the face, and then proceed with intraoral examination.)

Free ebooks ==> www.Ebook777.com

LOCAL INTRAORAL EXAMINATION

- Number?
- Site and extent?
- Margin, shape, size?
- Discharge, floor, edge, and surrounding area?

DIFFERENTIAL DIAGNOSIS

Traumatic ulcer (due to sharp tooth / ill-fitting denture, etc.)
– Painful
– Site: Opposite the traumatic agent like sharp tooth/ill-fitting denture
– Irregular. Instead of ulcer, there may be a crack or a fissure
– Size—0.5 to 1 cm
– Serosanguineous discharge
– Floor, in acute stage, is covered with slough or fibrinous pseudomembrane
– Edge:
  - In acute stage—edematous
  - In chronic stage—'heaped up', may resemble that of a malignant ulcer
– Surrounding mucosa—erythematous and edematous in early stages
– Base—slightly indurated in both acute and chronic stages
– Depth: Usually shallow. Chronic traumatic ulcer may be deep
– (Should) Heal within 2–3 weeks, once the cause is removed.

Viral ulcers
– Multiple ulcers affecting a particular region—e.g. soft palate in herpangina
– Widespread oral ulceration with systemic involvement—primary infection
– Small recurrent lesions on the lip ('cold sores') and occasionally intraorally—secondary infection.

Aphthous ulcers

Types—minor, major and herpetiform.
Differentiating points common to all types of aphthous ulcers:
– Usually occur in young individuals
– Painful and tender
– Floor—yellowish / white slough
– Edge—edematous
– Surrounded by a red border (halo)
– Depth—superficial, i.e. the ulcer is 'shallow'
– Regional lymphadenitis +.



Fig 51-2 Aphthous ulcer

Minor aphthous ulcers
– Site—nonkeratinized mucosa
– < 1 cm in diameter
– Disappear without scarring within 2 weeks.

Major aphthous ulcers
– (Unlike minor aphthae) may occur on keratinized (in addition to nonkeratinized) mucosa
– > 1 cm in diameter
– May last 'longer'
– May leave behind scar after healing.

Note: Major aphthae could be predisposed to by immunocompromized status.

Herpetiform aphthous ulcers
– Multiple pinprick sized; may coalesce to form larger ulcers
– No systemic upset (the fact that differentiates them from herpetic infection).

www.Ebook777.com

Tuberculous ulcers
- Extremely painful
- Multiple or solitary
- Sites—lips, tongue, floor of the mouth
- ~ 5 mm in diameter
- Pale granulating floor
- Undermined edge
- Regional lymphadenitis +
- Frequently associated laryngeal or pulmonary TB.

Primary syphilitic ulcer (Primary chancre)
- Age—sexually active years of life
- Sexual contact 1–3 weeks before, with a person suffering from infectious syphilis (chancre of primary syphilis and mucous patches, snail track ulcer of secondary syphilis)
- Starts as an indurated papule, the center of which breaks down → primary chancre
- Painless ulcer on lip or tongue (sites of inoculation). Usually solitary
- Round or oval
- 1–1.5 cm in diameter
- Discharge—serous (teeming with treponema)
- Floor—dull coppery red with pink granules; covered usually by a crust of thin necrotic material
- Edge—raised, erythematous, sloping towards center
- Base—indurated—gives a feeling of a 'buried button'
- Depth—superficial (i.e. ulcer is 'shallow')
- Regional lymphadenitis +.

Natural history: Ulcer regresses and heals even if not treated, usually within 3 weeks.

Secondary syphilitic ulcers
- Begin as grayish white pearly plaques 2–5 mm in diameter. Small shallow ulcers result on separation of the gray patch (dead macerated squamous epithelium)
- Adjoining ulcers coalesce to form 'snail track ulcers'.
- Associated manifestations, secondary syphilis being a systemic disease: Hepatitis, uveitis, low grade meningitis, skin rashes, generalized lymphadenopathy, etc.



Fig 51-3 Secondary syphilitic ulcers

Tertiary syphilitic ulcer (solitary gumma)

Basically,—a h/s/o primary and secondary syphilis 5–6 years earlier.
- Slowly enlarging painless swelling in the midline of the tongue or palate which has broken down → an ulcer
- Shape—round
- 1–2 cm in diameter
- Floor—covered with grayish-white wet slough looking like 'wash-leather'
- Base—indurated
- Deep crater-like ulcer.

Notes
- Manifestations of tertiary syphilis:
  - Gumma—after 5 years of infection
  - Cardiovascular syphilis and general paralysis of insane—after 10–15 years
  - 'Tabes dorsalis'—after 20 years.
- Tertiary syphilis manifests in the oral cavity in 3 ways:
  - Solitary gumma
  - Multiple small gummas
  - Chronic superficial glossitis.

Free ebooks ==> www.Ebook777.com

Torus
- A developmental anomaly
- In the midline of hard palate or lingual aspect of mandible
- Size and lobulation vary. Although alarming at first glance, it is harmless
- Does not increase in size
- May interfere with a denture. Otherwise, harmless
- **Hard** consistency.

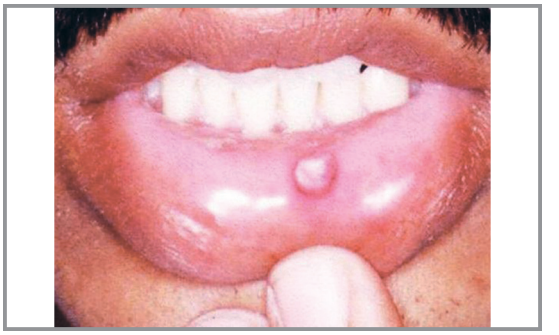


Fig 51-4 Mucous cyst

Mucocele (mucous cyst of minor salivary gland)
- Common sites: Lower lip > angle of mouth > 'bite-line' (mucous membrane of cheek opposite the level of occlusion of teeth)
- Pinkish / bluish / yellowish. Whitish if it has been repeatedly traumatized by teeth bite
- Well-defined. Globular
- Up to 1.5 cm in diameter
- Soft or tense
- Translucent
- Causes—1. Majority are 'extravasation' cysts resulting from (sudden unintentional) biting of mucosa → rupture of duct → escape of mucus into the tissues. 2. 'Retention' due to blockage of duct by minute calculi.

A swelling of this kind in the upper lip is likely to be a neoplasm.

Papilloma
- Usually situated at nonkeratinized sites
- Solitary or multiple
- Look whitish in the moist environment
- A mass of tiny close-set finger-like processes OR cauliflower-like having knobbly surface.

Fibroma
- Tend to arise at the sites of chronic irritation, e.g. buccal mucosa
- Normally sessile. Rounded. Can be pedunculated
- Smooth surface
- Firm.

Lipoma
Yellowish submucosal swelling in the cheek / floor of the mouth.

Teratoid cyst
- Teratoma-like tumors that lack a trigerminal or complex histological appearance
- Present at birth.

Kaposi's sarcoma
Reddish / purple sessile or only slightly raised or pedunculated swelling.

Orofacial granulomatosis
- Facial or lip swelling
- Angular cheilitis
- Oral ulceration
- Mucosal tags
- 'Cobblestoned' oral mucosa.

Possible etiology—Allergy, foreign bodies, Crohn's disease, sarcoidosis.

Gingival epuli—Refer to chapter 20.

www.Ebook777.com

CHRONIC SWELLING OF THE LIP

- AV malformation including lymphangioma
- Mucous cyst, papilloma, fibroma, salivary gland swelling/tumor
- Lupus vulgaris, SCC.

CHRONIC SWELLING OF THE JAW – Refer to chapter 20

CHRONIC SWELLING OF THE PALATE

- Mucous cyst, papilloma, fibroma, salivary gland swelling/tumor
- Alveolar abscess (lateral), gumma (midline), extension of CA maxilla, malignant melanoma, osteosarcoma.

CHRONIC SWELLING IN THE CHEEK

- AV malformation, AV fistula (acquired), lymphangioma
- Mucous cyst, papilloma, fibroma, neurofibroma, fibroepithelial polyp, lipoma
- Salivary gland swelling / tumor
- Facial lymphadenopathy
- Carcinoma
- Denture-induced granuloma.

DIFFERENTIAL DIAGNOSIS of selected chronic swellings in the cheek:

Acquired AV fistula

- Situated on the inner surface of the cheek opposite the occlusal plane
- Up to 0.5 cm in diameter
- Arises as a result of a bite which damages a submucosal vessel
- More massive AVFs surround the buccal pad of fat in the buccal space.

Lymphangioma

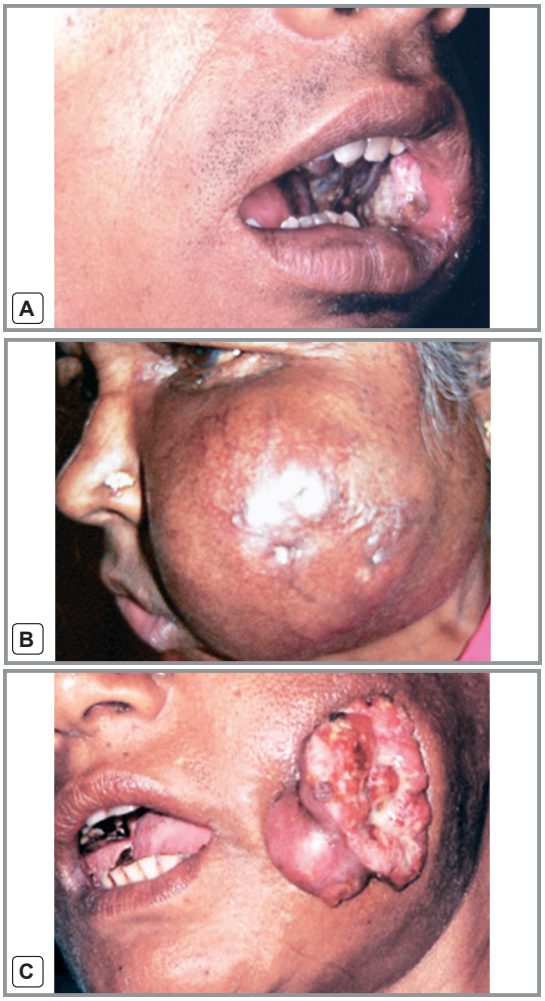
- Tends to occur posteriorly in the cheek
- Looks like brownish frogspawn.

Facial lymphadenopathy

A palpable swelling in the cheek or lower buccal sulcus. = enlarged facial lymph node—a lymph node which is an outlying member of the submandibular group of lymph nodes—found overlying the body of the mandible along the path of the facial artery as it proceeds towards the angle of the mouth.

Cancer of the cheek

- Squamous-celled carcinoma is the most common
- Malignant tumor of minor salivary gland may occur.



Figs 51-5A to C Examples of CA cheek

Free ebooks ==> www.Ebook777.com

- AV malformation, AV fistula (acquired), lymphangioma
- Ranula, dermoid cyst, mucous cyst, papilloma, fibroma, neurofibroma, fibroepithelial polyp, lipoma, teratoid cyst
- Sublingual / submandibular salivary gland sialomegaly often due to a stone in the duct
- Salivary gland swelling / tumor
- Carcinoma
- Denture-induced granuloma.

DIFFERENTIAL DIAGNOSIS of selected chronic swellings in the floor of the mouth:

Ranula

= extravasation cyst arising from a damaged sublingual salivary gland.

If mucus escapes from a posterior sublingual gland it may flow over the posterior margin of the mylohyoid down into the neck to form a 'plunging ranula'.

- Usual age: Children, young adults
- ~ 2–3 cm in diameter, however, may become large enough to interfere with speech and swallowing
- Site: One side of the floor of the mouth. May sometimes cross the midline
- Bluish gray in color
- Prominent blood vessels run over its surface
- Ill-defined
- Hemispherical / ovoid / irregular
- Soft. Fluctuant
- Brilliantly translucent.

Additional signs in case of a plunging ranula:

- Cross fluctuation present between the oral and cervical components of the swelling
- Pressure upwards on the cervical swelling makes the oral component more prominent.

Median sublingual dermoid

Grows in the midline in the floor of the mouth between the two genioglossus muscles. It enlarges towards the hyoid but remains above mylohyoid muscles; entirely in the oral cavity.

Although it lies above the mylohyoid muscle, it bulges downwards towards the submental region as the mouth is closed and the swelling is compressed by the tongue giving an appearance of 'double chin'.

Lateral sublingual dermoid

Lies in the floor of the mouth in the space between the hyoglossus and mylohyoid muscles. As it enlarges, it displaces the submandibular salivary gland backwards and emerges from behind the posterior border of mylohyoid muscle in the submandibular triangle.



Fig 51-6 Ranula

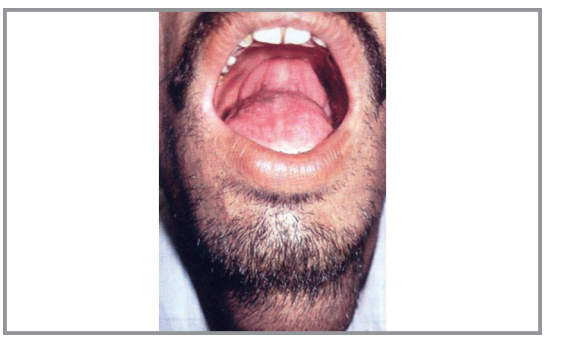


Fig 51-7 Median sublingual dermoid

www.Ebook777.com

Differentiating points common to both dermoids, except for the location :
– Usual age of presentation although congenital—10–25 years.
– Whitish, i.e. opaque
– Bidigitally palpable
– Well-defined. Hemispherical / ovoid
– 3–4 cm in diameter
– Soft or firm. Fluctuant
– Indentation (moulding) +ve
– Nontranslucent.

Neurofibromas → different morphological swellings.

1. Often produce a diffuse thickening of the gums spreading out into the cheek or floor of the mouth.
2. Ovoid / fusiform swelling along the line of a nerve, e.g. lingual nerve.
3. Pedunculated swelling.

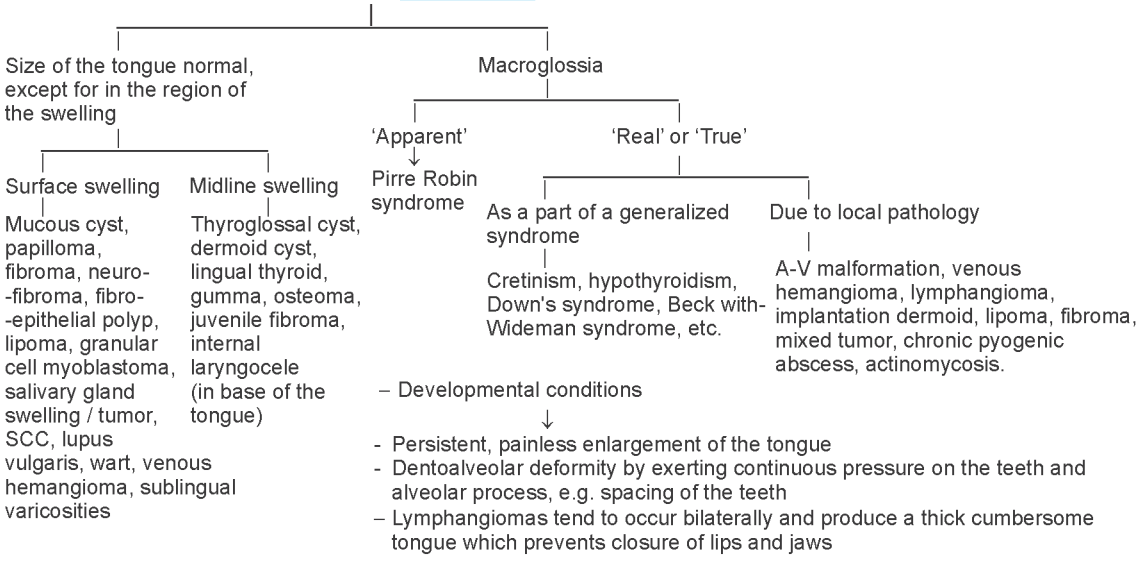
Fibroepithelial polyp

– Pedunculated, rounded, soft.
  If cheek is repeatedly traumatized at one place a thickened submucous scar is formed which may be pulled out onto a stalk by the suction of swallowing. A polyp of cheek may be sucked by a similar mechanism into a gap where a tooth is missing.

Denture-induced granuloma/s

– Fusiform fibrous masses in the buccal or lingual sulci of edentulous individuals
– Single OR several together parallel to one another
– Result from chronic irritation and ulceration from the flange of ill-fitting denture.

CHRONIC SWELLING OF THE **TONGUE**



Macroglossia = appearance of enlargement of tongue disproportionate to the size of the oral cavity.
'Apparent' macroglossia = false appearance of enlarged tongue (which is actually normal in size) within a small oral cavity.
'True' macroglossia is due to actual enlargement of the tongue, i.e. due to substance swelling in a normal-sized oral cavity.

Disorder	*Mechanism of enlargement of tongue*
– Cretinism, Down's syndrome	Unknown
– A-V malformation	SOL + increased blood flow leading to overgrowth
– Implantation dermoid, lipoma, fibroma, mixed tumor	SOL
– Lymphangioma, chronic pyogenic abscess, actinomycosis	Edema + inflammation

Free ebooks ==> www.Ebook777.com

– Squamous-celled CA is the most common
– Salivary gland cancer may occur
– Other types are rare

Clinical warning signs

1. **Color**:
   – Speckled leukoplakia
   – Erythroplakia
   – Leukoplakia
   – Intraoral pigmentation could be (rarely) due to malignant melanoma.
2. **Surface texture**: Early cancers are often smooth or granular.
3. A single **ulcer**, in the absence of any obvious injury that has been present for at least 2 weeks without any sign of regression.

Forms of presentation of oral cancer: Any lesion with or without changed color / an ulcer.

HISTORY

- Duration?
- Mode of onset?
- Progress?
- Numbness?
- Pain—where?
  – At the site of the lesion?
  – In the ear?
- Profuse salivation?
- Does the lesion bleed spontaneously?
- Tobacco consumption in any form, i.e. chewing / smoking / reverse smoking / pipe smoking / betel quid, etc?
  How many cigarettes / packs a day? For how many years?
- Alcohol?
  How many units a day?
  Smoking > 20 cigarettes and drinking > 6 units of alcohol per day increase the risk many folds.
  Someone who has stopped smoking for over 10 years has the same risk as a nonsmoker.
- Previous h/o any cancer in the region?
  Previous cancer in the region increases the risk for a second cancer in the upper aerodigestive tract.
- Any swelling in the neck?

LOCAL EXTRAORAL EXAMINATION (Examine the external aspect of the face, and then proceed with intraoral examination.)

LOCAL INTRAORAL EXAMINATION

1. Of a lesion with changed color—Refer to page 723 to 727.
2. Of a lesion without changed color—Number, site and extent, margin, shape, size, surface texture (smooth or granular)?
3. Of an ulcer—in the usual manner.

Careful palpation is extremely important. Oral cancers are usually locally invasive. Their external appearance is often elusive like 'the tip of an iceberg'. **The extent of induration should be included in estimating the size.**

EXAMINE THE NECK for metastatic lymphadenopathy.

***Notes* on oral cancer**

Morphological types *of squamous-celled CA*

1. Ulcerative – The commonest type.
2. Exophytic (*syns*. proliferative, cauliflower-like, fungating = ulcerated tumor / growth): Starts as a slowly growing plaque / nodule. Ulcerates sooner than later → ulcerated growth looking like a cauliflower.

www.Ebook777.com



Fig 51-8 SCC: Ulcerative

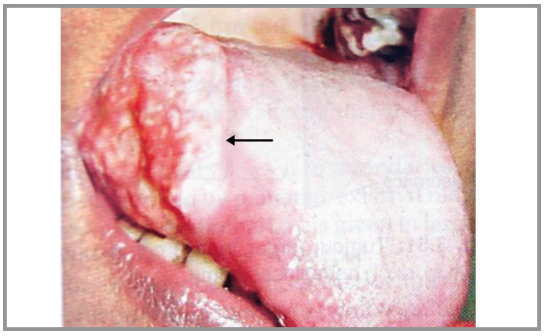


Fig 51-9 SCC: Exophytic



Fig 51-10 SCC: Verrucous (Squamous papilloma)

3. Verrucous (wart-like): Typically seen in elderly patients with poor oral hygiene, ill-fitting denture or other cause of chronic irritation. Indolent biological behavior. Late occurrence of local invasion of deeper tissues and lymph node metastasis.
4. Infiltrative: Seen in the tongue.
5. Fissure / crack: most commonly seen on the sides of the tongue.

*Manifestations of **clinically obvious disease** (often 'advanced')*

- Numbness—due to infiltration of sensory nerve
- Pain in the lesion itself—due to ulceration, infection
- Pain referred to the ear ⇒ irritation of lingual nerve (Referral through auriculotemporal nerve, both the nerves being branches of the mandibular division of trigeminal nerve)
- Profuse salivation—caused by pain. Pain-on-swallowing also prevents swallowing of saliva
- (Spontaneous) Bleeding—due to erosion of blood vessel/s
- Offensive smell—due to gross infection of the necrotic lesion
- Classical features of the lesion depending on morphological type
- Fixation to the underlying structures
- (Easy) Bleeding (on touch)
- Regional metastatic lymphadenopathy.

Terminal events

1. Inhalation bronchopneumonia—due to inhalation of infected material from necrotic tumor.
2. Cancer cachexia.
3. Starvation due to pain and dysphagia.
4. Bleeding:
   - From erosion of the lingual artery
   - In c/o tumors of the posterior third of tongue, from the internal carotid artery
   - Metastatic lymphadenopathy eroding internal / external carotid artery.
5. Asphyxia: Causes—metastatic regional lymph nodes, glottic edema.

Free ebooks ==> www.Ebook777.com

ORAL MANIFESTATIONS OF SYSTEMIC DISEASE

– Circumoral pigmentation ⇒ Anemia / Benign **intestinal polyps**
– Glossitis, oral ulceration ⇒ **Anemia**
Profound deficiency is required to produce obvious pallor of mucous membranes, the smooth tongue or the 'red beefy' tongue
– Gingival enlargement, bleeding gums, oral ulceration, petechiae, infection (viral or candidal) ⇒ **Leukemia**
– Swelling of lips, angular cheilitis, oral ulceration, mucosal tags and cobblestone appearance of buccal mucosa = 'Orofacial granulomatosis' ⇒ **IBD, coeliac disease**.



Fig 51-11 Circumoral pigmentation

Immunocompromized status (from any cause) may lead to the following oral manifestations:
– Severe gingivitis
– Periodontal disease. Dental radiographs may show rapid bone loss around the teeth, especially the posterior teeth, **sometimes in the absence of obvious poor oral hygiene**
– Oral ulceration appearing like major aphthae
– Candidiasis
– 'Hairy' leukoplakia
– Kaposi's sarcoma.

DISORDERS OF THE LIP

1. Biting injury.
2. Congenital cleft
   - Difficulty in feeding? Difficulty in sucking breastmilk?
   - Nasal regurgitation?
   - Difficulty in speech? Is there a difficulty in pronouncing B, P, M (consonants which require closure of lips)?
   - Nasal intonation?
3. Swelling.
4. Ulcer.

LOCAL EXTRAORAL EXAMINATION

Cleft lip: Look for the type of cleft lip, alae nasi and nasal septum.

- Exact site: Central OR lateral?
  – Unilateral or bilateral?
  – Does the cleft extend into the nasal floor?
  – Simple or compound?
- Alae nasi: Normal OR flattened?
  Alae nasi are flattened in complete cleft lip. Flattening occurs on the side of the cleft.
- Nasal septum: Normally placed in the midline OR deviated? – If deviated, to which side?
  In unilateral complete cleft, the septum may be deviated to the opposite side.

www.Ebook777.com

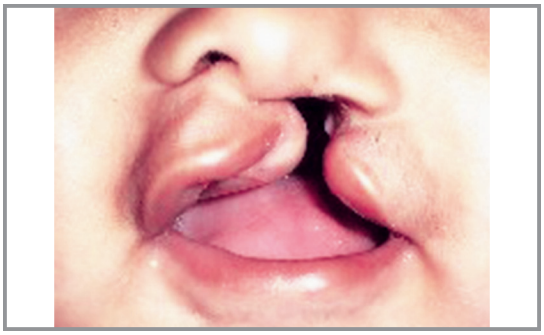
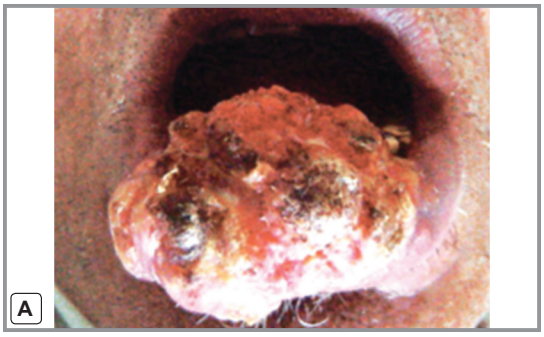


Fig 51-12 Congenital cleft lip





Figs 51-13A and B Examples of CA lip

LOCAL INTRAORAL EXAMINATION

Cleft lip: Examine the teeth, palate.

- Teeth:
  - Alignment of the teeth?
  - Are any teeth protruding?
- Palate: Any cleft?

Types of cleft lip:

- *Incomplete* = cleft in the lip does not extend into the nasal floor
- *Complete* = cleft in the lip extends into the nasal floor
- *Central* – Rare
- *Unilateral* = cleft on one side of the philtrum
- *Bilateral* = cleft on both sides of the philtrum, and the philtrum hangs freely in the midline
- *Simple* = cleft of only the lip alone
- *Compound* = cleft lip associated with cleft of the alveolus.

DISORDERS OF THE PALATE

1. Congenital cleft
   - Difficulty in feeding, particularly in suction?
   - Difficulty in speech?
     - Particularly a difficulty in pronouncing palatal consonants Ka, Kha, Gha?
     - Nasal intonation?
   - Difficulty in hearing?
   - H/s/o chronic ear infection?
   - Problems with dentition, e.g. delayed eruption of teeth.
   - Repeated respiratory tract infection?

Free ebooks ==> www.Ebook777.com

2. Perforation. Causes:
   - Syphilis.
   - Persistence of a hole after an operation for closure of a congenital cleft.
3. Ulcer.
4. Swelling.
   - H/o surgical correction of cleft palate?

LOCAL EXTRAORAL EXAMINATION (Examine the external aspect of the face, and then proceed with intra-oral examination.)

LOCAL INTRAORAL EXAMINATION

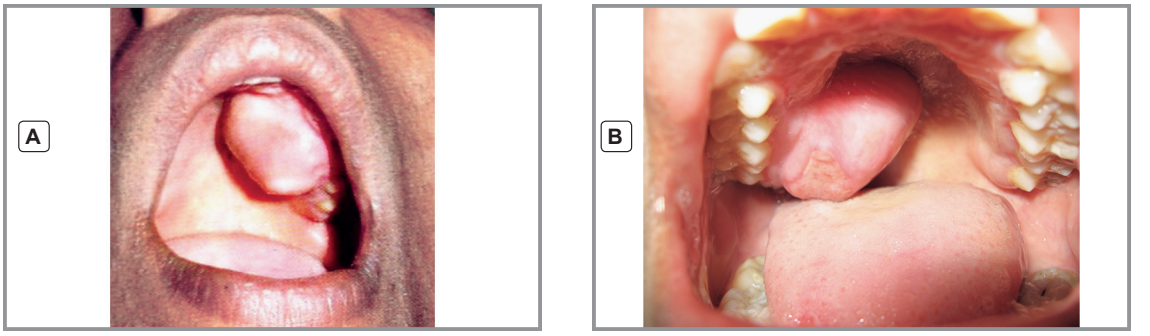
Examine congenital cleft / ulcer/perforation / swelling, any scar s/o surgical correction of cleft palate?

Details to be examined in a c/o **cleft palate**

- Exact site? – Premaxilla / hard palate / soft palate.
  |
  Incomplete OR complete?
- Hypodontia / hyperdontia?



Fig 51-14 Cleft palate



Figs 51-15A and B Examples of palatal swelling (Minor salivary gland tumor)

www.Ebook777.com

1. Developmental anomaly.
2. Glossodynia.
3. Unpleasant change in taste.
4. Color change.
5. Specific inflammatory conditions.
6. Ulcer.
7. Swelling.

HISTORY

The details need to be asked according to the disorder (after referring to 'differential diagnoses').

LOCAL EXTRAORAL EXAMINATION (Examine the external aspect of the face, and then proceed with intraoral examination.)

LOCAL INTRAORAL EXAMINATION

- Site and extent of the lesion? Note if an ulcer has extended to the floor of the mouth / jaw / tonsil
- Size? A massive size of the tongue (macroglossia) could be due to lymphangioma
- Color? Blue color ⇒ venous hemangioma
- Mobility:
  Ask the patient to put the tongue out and move it sideways. (Inability to protrude the tongue = Ankyloglossia)
  - In a child, inability to protrude the tongue, associated with a short frenum, and impaired speech ⇒ Tongue-tie
  - In an adult, inability to protrude, or deviation of tongue to the affected side may be noticed in advanced carcinoma
- Palpation: Ask the patient to keep the tongue within the mouth at rest in relaxed state. Palpate with a gloved (index) finger.
  In case of growth of the posterior third of the tongue, palpate the anterior part of the growth.
  Induration is present in squamous-celled carcinoma, and may be present in gummatous ulcer.
- Any additional clinical test may have to be performed according to the disorder [after referring to 'differential diagnoses' (below)].
- **IDL**: For assessment of the posterior third of the tongue
- **DL scopy ↓ GA may be essential** in suspected cancer of the posterior third of the tongue. The posterior third can be palpated, but induration due to tumor is not easily appreciated because of the lumpiness of the lingual tonsils.

DIFFERENTIAL DIAGNOSIS

A symmetrical developmental hyperkeratotic patch on the undersurface of the tongue
This is a premalignant condition.

Tongue-tie
Refer to chapter 88.

Congenital fissured tongue
– Fissures of varying depth running laterally from the median groove
– Surface between the grooves is covered with normal papillae.
Complication: Infection with *Candida albicans*.
DD: Syphilitic tongue—tends to be bald and with longitudinal fissures.

Free ebooks ==> www.Ebook777.com

Lingual thyroid
- A rounded swelling situated at the back of the tongue
- Reddish
- Lobulated.

Sequelae/complications: Dysphagia / difficulty in breathing / impairment of speech, bleeding.

Glossodynia
- Middle-aged and elderly women
- Often no cause can be found
- Sometimes predisposing factor may be present like DM, Sjögren's syndrome.

Unpleasant change in taste: Cause is unknown.

Color changes

General	Specifically occurring in the tongue
White lesion, red lesion, red-white lesion, etc.	– **Median rhomboid glossitis**: Refer to page. 725 – **Geographic tongue**: Refer to page. 725 – **Chronic superficial glossitis** (due to tertiary syphilis).

TB

TB affects the tongue in 2 ways – 1. Painful indolent ulcers (Refer to 'benign oral ulcers'), 2. Circumscribed interstitial tuberculoma.

Secondary syphilis
- Multiple shallow ulcers on the sides and undersurface of the tongue
- Mucous patches on the tongue and on the fauces
- Condyloma—always in the midline.

Chronic superficial glossitis of tertiary syphilis

Loss of papillae → 'beefy tongue' = plain raw red colored tongue.

Ulcers

Specifically occurring in the tongue	General
Post-pertussis – Typically found in patients suffering from whooping cough – Occur on the upper part of frenum and undersurface of the tip in patients who protrude the tongue over the lower incisors during bouts of coughing	Traumatic (dental, etc.), aphthous, tuberculous, syphilitic, malignant

SCC

Clinical types
- Infiltrative → 'Frozen tongue'—tongue is transformed into an indurated mass
- Fissured or cracked (indurated) type following chronic glossitis
- Ulcerative
- Fungating
- Nodular.

Presenting clinical features (may be of relatively early disease)
- Various forms of the (early presenting) tumor :
  - A papillated plaque with white keratin flecks on the surface
  - A typical ulcer: Necrotic floor, everted and indurated edge, and indurated base
  - A deep, often infected fissure with surrounding induration—little pain in the tongue itself

www.Ebook777.com

- A mass: Seemingly normal overlying mucosa. Lobulations ±. Pointing abscesses like appearance suggests yellow patches of submucosal necrosis. Indurated

– Pain at the back of the tongue or pain on swallowing may be suggestive of carcinoma of the posterior third
– Alteration in the voice may be the first indication of cancer of the posterior third.

Clinical manifestations of advanced disease

– Pain in the tongue itself—due to ulceration, infection
– Pain at the back of the tongue or pain on swallowing may be suggestive of carcinoma of the posterior third
– Pain referred to the ear ⇒ irritation of lingual nerve
– Profuse salivation (due to irritation of the nerves of taste). In the late stage, patient allows the saliva to dribble out of the mouth due to difficulty in swallowing
– Ankyloglossia, or deviation to the affected side when the patient attempts to protrude the tongue ⇒ infiltration of the muscles of the tongue
– Inability to articulate clearly
– Dysphagia—more pronounced with cancer of the posterior third
– Offensive smell—due to gross infection of the necrotic lesion.

Terminal events: Refer to page 735.

Osteoma of the tongue

– Beneath the foramen cecum or in the posterior third of the tongue
– Hard consistency.

It is a clinical curiosity.

Sublingual varicosities

– Purplish or blue black dilated and tortuous lingual veins on the undersurface of the tongue
– Not uncommon in elderly
– Should not be confused with AVM.



Fig 51-16 Lymphangioma of the tongue

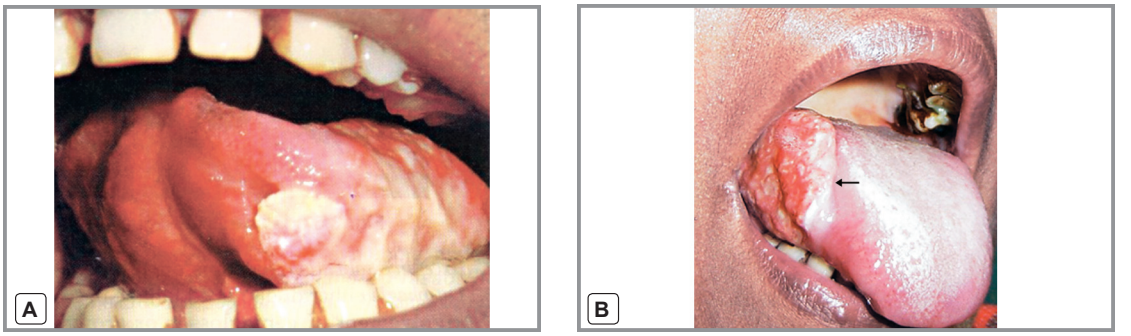


Fig 51-17 Lingual thyroid



Fig 51-18 Herpes simplex stomatitis

Free ebooks ==> www.Ebook777.com



Figs 51-19A and B Examples of CA tongue



Fig 51-20 Malignant melanoma of tongue

Gumma
- Starts as a slowly enlarging swelling
- Following necrosis of the overlying tissues the wash-leather slough separates to leave a deep, crater-like ulcer.

Differentiation from carcinoma:
- Carcinoma usually does not arise in the midline
- No tethering of tongue with a large gumma.

Granular cell myoblastoma
- Firm mass
- The mucous membrane overlying the mass—smooth or hyperkeratotic.

Juvenile fibroma of the tongue
- A slowly enlarging central swelling
- Soft consistency.

www.Ebook777.com

52 TEMPOROMANDIBULAR JOINT

Complaints:
- Pain in the joint
- Clicking sound
- Inability to open the mouth
- Inability to close the opened mouth.

CLICKING SOUND

Causes: Loose meniscus, osteoarthritis, septic arthritis.

Examination

1. Place the fingers on the joint just inferolateral to the tragus.
   Ask the patient to open and close the mouth repeatedly.
   - Feeling of **crepitus** ⇒ Osteoarthritis
   - Feeling of **clicking** ⇒ Loose meniscus.
2. Place the chestpiece of a stethoscope on the joint and ask the patient to open and close the mouth repeatedly. Try to listen for clicking sound.

INABILITY TO OPEN THE MOUTH

Causes

Ankylosis of the jaw
- Due to disorders of the joint—'true ankylosis'
- Due to fibrosis resulting from cancrum oris—'false ankylosis'

Spasm of muscles

Spasmodic clinching = 'Trismus'
- Any inflammatory condition in the neighborhood of the joint
- Tetanus → characteristic facies—painful sardonic smile

INABILITY TO CLOSE THE OPENED MOUTH

Patient presents (in the emergency room) with her/his mouth open, unable to close it.

Cause: Dislocation of the joint – Could be unilateral or bilateral.
Clinical signs are less pronounced in unilateral dislocation.

Examination

1. Palpate just behind the condyle. Feeling of a small hollow ⇒ dislocation.
2. Insert the little fingers into the external ears simultaneously with pulps of the fingers facing forwards (anteriorly).

Free ebooks ==> www.Ebook777.com

Ask the patient to try to open and close the mouth.
- Try to feel for movement of the condyles
- Inability to feel movement of the condyle ⇒ dislocation (on the concerned side).

Reduction
- Most patients do not need an anesthetic
- Make the patient sit forward in a chair
- Ask an assistant to stand behind and hold the patient's head
- Place gauze pieces over the lower posterior teeth on each side
- Press the premolar teeth downward, and simultaneously press the underneath of the chin upwards and backwards

  If the patient opens the mouth too wide again, dislocation may recur. So bandage the jaw to keep the mouth shut for 3 days. Allow to open the mouth (a little) only for eating.

www.Ebook777.com

53 BACKACHE

Definitions

- *Ankylosing spondylitis*: A condition affecting the central articulations of the body characterized by progressive inflammation of the spine, S-I joints, and the larger joints of the extremities particularly shoulder, hip and knee, leading to fibrous or bony ankylosis and deformity
- *(Cervical) Spondylosis*: A degenerative condition characterized by degeneration of intervertebral discs, and the formation of osteophytes which project backwards into the intervertebral foramen
- *Spondylolisthesis*: A condition in which a lower lumbar vertebra, usually the 5th, slips forward through the plane of the intervertebral disc below it, carrying with it the whole of the upper portion of the spine
- *Strain*: An overstretching of a part of the musculature
- *Sprain*: A joint injury in which some of the fibers of a supporting ligament are ruptured but the continuity of the ligament remains intact.

Normal movements of the spine	Region of the spine in which they occur
Flexion	Lumbar. Normal range—90°
Extension	Lumbar and thoracolumbar. Normal range of extension in lumbar region: 25–30°
Lateral flexion	Lumbar and thoracolumbar
Rotation	Cervical and thoracic
Nodding	Atlanto-occipital joint
Rotation of the head	Atlanto-axial joint.

Free ebooks ==> www.Ebook777.com

Causes of backache in general

- Due to disorder in the spine itself
  - Disorders of fascia / ligaments / muscles
    - Fibrositis of vertebral muscles: Occurs commonly in the back of the neck, shoulder, interscapular and lumbar region
    - Fibrositic nodules in subcutaneous tissue
    - Rheumatic fibrositis
    - Strain/sprain due to facet asymmetry, increased lumbosacral angle, defective posture, compensatory to leg causes,defective line of weight bearing (Refer to 3. (a), (b) and (c) under 'disorders of the bony spine'), or spondylosis
    - Severe muscle spasm
    - Myositis: Trichinellosis, collagen disease
  - Disorders of the spinal cord and meninges
    - Tumors
      - Extradural
      - Intradural
    - Radiculitis
    - Myelitis
    - Spinal meningitis
  - Disorders of the bony spine (including affection of vertebral joints)

1. *Congenital*
   - Spina bifida
   - Sacralization of L-5 (4 lumbar vertebrae)
   - Sacralization of the transverse process of L-5
   - Lumbarization of S-1 (6 lumbar vertebrae)
   - Sacrococcygeal teratoma
   - Spondylolisthesis
   - Spondylosis
   - Hemivertebra
   - Facet asymmetry
   - Increased lumbosacral angle
2. *Traumatic*: #, #-dislocation
3. *Faulty body mechanism (a sort of chronic trauma)*
   (a) Defective posture
   - Cervical region: Torticollis, defective eye sight
   - Thoracic region: Deformity of the rib-cage as in extensive fibrosis of lung or pleura, and rickets
   - Lumbar region: Pregnancy, pot belly abdomen, obesity, huge tumor, general ill health, defective posture due to occupation / habit, weakness of paravertebral muscles in poliomyelitis and myopathies

   (b) Compensatory to leg causes: Leg length discrepancy, flexion deformity of hip / knee, short hamstring muscles, and flat foot.
   (c) Defective line of weight bearing: due to deformities of the spine.
   (d) Chronic trauma to the bone as in body of the vertebra due to aneurysm etc.
4. *Inflammation*
   (a) Acute:–Osteomyelitis
   –Spinal meningitis
   –Spinal epidural abscess
   (b) Chronic:–Nonspecific osteomyelitis
   –TB, typhoid, syphilitic osteomyelitis
   (c) Vertebral arthritis:–Osteoarthritis—spondylitis deformans
   –Ankylosing spondylitis
   –Rheumatoid arthritis
   –Neuropathic spine
   –Brucellosis, syphilis, gonorrhea
   (d) Particular problems in S-I joint: Strain, sprain, ankylosing spondylitis, TB
   (e) Particular problem in sacrococcygeal joint: Coccydynia
5. *Tumor*
   - Metastatic deposits from leukemia, Hodgkin's disease, or other cancers
   - Myelomatosis
   - Rarely, sarcoma
6. *Deficiency diseases: Osteomalacia, rickets*
7. *Degenerative and metabolic conditions*
   - Spondylosis
   - PID
   - Osteitis deformans
   - Osteoporosis

   Causes of osteoporosis
   - Old age (senile osteoporosis)
   - Prolonged immobilization
   - Nutritional deficiency: Protein, vitamin C
   - Endocrine disturbances: Post-menopausal. Rare causes—hyperthyroidism, hyperparathyroidism, adrenal cortical hyperfunction, pituitary dysfunction like acromegaly gigantism, Cushing's syndrome
   - Tumors
   - Drugs: Excessive and prolonged cortisone therapy, hypervitaminosis A
   - Osteomalacia, rickets, renal rickets
     Osteomalacia affects the bones in the limbs producing deformities rather than #s while osteoporosis affects the spine and pelvis with frequent #
   - Idiopathic—may be due to defect in absorption of proteins
     Intravenous albumin helps in these cases
8. *Deformities of the spine:* Kyphosis, lordosis, scoliosis, spondylolisthesis
9. *Vertebral epiphysitis:* Osteochondritis

www.Ebook777.com

HISTORY

- Site (pinpoint with a finger)?
  - Does the pain radiate to the limb and distributed along particular dermatome?
  - Is the pain in the limb, segmental?
- Duration?
- M/o onset?
  Did it begin suddenly following a strain such as lifting a heavy weight?
- Progress/regress/fluctuation?
- Is the pain particularly less in the morning?
  Is the pain particularly more in the evening?
- Intensity?
- Nature: Dull and continuous / shooting (sharp) and intermittent / throbbing / twisting / colicky / constricting / burning / vague?
- Precipitating / Aggravating / Reliving factors?
  - Does the pain increase by bending forward?
  - Does the pain come after a long period of work in awkward position?
  - Does the intensity increase during working?
  - Is the pain aggravated by coughing/sneezing/any other act of straining?
  - Is the pain relieved by taking rest?
- *Specific points to be asked in cervical / thoracic backache*
  - Occupation involving strain on the back?
  - Fever / General ill health?
  - Does the pain (in the lower limb) increase by coughing/sneezing or by movement?
  - Paresthesias (in the lower limb)? (Almost as common as pain)
  - Motor weakness (in the lower limb)?

  } For radicular pain.
  - H/o trauma?
  - Refractive error?
  - General symptoms s/o metastatic malignant disease?
  - H/s/o nutritional deficiencies?
  - Chest symptoms? (Referred pain).
- *Specific points to be asked in low-backache*
  - Does your occupation involve lot of bending?
  - Do you have flat-foot?
  - Pain in the region of the S-I joint? Any pain while turning in the bed or jumping from some height (e.g. a stool)? (for affection of S-I joint)
  - Pain in the lower limbs? (- for PID)
  - H/s/o intra-abdominal (pelvic) pathology? (for referred pain)—e.g. bowel / urinary / gynecological complaints.
- Fever?
- Past h/o
  - Trauma?
  - Sudden catch during jerky movements?
  - TB / brucellosis / syphilis / typhoid / gonorrhea?

Specific points to look for in **GENERAL EXAMINATION**

- (A cold) Abscess in the neck, particularly in suboccipital region and posterior triangle of the neck?
- A retropharyngeal (cold) abscess?

} Applicable in c/o cervical pain.

Free ebooks ==> www.Ebook777.com

PHYSICAL EXAMINATION of the spine

Various positions in which examination is carried out: Standing, sitting, supine, and prone.

Inspection

- **Gait** (as the patient is entering the room)?
- Any obvious **attitude**?
- **Observe the back** carefully **from top to bottom**.
  - Position of the head?
  - The level of the shoulders: Is one side higher than the other?
  - The position of the scapulae: Elevated / Displaced?
  - Lateral margins of the body: Is one side flatter or more curved than the other?
  - Relative prominence of the iliac crests?
  - Any abnormal **curvatures**?—Level of the deformity?

    ↓

    Kyphosis:
  - Prominence of a single spinous process ⇒ collapse of one vertebra—called *Gibbus* (*syn. knuckle type*).
  - *Angular* deformity ⇒ collapse of 2 or 3 vertebral bodies.
  - *Round* deformity ⇒ involvement of several vertebrae. Causes—e.g. in the thoracic region of rapidly growing children, in old age (senile kyphosis).

  Lordosis is frequently compensatory, there being either kyphosis in the thoracic region or flexion deformity of the hip or bilateral congenital dislocation of the hip.

  In c/o *scoliosis*, ascertain to which side the convexity is directed. To determine this, mark the spinous processes with a skin pencil.
  - Any bulging / prominence? – On which side – On the side of the convexity or on the side of the concavity?
    - Bulging / prominence on the side of convexity ⇒ structural scoliosis
    - No prominence or a slight bulging on the side of concavity ⇒ postural scoliosis
  - Transverse furrow encircling the body between the costal margin and iliac crest?
  - Distinct prominence caused by the upper angle of the sacrum with a depression just above it?

    } *Spondylolisthesis*.
  - Any swelling/lesion?
  - Wasting/prominence of muscle?
- **Observe from front**
  - Anterior convexity of the sternum? – Seen in advanced cases of kyphosis. This occurs to compensate for diminished vertical measurement of thorax
  - Are the ribs crowded together?
  - In scoliosis, the chest diagonally opposite to posterior convexity is more prominent.

Palpation

- Local **temperature**?
- **Tenderness**
  - Cutaneous tenderness (Hyperesthesia): If present, it may be referred from the disease of the chest/abdomen, or the patient may be hysterical.
  - Bony tenderness: The methods mentioned below are all indirect methods for tenderness and consequently disease of the vertebral bodies.
    1. Apply gentle blows on each side of the spine: This method roughly locates the site of the disease.
    2. Press on the individual spinous processes with the thumb.
    3. Press on the transverse process of each side alternately of each vertebra in an attempt to rotate the vertebra.
    4. Press in the dimple situated just medial to the posterior superior iliac spine while the patient is asked to bend forward. Tenderness ⇒ affection of the S-I joint.
    5. Tenderness on pelvic compression and distraction ⇒ affection of the S-I joint.

www.Ebook777.com

- Feel for **wasting / rigidity of the erector spinae** muscle.
  ↓
  On finger-tip pressure, – Does the muscle feel firm or hard?
  – Can a depression be made in it?
- **Curvature**?
  Gently run the finger over the spine to note any projection and abnormalities of the curvature.
- **Edema**?
- Details of a swelling/ulcer/sinus, if any?

Movements (active and passive)

- Flexion:
  – In a child: Drop an object on the floor and ask to pick it up without bending the knees. Inability to do so (i.e. if the child has to bend its knees and hips) ⇒ rigid spine. Also, while raising the body, such a child puts her / his hand successively on the legs, knees and thighs as if she/he is climbing up her / his own legs
  – In an adult, ask the patient to lean forward while placing your hand on the spine to perceive the movement of the spinous processes. Note how much of the movement occurs at the spine and how much by hip flexion. Some patients, in spite of a stiff back, may touch their toes by excessive flexion of the hips
- During flexion, run a finger down the spine to note:
  – Tenderness (better elicited when the patient is bending forwards)
  – Normally increasing distance between the spinous processes
- Extension:
  – In a child: Make the child lie prone. Lift up the legs by holding the ankles while fixing the upper part of the thoracic spine with the other hand. Observe whether the vertebral column lifts up with bending or without bending.
    If the vertebral column lifts up as a whole without bending, it suggests rigidity due to a disease
  – In an adult: Ask the patient, in a standing position, to lean backward, i.e. to look up at the ceiling. Since extension occurs mostly in the lumbar region it is not expected to be affected in lesions of the upper thoracic region
- Lateral flexion:
  – In a child: Same as while testing extension except that the legs are carried first on one side and then to the other in order to bend the spine sideways. When rigidity is present, the spine will move as a whole without being bent
  – In an older child / in an adult: Hold the pelvis firmly from behind, and ask the patient, in a standing position, to slide the hand down the thigh, thus causing bending sideways
- Rotation and circumduction: Ask the patient to sit so as to fix the pelvis. Ask to keep hands on the iliac crests and turn the trunk to right and then to left, and later to carry out circumduction
- Cervical spine: Ask the patient to nod and rotate the head. If these movements are free and yet the neck is stiff, the pathology lies in the cervical spine below the upper 2 cervical vertebrae
- Costovertebral joints: Measure the difference in the chest girth between full inspiration and full expiration. Normal difference is ~ 7.5 cm. A marked reduction in the difference suggests ankylosing spondylitis.

Greatly **restricted mobility of the spine** ⇒ severe spasm of the muscles or ankylosis of the joints.

Measurements

Measure the length of the lower limbs to exclude shortening in one limb as a cause of scoliosis, if present.

If deformity of curvature is present, does it disappear or persist in different positions?

– Deformity apparent in standing position, disappearing on sitting ⇒ the problem lies in the lower limbs.
– Deformity apparent in sitting position, disappearing on lying down ⇒ postural deformity.
– Deformity persisting in all 3 positions ⇒ structural deformity.

Free ebooks ==> www.Ebook777.com

Special tests

- **Passive straight leg raising** test:
  Purpose: to diagnose PID.
  Procedure:
  – In a supine patient, raise the leg with knee extended by holding the foot and note when the patient complains of pain
  – Extend (the thigh) a little to relieve her/him of pain, and then forcibly dorsiflex the foot (to stretch the sciatic nerve only without stretching the hamstrings) and note if the patient complains of pain.
  Inference: Occurrence of pain ⇒ PID.
- (Active) **Straight leg raising** test:
  Purpose: to diagnose affection of S-I joint.
  Ask the patient to raise the limb straight up without bending the knee.
  Pain at the S-I joint ⇒ affection of the S-I joint.
  Principle: Pain occurs due to rotational strain produced by the tension of hamstring muscles.
- **Pump handle** test:
  Purpose: to diagnose affection of S-I joint.
  Procedure:
  – With the patient in supine position, flex the leg till the knee touches the abdomen
  – Put one hand on the ipsilateral shoulder to fix the trunk against the bed
  – With the other hand, push the flexed knee towards the opposite shoulder and note if the patient experiences pain on the affected side (the side being tested).
  Inference: Occurrence of pain ⇒ affection of the S-I joint.
- **Gilli's** test:
  Purpose: to diagnose affection of S-I joint.
  With the patient lying prone, hyperextend the thigh on the affected side with one hand while fixing the pelvis with the other hand placed on the unaffected S-I joint.
  Pain at the S-I joint ⇒ affection of the S-I joint.
- **Ober's** test:
  This test is done when lumbosacral strain is suspected.
  Principle: Contracture of the fascia lata and iliotibial tract is a common accompaniment of lumbosacral strain.
  Procedure:
  – Patient lies on the side, with the thigh in contact with the bed being flexed until the lumbar curve is obliterated
  – Stand behind the patient, steady the pelvis with one hand and flex the (upper) knee with the other hand
  – Abduct the thigh with the flexed knee as far as possible
  – Slowly extend the knee and allow the limb to drop by its own weight.
  Observation and inference:
  Failure of the thigh to drop completely ⇒ the presence of contracture.

Examine the **hip joint** as the apparent spinal problem may be secondary to pathology of the hip joint.
Restriction of extension alone without any limitation of other movements of the joint may be due to involvement of the psoas in a cold abscess and not due to arthritis of the hip joint.

Examine the **spinal cord and spinal nerves**
Retention of urine, brisk knee jerk, ankle clonus and Babinski's sign ⇒ cord compression.

Specific points to look for in **SYSTEMIC EXAMINATION**

- (A cold) Abscess in lateral aspect of the chest?
- Any e/o past affection of the chest by TB?—e.g. an old scar of drainage of empyema, collapse of the lung, etc.
- (A cold) Abscess in the iliac fossa / groin / lumbar triangle / buttock / ischiorectal region?

www.Ebook777.com

GROSS X-RAY (AP and lateral) FINDINGS of some selected conditions:

- Diminution of the intervertebral space with a hazy outline of the adjoining vertebral surfaces: The earliest and the most constant sign of caries of the spine.
  Wedging and gross destruction are late features of caries.
- In fracture, the vertebrae may be wedged but the intervertebral spaces remain unaffected.
- In secondary carcinoma, osteolytic changes are evident in bodies but the intervertebral spaces remain unaffected.
  In prostatic carcinoma however, metastatic deposits are osteoblastic.
- Characteristic posterior lipping ⇒ spondylitis deformans.
- Pertaining to S-I joint: It is extremely difficult to differentiate early bone changes from the normal irregularities of the S-I joint.

INFERENCE AND DISCUSSION

Age

In *children* backache is rare.

Common causes in children:	Ligamental strain due to faulty posture/compensatory to leg disorders, injury, deformities of the vertebral column, osteomyelitis, TB, tumor, leukemia, rickets.
Common causes during adolescence:	Sports injury, occupational strain, osteomyelitis, spondylolisthesis, Scheuermann's disorder.
Common causes in young adults:	Fibrositis, acute strain/sprain, congenital anomalies of the spine, trauma, defective posture, tuberculous osteomyelitis, acute PID, ankylosing spondylitis, rheumatoid arthritis, brucellosis and other causes of spondylitis, deformities of the spine, vertebral epiphysitis, referred pain, hysteria.
Common causes in middle age:	Degenerative disease, metastatic tumor.
Common causes in old age:	S-I sprain, spondylitis, osteoarthritis, non-articular rheumatism, osteomyelitis, metastatic tumor, spondylolisthesis, PID, osteoporosis, spinal deformities, referred pain.

Gender

Common causes in males:	Trauma, osteoarthritis, ankylosing spondylitis, PID.
Common causes in females:	Ligamental strain due to defective posture, osteoporosis, osteomalacia, pain referred from uterus/adnexa/kidney and psychogenic.

Causes of backache in cervical / thoracic region:

1. Fascitis, muscle strain.
2. Congenital disorders, post-traumatic, infection, tumor, deficiencies, and deformities of the spine: These disorders produce structural changes in the bone/joints, and are obvious clinically and radiologically.
3. Torticollis, defective eye sight, deformity of the rib-cage as in extensive fibrosis of lung or pleura, and rickets, defective posture due to occupation/habit.
4. Spondylosis, cervical disc prolapse.
5. Referred pain from intrathoracic disease.

Low (= Lumbosacral) backache

Low backache is the most common of all the chronic backaches particularly in the 3rd, 4th and 5th decade of life.

The conditions which produce structural changes in bones/joints form hardly 10% of the causes of low backache. *However, structural changes demonstrated by X-rays cannot always be taken as causative pathology of low backache in every case as equal groups of normal persons and backache patients show the structural changes in almost equal percentage.*

Free ebooks ==> www.Ebook777.com

Causes of low backache

In 90% cases	In 10% cases
– Fibrositis – Strain / sprain – S-I joint affections – PID – Osteoporosis – Referred pain from abdominal (including pelvic) conditions – Psychogenic	Congenital, post-traumatic, infection, tumor, deficiencies, deformities of the bony spine: These disorders produce structural changes in the bone / joints, and are obvious clinically and radiologically

Causes of **acute** backache

- Febrile backache
- Nonarticular rheumatism due to fibrositis
- Sprain
- PID
- Radiculitis
- Spinal meningitis, epidural abscess
- #s [traumatic, pathological (metastatic deposits / osteoporosis)]
- Acute infections: Osteomyelitis, brucellosis
- Referred pain from acute thoracic / abdominal conditions
- Psychogenic.

Other details of pain

- Referred pain = Pain referred to the limb and distributed along the dermatomes depending upon the compression of a particular nerve root. It is shooting in nature.
  Common causes: Spinal cord tumor, sacralization of the transverse process of L-5, spondylosis, PID, extensive deformity of the spine and conditions giving rise to collapse of vertebra.
- Segmental pain may suggest lesions of muscles giving muscle spasm and stimulation of interspinous ligaments
- Pain less in the morning ⇒ organic lesions of vertebrae
- Pain more in the evening ⇒ lumbosacral strain
- Shooting pain ⇒ radicular pain
- Aggravating factors
  - Pain coming after a long period of work in awkward position ⇒ postural strain
  - Intensity increasing during working ⇒ chronic strain
  - Pain increased by coughing/sneezing/any other straining act ⇒ radicular pain, most commonly due to PID
- Pain relieved by taking rest suggests a chronic inflammatory condition.

Fever ⇒ infection of soft tissue / bone / some types of arthritis.

Gait: A cautious gait; short steps and walking often on toes to avoid any jerking ⇒ caries of the thoracic spine.

Rigidity of the spine ⇒ severe spasm of the muscles or ankylosis of the joints.

DIFFERENTIAL DIAGNOSIS of selected disorders

Fibrositis

Localized tender spots and nodules are usually present.

Lumbosacral strain

- Pain over the top of the sacrum, often radiating to the front and outer aspect of the thigh
- Felt more in the evening
- Increased by bending forward

www.Ebook777.com

– Bending forward is restricted in both standing and sitting postures, whereas in S-I joint affections and functional pain, it is limited in standing but free when sitting as the pelvis is fixed in the latter posture
– Ober's test may be +ve.

Sacroiliac ligamentous strain

– In c/o an adult woman, low backache or sciatica is often noticed first after childbirth
– In c/o a male patient, a h/o twisting violence may be obtained
– Extremes of spinal movements are painful
– Special clinical tests for S-I joint affection are +ve.

Muscle spasm

– Spine appears straight in c/o spasm of the erector spinae
– Muscles seen to be standing out and bulging
– On finger-tip pressure, the muscle feels hard and no depression can be made in it
– Flexion movement—restricted.

Spina bifida: Refer to chapter 94.

Acute osteomyelitis of the spine is rare.

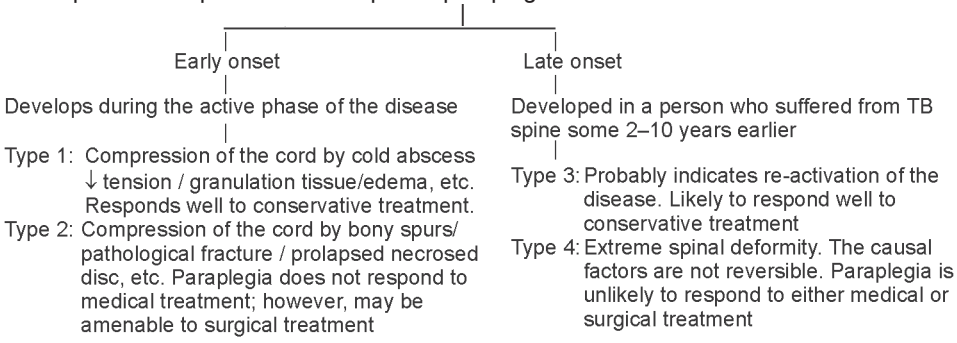
– Fever
– Severe local pain of sudden onset
– Collapse of vertebra does not occur owing to early recumbency, rapid new bone formation and paravertebral calcification
– Development of a pyogenic abscess, not a cold abscess
– Bony ankylosis; not fibrous ankylosis (*cf* in tuberculous osteomyelitis)
– Prognosis may not be good due to spread of infection to spinal meninges.

Tuberculous (chronic) osteomyelitis

– Young population is more commonly affected than older people.
– Constitutional symptoms are not very common in the early stages.
– The main symptom – Persistent pain in the back, which precedes any objective signs by several months. The sites of pain: local and referred according to the nerve roots involved. Shooting pain along the involved nerve roots usually precedes the onset of paraplegia.
– Stiffness in the back muscles and inability to bend forwards.
– Deformity begins to appear. Kyphosis develops at the level of maximum vertebral involvement.

Common complications: (Cold) Abscess formation and paraplegia.

– Cold abscess—initially develops within the spine and then spreads outward under the vertebral ligaments. Pus tracks along the nerves and presents as an abscess in the retropharyngeal region / posterior triangle of the neck / axilla / chest wall / psoas sheath / lumbar triangle / gluteal region / posterior aspect of the thigh, depending upon the location of tuberculous process from cervical spine downwards.
– Cold abscesses are seen radiologically in only ~ 50% of cases.
– Vertebral disease is virtually more extensive than that seen on an X-ray.
– An important complication of TB spine = paraplegia.



Free ebooks ==> www.Ebook777.com

– The disease ends in fibrous ankylosis which may proceed to bony ankylosis, unlike tuberculous arthritis of other joints in which fibrous ankylosis is the rule.

Locoregional specific clinical features according to the region affected: The most common site of involvement is the lower thoracic spine affecting T8–T11.

Cervical

– Pain—local and referred to the back of the head (high cervical) or arm (low cervical)
– Attitude: Patient supports the head with hand
– Rigidity of the head and neck
– Torticollis
– (Cold) Abscess:
  - Retropharyngeal (may → dysphagia and dyspnea)
  - Suboccipital
  - Posterior triangle of the neck whence it may travel down towards the mediastinum or along the brachial nerves to the axilla.

Thoracic

– Pain—local and referred to the chest or abdomen, sometimes girdle pain
– Rigidity
– Gibbus
– (Cold) Abscess:
  - Paravertebral
  - Lateral ± anterior thoracic due to pus burrowing along the cutaneous branches of the intercostal vessels and nerves
  - From the posterior mediastinum, it may gravitate down beneath the medial / lateral arcuate ligament to become a psoas or lumbar abscess respectively. Psoas abscess extends downwards. Lumbar abscess burrows superficially in lumbar triangle
– Paraplegia, retention of urine.

Lumbar

– Pain—local and referred to lateral aspect of thigh
– Limitation of movements, especially flexion and extension
– Deformity is slight as there is normal lumbar lordosis
– Iliopsoas abscess with fixed flexion deformity of the hip.
  The abscess may burrow into the thigh under the inguinal ligament or into the pelvis and perineum where a fistula may form on bursting, or it may travel to the buttock from the pelvis passing through the sciatic foramen
– Neurological symptoms are rare as the spinal canal is spacious.
  Causes of neurological symptoms, if they develop: 1. Backward extension of the disease forming a nodule under the posterior common ligament compressing the cord against the laminae. 2. Abscess burrowing backward and compressing the cord. 3. Tuberculous meningitis, myelitis, displaced sequestrum or pathological fracture compressing the cord.

Ankylosing spondylitis

– More commonly men are affected between 20 and 30
– Pain in the lower back and increasing stiffness
  Later, pain radiates down one/both lower limbs
– The spine gradually flexes forwards with marked restriction of all movements. Eventually the patient is bent double
– ESR is frequently raised.

Notes:

Ankylosing spondylitis is characterized by progressive ossification of the ligaments and capsules of joints, resulting in complete bony ankylosis of the spine and occasionally of the major joints of the limbs. The disease always begins in the S-I joints.

www.Ebook777.com

PID

– Usually occurs in the lumbar region. The disk between L-5 and S-1 or L-4 and L-5 is usually affected.
– *Predisposing factors*: Aging, trauma.
– *Precipitating factors*:
  - A strain such as lifting a heavy weight
  - Prolonged standing, e.g. standing in a queue. Pain is relieved only by sitting/lying down.

Acute PID:

– Mode of onset—sudden with agonizing pain in lumbar region.
  Pain is aggravated by coughing / sneezing.
– Characteristic attitude: Lumbar scoliosis with convexity to the affected side. Kyphosis and slight flexion at the hips and knees
– Local deep tenderness ~ 5 cm lateral to the midline on the side of the lesion
– Flexion and extension—greatly restricted
– Remission of symptoms occurs due to natural fibrosis of the protruded disk.

Chronic PID:

– Mode of onset—insidious with low backache, suggesting a small protrusion of the disc causing bulging of the pain-sensitive posterior longitudinal ligament
– Sciatic pain ⇒ a large protrusion passing through the posterior ligament and impinging upon the nerve. Sciatica is at first located in the buttocks, but it soon spreads to the thigh, leg and toes.

S-I joint affections

– Pain and tenderness in the region of the joint
– Pain while turning in bed or jumping from a height (a stool for example)
– Bending forward is limited in standing but free when sitting as the pelvis is fixed in the latter posture.

Coccydynia

– Pain over the sacrococcygeal area when sitting or while defecation, and tenderness over this area
– It is a painful condition in the region of the coccyx, caused by injury to or strain of the sacrococcygeal joint.

Osteoporosis

Usually occurs after 50, particularly in women.

Psychogenic backache

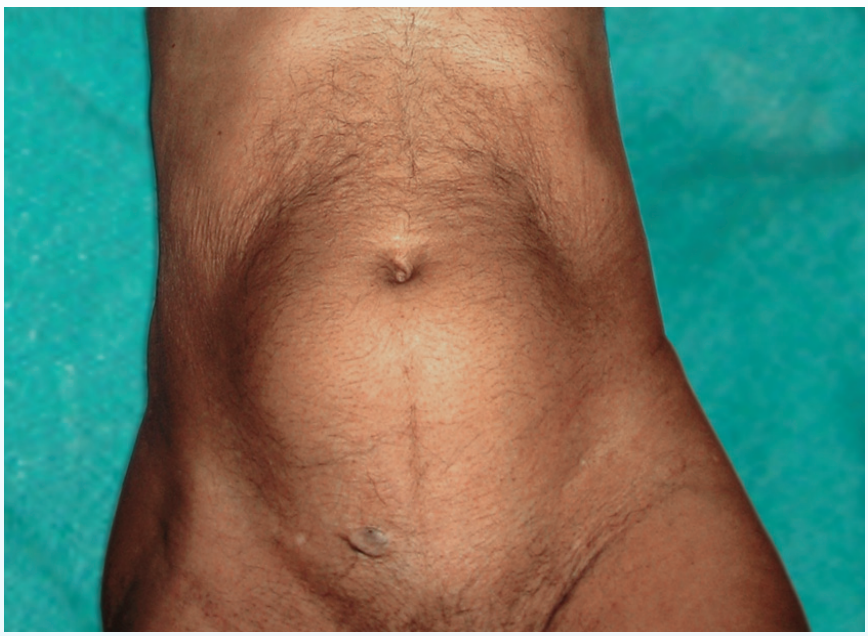
– Distribution of pain may be unusual
– Even though the patient complains of severe pain, she / he does not seem to be in agony and usual accompaniments of severe pain are absent with hardly any disturbance of function
– Symptoms and mode of onset—vague
– Depression may be seen on the face and sensed in the attitude
– If the patient is observed in the waiting room or while entering the consulting room, she / he may not appear in distress due to pain, but complains of pain when the examination begins.

Free ebooks ==> www.Ebook777.com

www.Ebook777.com

SECTION 2

Urogenital Case



Free ebooks ==> www.Ebook777.com

www.Ebook777.com

54 UROGENITAL CASE

(For disorders of the scrotum, refer to chapters 25, 26, 85)

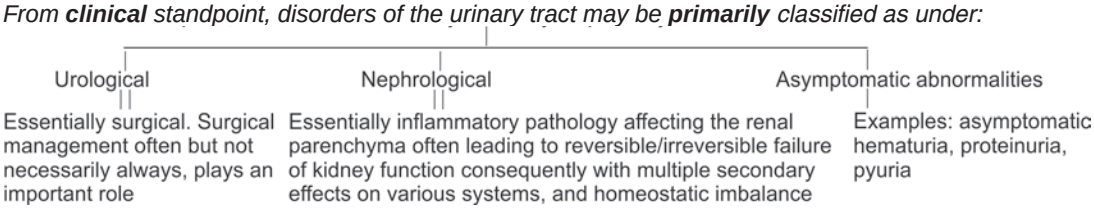
GENERAL REMARKS

From clinical standpoint, urinary tract is divided into upper tract and lower tract:

- The upper urinary tract (UUT) consists of kidney, pelvis and ureter
- The lower urinary tract (LUT) consists of urinary bladder, bladder neck (trigone, prostate and prostatic urethra), and urethra distal to the external sphincter (rhabdosphincter).

Various disorders which may affect the urinary tract: Obstruction, inflammation, infection, stone formation, fistulization, functional, tumor, incontinence and congenital.

*From **clinical** standpoint, disorders of the urinary tract may be **primarily** classified as under:*



Because of the large reserve of renal tissue, considerable structural damage can occur before functional damage becomes apparent.

Kidney function can be impaired in 3 principal ways	*Example of the pathology responsible*
1. A reduction in renal plasma-flow	Essential HT
2. Glomerular damage	Glomerulonephritis
3. Impairment of tubular function	Pyelonephritis
4. Impairment of all the functions	Back pressure due to obstruction

Many patients presenting with symptoms of urinary tract disorder require further investigation.
Hematuria always requires full urological assessment.

DEFINITIONS

Dysuria may be defined as difficulty in micturition (of some or the other kind relating to the lower tract, and the pathology being usually though not necessarily obstructive in nature).

Frequency = the number of times urine is passed in a day (24 hours). Conventionally, it is taken for granted to mean increased frequency.

Nocturia = an urge to urinate that interrupts sleep.

Urgency = an overwhelming desire to void which can be postponed for only a short time.

Urge incontinence: Urgency is so severe that the onset of micturition occurs immediately after warning.

Hesitancy = delay in starting the act of micturition.

Intermittency = interruption of the stream of urine.

Free ebooks ==> www.Ebook777.com

Oliguria = a urine output of < 0.5 mL/kg/hr, or < 100–400 mL/24 hours.
Anuria = 0 output over 12 hours (practically < 30 mL on catheterization).
Polyuria = an abnormally increased volume of urine output of > 2 L/24 hours in children, and > 3 L/24 hours in adults.
Hematuria = the presence of blood in urine.
Essential hematuria = episodic or continuous gross hematuria, the cause of which cannot be determined by radiological or hematological investigation.
Gross hematuria is obvious to the naked eye and appears as red, brown or smoky urine.
Hematuria is called ***microscopic*** when more than 3–5 RBCs are detected per high power field of a microscope.
Chyluria = milky urine because of admixture with intestinal chyle.
Hydatiduria = passage of fragments of hydatid membrane and daughter cysts in urine.
Strangury = frequent attempts at micturition with severe sharp stabbing pain, passing only a small quantity each time.
Total (True) incontinence = continuous leakage of urine with bladder being unable to retain any amount of urine.
Overflow incontinence = involuntary leakage of urine after the bladder having got decompensated to its limits and incapable of contraction sufficient to generate a stream of urine.
Stress incontinence = intermittent leakage of urine that occurs due to sudden physical strain like coughing, laughing, sneezing, etc.
Enuresis = involuntary voiding of urine during sleep without any detectable urological or neurological disease in children.
Hematospermia = passage of blood in the seminal fluid.
Azotemia = a biochemical abnormality of elevation of BUN and S Cr levels.
Uremia: When azotemia becomes associated with a constellation of symptoms and signs and (other) biochemical abnormalities, it is termed uremia.

URINARY TRACT OBSTRUCTION

presents with variable symptoms depending on whether it is acute or chronic, unilateral or bilateral, complete or partial, and on the underlying etiology.
It is an important reversible cause of unexplained renal failure. The possibility should be considered in all cases of ARF or when CRF worsens abruptly.
Early (hours) obstruction results in urinary indices identical to those of prerenal failure; later, obstruction will resemble the indices of ATN.
An acutely obstructed kidney does not necessarily cease to function. Return to useful function is then dependent on:

– the length of time that the kidney is obstructed,
– the degree of obstruction,
– the presence or absence of infection above the obstruction.

Recovery of renal function after relief of obstruction usually occurs within 2 months but can take up to 6 months. Therefore, renal function 2 months after the relief of obstruction reflects the degree of potential renal recovery.
After relief of bilateral obstruction a postobstructive diuresis may occur. In this event, it is very important to monitor fluid balance carefully, otherwise the patient can become hypovolemic and hypotensive with further renal damage occurring. Sodium and potassium conservation usually return after 48–72 hours but concentrating ability can take up to 7–10 days to return to normal. These postobstructive changes do not occur to any great extent when one normal kidney remains unobstructed and retains its normal homeostatic responses.
Patients at risk of developing an obstruction:

– Women, due to pelvic tumor,
– Elderly men, due to prostatic enlargement,
– Diabetics, due to papillary necrosis, neurogenic bladder,
– Patients with stones, vesicoureteral reflux, retroperitoneal disease, or functional urinary retention.

Urinary tract obstruction should be considered in patients with UTI, stones and in all patients with unexplained azotemia.

www.Ebook777.com

Location of disease	**Causes of obstructive uropathy**
Pelviureteric junction	Congenital PUJ obstruction. Stone/Tumor blocking the PUJ.
Ureter	Luminal/Mural: Stone, stricture, tumor, clot, sloughed papilla. Extrinsic: Retroperitoneal—tumor, fibrosis, inflammation, bleed. Bladder cancer/Prostatic cancer blocking the VU junction.
Bladder outflow obstruction	Benign/Malignant enlargement of prostate. Bladder neck stenosis.
Urethra	Stricture, cancer, phimosis.

BPH is a histopathological diagnosis which should not be used as a clinical descriptor.
BPE (Benign Prostatic Enlargement) = the presence of enlarged gland on clinical and ultrasound examination, without suspicion of cancer.

UROLITHIASIS

→ Pain / UTI / hematuria / dysuria / unexplained pyuria.
Unsuspected stones may be found on routine X-ray.

Predisposing factors

Urinary pH (urate and oxalate stones form in acidic urine; phosphate stones in alkaline urine), stasis often due to obstruction, infection, metabolic factors (hypercalcuria, hyperphosphaturia, urate excess, cystinuria, xanthinuria).

Etiology of urolithiasis

- Idiopathic: account for a large number of cases.
- Hypercalcuria: - The most common abnormality found on routine investigation.
  - Causes: Idiopathic, increased intestinal absorption, renal leak, bone demineralization.
- Hyperparathyroidism.
- Inherited enzyme disorders such as cystinuria, renal tubular syndrome.
- Hyperoxaluria: - Primary—a rare genetic disorder.
  - Secondary to short gut syndrome or fat malabsorption.
- Hyperuricosuria due to increased dietary purine intake, defective renal tubular handling of uric acid, or rare hereditary enzyme deficiency.

Types of urinary stones

- Calcium oxalate: Most common.
- Calcium phosphate: Pure calcium phosphate stones are rare and usually seen in renal tubular acidosis.
- Struvite = infective = triple phosphate stones. Composed of calcium, magnesium, ammonium and phosphate.
  Infection with urease-producing bacteria; usually Proteus is needed to form these stones.
- Uric acid: Form in acidic, concentrated urine.
- Cystine: Occur in cystinuria, an autosomal recessive disorder.
- Rare: Xanthine, silicate, triamterene, indinavir.

UTI

Generally defined as bacteriuria > 10 bacteria/mL of urine.
Levels > 100 may indicate infection in some symptomatic patients but are commonly due to poor sample collection.
Adults at risk: Sexually active women, pregnant women, or anyone with UT obstruction, reflux, catheterization, and neurogenic bladder.

UTI is a common cause of sepsis.
Increased frequency, urgency, cloudy offensive urine and suprapubic discomfort ⇒ Lower UTI.
Loin pain, fever, rigors and malaise ⇒ Upper UTI (Complicated infection). Upper UTI should be taken seriously as serious sepsis can ensue.

Free ebooks ==> www.Ebook777.com

Infections of the kidney

Pain in the flank, nausea, vomiting and fever ⇒ renal infection.

Renal infections arise in the following ways:

1. Ascending the lumen of the ureter from the bladder – the commonest route.
2. Ascending the periureteral lymphatics from the cervix or prostate.
3. Hematogenous: from a primary focus somewhere else in the body, e.g. tonsillitis, carious teeth, cutaneous infections particularly boils and carbuncles, pulmonary TB, etc.

Acute pyelonephritis

– usually, but not necessarily always, an acute ascending pyogenic infection usually associated with lower UTI (obstruction and/or vesicoureteric reflux often being present).

Large numbers of bacteria are found in the urine, several hundred thousand per ml in the acute phase.

Natural history: Healing / perinephric abscess / papillary necrosis / recurrence / chronic pyelonephritis.

Chronic pyelonephritis—essentially the result of repeated attacks of inflammation and healing.

Depending on the cause, only one or both kidneys may be affected to variable degrees.

It is possible, but unusual, for chronic pyelonephritis to remain asymptomatic until advanced renal insufficiency appears.

Symptoms / *ways of presentation*: one or more of the following:

– Chronic dull ache in the loin.
– Increased frequency of micturition.
– HT. Develops slowly, hence, the presence of HT suggests long-standing case. Occasionally, HT could be accelerated type.
– Constitutional symptoms: anorexia, nausea, malaise, headache, lassitude.
– Attacks of fever exceeding 100°F. The need to explain these attacks often leads to a thorough investigation including that of the urinary tract, then bringing the disease to light.
– A normochromic anemia.

Pyonephrosis

The kidney is converted into a multilocular sac containing pus or purulent urine.

It may complicate hydronephrosis, acute pyelonephritis, or renal stone.

UTI in childhood

C/Fs in young children

– There may be no obvious urinary symptoms, although the mother may notice a cloudy/offensive urine.
– Pain/screaming on micturition, refusal to eat, high-grade fever with rigors ± convulsions, and failure to thrive: think of the possibility of UTI.

C/Fs in older children

– Pain in the loin.
– Sudden development of increased frequency of micturition and enuresis.

Many cases are associated with *underlying anatomical or congenital abnormalities*.

It is important to recognize UTI because it frequently involves the kidneys. If recurrent, it may lead to severe renal impairment or renal failure in early adult life.

Cystitis

Until after middle age, cystitis is very much commoner in women than in men.

Indications for investigation to discover and treat the predisposing cause; though sometimes no cause can be found:

– Repeated attacks of cystitis in women.
– Single attack of cystitis in a man.
– Single attack of cystitis in a child of either sex.

Acute cystitis

– Common in healthy women, especially frequent in pregnancy, and also associated with intercourse.
– In males it is usually secondary to obstruction.
– Obstruction encourages infection, enhances its effects and prolongs the inflammatory process. Mixed infections, and infection with *Proteus*, *Staphylococcus* are common.
– In non-obstructive cases, infection is usually due to *Escherichia coli*.

www.Ebook777.com

Chronic cystitis

Causes/predisposing factors: Recurrent attacks of acute cystitis, obstruction, specific chronic infections like TB, schistosomiasis.

Tuberculous cystitis is always secondary to a tuberculous infection elsewhere, usually in the kidney.

(Nonspecific) **Prostatitis**

– In both acute and chronic prostatitis the seminal vesicles and the prostatic urethra are also inflamed; thus there is a triad of pathological conditions viz. prostatitis, posterior urethritis and seminal vesiculitis
– As a rule, the infection is hematogenous from a distant focus. In a minority of cases the infection ascends from the urethra, or descends from the bladder or kidneys.

TUMORS OF THE URINARY TRACT

– Truly benign tumors of the kidney are rare; examples—fibroma, adenoma (smaller than 2 cm)
– All tumors of the kidney which can be recognized clinically should be considered malignant and treated as such.

• *Tumors of the kidney are seen to occur in 2 groups*

– 1. in children between 1 and 7 years of age, and 2. In adults after 40
– Malignant tumors are unusual between the ages 7 and 40
– Urothelial tumors are often multiple and recurrence is common. Long term surveillance is important
– *Renal adenocarcinoma* probably originates from proximal tubular cells
– *Transitional cell carcinoma* arises from the urothelium that lines the whole of the urinary tract from collecting system to urethra
– *Nephroblastoma* (Wilm's tumor) is the most common childhood urological malignancy.

ACUTE RENAL FAILURE (ARF) may be defined as rapid onset of renal impairment that results in accumulation of nitrogenous waste products, most notably urea and creatinine in the body.

ARF is often potentially reversible but in few cases it may not be so due to a pre-existing renal damage (acute-on-chronic renal failure) or irreversible mechanism of renal damage or if the treatment is delayed. Conventionally, ARF is often equated to acute tubular necrosis (ATN), where there is intrinsic but reversible damage to the kidney.

Clinical course

– ATN begins with diminished urine output apparent within a day of the insult; anuria may be present in some cases
– Oliguria lasts for 10–14 days
– If oliguria persists for > 2–3 weeks, other diagnoses should be considered
– The daily increments in BUN and serum creatinine average 10–20 and 0.5–1 mg%, respectively, but will be greater in catabolic patients
– During the recovery phase, urine output increases progressively; BUN and serum creatinine plateau, and then begin to fall. Major complications of ARF may first appear during this stage.

Features (effects) of ARF

– Volume expansion leading to edema, HT, CHF
– Hyperkalemia, hyponatremia and acidosis
– ↑ BUN and serum creatinine

Causes of ARF

These are classified essentially into 3 groups.

i. *Prerenal*—due to inadequate perfusion of kidneys with arterial blood
ii. *Renal*—due to renal disease which results in destruction of excretory function
iii. *Postrenal*—due to obstruction of the outflow from kidneys
iv. *Overlap*—long-continued renal hypoperfusion or renal outflow obstruction resulting in secondary renal damage which after a time may become irreversible.

A point to note: Although renal artery embolism/thrombosis, and renal vein thrombosis are technically prerenal causes, they may be clinically grouped with the renal causes as they do not present with shock.

Free ebooks ==> www.Ebook777.com

Prerenal causes of ARF
- Reduced cardiac output: Acute MI, cardiac arrest, cardiac failure, cardiac tamponade
- Depleted effective circulating volume: Sepsis, bleeding, hypoalbuminemia
- Depleted extracellular fliud: Diarrhea, vomiting, excessive diuresis, loss from skin/body surface associated with sweating and burns.

Causes of intrinsic ARF
- Hypercalcemia
- Hepatorenal syndrome
- Toxins:
  - Primary ATN (Aminoglycosides, cephalosporins, paracetamol, salicylates, heavy metals)
  - Tubular obstruction (Myeloma)
  - Myoglobinuria (Muscle injury, snakebite, heroin, barbiturates)
  - Hemoglobinuria (Mismatched BTF, nitrofurantoin).
- Acute interstitial nephritis:
  - Drug-induced, e.g. some antibiotics, NSAIDs, diuretics, etc.
  - Infections, e.g. mononucleosis, measles, leptospirosis, brucellosis, etc.
- Vascular (endothelial damage): Vasculitis, hemolytic uremic syndrome, DIC, accelerated HT
- Rapidly progressive glomerulonephritis:
  - Primary
  - Secondary to systemic disease: Infection (bacterial endocarditis), 'shunt' nephritis (usually associated with ventriculoatrial shunts), hepatitis B-associated nephritis
  - Vasculitis
- Renal artery embolism/thrombosis, renal vein thrombosis.

Postrenal causes of ARF: Basically, obstructive uropathy.
- Renal: Papillary necrosis
- Congenital PUJ obstruction, pelviureteric stone, crystals, clot, tumor, retroperitoneal fibrosis, accidental ligation of both ureters during operation like hysterectomy, etc.
- Lower urinary tract obstruction: Prostatic enlargement, bladder neck obstruction, urethral stricture, posterior urethral valves, etc.

Urinary indices to differentiate the type of ARF

Type (cause) of ARF →	*Prerenal*	*Renal*	*Postrenal*
Urine volume	Low	None	Low
Plasma BUN: Serum Creatinine ratio	> 20	< 20	> 20
Urine Na	< 20 mEq/L	> 40 mEq/L	< 20 mEq/L
Osmolality	> 500	< 400	> 500
Fractions excretion of Na %	< 1	> 1–2	< 1
Ultrasound blood flow	Diminished	Diminished or normal	Normal
DTPA, DMSA scan	Diminished perfusion	Scarred kidney	Normal or scarred

CHRONIC RENAL FAILURE (CRF)

- Unlike ARF, it is irreversible, although the rate at which the function is lost is variable and dependent on a number of factors. Progressive loss of renal function occurs over months to years. Once a significant proportion of renal tissue is scarred, progression to end-stage renal failure becomes inevitable
- There is no widely acceptable definition of the point at which CRF becomes established.

A guide to the degree of severity of CRF	*GFR* (mL/min)	*Features*
Mild	40–50	Asymptomatic / mild HT ±
Moderate	20–39	Anemia, lassitude, HT, early osteodystrophy
Severe	10–19	Anorexia, vomiting, pruritus, edema
End-stage	< 10	Pulmonary edema, pericarditis, convulsions, coma

www.Ebook777.com

- Measurement of serum or plasma creatinine, which varies inversely with the GFR, is the simplest and the most commonly used measurement. However, the value of this measurement is limited by a wide range of normality, and considerable renal damage (with over 50% nephron loss) can occur before the plasma creatinine exceeds the upper limit of the normal range
- Little or no change in arterial pH, plasma HCO_3, or P_{CO_2} occurs until CRF is far advanced
- Uremia will not occur in unilateral renal failure, the contralateral kidney being normal
- Uremia is a clinical syndrome. It is characterized not only by failure of renal excretory function but also by a host of metabolic endocrine alterations incident to renal damage. There are, in addition, secondary cardiovascular, neuromuscular and gastrointestinal involvements which are usually necessary for the diagnosis of uremia.

Presentation of chronic renal disease

Symptoms are often a late feature of CRF. Because of adaptive mechanisms, symptoms of uremia do not appear until GFR is reduced to < 25% of normal.

Clues to the presence of renal disease:

– *Persistent asymptomatic microscopic hematuria and/or proteinuria*
– *Persistent asymptomatic elevation of serum creatinine*
– *In case of a pregnant woman*, elevation of BP particularly before the last trimester
– *HT accompanied by proteinuria, hematuria or elevated serum creatinine. Severe HT associated with bilateral retinal bleeds, exudates and/or papilledema*
– *Macroscopic hematuria*: Often intermittent. 'Smoky' urine and reduction in urine output may be suggestive of acute nephritic syndromes
– *Dysuria*
– *Nocturia, polyuria*
– *Edema*.

Most common causes of CRF: Chronic GN, chronic pyelonephritis and interstitial nephritis, polycystic kidney disease, HT, DM, obstructive uropathy and idiopathic. In many cases, the underlying cause cannot be determined.

The severity of renal failure is monitored by BUN, serum creatinine and GFR (normal GFR is ~ 100 mL/min).

TWO WELL-RECOGNIZED MODES OF PRESENTATION OF RENAL DISEASE

– The nephrotic and nephritic syndromes. Both are associated with many different renal diseases.

– Gross edema
– Normal urine output
– Hypovolemia
– Proteinuria > 3 g/24 h
– Hypoalbuminemia < 3 g%

⇒ **Nephrotic syndrome**. A typical presentation of minimal change and membranous GN, renal amyloidosis, and diabetic nephropathy.
Causes: Idiopathic, drug-induced, infections, tumors, multisystem / hereditary diseases.
Complications: Protein malnutrition, ↑ed risk of VT and infection.
Patients are almost always hypercholesterolemic.

– Mild edema
– Oliguria
– HT
– Hematuria, often microscopic
– Hypervolemia
– Proteinuria < 3 g/24 h
– Impaired renal function

⇒ **Nephritic syndrome**. The usual presentation of acute nephritis, rapidly progressive GN, infective endocarditis, hemolytic uremic syndrome.

GLOMERULAR DISEASES

→ 4 main clinical syndromes: Nephritic syndrome, nephrotic syndrome, CRF and asymptomatic hematuria or proteinuria.

The main causes: Immune damage, DM, vascular disease, e.g. HT.

Free ebooks ==> www.Ebook777.com

Examples
Acute diffuse proliferative GN (classically following 2–3 weeks after an infection like Group A hemolytic streptococcal pharyngitis), rapidly progressive GN (progress to end-stage renal failure in weeks/months), membranous GN, membranoproliferative GN, focal GN, IgA nephropathy, minimal change GN, chronic GN (the end, stage of many forms of GN), parasitic glomerulopathy (malaria, schistosomiasis), etc.

TUBULOINTERSTITIAL DISEASES

Examples
Acute tubular necrosis, pyelonephritis, acute tubulointerstitial nephritis (developing 10–14 days after exposure to drugs like methicillin, NSAIDs, etc.), chronic tubulointerstitial nephritis (often idiopathic), analgesic nephropathy (a distinctive disorder caused by long-term exposure to analgesics), renal tubular acidosis, etc.

DIABETIC NEPHROPATHY

- *Clinical features* include HT, bacteriuria, proteinuria and azotemia
- Duration of diabetes is variable, but proteinuria may develop 10–15 years after the onset of diabetes, progress to nephrotic syndrome, and then progress to renal failure over 3–5 years
- Tight glucose control may delay the onset of nephropathy.

ACUTE RENOVASCULAR OCCLUSIVE EVENTS

Acute renal artery embolism, renal artery thrombosis, renal artery dissection, acute renal vein thrombosis.

ASYMPTOMATIC URINARY ABNORMALITIES

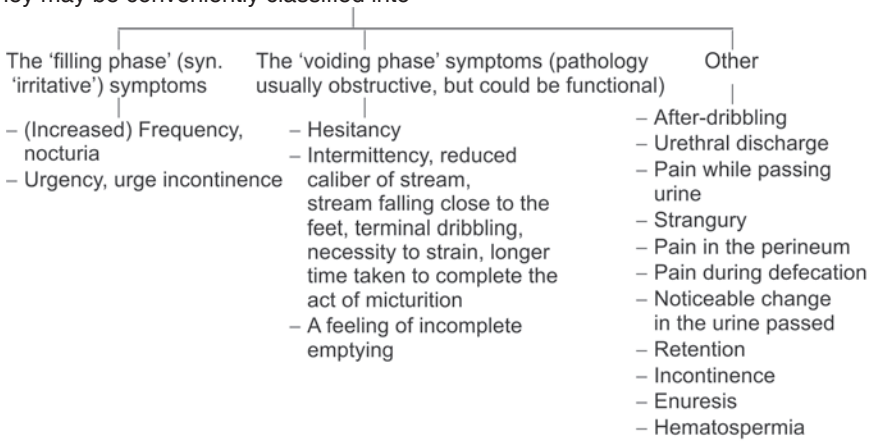
– **Asymptomatic hematuria**
  - Possible causes: Stones, tumor, infection at any level in the urinary tract, sickle-cell disease, analgesic abuse
  - Pattern of gross hematuria may help in localizing the site
– **Hematuria with low-grade proteinuria** ⇒ benign recurrent hematuira or IgA nephropathy
– **RBC casts, proteinuria, or dysmorphic RBCs in urine** ⇒ renal parenchymal disease
– **Modest proteinuria**
  - As an isolated finding it may occur due to fever, CHF, exertion, upright posture
  - Renal causes: DM, amyloidosis, other mild forms of glomerular disease
– **Pyuria** as a sole finding
  - Mild renal inflammation—interstitial nephritis, SLE, transplant rejection
  - UTI
  - 'Sterile' (on ordinary culture media): Due to treated infection, TB, prostatitis, urethritis, unusual urinary pathogens including *Entamoeba histolytica*.

SYMPTOMS OF URINARY TRACT DISORDER

IT MAY BE PRACTICALLY USEFUL TO CLASSIFY THE PRESENTING SYMPTOMS IN 5 GROUPS.
1. Typical symptoms suggesting specifically a urinary system disorder.
2. Abdominal symptoms. It may be necessary to differentiate urological disorder from retroperitoneal disorder (other than urological), intraperitoneal disorder, or musculoskeletal disorder.
3. Symptoms related to the external genitalia and perineum; disorder easily amenable to physical examination.
4. Nonspecific symptoms due to the effect of a chronic renal disease: Anorexia, nausea, vomiting, anemia, fever, fatigue, lethargy, etc.
5. Symptoms of sexual dysfunction, infertility.

www.Ebook777.com

– Each symptom should be evaluated to find a clue with respect to anatomical location as well as pathological nature of the disease. Some symptoms however, will give a clue to only the possible anatomical site or the possible pathological nature of the disease
– Seemingly unrelated symptoms may also be urological, e.g. backache, fever of unknown origin, anemia, lethargy, etc.
– One-half of all men over 65 have some urinary symptoms. Many symptoms are caused by age-related changes in the bladder unrelated to prostatic disease or to outflow obstruction
– Symptoms caused by disorder of the lower urinary tract are specific, and are called 'lower urinary tract symptoms'. They may be conveniently classified into



A SIMPLE AND VALUABLE CLINICAL 3-GLASS URINE TEST

– During one act of micturition, ask the patient to collect urine in three glasses successively such that approximately equal amount of urine is collected in each glass
– Examine all the three glasses particularly for—if the urine is clear or cloudy/turbid, and if it contains blood/blood clots and (prostatic) threads
– Pour the urine in a flat tray containing water and examine the clots and also look for (prostatic) threads and pieces of growth (if found, send for histological examination).

HISTORY

- **Occupation**?
  – Chronic exposure to organic solvents, lead, cadmium may lead to renal disease
  – Bladder tumors are an industrial hazard. Examples—rubber industry, β-naphthylamine (aniline dye manufacturing), benzene, etc.

Typical symptoms suggesting a urinary system disorder

- **Frequency** of micturition: Increased OR decreased?
  ↓
  – Duration?
  – How many times during the day and how many times during the night? (Record as D … / N …)
  – Volume of urine—increased or normal?
  – Did you drink a lot of water?
  – Have you been suffering from DM?—controlled or otherwise?
  – Have you been taking diuretic?
  – Known to have any kidney disease?
- **Urgency**?

Free ebooks ==> www.Ebook777.com

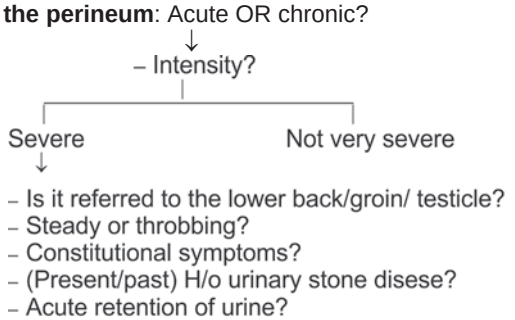
- **Urge incontinence**?
- **Hesitancy**?
- **Intermittency**?
- How is the **caliber of stream**? Has it reduced than before?
- Does the **stream fall close to the feet** rather than away unlike before?
- **Terminal dribbling**?
- Do you have to **strain to pass urine** (by contracting abdominal muscles)?
- Does straining improve the stream or weaken the stream?
  Improvement ⇒ urethral obstruction. Weakening ⇒ enlarged prostate (due to further kinking of the prostatic urethra).
- How much **time do you take to complete the act of micturition**?
- Does it take longer than before?

One/more of these symptoms qualify for '**poor flow**'.

- Feeling of **incomplete emptying**?
- **After-dribbling**?

  This must be distinguished from terminal dribbling. After-dribbling is not a symptom of bladder outflow obstruction (except occasionally a urethral stricture).
- **Urethral discharge**?
  - Quantity?
  - Consistency?
  - Color—yellow / gray / watery / bloody?

  INTERPRETATION:
  - Profuse, thick, yellow-gray colored purulent ⇒ (acute) gonococcal urethritis
  - Scanty, watery or gleet (mucoid or purulent discharge noticed at the external urethral meatus especially in the morning) ⇒ nonspecific (chronic) urethritis, prostatitis
  - Bloody ⇒ urethral cancer.
- **Pain while passing urine**?
  - Is the pain experienced at the end, or is it experienced throughout the act of micturition?
  - Is it referred to the tip of penis / labia majora / perineum / suprapubic region?
  - Nature? Is it scalding?
  - Does the pain disappear after passing urine?
- Frequent attempts at micturition with severe sharp stabbing pain, passing only a small quantity each time?
  – Strangury
- **Pain in the perineum**: Acute OR chronic?



- **Sensation of fullness/weight in the perineum**? ⇒ prostatitis
- **Pain during defecation**?
- Any **noticeable change in the urine passed**
  - Color: Red / brown / stale tea-like / smoky / 'cloudy' / turbid / white?
  - Presence of gravel/stone, fragments of a growth, (prostatic) threads, gas or fecal matter / worms?

www.Ebook777.com

*Details to be asked in c/o **hematuria*** (red/brown-colored or smoky urine):

- Does the urine contain blood OR Does pure bleeding occur from the urethra independent of micturition?
- Do the underclothes get stained with blood? ⇒ bleeding occurring independent of micturition.
  Bleeding occurring independent of micturition ⇒ essentially a urethral lesion (urethra distal to rhabdosphincter).

Continue with details to be asked for hematuria 'proper':

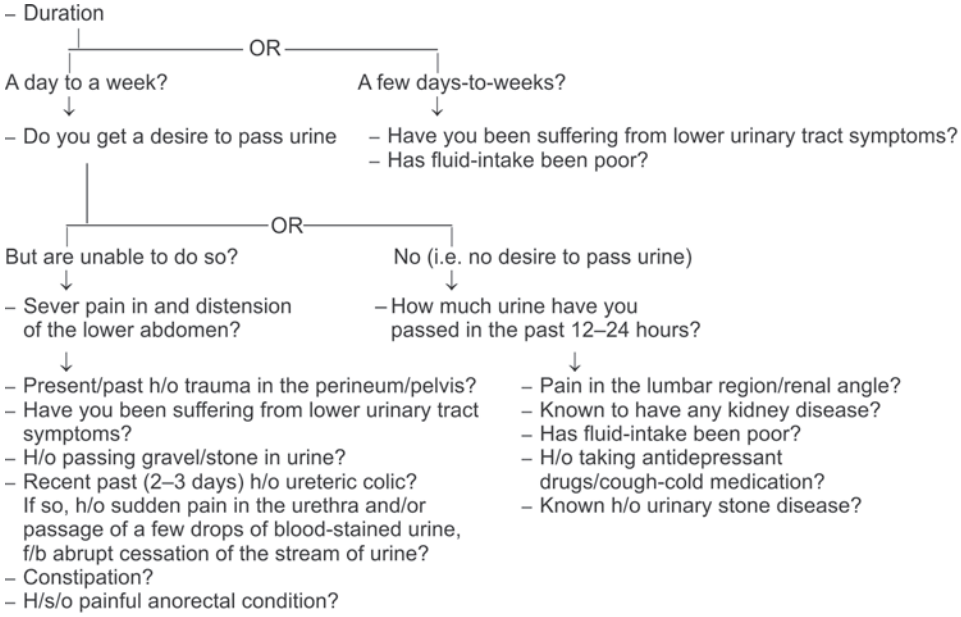
- Initial / terminal / total?
- Amount?
- Clots—plaque-like / thread-like?
  The presence of clots usually indicates a more significant degree of hematuria, and consequently the probability of identifying significant pathology increases.
- Does exercise initiate episodes of hematuria? ⇒ stone.
- Pain? Where?—in the lumbar region/renal angle, OR in the suprapubic region?
- Any lump in the abdomen? Where?—in the lumbar region/renal angle, OR in the suprapubic region?
- Are you suffering from any bleeding disorder?
- Have you been consuming anticoagulants?
- Have you been consuming medicines like quinine, sulpha, aspirin, etc?
- Did you consume beet root, vitamin B-complex, or any other unusual-colored food/medicine?

*Details to be asked in c/o **turbid** urine*:

- Is it 'initial'? i.e. is only the initial portion of urine passed during any act is turbid?
- Any predominant time at which it is more; particularly is it more in the morning?
- Does pus come out of urethra spontaneously without trying to pass urine (i.e. apart from micturition)?
- Severe burning (in the penis) during micturition?
- Difficulty in passing urine?
- Pain? Where?—in the perineum / mid-sacral region / suprapubic / lumbar?
- Any increase in frequency of micturition?—severe or otherwise?
- Fever? High grade with rigors, or otherwise?
- Lump? Where?—suprapubic / lumbar.

*Details to be asked in c/o h/o passing **gravel/stones***:

- Is this the 1st episode OR is this a recurrent episode?
- Details of the past episodes, if any?
- **'Not passing enough urine (*with regard to frequency / quantity / both*)**'?



Free ebooks ==> www.Ebook777.com

- **Incontinence**?
  - Duration?
  - Is it experienced since childhood?
  - Despite (continuous) dribbling, do you also get normal desire to void and consequently can void normally?
  - Does it occur through the urethra or somewhere else such as from the vagina, rectum or any abnormal opening?
  - Did it start spontaneously or did it follow an operation (prostate, pelvic) or did it follow difficult delivery?
  - Is it continuous? OR Does it occur periodically?

    ↓

    - Automatically, i.e. spontaneously at a regular frequency of ~ every 2–4 hours with complete evacuation?
      OR
    - Irregular small evacuations? Is evacuation assisted by manual pressure fruitful? H/s/o chronic retention?
      OR
    - Does it occur in the form of dribbling, the bladder being fully distended; following initial h/s/o retention?
      OR
    - Does it occur only on straining (coughing/laughing/sneezing, etc.)?
      OR
    - Does the patient seem to pass urine at inappropriate places, being unaware of wet clothes/linen? Incontinence of feces as well?
  - Any other urological symptoms?
  - H/o cancer of a pelvic organ?
  - Past h/o RT to the pelvis?
  - Present/past h/o injury/disease to the spine?
  - Any disease of CNS?

History to be asked in a **suspected c/o neurogenic bladder dysfunction**

- Acute and very severe low back pain?
- Have you been suffering from a chronic (degenerative) low back pain?
- Pain in the lower limb?
  - Extent? i.e. does it extend all the way down as far as the foot, or Does the pain stop short of the knee?
  - Is the pain increased by coughing or by movement?

  } ⇒ Radiculopathy.
- Paresthesias?
- Motor weakness?
- **Enuresis**?
  - Is it continuously present since birth? = Primary enuresis.
  - Has it recurred after being previously dry for sometime? = Secondary enuresis.

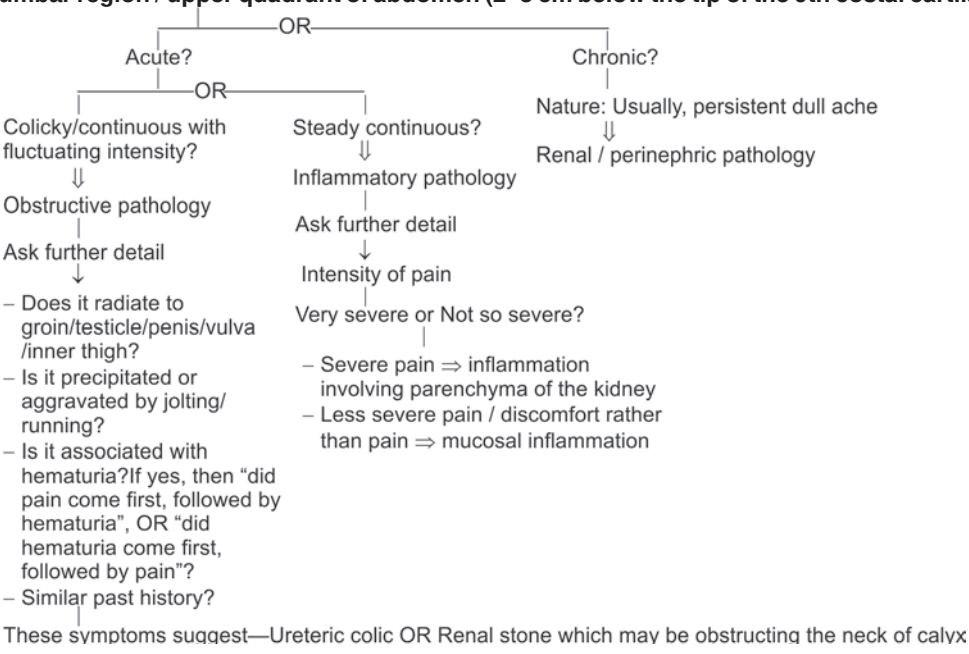
    ↓

    – Is there any psychological problem?
- **Hematospermia**?
  - Duration?
  - Associated with increased frequency/urgency of micturition?
  - Hypertension?
  - H/s/o urogenital TB?
  - H/s/o cancer?

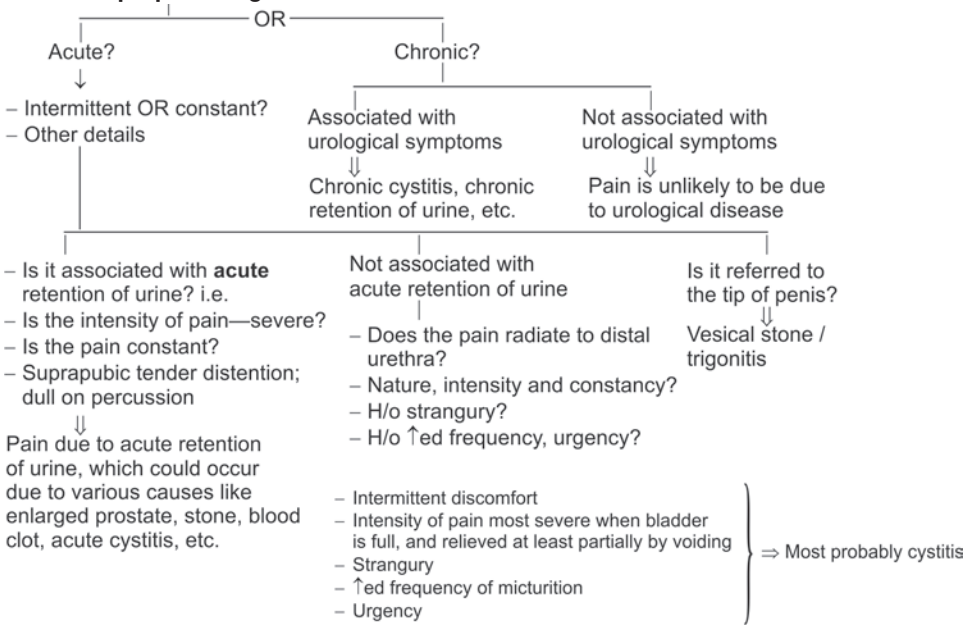
www.Ebook777.com

Abdominal symptoms

- **Pain in lumbar region / upper quadrant of abdomen (2–3 cm below the tip of the 9th costal cartilage)**?



- **Pain in suprapubic region**?



Sudden screaming and pulling at the prepuce during micturition by a child ⇒ bladder stone.

- **Lump**—lumbar / suprapubic?: (Also) Refer to the chapter 35—'Lump in abdomen'.

Free ebooks ==> www.Ebook777.com

Symptoms directly pointing at the external genitalia and perineum

Foreskin cannot be retracted behind the glans ⇒ Phimosis.

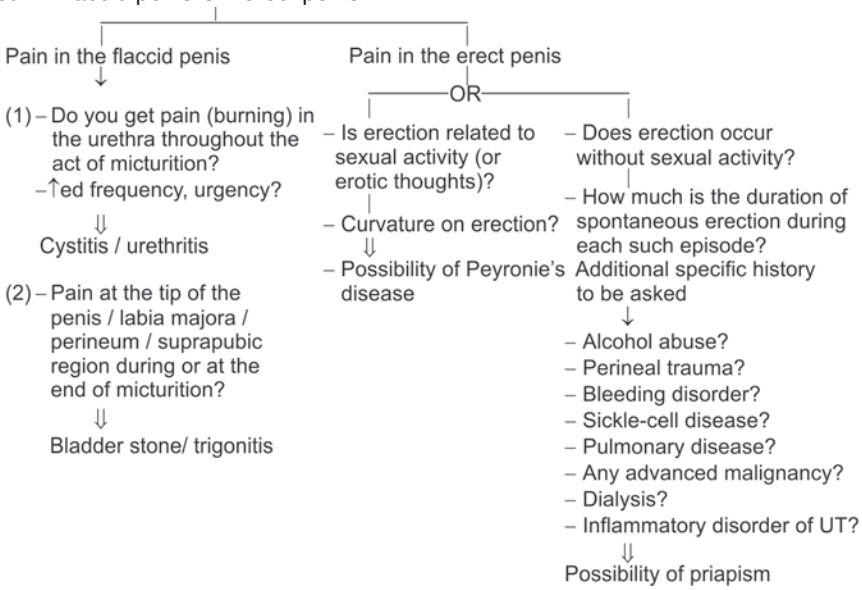
- Was it previously possible?
- Duration?
- Any pain in the penis?
- Any difficulty in passing urine? Does the foreskin get ballooned while passing urine?
- Acute painful and tender lump in the suprapubic region associated with inability to pass urine? (– indicating acute retention of urine)
- Discharge?
  - Details: Amount, blood-stained or otherwise, purulent, foul-smelling?
- DM?
  - Controlled / uncontrolled / not aware?
- Any swelling in the groin (indicative of lymphadenopathy)?
- Symptoms s/o uremia?

Acutely painful swelling of the retracted rolled-up prepuce ± the glans, and inability to reposition the retracted prepuce ⇒ Paraphimosis.

- Previously known to have phimosis? In other words, previously (i.e. before this episode developed), could the prepuce be retracted behind the corona with ease as for example while cleansing during bath, or during intercourse?
- Did you forcibly retract the prepuce yourself, OR was it retracted by someone else as for example by a professional health care provider, while either cleaning the prepucial sac, or introducing a catheter, etc; and then having forgotten to reposition the prepuce?

Pain in the penis

- Does it occur in flaccid penis OR erect penis?



A nonhealing progressively enlarging / progressively destructive (ulcerated) lesion on the glans / prepuce

- Duration, mode of onset, progress?
- Discharge?
- Difficulty in micturition?
- Any swelling in the groin (– indicative of lymphadenitis or metastatic lymphadenopathy)?

www.Ebook777.com

- Fever?
- Symptoms s/o distant metastatic disease?

Abnormal size of the penis

– Abnormally small size = *Micropenis*: Caused by fetal testosterone deficiency
– Abnormally big size—*Macropenis*: May be caused by interstitial cell tumor of the testis.

Congenitally abnormal position of urethral meatus ⇒ Hypospadias / epispadias.

- Is it on the undersurface or upper surface?
- Is it possible to urinate in standing position, though with some difficulty understandably?
- H/o undescended testicles (on both sides)?
- H/o any other congenital anomaly?

Passage of urine from abnormal opening/openings on the undersurface of the penis

- Since birth OR acquired later?—duration?
- Did the problem begin after a trauma / operation on the lower urinary tract / following bursting or I and D of a periurethral abscess?
- H/s/o acute gonococcal urethritis preceding an abscess on the underside of the penis?
- Long-standing h/s/o stricture of urethra?
- How much urine do you pass from the abnormal opening(s)?—A small amount OR most, practically all?

Scrotal symptoms

Refer to chapters 25, 26, 85.

***Nonspecific symptoms / effects** of (chronic) renal disease*

Symptom(s)/effects	*Cause(s)*	*Pathology/altered physiology*
– Anorexia, vomiting	CRF	-
– Anamia	CRF	Erythropoetin deficiency when GFR drops below 30%, Iron deficiency anemia
	Renal tubular acidosis	RTA → ↓ RBC survival
	Uremia	GI bleeding
– Weakness	CRF	Poor muscle mass
– Fever	Acute pyelonephritis,	
	Renal abscess	Release of inflammatory mediators
– Convulsions	Hypertensive encephalopathy	Acute poststreptococcal GN, renal parenchymal diseases, renovascular disease
– Sick look	CRF, UTI	-
– Drowsiness , lethargy	Uremia	-
– S/o electrolyte disturbances	CRF	Electrolyte disturbances

Symptoms of sexual dysfunction

Loss of libido

- Do you also have decreased or absence of ejaculation?
- How much is the semen volume—normal or decreased?

Impotence

- Duration, mode of onset, and progress?
- Any marital stress?
- Loss or change of sexual partner?
- Is the problem consistent?
- Can you achieve normal erections while masturbating / watching erotic pictures or videos?
- Do you get nocturnal / early morning erections?

Free ebooks ==> www.Ebook777.com

Failure to ejaculate

- How much is the semen volume—normal or decreased?
  Decreased seminal volume ⇒ androgen deficiency.
- Drug history: Alpha adrenergic antagonist?—may interfere with bladder neck closure at the time of orgasm and result in retrograde ejaculation
- Past h/o specific surgery?
  1. Sympathectomy / extensive retroperitoneal surgery, in particular retroperitoneal lymphadenectomy for testicular cancer. → interference with autonomic innervation of prostate and seminal vesicles → absence of smooth muscle contraction → absence of seminal emission at the time of orgasm.
  2. Surgery upon bladder neck/prostatic urethra, e.g. TURP may interfere with bladder neck closure at the time of orgasm and result in retrograde ejaculation.
- Are you diabetic?
  DM may spontaneously → retrograde ejaculation.

Lack of orgasm

- Are you diabetic?
- How is the penile sensation—normal or decreased?
- Are you on antipsychotic drugs?

Infertility

Clinical evaluation of only male factors is discussed here.

- Does your occupation involve working in a hot environment?
- Have you been taking medicines like for example nitrofurazone / cyclophosphamide / tranquilizer, etc?
- Did you undergo any operation upon the inguinoscrotal region?
- Did you suffer from mumps / TB / gonorrhea?
- Do you smoke tobacco / consume alcohol rather excessively?
- Any other addiction?

} These factors lead to suppression of spermatogenesis

- Sexual habits:
  - Frequency of intercourse?
  - Do you have an understanding of ovulatory cycle? Does the timing of intercourse coincide with proper timing of the cycle?
  - Does physiological intercourse take place with ejaculation occurring in the right place?
  - Do you use lubricants during intercourse? Some lubricants may have spermicidal effect.
- H/s/o sexual dysfunction?—refer to the section above.
- Previous infertility evaluation and treatment?
- Reproductive history from previous marriage if any?

Review of systems

- TB: May lead to impaired renal function, ureteral obstruction, chronic unexplained UTI
- HT: May lead to sexual dysfunction due to PAOD
- Antihypertensive medicines may → impotence
- Sickle-cell anemia: May lead to erectile dysfunction secondary to recurrent priapism
- DM: May lead to autonomic dysfunction → impaired urinary and sexual function
- Multiple sclerosis: May lead to urinary and sexual dysfunction
- Pre-existing detrusor hyperreflexia. Surgical treatment of bladder outflow obstruction in the presence of detrusor hyperreflexia may → increased urinary incontinence postoperatively.

H/o medications

Obtain accurate and complete list of medications.

- Most of the antihypertensives → erectile dysfunction
- Many psychotropic drugs → interference with emission and orgasm.

www.Ebook777.com

Past h/o any surgical procedure upon the urinary tract?
Previous operation upon the urinary tract may make subsequent surgery more difficult.

At this juncture it is worthwhile summarizing the symptoms and analyzing them quickly with respect to the possible site of disease and the possible pathological nature/altered physiology, so that physical examination can be focused.

Symptom	*Possible anatomical site / physiological system of disease*	*Possible (primary) pathology / altered physiology*
Increased frequency	Essentially LUT	– Inflammation (Infection) – Functional (1. Detrusor instability. 2. Reduced bladder compliance (due to old age, obstruction)) – Obstruction (→ reduced bladder compliance) – Incontinence
Polyuria	– Kidney, metabolic system – CNS	– (Physiological cause having been ruled out,) CRF, recovery from ARF, DM, DI, drug-induced – Injury, infection, tumor
Nocturia	– LUT – Kidney – Heart – Fluid compartment	– Causes: Refer to 'increased frequency' above – Significant chronic renal impairment – CHF – Old age
Urgency	LUT	This symptom is not fully understood, but is probably due to detrusor instability
Urge incontinence	LUT	Basically ⇒ severe degree of urgency
Hesitancy	LUT	– Obstruction (BOO most commonly due to BPE) – Functional (DSD)
Poor flow	LUT	– Obstruction – Functional (Old age (in both genders) – due to reduction in the power of detrusor muscle, detrusor decompensation secondary to chronic obstruction)
Feeling of incomplete emptying	LUT	– Low-grade inflammatory changes in the bladder commonly seen in BPE – Detrusor instability where 'after-contractions' may produce a desire to void
After-dribbling	LUT	– Weakness of bulbospongiosus – Could be due to urethral stricture
Urethral discharge	Urethra	Inflammation, cancer
Terminal pain while passing urine	– Bladder neck – Bladder – Lower end of ureter	– Stone, inflammation, cancer – Adhesions of bladder to surrounding structure(s) → dragging pain – Stone
Pain throughout the act of passing urine	– Urethra – Glans/prepuce – Bladder	– Inflammation – Inflammation – Inflammation, advanced cancer
Strangury	– Bladder neck – Lower end of the ureter (intramural portion) – LUT in general	– Obstruction – Impacted stone – Severe infection (inflammation)

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

Symptom	*Possible anatomical site / physiological system of disease*	*Possible (primary) pathology / altered physiology*
Acute pain in the perineum	LUT	Inflammation, stone, extravasation of urine.
Chronic pain in the perineum	– Urethra – Prostate	– Inflammation, stricture – Inflammation, cancer
Pain during defecation	Prostate, seminal vesicles	Inflammation
Retention of urine	LUT	Obstruction, inflammation, neurological including reflex spasm due to any painful pathology in the perineum/pelvis, trauma
Oliguria / anuria	– Essentially UUT – CNS (pituitary) – Lung – Heart – Other systems	– Parenchymal inflammation, obstruction, drug-induced – Inflammation, tumor, vascular lesion – Cancer / other pulmonary diseases – Failure – Shock, hemolytic uremic syndrome
Hematuria	– Anywhere along the UT from renal parenchyma to urethra – Adjacent organs – Blood – Miscellaneous	– A large variety of pathological processes – Inflammation, malignant infiltration – Bleeding disorder, leukemia – Drug-induced, gnathostomiasis (a worm infestation)
Pyuria	Anywhere along the UT	Infection
Chyluria	Lymphatics in calyces, pelvis, ureter, and at times in the urinary bladder	Basically, lymphatic hypertension presumably due to widespread fibrosis and obliteration of lymphatic vessels caused by filariasis. Episodic chyluria is produced by fresh attacks of lymphangitis (which probably produce periodic increase in lymphatic hypertension)
Passage of gas	– Intestine – Immune system	– Fistula – Infection with gas-forming organisms
Passage of fecal matter / worms	Intestine	Fistula
True incontinence	– Sphincter – Pudendal/pelvic nerves – CNS – Abnormal opening	– Injury during operation (e.g. prostatic adenomectomy), cancer – Injury during (a pelvic) operation – Complete destruction of spinal centers of micturition – Fistula (congenital / post-difficult labor / postoperative / post-traumatic)
False incontinence	– Sphincter – Bladder neck – Ectopic ureter – Nervous system	– Weakness (Stress incontinence) – BOO – In the female, ectopic ureteric orifice – Spinal cord lesions
Hematospermia	– Urethra, prostate, seminal vesicles, ejaculatory ducts – CVS – Miscellaneous	– Inflammation, stones, cyst – HT – Idiopathic

Contd...

www.Ebook777.com

Contd...

Symptom	*Possible anatomical site / physiological system of disease*	*Possible (primary) pathology / altered physiology*
Pain in the lumbar region	– UUT – Esophagus (Lower end) – Costal nerves	– Obstruction, inflammation – Inflammation – Compression, inflammation
Acute pain in the suprapubic region	Bladder	Obstruction, inflammation
Chronic pain in the suprapubic region	– Bladder – Nonurological organs	– Obstruction, inflammation
Loss of libido	Testes, pituitary	Androgen deficiency
Failure to ejaculate	– Bladder neck, prostatic urethra – Testes, pituitary – Autonomic NS – Metabolic system – Miscellaneous	– Postoperative (→ retrograde ejaculation) – Androgen deficiency – Postsympathectomy / postextensive retroperitoneal surgery (→ absence of seminal emission) – DM (→ retrograde ejaculation) – Drug-induced (→ retrograde ejaculation)
Premature ejaculation	Mind	Almost always psychogenic
Absence of orgasm	– Metabolic system – Mind – Miscellaneous	– DM – Psychogenic – Drug-induced

In GENERAL EXAMINATION, specifically look for the following:

- Drowsiness/marked delirium—may suggest uremia
- Dry tongue covered with brown fur ⇒ uremia.

ABDOMINAL EXAMINATION (including examination of lump): Refer to chapters 33, 34, 35.

EXAMINATION OF PENIS

In a case suspected of having **paraphimosis**

Look for – Engorgement of the glans due to vascular congestion
– Tightly constricting ring of foreskin, which has been retracted behind the corona
– Severe tenderness.

While inspecting the **penile skin in general**,

Look for – Superficial vesicles indicating herpes simplex
– Venerial warts (condylomata acuminata)—irregular, papillary velvety lesions
– Ulcers—could be due to venereal infection or tumor.

In **suspected priapism**

- Inspect the glans with respect to the shaft. Does the glans look erect or flaccid?
- Palpate the shaft. Feel for tenderness and consistency.

Examine (Inspect and palpate) **the penile and bulbar urethra** (undersurface of the penis, and perineum) for

- (Periurethral) Abscess
- Stone—felt as a hard fixed or slightly mobile nodule
- Stricture—felt as a hard fixed nodule
- Fistula(e).

Free ebooks ==> www.Ebook777.com

In **suspected** c/o **hypospadias / epispadias**
– Note the exact position of the urethral meatus
– Look for other congenital anomalies.

Palpate the dorsal aspect of the shaft.
Look for fibrous plaques/ridges—they indicate Peyronie's disease.

Retract the foreskin.
Examine:
1. The glans and prepuce for balanoposthitis, tumor (most commonly squamous-celled carcinoma). Morphological types of cancer: (a) Velvety raised lesion on the glans or inner surface of the prepuce. (b) Ulcerative lesion.
2. Urethral meatus for abnormality of position (hypospadias / epispadias).
3. Separate the lips of urethral meatus between thumb and index finger. Look for redness (urethritis), tumor (papilloma), and any stone in the navicular fossa.

Inability to retract the foreskin = Phimosis.
Criterion of 'tight phimosis', which is an absolute indication for (urgent) circumcision: Ballooning of the prepucial sac by urine.

Palpate the prepuce and glans (glans—from outside, as the prepuce cannot be retracted). Look for:
– Prepucial swelling/thickening and any swelling of the glans
– Note the shape, surface and consistency of such a thickening/swelling
– Irregular shape and surface, and induration ⇒ malignancy OR **chronic** balanoposthitis. Stony hard consistency would go more in favor of malignancy than balanoposthitis.

Phimosis accompanied by bloody discharge (in adult patient) may be due to cancer of the penis (prepuce/glans). – Dorsal slit / circumcision with biopsy at the earliest is indicated in such a situation to relieve difficulty (occasionally retention of urine) and prove/rule out cancer.

EXAMINATION OF PERINEUM

Look for abscess, extravasation, fistula.

EXAMINATION OF SCROTUM: Refer to chapters 25, 26, 85.

SYSTEMIC EXAMINATION

Specifically look for
– Pulmonary TB. (However, remember that pulmonary TB may have healed by the time genitourinary TB presents)
– Metastases to chest (from genitourinary cancer)
– Infective endocarditis, which may cause infarction of kidney (and cause hematuria)

Do not forget to examine the nervous system, vertebral column, and parathyroid.

PELVIC EXAMINATION IN A SUSPECTED C/O VESICOVAGINAL FISTULA

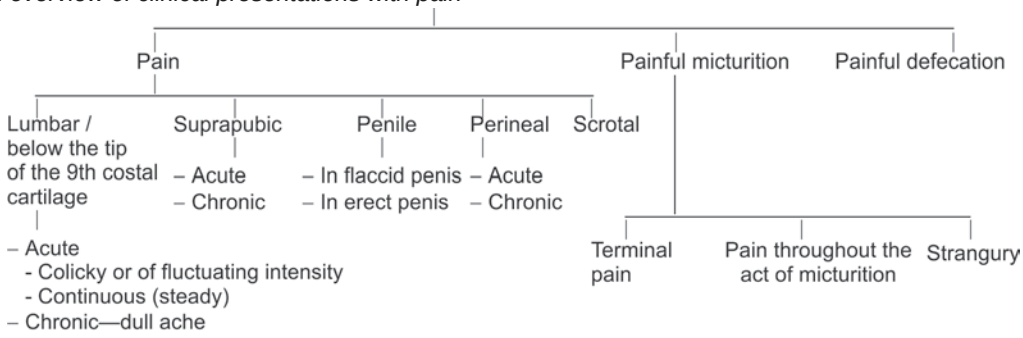
Look for the following findings:
– On inspection: Excoriation of the vulva
– On PS/E: Urine escaping from an (abnormal) opening in the anterior vaginal wall or the vault
– On digital examination: A localized thickening on the anterior vaginal wall or the vault. If big enough, actual opening of the fistula may also be felt.

A vesicovaginal fistula may have to be differentiated from a ureterovaginal fistula.
Clinical method to differentiate:
– Place a swab in the vagina
– Instill a solution of methylene blue into the bladder via a catheter
– Remove and observe the vaginal swab after a while
– Staining of the swab confirms the diagnosis of vesicovaginal fistula
– The absence of staining of the swab suggests ureterovaginal fistula.

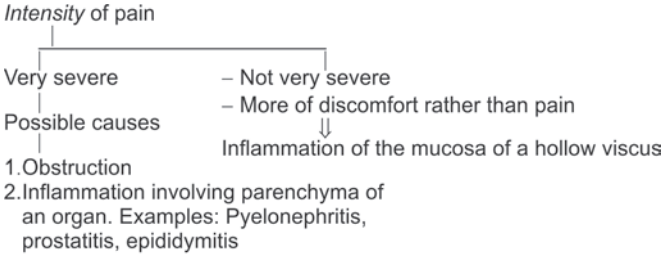
www.Ebook777.com

PRINCIPLES AND PITFALLS

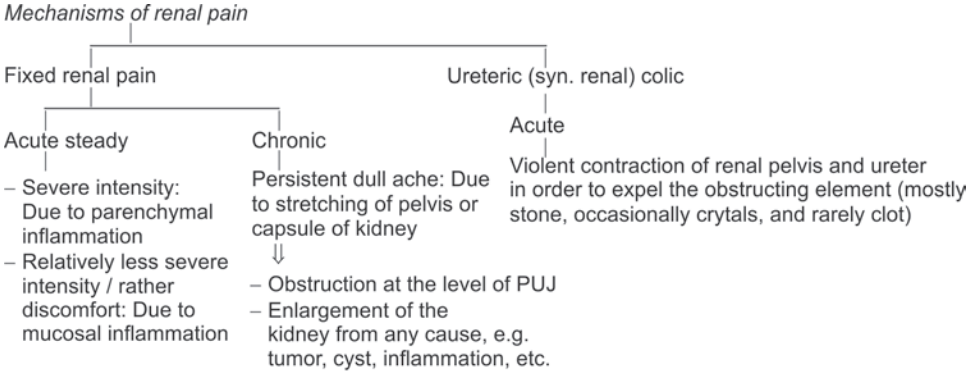
- **Analysis of pain**
  An overview of clinical presentations with pain



Usual causes of urological pain: Obstruction and inflammation.



Mechanism of pain caused by inflammation: Inflammatory edema → stretching (distension) of capsule of the organ → pain.



Mechanisms of vesical pain: 1. Overdistention of bladder due to **acute** retention. 2. Inflammation.

- **Abacterial pyuria in urine of acidic pH—**almost always means urinary TB.
  Nevertheless, a small group of cases may be nontuberculous. Special investigations in these patients fail to reveal *Mycobacterium tuberculosis*. Cystoscopy reveals a severe cystitis. Occasionally amebiasis is responsible.
- Diverticulum of the seminal vesicle is one of the causes of **persistent urethritis**

Free ebooks ==> www.Ebook777.com

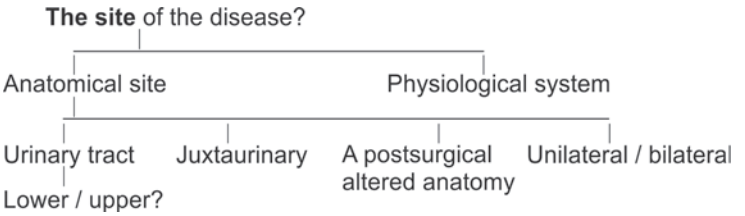
- Almost three quarters of all **women suffering from gonorrhea** attend initially as a result of the diagnosis of their male partners, and have neither symptoms nor significant signs
- In the **young prepubertal girls, a primary gonococcal vaginitis** can occur. It may be transmitted by the use of infected mother's towel.

STEPWISE ANALYSIS OF DATA

Step 1

Acute / chronic?

Step 2



Step 3

Pathological nature of the disease?

- *Congenital anomaly*—produces its effects by one/more pathological processes mentioned below
- *Abnormal position*: Ectopic kidney, ptosis
- *Abnormal size:*
  - Reduction in size (e.g. due to atrophy)
  - Enlargement: Could be due to some of the following
- *Distension / dilatation*: Hydronephrosis
- *Congenital swellings,* e.g. polycystic kidneys, hamartoma
- *Collection* (of fluid, blood, pus, etc.): Cyst, pyonephrosis
- *Inflammation* / end *result of inflammation* (suppuration / ulceration / necrosis / fibrosis)
- *Infection*: Nonspecific / specific
- *Tumor*
- *Obstruction*:
  - Continuous / intermittent
  - Structural / functional
  - Complete / incomplete
  - Degree of its effect: The degree of loss of physiological function
- *Stone formation*
- *Functional*: Reflux, reduction in power of muscle which may be age-related, detrusor-sphincter dyssynergia.
- *Fistula formation*
- *Incontinence*
- *Injury*
- *Presence of foreign body*
- **One or more** of the above-mentioned pathological processes may occur in **combination**
- **There can be an interplay between various pathological processes with respect to cause and effect**, for example:

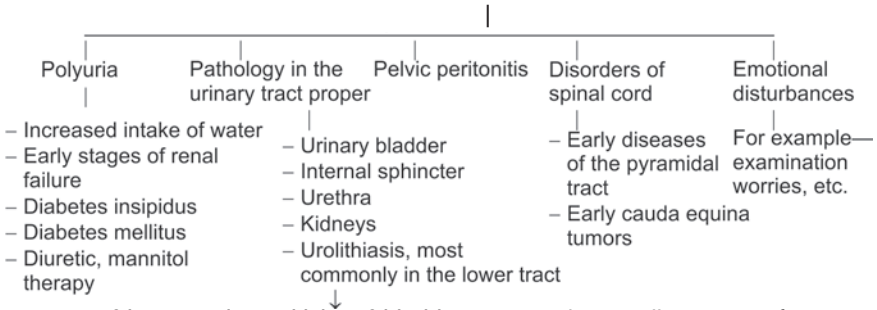
– Obstruction predisposes to stone formation
– Stone can lead to obstruction
– Stone → obstruction → stasis → infection
– Infection predisposes to stone formation, etc.

Hence, do not be satisfied on finding one pathology. Try to detect as many as possible.

www.Ebook777.com

(INCREASED) FREQUENCY OF MICTURITION

Causes of increased frequency of micturition



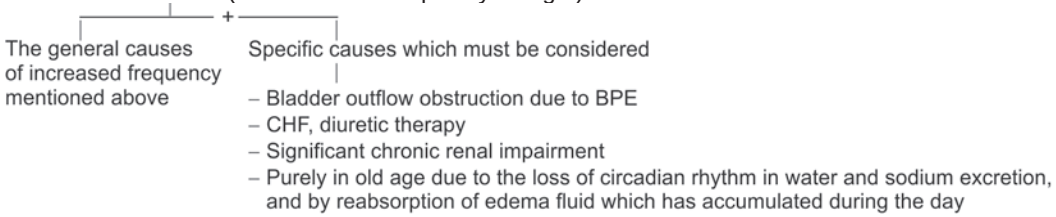
Causes of increased sensitivity of bladder mucosa (∴ Small quantity of urine will start the voiding reflex): Cystitis, ulcer, cancer, decreased capacity of bladder as in tubercular cystitis or after partial cystectomy
Causes of incompetent internal sphincter: Enlargement of prostate, stone impacted in the internal meatus
Mechanism of obstructed urethra causing increased frequency of micturition: Because of obstruction *per se*, and severe straining that is required to pass urine, evacuation of bladder remains incomplete during each act of micturition → 'residual urine'. A small quantity of newly excreted urine will add to the residual urine shortly to make up 300 mL of urine in the bladder → initiation of voiding reflex
Common causes of obstruction of urethra: Enlarged prostate, stricture
In the later stages, when bladder becomes atonic and fully distended, 'overflow incontinence' will ensue

Renal causes of increased frequency: Pyelitis, tuberculosis of kidney.

Disorders of spinal cord:

1. Early diseases of the pyramidal tract. Mechanism: Ability to inhibit micturition by the brain depends on continuity of the pyramidal tract. In (early) diseases of the pyramidal tract, this ability is lost. Consequently, patient may have to run to pass urine even before less than 300 mL of urine is collected in the bladder.
2. Cauda equina tumor. In early stages, irritation of pelvic nerves → increased frequency. In late stage, incontinence follows.

Causes of nocturia (i.e. increased frequency at night)



Nocturia caused by bladder outflow obstruction can be easily differentiated from the rest of the causes by keeping a frequency-voiding chart for a few days.

Polyuria

Causes of polyuria	*Pathological process / altered physiology*
– (Psychogenic) Polydipsia, excessive infusion of IV fluids	Physiologic response to fluid overload
– CRF due to glomerular and/or tubular affection	Augmented Na excretion due to relatively narrow range of urine osmolality which necessitates a large daily urine volume to excrete solute load
– Recovery from ATN, diuretic therapy	Diminished tubular reabsorption
– Diabetes insipidus*—central or nephrogenic	Diminished ADH secretion or diminished sensitivity to ADH
– Diabetes mellitus, mannitol administration	Excessive solute overload
– Cold weather	to compensate for decreased fluid-loss from diminished sweating

**Causes of*
– Central DI: Idiopathic, hypophysectomy, head injury, tumor, sarcoid, infection
– Nephrogenic DI: Hypokalemia, hypercalcemia, obstructive uropathy, analgesic abuse, pyelonephritis, drugs.

Free ebooks ==> www.Ebook777.com

POOR FLOW

⇒ bladder outflow obstruction most commonly due to enlargement of prostate, **provided 2 conditions are ruled out -** 1. Detrusor hypotonia which commonly occurs with aging, and 2. Severe frequency and/or urgency significantly reducing the functional bladder capacity. Low volume of voided urine logically reduces the flow.

PAINFUL MICTURITION, i.e. pain while passing urine

*Causes of **terminal** pain*

1. Pathology in the bladder neck (including prostate): Stone, trigonitis, tuberculosis, malignancy, acute prostatitis, acute prostatic abscess.
2. Stone impacted in the lower end of ureter.
3. Adhesion of bladder to surrounding structure → dragging pain.

*Causes of pain **throughout the act of micturition***

1. Pathology in urinary bladder: Cystitis, advanced cancer.
2. Urethritis. Pain typically scalding in nature.
3. Meatal ulcer.
4. Balanitis / balanoposthitis.

*Causes of pain **referred***

– To the tip of the penis/labia majora/perineum: Stone in the bladder (causing irritation of trigone) / trigonitis.
– To the suprapubic region: Inflammation of the dome of the bladder.

Pain relieved after micturition ⇒ urethritis, cystitis.

On **3-glass urine test**

– Cloudy/turbid urine in the second/third glass confirms cystitis
– The presence of 'threads' in the first glass ⇒ prostatitis

Causes of **strangury**

– Obstruction of the bladder neck, e.g. by a stone, cancer
– A stone impacted in the lower end (intramural portion) of the ureter
– Severe urinary infection.

Urological causes of **PAINFUL DEFECATION**

– Acute inflammatory conditions of prostate / seminal vesicle / bladder neck

Examples—acute prostatitis, prostatic abscess, (acute seminal vesiculitis)

RED / BROWN / STALE TEA-COLORED / SMOKY URINE = HEMATURIA

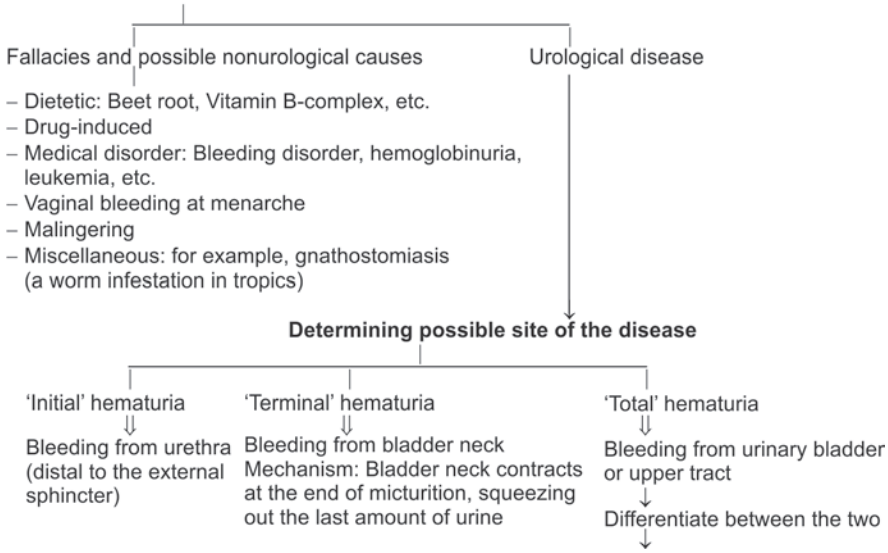
On **3-glass urine test**

1. – Blood in only the first glass ⇒ 'Initial' hematuria
– Blood in only the last glass ⇒ 'Terminal' hematuria
– Blood in all the three glasses ⇒ 'Total' hematuria
(Logically, it is not possible to get blood in only the second glass!).

2. Pour the urine in a flat tray containing water and examine the clots and also look for pieces of growth (if found, send for histological examination).

www.Ebook777.com

Causes of hematuria



Feature	*Urinary bladder*	*Upper tract (kidney-pelvis-ureter)*
Color of urine	(Bright) Red	Smoky (or dark red)
Clots	Plaque-like	Thread-like (worm-like) suggest that the clots are formed in the ureter
Amount	'Moderate-to-severe'	'Slight-to-moderate'
Site of pain, if any	Suprapubic	Lumbar
Lump, if any	Suprapubic	Lumbar

Site of the disease	**Pathology**
– *Urethra* distal to rhabdosphincter	Stone, tumor (papilloma, angioma), infection, external mechanical trauma, instrumentation
– Bladder neck	Stone, tumor, infection, trauma, enlarged prostate
– *Urinary bladder*	Stone, tumor (papilloma, carcinoma, etc.) Infection (cystitis, diverticulitis, bilharziasis) External mechanical trauma, instrumentation Varicosity (bladder piles) Bleeding disorder
– *Kidney-ureter*	Stone Tumor (papilloma, renal cell carcinoma, nephroblastoma, etc.) Inflammation (acute nephritis, chronic interstitial nephritis, TB) Leak of RBCs from glomerular filtration barrier (glomerulonephritis) Trauma Congenital disorder (polycystic disease) Infarct, collagen disease Dietl's crisis Drugs: Quinine, sulfa, aspirin, anticoagulants, etc.
– *Adjacent organs*	Inflammatory or malignant disorder, e.g. acute appendicitis, salpingitis, pelvic abscess, CA rectum/uterus

***Clinicopathological correlations** worth remembering:*

– Profuse bleeding suggests papilloma/angioma; however, sometimes may occur in polycystic kidney
– Exercise precipitating episodes of hematuria ⇒ stone

Free ebooks ==> www.Ebook777.com

– Painless hematuria is a distinct clinical entity and is usually associated with tumor (papilloma, carcinoma etc.)
– Colicky pain (lumbar / loin-to-groin) preceding hematuria ⇒ ureteral stone
– Hematuria preceding colicky pain (lumbar / loin-to-groin) ⇒ 'clot colic'
– Causes of increased frequency: Stone, early renal tuberculosis, cystitis, BPE.

A note on hematuria

– Gross v/s Microscopic hematuria: The significance is simply that the chances of identifying significant pathology increases with the degree of hematuria. Thus, the chances of identifying significant pathology are more in a case presenting with gross hematuria as compared with the case presenting with microscopic hematuria.
– Hematuria of any degree should **never be ignored**, and evaluated thoroughly until diagnosis is reached
– Hematuria, particularly in the adult, should be regarded as a symptom of cancer until proved otherwise. The most common cause of gross hematuria in a patient over 50 years old is bladder cancer.

PURE BLEEDING PER URETHRA INDEPENDENT OF MICTURITION

– May suggest carcinoma of the urethra.

CLOUDY URINE

Clarify as to whether it is turbid, or white-colored

– Turbidity ⇒ infection
– White-colored urine ⇒ (otherwise normal) alkaline urine OR chyluria.

Alkalinity predisposes precipitation of phosphates, which in turn impart white color to urine.

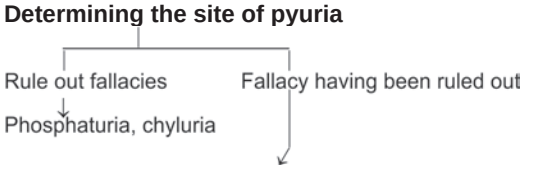
TURBID URINE

Most common cause: Pus in the urine.

Relevant points in **EXAMINATION**

- Tenderness in lumbar region OR suprapubic region?
- Lump in lumbar region OR suprapubic region?
- Tenderness on the undersurface of penis (i.e. along the urethra)?
- Redness of the external urethral meatus?
- Milking of penis → any flow of pus per urethra?
- DRE: Prostatic tenderness?
  Prostatic massage → any flow of pus per urethra?

Determining the site of pyuria



1. – 'Initial' pyuria, i.e. turbidity essentially at the beginning of micturition
– More often in the morning
– Pus may flow spontaneously per urethra, apart from micturition
– Severe burning during micturition
– Redness of external urethral meatus
– Tenderness on the undersurface of penis (i.e. along the urethra)
– Milking of urethra may bring out pus
⇒ the site of pathology—**urethra**.



Fig 54-1 Pus per urethra

www.Ebook777.com

2. – Difficulty in micturition
   – Pain in the perineum/mid-sacrum
   – On DRE: Tender prostate. Prostatic massage may bring out pus
   ⇒ the site of pathology—**prostate**.
3. – 'Severe' frequency of micturition
   – Slight fever ±
   – Tenderness in suprapubic region
   – Lump if present, in suprapubic region
   ⇒ the site of pathology—**urinary bladder.**
4. – Increased frequency of micturition present, but not as 'severe' as in the case of pathology of urinary bladder
   – Fever—high grade with rigors
   – Tenderness in lumbar region
   – Lump if present, in lumbar region
   ⇒ the site of pathology—**kidney/pelvis/ureter**.

WHITE-COLORED/MILKY URINE

Causes: Phosphaturia (due to alkaline pH), chyluria.

Chyluria

Basically, passage of milky white opalescent urine. Urine on standing may form a jelly-like clot.

– 20–50 years. Men > women
– Episodic. Mode of onset—sudden, often heralded by an attack of fever with rigors; duration of each episode—weeks/months. Episodes subside spontaneously
– Total duration of the history may be anything from a few months to about 10 years or so
– May be associated with hematuria with its sequelae like clot colic, retention of urine, etc.
– Pain in abdomen, backache
– Weakness, considerable loss of weight (due to marked albuminuria, anemia)
– Other clinical manifestations of filariasis may be present, e.g. chronic epididymo-orchitis, tunica vaginalis hydrocele, inguinal lymphadenitis, very rarely elephantiasis of lower limbs

Most commonly caused by filarial infestation.

Causes other than filariasis: TB, trauma, retroperitoneal tumor causing rupture of lymphatics above the level of cisterna chyli.

Causes of **HYDATIDURIA**

– Hydatid cyst of the kidney
– Rarely, a cyst in the pelvis may adhere to the urinary bladder and develop a fistula.

'NOT PASSING ENOUGH URINE (With regard to frequency / quantity / both)'

This symptom relates to 3 different clinical conditions: Oliguria, anuria, and retention of urine (acute/chronic).

Relevant points in **EXAMINATION**

- Does the patient appear restless?
- Is the bladder palpable (per abdomen)?

With reference to catheterization of bladder (↓ *strict aseptic precautions)*

– Any difficulty during catheterization?
– Amount of urine drained?

Free ebooks ==> www.Ebook777.com

INTERPRETATION AND DISCUSSION

- Complete/near-complete inability to pass urine in conjunction with a palpable bladder ⇒ Retention of urine
- H/o not having passed urine (almost at all) for 12 hours in conjunction with impalpable bladder ⇒ Anuria
- H/o having passed small quantity (< 100–400 mL) of urine in 24 hours in conjunction with impalpable bladder ⇒ Oliguria. This clinical situation is usually met with practically in inpatients often in postoperative period.

Clinical features	*Anuria*	*Acute retention*	*Chronic retention*
– Site of pain and tenderness	Lumbar region and renal angle	Suprapubic	Often painless
– Restlessness	Absent	Present Patient cries in agony constantly begging to be relieved	Absent Patient may be absolutely ignorant about the condition, and may look cheerful even with the bladder distended up to umbilicus
– Drowsiness	±	-	May be present due to uremia
– Bladder	Not palpable	Palpably enlarged	Palpably enlarged (Fig 35-2)
– Catheterization	No difficulty	May be difficult	May be difficult
– Amount of urine drained	< 30 ml	Large, often > 1 l	Large, often > 1 l

Causes of oliguria/anuria	*Pathological process / Altered physiology*
– Hypovolemia, shock, CHF	Hypoperfusion of kidneys → ↓ ed GFR
– AGN, CGN, hemolytic uremic syndrome, etc.	Diminished filtration due to glomerular block
– Obstructive uropathy	Obstruction to the free flow of urine
– SIADH	Excessive secretion of ADH

Suppression of urine is seldom complete (anuria), except in postrenal (i.e. ureteric) obstruction.

Causes of **retention** of urine **(etiological perspective)**

Mechanical

- Phimosis, (rarely) paraphimosis
- Urethral: Rupture, pinhole meatus, acute periurethral abscess, acute urethritis, stricture, stone, tumor, foreign body, posterior urethral valves, spasm of a stretched urethra caused by an incarcerated ovarian cyst
- Prostatic: Enlarged prostate, prostatitis, prostatic abscess, prostatic stones
- Vesical: Rupture, stone, tumor, blood clot
- Pressure on the bladder outlet from outside: for example, fecal impaction in the rectum, retroverted gravid uterus, impacted fibroid, cancer of the cervix/rectum, pelvic growth

Neurological

- Following operations in the perineal region or lower abdomen including groin
  Mechanisms: 1. Reflex spasm of the urethral sphincters due to pain, 2. Hesitation arising out of compulsion to pass urine in an unaccustomed posture and/or environment
- Spasm due to acute painful anorectal conditions
- Transverse myelitis, disseminated sclerosis, tabes dorsalis
- Fracture-dislocation of spine, Pott's disease
- Cauda equina compression, e.g. from a central disk prolapse

- Persistent retention following resection of the rectum. In some patients, retention may persist for 3 months or more
  Mechanism: Sagging of the bladder into the space formerly occupied by the rectum

www.Ebook777.com

A guideline to determine the cause of retention of urine

The most probable cause to be considered at the outset
- In children: Phimosis, pinhole meatus, posterior urethral valves, vesical stone
- In young adult: Acute urethritis.
- In adult: Stricture of urethra*
- In old age: BPE**
- In women: Retroverted gravid uterus, fibroid, ovarian cyst, CA cervix

If previous h/o 'difficulty in micturition' (lower urinary tract symptoms/stone disease)
OR
Recent past (2–3 days) h/o ureteric colic
- Stone in the bladder obturating the bladder outlet
- Migratory stone in the urethra
- Stricture of urethra*
- BPE

Think about the possible cause based on the specific clinical background
- Trauma, e.g. fall astride on the perineum, fracture of pelvis, etc.
- Pregnancy
- Patient having been operated upon the perineum / lower abdomen
- Acute painful anorectal condition
- Acute exacerbation of low back pain†
- Neurogenic bladder dysfunction††

***** A stricture is seldom complete; acute retention occurs when congestion and spasm supervene.
****** BPE often causes chronic retention.
† In the context of known degenerative low back pain the commonest cause of inability to pass urine is an acute exacerbation of the pain. Bladder sensation is preserved, but patients find it difficult to pass urine because straining increases their pain—whether in the low back, referred or radicular.
†† Cauda equina compression, e.g. from a central disk prolapse, is a much-feared cause of bladder dysfunction because the diagnosis is often made too late. The nerve roots are exquisitely sensitive to compression, and if this is allowed to go on for > 24 hours after sphincter control has been lost then even prompt decompressive surgery is unlikely to restore lost bladder and bowel and sexual function.

- The hallmark of neurogenic bladder dysfunction is painless retention of urine, associated almost always with perineal sensory disturbance (usually bilateral)
- Low back pain is very common, as are disorders of pelvic musculature in women and prostatic symptoms in men, so the potential for overlap for these potential causes of bladder dysfunction is great
- During the examination of a **suspected c/o neurogenic bladder dysfunction**, look for areflexia and perineal sensory loss.

INCONTINENCE OF URINE

Incontinence without normal voiding
Leakage usually per urethra
Usually suggests failure of the sphincter mechanism

Continuous dribbling despite getting normal desire to void and consequently being able to void normally
⇓
Fistula bypassing the normal sphincter mechanism
- Dribbling per urethra ⇓ Opening of an ectopic ureter beyond the rhabdosphincter—usually in females
- Dribbling per vaginum ⇓ Vesicovaginal fistula

- **True incontinence**: Urine dribbles continuously and bladder is (usually) empty
- However, in a female having an **ectopic ureter**, the ectopic ureter opens either into the urethra distal to the rhabdosphincter, or into the vagina, and causes intractable incontinence. Classical history in a girl or a woman: Intractable incontinence of urine for as long as she can remember, despite the fact that she has a desire to void, and does urinate
- **False incontinence**: Urine dribbles involuntarily through urethra periodically but bladder does not become completely evacuated; small or large amount of residual urine remains
- **Incontinence should not be mistaken for increased frequency** of micturition, which is associated with expulsive act of passing urine

Free ebooks ==> www.Ebook777.com

Causes of 'true incontinence'

- Per urethra
  - Injury to sphincter during operation on prostate
  - Paralysis of sphincter due to injury to pudendal nerve and/or pelvic nerves
  - Severe epispadias
  - Extension of malignancy
    Mechanisms: 1. CA-cervix, prostate, etc. invading the sphincter mechanism causing incontinence
    2. Fistula formation
  - Complete destruction of spinal centers of micturition
- Through an abnormal opening
  - Congenital ureterovaginal fistula
  - Vesicovaginal or urethrovaginal fistula following trauma/operation/difficult labor
  - Ectopia vesicae

Causes of false incontinence

1. **Automatic bladder** (Reflex bladder)

Causes:

– Normal in infants

– Lesion above the lumbar segment of spinal cord

Pathophysiology: When the urinary bladder gets filled with certain amount of urine, detrusor contracts reflexly and unconsciously from time to time (roughly every 2 to 4 hours). Sensory impulses reach the spinal centers for micturition but do not reach to level of consciousness in the cerebrum. These sensory impulses periodically stimulate the motor centers in the sacral segments → reflex contraction, and almost complete evacuation of bladder results.

2. **Autonomous bladder** (Isolated bladder)

– Clinically, there is chronic painless distension of bladder with irregular small evacuations and a large amount of residual urine leading to hydroureter, hydronephrosis and infection

– Evacuation can be assisted by manual pressure

Pathophysiology: Bladder is completely cut off from the CNS, and depends for its activity on single neurone reflexes through ganglia situated in the bladder wall. This gives some tone to the bladder muscle and makes it capable of some functional activity.

3. **Overflow incontinence**

Causes:

– Bladder outflow obstruction

– Transection of spinal cord due to any lesion leads immediately to retention followed by overflow incontinence.

This is a temporary phase and will proceed to either automatic or autonomous bladder.

Pathophysiology: Bladder becomes fully distended as there is no muscle tone at all. Overflow incontinence producing dribbling of urine with more or less fully distended bladder results.

4. **Stress incontinence**

5. **Urge incontinence**

a. Simple cystitis will often be sufficient, particularly in women, to induce incontinence.

b. Sometimes thyrotoxicosis may → frequency and incontinence.

6. **Frontal lobe incontinence**

Patient passes urine at inappropriate places, and has no idea about his immediate surroundings, unaware of wet clothes and bed linen. May also be accompanied by incontinence of feces.

Causes: Frontal lobe tumors, cerebral lesions with mental confusion.

ENURESIS

- Primary
  - Normal up to the age of 3 years, but persists in about 15% children up to the age of 5, and in about 1% children up to the age of 15, without any significant detectable urological abnormality
- Secondary
  - In children
    - Psychogenic
  - In teenagers and young adults
    - Very deep sleepers
    - Unstable bladder contraction

www.Ebook777.com

BLADDER OUTFLOW OBSTRUCTION

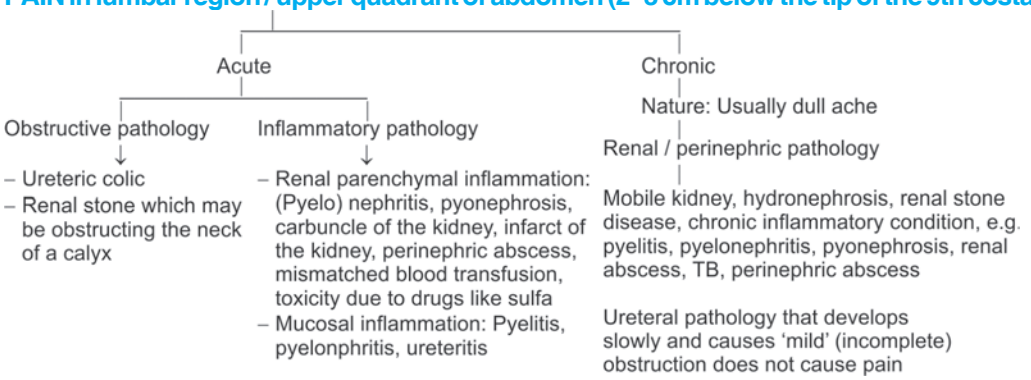
Differentiating points: H/o hesitancy and/or poor flow OR presentation with acute/chronic retention of urine
Additional symptoms which may be present: Frequency, urgency
Complication: Back pressure changes → hydroureter, hydronephrosis, renal failure, uremia
Causes of bladder outflow obstruction: BPE / BNH / BNC / carcinoma of prostate / urethral stricture / stone impacted in the urethra / sphincter spasm caused by neurological disease especially multiple sclerosis / Fowler's syndrome (excess rhabdosphincter activity in young women).

Factors which may help in fixing the cause of bladder outflow obstruction:
- Age
- H/o 'after-dribbling' present or absent
- Clear-cut h/s/o etiology of urethral stricture present or absent
- Physical findings.

HEMATOSPERMIA

Causes
1. Pathology of urethra / prostate (/ seminal vesicles) / ejaculatory ducts. Examples—nonspecific inflammation, TB, stones, cysts, etc.
2. Severe HT.
3. Unknown.

PAIN in lumbar region / upper quadrant of abdomen (2–3 cm below the tip of the 9th costal cartilage)



Causes and differentiating features of **acute colicky pain in lumbar region**
1. *Calculous colic* (caused by a ureteral stone)
   - Pain restricted to the loin ⇒ the stone just coming out of the pelvis
   - Pain radiating from loin to the lower quadrant of abdomen, and sometimes even down to the level of scrotum / labium ⇒ obstruction of the mid-ureter
   - Pain radiating along the urethra to the tip of penis or inner thigh ± symptoms of vesical irritability (increased frequency, urgency, suprapubic discomfort) ⇒ obstruction of the lower ureter
   - May be precipitated/aggravated by jolting/running
   - Pain comes first, which may be followed by hematuria. Hematuria is slight
   - Pain from 'loin to groin' may be caused apart from obstructive pathology of the ureter, by rupture of AAA, provided other clinical features agree.
2. *Crystal colic*: caused by the passage of a shower of oxalate crystals, e.g. after eating excess of strawberries.
3. '*Clot colic*'.
   - Rare
   - May occur on the background of operated kidney
   - Hematuria occurs first, then followed by pain.
4. ***Acute colicky pain may also be encountered in hydronephrosis*** (Dietl's crisis) ***and pyonephrosis***.

Free ebooks ==> www.Ebook777.com

Differential diagnosis of lumbar pain

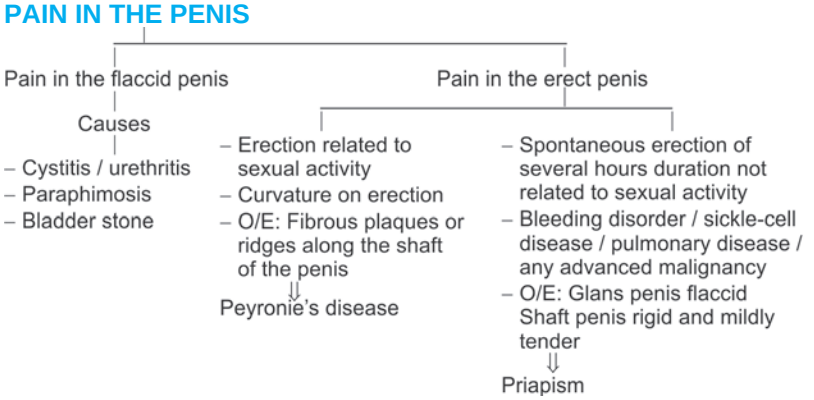
1. *Intraperitoneal pathology*
   - Patient preferring to lie motionless to minimize pain ⇒ intraperitoneal pathology.
   - Patient who may be more comfortable moving around and holding the flank is more likely to have a renal pathology.
2. *Radicular pain* (due to irritation of costal nerves, most commonly T_{10}–T_{12})
   - Hyperesthesia of the overlying skin, and alteration of intensity of pain due to changing position ⇒ radicular pain
   - Intensity of pain arising from renal pathology should not alter with changing position. Moreover, if renal pain is caused by obstructive pathology, then the pain will be colicky in nature.
3. *Pain experienced during the pre-eruptive phase of herpes zoster involving any of the segments between T11 and L2* may simulate pain of renal origin. Hyperesthesia of skin may favor the diagnosis against renal disease.

The side of renal pain does not necessarily imply the side of disease. Pain could be due to physiological compensatory hypertrophy caused by a painless disease of the contralateral kidney. Hypertrophy → stretching of capsule → pain.

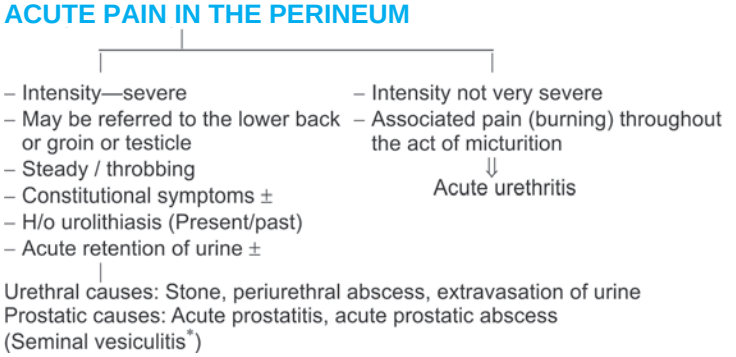
Essentially painless disorders of the kidney: Polycystic disease, cysts, hydronephrosis, tuberculosis and tumors (both benign and malignant).

Essentially painless disorders of the urinary bladder: Diverticula, tumors (papilloma, carcinoma).

PAIN IN THE PENIS



ACUTE PAIN IN THE PERINEUM



www.Ebook777.com

CHRONIC PAIN IN THE PERINEUM

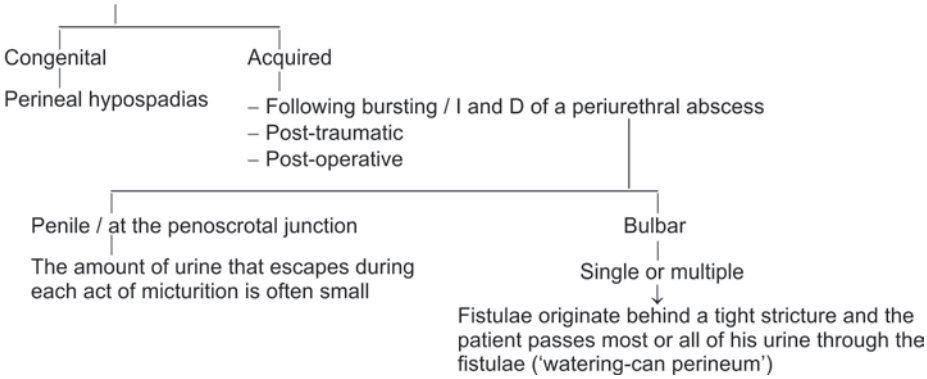
- Urethral causes: Chronic urethritis, stricture
- Prostatic causes: Chronic prostatitis, cancer of prostate.
  (Seminal vesiculitis*).

Prostatic Pain

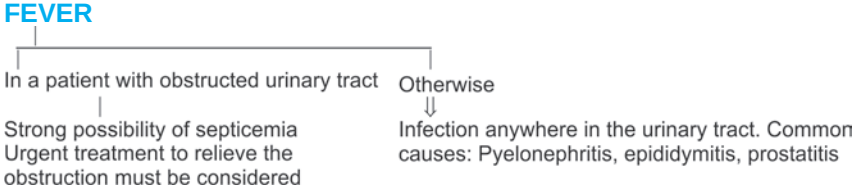
– *Mechanism*: Inflammation leading to edema and stretching (distension) of prostatic capsule
– Site: Primarily in the perineum. May be referred to the mid-sacrum, groin or testicles
– Intensity can be severe
– Continuous
– Frequently associated with frequency and dysuria.

Essentially painless disorders of urethra: Papilloma, cancer.

URETHRAL FISTULA



FEVER



PASSAGE OF GAS IN URINE

Causes

1. Fistula between intestine and bladder, usually due to pathology in the intestine.
   Examples: Diverticulitis, regional enteritis, carcinoma of sigmoid colon.
2. Rarely, in diabetic patients. Mechanism—infection with gas-forming microorganisms. Carbon dioxide is formed from fermentation of high concentration of sugar in the urine.

PASSAGE OF FECAL MATTER / WORMS IN URINE

Cause: Fistula between intestine and bladder, usually due to pathology in the intestine.
Examples: Diverticulitis, regional enteritis, carcinoma of sigmoid colon.

* **Seminal vesiculitis**: Bacterial infections of seminal vesicles generally cannot be proved clinically. Whether bacterial seminal vesiculitis occurs without concomitant prostatic infection is unknown.

Free ebooks ==> www.Ebook777.com

LOSS OF LIBIDO

Possible causes:
– Basically, androgen deficiency arising from either pituitary or testicular dysfunction
– Normal semen volume suggests testicular dysfunction as more likely cause.

IMPOTENCE

Basically, there are two possible etiologies: Psychogenic or organic.
– Quick development (sudden mode of onset with rapid progress) secondary to a precipitating event like marital stress / loss or change of sexual partner ⇒ psychogenic cause
– Slow development (insidious mode of onset with gradual progress) ⇒ organic cause
– Frequently linked to advancing age / other risk factors
– Inconsistency ⇒ psychogenic cause
  In general, patients who can achieve adequate erections in some situations but not in other have psychogenic rather than organic impotence.

ABSENCE OF ORGASM

Causes:
1. DM. → Peripheral neuropathy → impaired function of pudendal nerve → decreased penile sensation.
2. Psychogenic.
3. Antipsychotic drugs.

PREMATURE EJACULATION

Criterion: Achievement of orgasm within less than one minute of initiation of intercourse
This is a highly subjective symptom. History should be taken carefully. Many men actually have normal sexual function with abnormal sexual expectations
Cause: Almost always psychogenic.

INFERTILITY

Definition: Primary infertility is defined as absence of conception after 1 year of marriage provided the husband and wife are staying together and not using contraception.
Both the husband and wife should be evaluated simultaneously.

Causes of male factors leading to infertility

A. *Pretesticular* causes
  – Isolated GnRH/LH/FSH deficiency
  – Pituitary insufficiency
  – Excess of steroid → ↓ spermatogenesis
  – Hyper-/hypothyroidism
  – Excess of estrogen/androgen.

B. *Testicular* causes
  – Bilateral incompletely descended or undescended testicles
  – Bilateral anorchia due to trauma, torsion, infection, etc.
  – Orchitis—mumps, leprosy, etc.
  – Trauma:
    - Accidental
    - Surgical, e.g. damage during inguinal/scrotal surgical procedures: Direct trauma to the testis / interference with blood supply of the testis
    - Compression of the testis due to a long-standing large bilateral hydrocele
  – Varicocele—only if bilateral and long-standing.
  – Gonadotoxins:
    - Drugs—spironolactone, cancer chemotherapeutic drugs, alcohol
    - RT. Germ cells are sensitive. Single exposure of < 600 rads → reversible damage. Leydig cells are relatively resistant.

www.Ebook777.com

- Chromosomal abnormalities—XX, XYY, Klinefelter syndrome
- Defective androgen synthesis and action. Examples—enzymatic defects, androgen resistance.

C. *Post-testicular* causes: Disorders of sperm transport, and other causes.
- Congenital disorders of ductal system (epididymis ↔ ampulla, including seminal vesicles): Absence of some portion of the ductal system, atresia, stenosis.
- Acquired disorders of ductal system:
  - Chronic epididymitis → fibrosis → obstruction
  - Accidental ligation of/damage to ductus deference during surgery in the inguinoscrotal region, e.g. hernia / hydrocele / varicocele, etc.
  - Voluntary sterilization
  - Postprostatic adenomectomy leading to retrograde ejaculation
- Functional disorders of ductal system:
  - Damage to the sympathetic nerve → aperistalsis of the ductus. Causes: Retroperitoneal lymphadenectomy, extensive pelvic surgery
  - Spinal cord damage → aperistalsis of the ductus, altered testicular temperature regulation
- Other:
  - Motility defects—immobile cilia syndrome, infection of semen with *Escherichia coli*
  - Immunological defects—breakdown of blood-testis barrier due to trauma or infection
  - Maturation defects
  - Sexual dysfunction.

D. *Idiopathic* cause—Constitutes at least **40%** of the cases of infertility.

Pitfalls in arriving at the diagnosis:
1. On clinical examination testes of men whose azoospermia is due to germinal cell aplasia can be normal in size and consistency.
2. Tenderness elicited by pressing the prostate does not necessarily indicate that the gland is diseased. Enlarged / boggy prostate ⇒ infection / congestion / both.

ANALYSIS AND INTERPRETATION OF URINE EXAMINATION

- *Crystal clear urine is not infected*; it is a waste to send it for culture
- If protein, nitrites and leukocytes are all absent, the urine is not infected and should not be sent for culture
- Differentiating small amount of blood (+) from greater amounts (++, etc.) is of no value
- A negative result for nitrites does not rule out infection

Feature	*Test, and normal value*	*Interpretation of abnormal findings*
Quantity	24 hour collection	• < 0.5 mL/kg/h or < 100–400 mL/24 hours ⇒ Oliguria • < 100 mL in 12 hours ⇒ Anuria • > 2 l/24 hours in children > 3 l/24 hours in adults } ⇒ Polyuria
Appearance / color	Collect urine in a test tube and see against white background Normal**:** Clear, colorless or pale yellow	*Cloudy* due to phosphates, urates, cells, bacteria, crystals *Smoky* due to hematuria *Red* due to – Hematuria/Hb. Cola colored – Beet root. Bright red – Drugs – Rifampicin, chloroquin, phenytoin. *Brown* due to – Hematuria – Metronidazole, antimalarials, salazopyrine, vitamins B complex *Brown black* due to alkaptonuria, hemosiderosis, myoglobinuria *Purple* due to porphyrinuria *Blue* due to pseudomonas UTI, blue diaper syndrome

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

Feature	*Test, and normal value*	*Interpretation of abnormal findings*
Odor	Normal urine is odorless	Unusual odor—due to unusual metabolite *Sweet* due to maple syrup disease *Mousy* due to phenylketonuria *Fishy* due to hypermethioninemia
Blood	Normally absent	The presence of blood ⇒ RBCs, Hb or myoglobin The absence of RBCs on microscopy ⇒ Hemoglobinuria or myoglobinuria
pH	Normal: 5–7.5 Usually acidic	Alkaline pH ⇒– Proteus infection – Patient taking alkalinizing agent – Renal tubular acidosis
Protein	Normally absent	– Moderate levels are found in infections, stone, tumor, nephritis – A result > 'trace' should be further investigated by 24 hours urine protein measurement – False +ve: Alkaline urine – False –ve: Acidic urine nonalbumin proteinuria
Sugar	Normally absent	– Renal glycosuria (normal BSL) – DM (elevated BSL) – False +ve for reducing substances due to the presence of ascorbic acid in urine
Ketones	Normally absent	– Ketoacidosis due to fasting state or diabetic ketoacidosis – Neonatal ketoacidosis may suggest metabolic defect like organic acidemias or glycogen storage disease
Nitrites	An indirect method of detecting bacteria in the urine. Bacteria contain enzymes which convert nitrates in the urine into nitrites (mostly Gm –ve organisms) Normally absent	– UTI (by Gram –ve bacteria) – The presence of nitrites and > 5 WBCs/hpf indicate further testing of urine with culture – A positive test does not rule out the presence of bacteria
Fats (long chain triglycerides)	Normally absent	Chyluria
RBCs	Normal: < 5/hpf Few RBCs can pass through glomerular filter	**> 5**/hpf ⇒ hematuria Color of urine, RBC morphology and casts collectively help to localize the site of bleeding – Brown urine and deformed RBCs with casts ⇒ glomerular pathology – Red urine, normal RBCs, without casts ⇒ non-glomerular pathology
WBCs	Fresh clean catch sample Normal: < 5/hpf	**> 5**/hpf – Inflammation most commonly caused by infection – Stone, even in the absence of infection – Interstitial cystitis, papillary necrosis – After surgery on the urinary tract > 5 WBCs/hpf and the presence of nitrites indicate further testing of urine with culture
Bacteria	Fresh sample	*Motile* bacteria ⇒ UTI

Contd...

www.Ebook777.com

Contd...

Feature	*Test, and normal value*	*Interpretation of abnormal findings*
Casts	Fresh or preserved urine with acidic pH Casts are formed within renal tubules from cellular debris Occasional hyaline/granular cast is seen in normal urine Other casts are not seen in normal urine	– *RBC* casts: Always pathological and indicates GN – *Granular* casts are nothing but disintegrated RBCs Their presence ⇒ glomerular inflammation – *Hyaline* casts originate from tubular desquamated epithelial cells indicating tubular injury – *WBC* casts ⇒ pyelonephritis / tubulointerstitial nephritis
Crystals	Normally absent Seldom of clinical importance as most crystals are formed when urine cools down from body temperature	– *Persistent presence* of crystals arouse suspicion of renal stones – *Uric acid* crystals are formed in acidic urine, *phosphate crystals* in alkaline urine – Biochemical investigations are required for further evaluation
CFU, i.e. colony forming units on urine culture	Urine culture is done to identify the organism(s) and determine antibiotic sensitivity Freshly voided urine is transferred onto a culture plate, incubated for 24 hours to obtain colony count, and a further 12–24 hours for antibiotic sensitivity Interpretation of CFU differs according to method of collection of urine	1. *Midstream clean catch specimen* – Asymptomatic patient: > 100000 CFU/mL of single organism – Symptomatic patient: As few as 1000 or even 100 organisms/mL with WBCs in urine may be diagnostic 2. *Catheterization* > 50000 CFU/mL of single pathogen. 3. *Suprapubic bladder aspiration* Growth of Gm –ve pathogen in any number (except coagulase –ve staphylococci).
Osmolality and specific gravity	Provides a measure of concentration of urine Normal osmolality: 300–800 mOsm/L Normally urine is hyperosmotic to plasma. Normal specific gravity: 1.010–1.030 Fluid is withheld × 12 hours overnight. The specific gravity of the first 2 morning specimens should reach 1.020. Then, a liter of water is given orally Within 4 hours the specific gravity should go as low as 1.002 A fixed specific gravity of 1.010 under these varying circumstances is a good presumptive evidence of impaired function of distal tubules	1. Urine osmolality determines *causes of polyuria* – < 150 ⇒ DI or primary polydipsia – > 300 ⇒ osmotic diuresis, e.g. DM. 2. *Fixed* osmolality of 300 or specific gravity of 1.010 ⇒ renal (distal tubular) impairment. 3. – Urine and plasma osmolality is same in renal causes of ARF – Urine osmolality > plasma osmolality ⇒ prerenal cause of ARF 4. Urine osmolality > plasma osmolality ⇒ SIADH.
Urinary electrolytes	Spot sample of urine can be used to estimate urinary electrolytes Normal values: – Urine Na 40 mEq/L – Urine Cl 40 mEq/L – Urine K 20 mEq/L Interpretation depends upon the clinical situation	1. Urinary Na concentration is useful in determining the cause of hyponatremia < 20 mEq/L ⇒ Na conservation by kidneys in ECFV depletion and edematous states 2. Urinary K concentration is useful in determining the cause of hypokalemia – < 20 mEq/L ⇒ extrarenal cause – > 20 mEq/L ⇒ renal cause of K loss

Free ebooks ==> www.Ebook777.com

POINTS OF PRACTICAL IMPORTANCE REGARDING SOME KEY INVESTIGATIONS

BUN and Serum Creatinine

– BUN is more dependant on the level of hydration (severity of dehydration)
– Creatinine concentration does not rise until > 50% glomerular function has been lost. However, a normal serum creatinine does not necessarily indicate normal renal function.

Serum electrolytes

– The most important electrolyte is potassium
– A very high serum creatinine with a normal potassium level indicates that renal failure is chronic. This distinction may be important in the immediate management of patients admitted with obstructive uropathy
– In the 'TUR syndrome' (essentially water intoxication) Na may fall to dangerous concentrations; recovery is rare if serum Na falls below 120 mmol/l.

Creatinine clearance is a convenient method of measuring the GFR.
One sample of blood and an accurately timed collection of urine for a period of 24 hours are all that is required. Normal clearance: 90–130 mL/min. It decreases in old age.

Urine culture

– Correct methods of collection and transportation are crucial. The patient must be given adequate instructions to avoid contamination. This is especially important in elderly women. The introitus/vulva should be prepared with normal saline
– Urine should not *routinely* be cultured from indwelling catheters, which are always colonized with organisms, mostly of no clinical significance
– The classical cause of 'negative' culture (on ordinary medium) in the presence of WBCs on urine microscopic examination (i.e. 'sterile pyuria') is tuberculosis
  Causes of sterile pyuria other than TB: Papillary necrosis, interstitial cystitis, after surgery on the urinary tract.

Urine cytology

– Positive cytology is diagnostic of cancer. Negative cytology does not exclude cancer
– Results may be equivocal in the presence of stones, infection, and after instrumentation.

DIFFERENTIAL DIAGNOSIS

Bladder outflow obstruction

Differentiating points

– H/o hesitancy and/or poor flow OR presentation with acute/chronic retention of urine
  |
  +
  - Increased residual volume of > 100 mL on repeat examination (often preferably on US scan examination; alternatively on catheterization)
  - Peak flow rate of < 15 mL/s, provided voided volume is at least 150 mL, preferably 200 mL, in the absence of fear of interruption (on uroflowmetry)

Additional symptoms which may be present: Frequency, urgency
Complication: Back pressure changes → hydrourter, hydronephrosis, renal failure, uremia
Causes
BPE / BNH / BNC / carcinoma of prostate / urethral stricture / stone impacted in the urethra / sphincter spasm caused by neurological disease especially multiple sclerosis / Fowler's syndrome (excess rhabdosphincter activity in young woman).

BPE

Modes of clinical presentation

1. Lower urinary tract symptoms suggesting bladder outflow obstruction + irritative symptoms.
  – Increased frequency of micturition at first during the night and later by day is one of the earliest symptoms
  – Hesitancy (several seconds or even minutes)

www.Ebook777.com

- Straining weakens the stream:
  - in c/o median lobe enlargement, due to further kinking of the prostatic urethra
  - in c/o lateral lobe enlargement, due to their approximation
- The symptoms may progress very slowly, or may remain unchanged
- If nocturia is present, frequency-voiding charting may help to differentiate the real cause of nocturia such as old age, CHF, etc; from BPE.

2. Acute retention of urine.
- This may happen with little or no warning, or after a long period of troublesome symptoms
- Precipitating factors: Constipation, exposure to cold, enforced holding of urine, over-drinking; drugs (tricyclic antidepressants, α-stimulants (including some in proprietary cough and cold medicines)); surgical operation, immobilization.

3. Chronic retention of urine especially in elderly men.

Modes of presentation:
- Gradually increasing painless overdistension of bladder leading to increasing abdominal girth
- UTI
- Uremia

4. Hematuria, UTI. These are the unusual modes of presentation
- Hematuria may be terminal if caused by prostatic congestion, or profuse if caused by rupture of 'vesicle piles'.

Supporting points
- Age > 65
- Findings on DRE

In c/o lateral lobe enlargement
- Size of the prostate is enlarged; its upper limit cannot be reached. Try to pull the gland down. Benign gland possesses some degree of mobility.
  Approximate weight: Height × breadth × depth = volume in cm^3. 1 cm^3 is equivalent to 1 gm
- Deepening of the lateral grooves. Median sulcus is maintained
- Surface—smooth. Consistency—elastic. Firm consistency ⇒ more fibrous content in the enlarged gland
- On US-scan examination: Size of prostate is enlarged Criterion: > 20 cm^3 (i.e. > 20 g)

In c/o median lobe enlargement → Normal

Notes
- Benign prostatic hyperplasia as a histological condition begins at about 35 years of age. It does not necessarily cause obstruction or symptoms
- There is little correlation between the overall size of the gland and the degree of obstruction, or the severity of symptoms.

Bladder neck hypertrophy

Basically, lower urinary tract symptoms suggesting bladder outflow obstruction in the neonate, young children or early adult life.

Other effects
- Visible / palpable distension of bladder
- (Recurrent) UTI
- Pyelonephritis due to V-U reflux.

Caused by congenital hypertrophy of the smooth muscle at the bladder neck.

Bladder neck contracture

Basically, lower urinary tract symptoms suggesting bladder outflow obstruction above 45 but below the age at which BPE presents.

Free ebooks ==> www.Ebook777.com

Differentiating points
- Younger age than that at which BPE is more common
- Straining improves the urinary stream (*cf* weakening in BPE)
- Past h/s/o chronic prostatitis in men / urethrotrigonitis in women
- No prostatic enlargement.

Other modes of presentation: Acute or chronic retention of urine, hematuria, (recurrent) UTI

Caused by fibrosis of tissues at the bladder neck, as an aftermath of chronic prostatitis in men, and urethrotrigonitis in women.

CA prostate

Modes of presentation of early local (organ-confined) disease:
1. Minimal or no symptoms. PSA ↑.
2. On histopathological examination of the tissue resected at TURP for clinically BPE.

Modes of presentation of locally advanced disease:
1. Lower urinary tract symptoms.
2. Chronic pain in the perineum.
3. Hematuria.
4. Uremia due to bladder outflow obstruction or (bilateral) ureteric obstruction.
5. Venous or lymphatic congestion of lower limbs by direct mechanical pressure.

Possible findings on DRE:
- Hard consistency often with bulging and irregularity of the contour of the gland
- 'Suspect' if there is asymmetry of the gland / excessive firmness, induration or extension of a lobe / obliteration of the median sulcus / obliteration of the lateral grooves
- NAD. A normal DRE does not exclude CA prostate.

Conditions which may simulate CA prostate: Multiple prostatic calculi, granulomatous prostatitis.

Urethral stricture

Modes of presentation
1. Lower urinary tract symptoms suggesting bladder outflow obstruction. Straining improves the urinary stream (*cf* weakening in BPE).
2. UTI.
3. Acute retention of urine.

On palpation of penile/bulbar urethra: The stricture may be felt as a hard fixed nodule.

Criterion of stricture in an adult female urethra: Failure to admit freely a No. 20 F bougie.

Causes
- Post-traumatic: Iatrogenic trauma (urethral instrumentation particularly using large caliber instruments, operations upon the lower urinary tract), difficult labor, external urethral injury
- Postgonococcal urethritis, post-tuberculous urethritis.

Complications:
- Back-pressure changes.
- Periurethral abscess. Rupture of abscess → fistula or extravasation of infected urine.

Congenital urethral stricture

Very rare. Usually the symptoms are delayed until adolescence.

Detrusor-sphincter dyssynergia
- A long h/o hesitancy of micturition, especially in unfamiliar surroundings
- *Caused by* lack of co-ordination between contraction of detrusor and relaxation of rhabdosphincter
- No serious consequences
- *Uroflowmetry* shows a reduced peak flow and a rapid 'fluttering'.

www.Ebook777.com

Bladder stone

– Increased frequency of micturition—the earliest symptom
  ± one or more of the other lower urinary tract symptoms.
– Terminal hematuria—A few drops of bright red blood at the end of micturition
– Pain during or at the end of micturition that is referred to the tip of the penis/labia majora, and rarely to the perineum/suprapubic region
– In children, sudden screaming during micturition and pulling at the prepuce is characteristic
– No physical signs unless complication occurs like acute retention of urine
  However, a large stone may be palpable bimanually during pelvic examination, especially in children.

Papilloma of urinary bladder

– Gross hematuria—spontaneous, painless, profuse and paroxysmal

± one/more of the lower urinary tract symptoms

± fragments of the growth passed in urine

Painful hematuria that is constant (rather than paroxysmal) ⇒ malignant transformation of papilloma.

Cancer of urinary bladder (mostly a TCC)

Modes of presentation/detection

1. Invariably—gross hematuria—painful and constant
  ± one/more of the lower urinary tract symptoms.
2. Unusual:
  – Incidental finding during TURP
  – Recurrent UTI. Papillary fronds become necrotic and act as a nidus for infection
  – Advanced disease: Pelvic mass, renal failure due to bilateral ureteral obstruction, bone pains, etc.

Predisposing factors: Tobacco smoking, exposure to industrial carcinogens, chronic cystitis due to indwelling catheter / stones / large residual urine volumes, Bilharziasis, pelvic irradiation.

Notes

– Papillary tumors are usually well-differentiated
– Solid nodular invasive tumors are usually poorly differentiated
– Different grades of differentiation may be found within the same tumor
– Squamous cell carcinoma and adenocarcinoma can occur as metaplastic elements within a predominantly TCC. The presence of these changes does not alter the classification of the tumor, or its biological behavior
– Pure squamous cell carcinoma and adenocarcinoma behave differently.

Ectopia vesicae (*syn. extroversion of the bladder*)

= a congenital anomaly due to impaired development of the anterior wall of the allantois and the lower anterior abdominal wall.

– Umbilicus—absent
– The posterior wall of the bladder is exposed to the exterior. Subsequently, it bulges outwards due to intra-abdominal pressure. The everted vesical mucosa is ulcerated and painful
– True incontinence through the ureteric orifices that are exposed. The odor of urine always accompanies the patient
– Penis
  - Ill-developed / may be cleft / has epispadias
  - Often drawn upwards, and has to be pulled down to expose the ureteric orifices
– Clitoris is cleft, and the labia minora are separated anteriorly, exposing the vaginal orifice.
– Symphysis pubis is absent. The separated pubic bones are connected by a strong ligament. Waddling gate due to this structural deformity.



Fig 54-2 Ectopia vesicae

Free ebooks ==> www.Ebook777.com

Complications
– Ascending pyelonephritis
– Adenocarcinoma of the bladder.

Bilharziasis of the bladder

- *Basically*, intermittent painless terminal hematuria in a patient from the endemic area (Africa, throughout Israel, Syria, Arabia, Iran and Iraq, Portugal, Greece, Madagaskar, Mauritius, Cyprus)
- Past h/o high-grade evening fever, sweating, asthma, urticaria many months ago ±.

Cystitis

Symptoms
– Increased frequency both by D&N—every hour to every few minutes
– Urgency, urge incontinence
– Intermittent discomfort in the suprapubic region. Intensity of discomfort/pain most severe when bladder is full, and relieved at least partially by voiding
– Pain during micturition, referred to the suprapubic region or tip of the penis/labia majora/perineum
– Hematuria—total or terminal. Total ⇒ generalized cystitis. Terminal ⇒ trigonitis.

Signs
– The patient looking drawn due to lack of sleep ⇒ severe infection
– Tenderness—in the suprapubic region, on DRE and on PV/E.

Predisposing causes
1. Incomplete emptying of bladder due to bladder outflow obstruction / diverticulum of the bladder / injury or disease of the spinal cord.
2. Bladder—stone/tumor/foreign body.
3. Immunodeficiency in general.

Avenues of infection
1. Ascending from the urethra: Spontaneously or after instrumentation.
2. Descending from the kidney.
3. From adjoining diseased (usually infected) organs, e.g. uterine tube / vagina / intestine especially diverticulitis / cancer of the pelvic colon / fistulae.
4. Hematogenous, lymphogenous.

Complication: Vesicoureteral reflux may lead to pyelonephritis.

For differential diagnosis of various special forms of cystitis (acute abacterial *syn*. hemorrhagic cystitis, urethrotrigonitis, interstitial cystitis, alkaline encrusting cystitis, etc.), please refer to authentic textbook of General Surgery/Urology.

Acute gonococcal urethritis in the male

– Incubation period—2–10 days (after intercourse with an infected partner)
– Itching, redness and swelling of the external urethral meatus
– Scalding pain throughout the act of micturition
} – The early symptoms
– In a day or so, profuse, thick, yellow-gray colored purulent urethral discharge begins
– Inguinal lymphadenitis.

Local complications: Posterior urethritis, prostatitis, epididymo-orchitis, periurethral abscess, urethral stricture.

Systemic complications: Arthritis, iridocyclitis, endocarditis, septicemia.

Chronic urethritis

– Chronic pain in the perineum
– Scanty watery, or gleety urethral discharge at the external urethral meatus especially in the morning
– Lips of the external urethral meatus are glued together by sticky discharge.

Acute gonococcal urethritis in the female

- Gonorrhea affects primarily the urethra and the cervix, and is often asymptomatic.
- It can never be diagnosed on clinical grounds alone.

www.Ebook777.com

Symptom if at all: A slight dysuria.
Signs
– Slight to moderate vaginal discharge unnoticed by the patient. A copious vaginal discharge is more likely to be due to concomitant trichomonal vaginitis
– Reddening or erosin of the cervix with a mucopurulent cervical plug
– A bead of pus at the meatal orifice on milking the urethra down against the posterior aspect of the ramus of pubis.

Local complications: Proctitis, salpingitis, bartholinitis/abscess.
Systemic complications:—Same as in males.

Nongonococcal (*syn*. Nonspecific) urethritis in the male

Symptoms
– Same as in gonococccal urethritis, but usually milder
– Incubation period: 2–30 days; even up to 6 weeks after intercourse.

Causes of relapse: Reinfection / prostatitis.

Nongonococcal (*syn*. Nonspecific) urethritis in the female
– Increased frequency, urgency
– Terminal hematuria
– Low backache
– Urethral tenderness.

Causes of nongonococcal (nonspecific) urethritis
– Physiological cause in the male: Mucus from unrelieved sexual stimulation
– Traumatic: Accidental injury, iatrogenic insult (catheterization, instrumentation), foreign body
– Metabolic: Oxaluria, urinary stones, gout
– Urinary tract or prostatic (nonvenereal) infections from any cause
– Malignancy.

Urethral stone
– Recent past (2–3 days) h/o ureteric colic
– Sudden pain in the urethra and abrupt cessation of the stream of urine
– A few drops of blood-stained urine f/b retention of urine
– A stone readily palpated through the floor of the penile urethra
– If arrested in the prostatic urethra, then palpated on DRE as a tender hard nodule in the midline, generally near the apex of the prostate

⇒ Migratory stone

– Development of painful micturition / retention of urine in a k/c/o stricture urethra
– A stone readily palpated through the floor of the urethra if it has attained a considerable size

⇒ Primary stone

In some cases, the presence of a primary stone is not suspected if the stone is not palpable owing to its small size, or owing to periurethral thickening. In that case, the stone is detected incidentally during urethroscopy or bouginage.

Notes
– A migratory urethral stone = basically a urethral stone which migrates to and gets arrested in the urethra
– A primary urethral stone = a stone that forms in the urethra behind a stricture or in an infected urethral diverticulum.

TUMORS OF THE MALE URETHRA: Polyp, papilloma, angioma, carcinoma.

Papilloma (male urethra)
– Single or multiple
– Protrude from the external urethral meatus
– May be associated with penile papillomas / papillomas of the bladder.

Free ebooks ==> www.Ebook777.com

Angioma (male urethra)
→ profuse hematuria or bleeding per urethra.

Carcinoma (male urethra)
– Usually a stricture is antecedent.
– Metastatic spread to inguinal lymph nodes. Blood-borne distant metastases ⇒ infiltration of cavernous tissue of the penis
– Profuse urethral discharge—the first and only symptom. Hence, initially it may be misdiagnosed as urethritis. Later, the discharge becomes blood-stained
– Palpable induration
– In the deeper parts of the urethra, sometimes the first manifestation is an indolent periurethral abscess that after I & D not only fails to heal, but the periurethral induration increases.

ABNORMALITIES AFFECTING THE FEMALE URETHRA: Prolapse, diverticulum (urethrocele), caruncle, carcinoma.

Prolapse of the urethral mucosa (female urethra)
– Congenital / acquired. Acquired prolapse is more common before menarche, and past menopause
– Asymptomatic, unless associated with (senile) urethritis, or more than half of the circumference prolapses
– Local discomfort, especially on walking
– Appears as a swollen red ring around the urethral meatus
– Central position of the external urethral meatus ⇒ complete prolapse
– Eccentric position of the external urethral meatus ⇒ partial prolapse.

Urethral diverticulum (female urethra)
– Inability to pass all the urine at one time
– After-dribbling
– Hematuria ± ⇒ formation of a stone in the diverticulum
– Recurrent attacks of cystitis (perpetuated by reinfection from the diverticulum) in spite of consumption of antibiotics / chemotherapeutic agents ⇒ infection of the diverticulum

On PV/E
– A swelling is palpable on the anterior vaginal wall in the line of the urethra
– On compressing the swelling—urine, usually obviously purulent, is expressed.

Urethral caruncle (female urethra)
– Common in middle-aged and elderly women
– A raspberry-like, pedunculated, granulomatous, soft mass about the size of a pea, attached to the posterior urethral wall near the external meatus
– Usually asymptomatic
– Symptoms when present: Increased frequency, pain during and after micturition, blood-stained discharge independent of micturition.

DD
1. Carcinoma of the urethra: Thickened or nodular indurated mass.
2. Prolapse of the urethral mucosa: Insert a probe very gently. The mass is exquisitely tender, and bleeds readily.

Carcinoma (female urethra)
– A visible bleeding protrusion at the external meatus (the most common site)
– Induration distinguishes it from a benign tumor.

Acute bacterial prostatitis
– Lower urinary tract symptoms and acute pain deep in the perineum/rectum or suprapubic region or one or both lower quadrants of the abdomen + severe constitutional upset. A high-grade fever
– Usually a complication of acute gonococcal urethritis. Occurs in 2nd/3rd week after an attack of urethritis, when urethral discharge ceases
– On DRE: Prostate exquisitely tender and tense.

www.Ebook777.com

May → acute retention of urine.
Failure to show improvement within 24 hours of institution of treatment ⇒ **abscess** formation
↓
Digital ± ultrasonic rectal examination ↓ GA indicated for confirmation of the diagnosis.

Recurrent subacute/chronic bacterial prostatitis

– May or may not follow an acute episode. Clinical features are extremely varied.
  1. Disabling chronic pain in the perineum radiating along the urethra, to the sacral area and scrotum, and in a sciatic distribution, and/or in suprapubic region ± one or both lower quadrants.
     Pain aggravated by sitting on a hard chair / after ejaculation.
  2. Lower urinary tract symptoms—particularly increased frequency.
  3. Scanty watery, or gleety urethral discharge suggesting urethritis. Prostatitis should not be diagnosed unless, after irrigating the urethra and massaging the prostate, the resultant specimen shows ≥ 15 pus cells/hpf.
  4. Sexual dysfunction—premature ejaculation, impotence.
  5. Recurrent attacks of mild fever lasting about 3 days and accompanied by malaise.
  6. 'Silent' prostatitis: Arthritis, myositis, neuritis, iritis, conjunctivitis.

On DRE: Size of the gland may be normal, smaller or larger. Surface—irregular.

– Normal features ⇒ mild prostatitis
– OR may be soft and boggy
– OR may be smaller and harder
– Expressed prostatic secretion (by massage through rectum) contains an excess of leukocytes and ≥ 15 pus cells/hpf
– On 3-glass urine test after prostatic message: Prostatic threads are found in urine in the first glass.

Chronic nonbacterial prostatitis

– This is not always distinct from chronic bacterial prostatitis, since specific organisms are often absent in both conditions
– Some cases may be caused by chlamydia or other fastidious organisms.

Tuberculous prostatitis

Presents with one/more of the following *symptoms*:

1. Urethral discharge. Prostatitis needs to be differentiated from gonorrhea and abacterial urethritis based entirely on bacteriological findings. Prostate on DRE at this stage often feels normal.
2. Mild ache in the perineum.
3. Painful, sometimes blood-stained ejaculations.
4. LUT symptoms, terminal hematuria ⇒ posterior urethritis.
5. Infertility.

Findings on DRE

One or more well-defined nodules consistency of which is not stony hard, or a large solitary mass occupying a more central position.

Cold abscess in the prostate is felt as a slightly tender, soft swelling.

Oliguric phase of **ARF** (Acute renal failure)

– The average duration of the oliguric phase—10 to 14 days
– Dark urine in small amounts (< 100–400 mL/24 hours)
– Anorexia
– Hiccups
– Within 4–5 days, copious effortless vomiting
– Abdominal distension
– Fatigue, SOB, orthopnea, paroxysmal nocturnal dyspnea suggesting CHF
– Jugular venous distension
± Systolic HT (~ 200 mm Hg) after 2–3 days
– Hepatomegaly
– Lung crepitations
⇒ CHF.

Free ebooks ==> www.Ebook777.com

Calculous anuria

Mechanisms

1. A stone impacted in the ureter of the only functioning kidney, the other kidney being congenitally absent, previously removed, or destroyed by disease.
2. Both ureters simultaneously getting obstructed by stones or crystals.

Anuria due to uric acid crystalluria can occur in rapidly breaking-down tumors—more particularly lymphomas. The pelvis and ureter can become completely blocked by uric acid and urate crystals. In patients undergoing CT/RT, estimate serum uric acid levels periodically, and also maintain the pH of urine alkaline. The solubility of uric acid is vastly increased at pH 7-8.

Clinical features and course

– A long history of urinary lithiasis ±, OR without any previous clinical evidence of a kidney disease
– Stage of tolerance:
  - Duration 3–6 days. Patient may feel comparatively well during this period
  - Nonspecific symptoms may be complained of, e.g. headache, sleeplessness, constipation, lassitude
  - BUN continues to mount steadily during this period
– Modes of onset: Sudden or insidious
  - In c/o sudden onset: An attack of ureteric colic, f/b oliguria/anuria. Colicky pain is later replaced by a continuous severe pain in the loin
  - In c/o insidious onset: Several days' history of increasing oliguria, f/b anuria. Pain absent or little, if any
– Uremia
– Tenderness in the loin. Guarding in the upper quadrant/lumbar region ±
– A recently obstructed kidney is usually impalpable
– A palpably enlarged kidney ⇒ hydronephrosis/pyonephrosis, and may be probably functionless.

Renal stone

Modes of presentation

– Acute ureteric (renal) colic as a result of the stone causing complete or partial obstruction
– The commonest site of impaction: Uretrovesical junction—the narrowest part of the upper tract
– Other common sites of impaction: At the pelvic brim (where ureter crosses the iliac blood vessels), PUJ
– Fixed renal pain in the loin due to stone impacted in the neck of a calyx
– Hematuria
– Recurrent UTIs
– Pyonephrosis
– Sepaticemia
– Incidental finding on imaging investigation in asymptomatic (with reference to urinary tract) patient
– End-stage renal failure secondary to bilateral disease.

O/E: No abnormal finding unless a complication has supervened.

PUJ obstruction

Modes of presentation

1. In children:
   – Lump in abdomen (hydronephrosis)
   – Nonspecific pain in abdomen, often simulating GI disease with episodes of nausea and vomiting.
2. In adults: Pain in the loin classically after drinking large volumes of fluid.
3. Occasionally: pyelonephritis, pyonephrosis.

- *Cause of primary PUJ obstruction* in childhood: Incomplete recanalization during fetal development
- *Cause of primary PUJ obstruction* in adulthood: A lower pole crossing vessel
- *Causes of secondary PUJ obstruction*: Stone, previous surgery, rarely due to a TCC of the pelvis.

Childhood malignant tumor of the kidney, most probably a nephroblastoma (Wilm's tumor)
Refer to chapter 35.

www.Ebook777.com

(In an adult patient,) **A tumor of the kidney**, most probably a RCC
Refer to chapter 35.

TCC of the upper urinary tract (renal pelvis / ureter)

Modes of presentation/detection

1. Hematuria. 90% tumors are detected during investigation for hematuria.
2. During routine monitoring of the upper tract, of patients with bladder cancer.
3. During investigation of loin pain.

Kidney rarely becomes palpably enlarged, except in rare cases where the PUJ becomes occluded and hydronephrosis results.

Predisposing factors: Tobacco smoking, exposure to industrial carcinogens, Balkan nephropathy, abuse of phenacetin.

TB of the kidney

– A young adult
– Progressively increasing frequency both by D and N: This is the earliest and often the only symptom. Duration—in months.
  Possible mechanisms of increased frequency—tuberculous cystitis OR polyuria due to renal tubular inflammation.
– Pain in the suprapubic region, scalding pain in the urethra throughout the act of micturition ⇒ (tuberculous) cystitis
– Strangury if cystitis is severe
– (Painless) Hematuria. Site of bleeding: Ulcer situated on a renal papilla
– Vague discomfort/pain in the loin ±
– A renal lump ±
– Manifestations of tuberculous epididymitis ±
– Nonspecific constitutional symptoms of TB viz. loss of appetite, loss of weight and low-grade evening fever with night sweats.

Urine examination report on which renal TB should be suspected:

– Abacterial pyuria in urine of acidic pH
– Sterile urine on routine culture
– In special culture, tubercle bacilli can be demonstrated after 3 weeks. If, however, the ureter is blocked by caseous material or stricture, the urine coming solely from the uninfected kidney may be sterile.

Notes

– The disease is commonly unilateral. However, infection of one kidney may spread to the contralateral kidney
– Modes of spread of infection to the kidney: (1) Hematogenous spread from another site, e.g. lung. However, lesions in the lung may have healed and disappeared by the time kidney damage is clinically apparent. (2) Ascending infection from some other part of the genitourinary system, e.g. epididymis. This mode of spread is much less common.

Acute renal artery embolism

Sources of emboli: Heart/aorta
Incidence of bilateral embolism: ~ 30%.
Clinical presentation:

– Nonspecific clinical features: Pain in abdomen / flank / back, nausea / vomiting, low-grade fever, high BP
– Hematuria
– Anuria—acute renal failure.

Routine investigation findings (even though they are nonspecific too):

– Leukocytosis
– Elevated lactate dehydrogenase levels
– Urine: RBCs, WBCs, proteinuria.

Renal artery thrombosis

(*Modes of) Clinical presentation*

– May be clinically silent if adequate collaterals have been developed
– Severe HT
– Acute onset of oliguric renal failure.

Free ebooks ==> www.Ebook777.com

Renal artery dissection

Primary = associated with underlying renal artery disease, e.g. fibromuscular dysplasia, atherosclerosis, etc; and occurs spontaneously.

Secondary = result from blunt trauma / interventional procedures involving selective renal artery catheterization. Aortic dissection can extend into the renal artery.

(*Modes of) Clinical presentation*

– Pain in abdomen / flank / back, nausea / vomiting
– Hematuria
– Severe HT that may be difficult to control; usually requires multi-drug therapy
– Anuria—acute renal failure
– May be silent.

Acute renal vein thrombosis

Predisposing disorders

– Nephrotic syndrome
– Neonates with severe dehydration due to vomiting / diarrhea / septic states / diabetic mother.

Clinical presentation in adults

– Pain in abdomen / flank / back, nausea / vomiting
– Hematuria
– Impairment of renal function
– Proteinuria
– Rarely, venous congestion of kidney can cause hemorrhagic infarction and rupture, leading to severe retroperitoneal bleeding which may require urgent nephrectomy.

Clinical presentation in children

– Sudden onset of abdominal distension
– Hematuria
– A mass in the flank
– Proteinuria
– Progressive renal insufficiency.

Extrinsic ureteric obstruction

Modes of presentation

1. An incidental finding in patient presenting with features of the underlying primary condition.
2. Flank pain.
3. Uremia, septicemia

Hypospadias

A common developmental abnormality in which the urethra opens in an abnormal position on the underside of the penis. The most common associated abnormalities: Maldescended testes and inguinal herniae.

– In glandular variety there is a depression at the normal expected site of external urethral opening
– In perineal variety the scrotum is split and the urethra opens between its two halves. If associated with bilateral maldescended testes, the sex of the child may be difficult to be determined.

In all except the glandular variety the penis is curved (bowed) in a downward direction ('chordee'). The farther away the opening from the normal position, the more pronounced is the bowing. In all cases, the inferior aspect of the prepuce is poorly developed giving it an appearance of a hood ('hooded prepuce'). The hooded prepuce can be effectively used for repair of hypospadias.

Disabilities due to hypospadias:

– The stream of urine may be deflected downwards or splash and drip back along the shaft of the penis
– Penoscrotal/perineal hypospadias compels the boy to void in sitting position
– Uncorrected chordee interferes with normal intercourse, and semen from a penoscrotal/perineal hypospadias may not be directed into vagina.

www.Ebook777.com

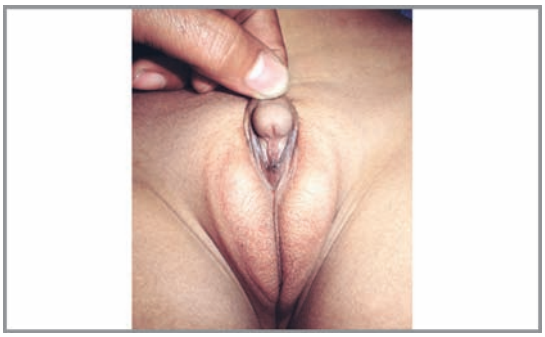


Fig 54-3 Hypospadias: Scrotal variety

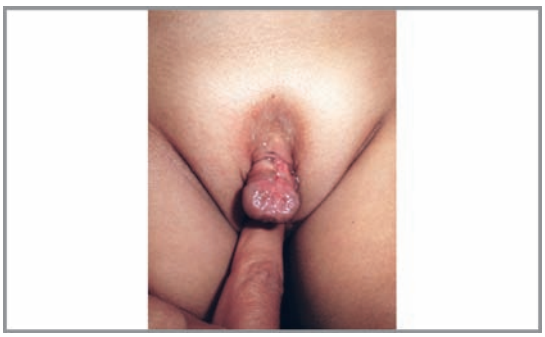


Fig 54-4 Epispadias

Epispadias

A developmental abnormality in which the urethra opens in an abnormal position on the dorsal aspect of the penis. This abnormality may occur in isolation, or in conjunction with bladder extrophy. In the glandular or penile variety the bladder neck is intact and continence is preserved. In the more posterior penopubic or subsymphyseal variety the bladder neck is incompetent and dribbling incontinence is present.

Phimosis

Variables to be considered in arriving at the diagnosis:

1. Age.
2. Duration

Has it been present all throughout since birth?	Has it developed recently? Other way to put it: Has phimosis developed in a previously normal retractable foreskin?
⇓	⇓
Primary phimosis	Secondary phimosis
Considered normal in children up to 4 years of age	Inguinal lymphadenopathy if present, could be secondary to cancer, or chronic balanoposthitis

Primary phimosis

Separation of the prepuce or circumcision before 4 years of age is not recommended unless strongly indicated, as exposure of the glans to urine may result in ammonical dermatitis. As the child grows, usually prepuce can be gradually retracted and by adolescence there would be no phimosis any more. Only in few children primary phimosis may persist through adulthood.

Causes of development of **secondary** phimosis: 1. Balanoposthitis. 2. Cancer of the glans/undersurface of prepuce.

Paraphimosis

An acute condition in which tight prepuce cannot be repositioned after having been forcibly retracted behind the corona.

It can occur in two ways:

1. Patient himself may make a rather forcible attempt to retract the foreskin who had been having a degree of phimosis.
2. Iatrogenic: A health professional having forgotten to reposition the foreskin after initially having retracted it for a purpose such as examination of the penis, urethral catheterization, etc.

The condition → vascular congestion and edema of the retracted rolled-up prepuce. Eventually may lead to ischemia of the glans if not reduced expeditiously (manually, or surgically if gentle manual reduction seems impossible or has failed).

Free ebooks ==> www.Ebook777.com



Fig 54-5 Paraphimosis



Fig 54-6 Balanoposthitis. Note that it is also associated with phimosis

Balanoposthitis

= inflammation of the opposing surfaces of the glans and the prepuce. Nearly always associated with some degree of phimosis.

– Mild inflammation → itching and some discharge
– More severe inflammation → pain in the distal portion of the penis and purulent foul-smelling discharge from inside the narrowed prepucial opening
– The prepuce is red, swollen and tender.

Candidiasis is very common.

Causes of **balanitis**

– Failure to maintain hygiene
– Phimosis, by precluding maintenance of hygiene
– Uncontrolled DM
– Fixed drug reaction (tetracycline, sulfonamide, etc.), scabies, dermatitis, psoriasis, lichen planus.

Gangrenous balanitis

This may result from combined infection by spirochetes and vibrio organism. If untreated, may result in extensive destruction of the glans and shaft of the penis.

Balanitis xerotica obliterans

– Typical white firm prepuce which bleeds easily. Dense phimosis.
– Meatal stenosis ±.

The cause of this condition is unknown. It is not premalignant but may require circumcision and meatotomy/meatoplasty.

Prepucial stones

Usually seen late in life; chronic posthitis having resulted in long-standing phimosis. When the precpuce has not been retracted nor its interior cleansed for many years, prepucial stones result.

Types: 1. Resulting from inspissated smegma. 2. Mixture of smegma and urinary salts. 3. Consisting entirely of urinary salts.

Indications for circumcision

1. 'Tight' phimosis—Criterion of tight phimosis—ballooning of prepuce occurring at the time of micturition.
2. Recurrent attacks of balanoposthitis.
3. Primary phimosis persisting through adulthood (To enable satisfying intercourse).
4. Secondary phimosis.
5. Phimosis which precludes cystoscopy when indicated.
6. In c/o zipper injury, if zipper cannot be removed by manipulation.
7. In c/o paraphimosis, if gentle manual reduction seems impossible or has failed.

www.Ebook777.com



Fig 54-7 'Tight' phimosis. Note ballooning of the prepuce

Priapism
= abnormally prolonged erection without sexual stimulation or desire.
Erection is confined to the corpora cavernosa; so the glans is not erect as the corpus spongiosum is spared.
May be associated with bleeding disorder / sickle-cell disease / pulmonary disease / any advanced malignancy.

Types: Low flow and high flow.

– '*Low flow*' priapism: Associated with venous occlusion. Blood gas analysis will reveal readings similar to venous (deoxygenated) blood
– '*High flow*' priapism: Usually follows trauma to the corpora and is associated with increased inflow of arterial blood through AV fistula.

Peyronie's disease
– Age—over 40
– Pain and/or curvature on erection of penis; erection being associated with sexual stimulation or desire. After a few weeks, pain disappears
– (Otherwise) Asymptomatic swelling in the penis due to fibrotic plaque (on dorsal / lateral / ventral aspect)
– Progress of the disease: Slow over years, OR rapid producing marked angulation of the erect penis preventing intercourse.

Notes:
– An idiopathic disorder causing fibrotic changes in the elastic tissue of the tunica of the corpora cavernosa
– ~ 10% patients have associated Dupuytren's contracture
– *DD*: Secondary carcinoma of the penis. Differentiation may not be difficult as the (secondary) carcinoma of the penis occurs late in the course of the primary disease.

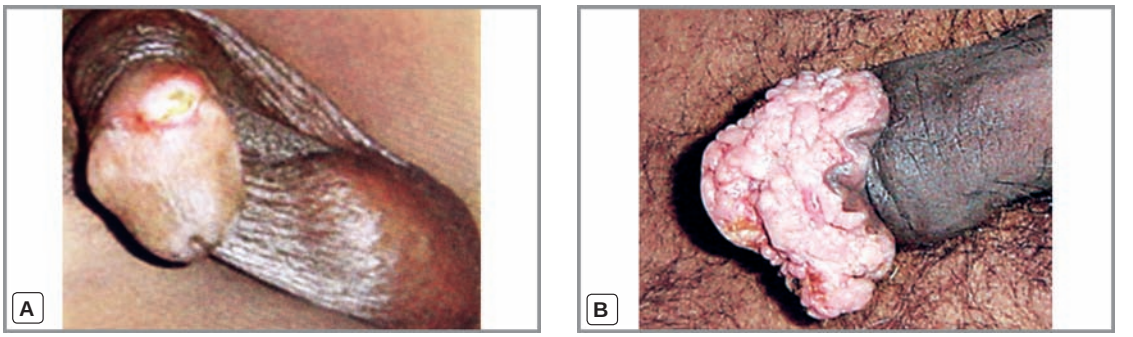
(Primary) **Carcinoma of the penis** (SCC of the glans/inner surface of the prepuce)
– Mild irritation and a purulent discharge from under the prepuce—the first symptom
– Duration—long—in months. Progress—slow
– Discharge later becomes blood-stained and foul-smelling
– In most cases, the prepuce cannot be retracted, and in order to view the lesion, the prepuce must be slit ↓ anesthesia (a biopsy is performed during the procedure)
– Morphology: Velvety raised lesion / a typical ulcer with characteristics of squamous-celled carcinoma
– Inguinal enlarged lymph nodes could be due to infection, or due to secondary metastatic deposits.

Notes:
– Not necessarily a disease of only old age. It is seen even in men under 40
– Common in uncircumcised males
– Precancerous lesions: Leukoplakia, erythroplasia, long-standing genital warts.

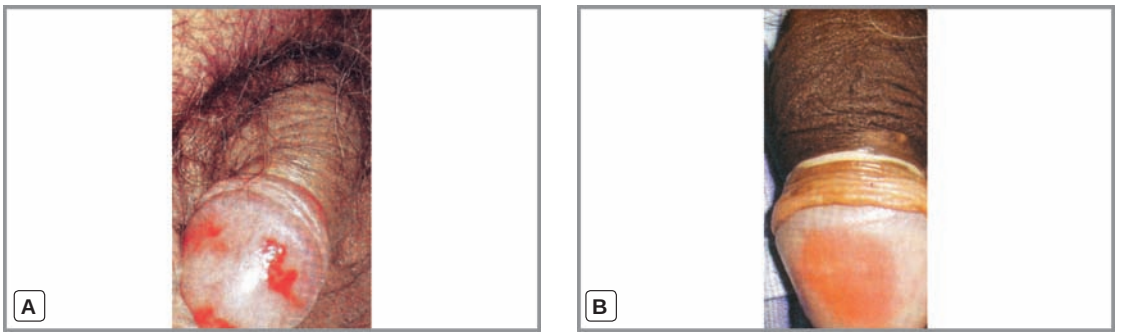
Secondary carcinoma of the penis
Primary sites—usually the bladder / rectum / prostate.

Amoebic ulcers on the penis (glans and prepuce)
Multiple irregular punched out ulcers with yellowish slough.

Free ebooks ==> www.Ebook777.com



Figs 54-8A and B Examples of CA penis



Figs 54-9A and B Examples of erythroplasia

Modes of spread of amoebae to the penis

1. Autoinfection from feces.
2. Anal intercourse.

Chordee

= a fixed (downward) bending or bowing of the penis.

Causes:

1. Hypospdias (all varieties except for glandular).
2. Acute chordee may result from general urethritis. Erection is grotesque and very painful.

Elephantiasis of the penis

- Most common cause—filariasis.
- May or may not be associated with scrotal elephantiasis
- When penis alone becomes elephantiatic, it does not get buried into the scrotum (= 'Ram's horn penis')
- The erectile tissues of the glans and the body do not become elephantoid as they are devoid of lymphatics
- The elephantiatic changes take place under the skin covering the shaft and the outer layer of prepuce. The inner layer of the prepuce is always normal.

Meatal stenosis

- Congenital stenosis is associated with phimosis
- Acquired stenosis may complicate a meatal ulcer.

Pinhole meatus is an extreme degree of meatal stenosis which can result in back-pressure changes in the whole of the urinary system.

Ulcer of the urethral meatus

- This may occur in young male children, 3 months to a year or so after performing circumcision. Never occurs in uncircumcised children
- A scab which is formed on the ulcer may block the meatus. Removal of the scab to be able to pass urine → pain and passage of a few drops of blood
- *Complication*: Cicatricial contracture leading to acquired pinhole meatus.

www.Ebook777.com



Fig 54-10 Lymphedema



Fig 54-11 Genital herpes

Acute penile periurethral abscess—an infection of one of the urethral glands.
– Usually a complication of acute gonococcal urethritis
– A tender induration on the undersurface of the penis.

Natural history: Bursts externally → fistula.

Acute bulbar periurethral abscess / acute periurethral phlegmon

= cellulitis in the same plane in which superficial extravasation of urine occurs viz. into the scrotum, beneath the superficial fascia of the penis, and up the abdominal wall beneath the deep layer of the superficial fascia.
– May or may not be predisposed to by stricture of the urethra
– Pain in the perineum
– High-grade fever with rigors
– Initially, tenderness and swelling are limited to the perineum. Within hours, cellulitis spreads to the scrotum, penis and the abdominal wall.

Chronic periurethral abscess
– Nearly always situated in the perineum
– Almost invariably the result of a long-standing stricture of the bulbous urethra.

Genital herpes
– Usually sexually transmitted
– Recurrent attacks ±
– Pain along the distribution of sensory nerve, usually the genitor-femoral, precedes the eruption by 2 days. Pain around the perianal region may be exceptionally severe
– A group of tiny vesicles appears—they rapidly erode to form shallow red or yellow based ulcers which may coalesce to form a serpiginous outline.
– In the females the lesions may spread on to the thighs during the first attack.
– Involvement of the urethra may → retention of urine (due to intense dysuria). Longer-lasting (10–14 days) retention ⇒ radiculitis of the 2nd and 3rd sacral nerves.

Granuloma inguinale

= a chronic slowly progressive ulcerative tropical disease affecting the genitalia and surrounding tissues but occasionally occurring on extragenital sites.
– Average incubation period: 1 to 4 weeks, but may be shorter or very much longer
– A painless vesicle or indurated papule—gradually erodes into a slowly extending painless ulcer with a beefy red/grayish granulomatous floor
– The ulcer bleeds easily on touch.

Natural history: 1. Partial healing and formation of keloid scar. 2. Malignant transformation.

Chancroid (Soft sore)
– Caused by Gram negative *Streptobacillus* of Ducrey
– Sexually active adults. More common in men

Free ebooks ==> www.Ebook777.com

- Incubation period: 2–5 days
- (Often) Multiple acute painful inflamed ulcers on the genitals (penis, vulva)
- Rounded 0.2–1 cm in diameter
- Copious purulent discharge
- Yellowish slough
- Edematous undermined edge
- Base—soft
- Easily bleed on touch
- Inguinal lymphadenitis—tender, hard and matted—causing a feeling of stiffness in the groin. May suppurate → abscess
- Phagedena sometimes occurs.



Fig 54-12 Chancroid

PRINCIPLES OF MANAGEMENT OF 'EMERGENCY' CLINICAL SITUATIONS

STRANGURY

If severe UTI is suspected
↓
- Commence antibiotic treatment
- Cystoscopy later (after a few hours or a day)
  Reason for waiting x a few hours or a day: Urethral instrumentation during UTI without an antibiotic cover may lead to pyelitis, septicemia

Otherwise
↓
Urgent cystoscopy

Specifically look for
- CA bladder neck (which on HP examination may prove to be of bladder/prostatic origin)
- Stones impacted in the bladder neck

RETENTION OF URINE

Attempt at catheterization

Successful
i.e. catheter passes into bladder and urine flows to the exterior
⇓
The most probable level of obstruction: Bladder neck

In men under 55, who have not had much dysuria before the acute episode of retention
↓
- 'Routine' blood investigations
- US scan

If no significant abnormality
↓
A trial of catheter removal may be considered after 24–48 hours (during which time edema in the distal urinary tract subsides)

Successful
If not successful, investigate further

If significant abnormality is disclosed

Not successful

If enlarged prostate is suspected
↓
SPC is safer than repeated attempts of introducing a catheter
Definitive management subsequently

If urethral stricture is suspected
↓
- SPC
  OR
- SPC if initial attempts at passing filiform-follower fail

Definitive management subsequently

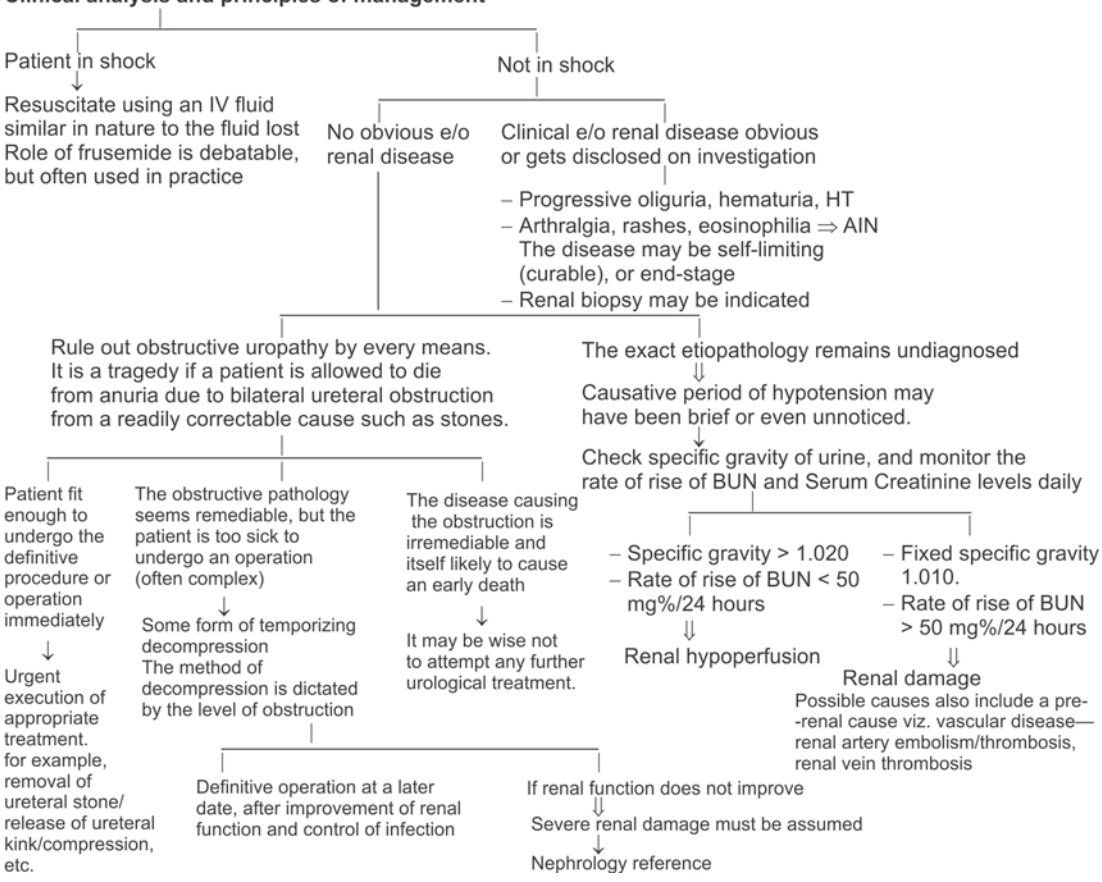
In vast majority of patients
↓
An operative treatment will be required [either with the catheter still in, or (preferably) after a trial of catherter removal]

The timing of operation (prostatic adenomectomy, for example) will depend on whether retention was acute or chronic

www.Ebook777.com

OLIGURIA / ANURIA

Clinical analysis and principles of management

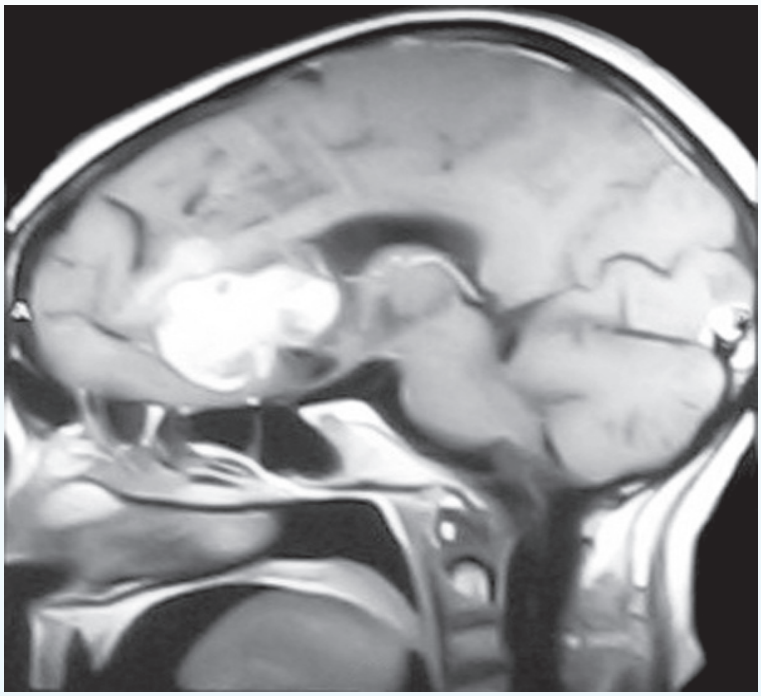


Free ebooks ==> www.Ebook777.com

www.Ebook777.com

SECTION 3

Neurosurgery



Free ebooks ==> www.Ebook777.com

www.Ebook777.com

55 INTRACRANIAL SPACE-OCCUPYING LESION

Causes of intracranial SOL

Insidious mode of onset and gradual progress

– Tumor: Primary/secondary
– Trauma: - Chronic SDH
- Abscess complicating a penetrating injury
– Infections: - Tuberculoma
- Brain abscess (pyogenic)
- Complications of HIV: Toxoplasmosis, NHL
- Fungal infections
- Cryptococcoma: Single/multiple lesions
- Other parasitic infestations: Cysticercosis, hydatid disease, etc.
– Vascular: An AVM stealing blood from adjacent eloquent brain, especially the sensorimotor areas

Sudden mode of onset and rapid progress

– Vascular: - Intracerebral hematoma/infarction
- Rupture of an AVM
– Infection: - Brain abscess (pyogenic) often secondary to sinusitis/mastoiditis

Effects of an SOL depend on the site, progress and stage of the lesion.

↓

One or more of the following:

– Vague disturbances of higher functions
– Emotional instability
– Epilepsy
– Localizing symptoms and signs (Focal syndromes)
– Raised ICP
– 'Coning'.

Frontal lobe is a relatively silent area of the brain and lesions should usually must reach a large size before causing any symptoms. The localizing symptoms and signs s/o frontal lobe lesion may be low and subtle in developing, and therefore difficult for acquaintances of the patient to recognize them until they are quite obvious.

Occipital lobe is also a relatively silent area.

HISTORY

- Forgetfulness?
- Slow comprehension?
- Decreased intellectual power?
- Inability of sustained mental activity?
- Undue irritability?
- Social lapses?

} **Vague disturbances of higher function**

- **Emotional instability**?
- **Epilepsy**?
  – Duration?
  – Type?
  – eneralized epilepsy preceded by aura of flashing lights?
  – Generalized epilepsy preceded by hallucinations of smell and taste?

Free ebooks ==> www.Ebook777.com

- **Localizing symptoms and signs** (**Focal syndromes**)?
  - Disturbances of higher functions. Lapses in memory
  - A mute state, aphasia, lack of interest in surroundings, lack of drive and initiative, a passive immobile state
  - Sometimes disinhibition with hypersexuality and/or aggression with lack of control over social graces, a lack of care with appearance
  - Incontinence of urine and/or feces.

  } for frontal lobe lesion

  Subfrontal lesions may impinge on the olfactory tract and cause loss of sense of smell either unilaterally or bilaterally.
- – Dreamy state of mind, lapses in memory
  - Superior quadrantic visual field defects
  - Progressive aphasia (due to pressure on Broca's area)
  - Generalized epilepsy preceded by hallucinations of smell and taste.

  } for lesion in the temporal lobe
- – Lower limb weakness (on contralateral side)
  Bilateral lower limb weakness ± (⇒ parasagittal SOL)
  - Sensory deficit.

  } for upper sensorimotor area
- – Weakness in the arms, clumsiness of fingers, slurred speech
  - Sensory deficit.

  } for lower sensorimotor area
- – Lack of orientation in space, e.g. getting lost in a familiar environment
  - Sensory disturbance with lack of appreciation of what is being held in the hand (astereognosis), sensory neglect (on the opposite side)
  - Eye movement disorder, with difficulty in following objects
  - Lack of co-ordination
  - Jacksonian epilepsy.

  } for lesion in the parietal lobe
- – Flashes of light and color (in opposite visual field with visual field defects)
  - Generalized epilepsy preceded by aura of flashing lights.

  } for lesion in the occipital lobe
- – Multitude of cranial nerve deficits
  - Pyramidal weakness on one/both sides
  - Sensory deficit on one/both sides
  - Inco-ordination due to disruption of cerebellar pathways.

  } for brain-stem lesion
- – Spontaneous pains
  - Slight paresis
  - Decreased superficial and deep sensations (on opposite side of the body).

  } for lesion in the thalamus
- – Adiposity
  - Disturbances of sex
  - Hyperthermia
  - Diabetes incipidus.

  } for lesion in the hypothalamus.
- – Giddiness
  - Stumbling forwards
  - Drunken gate.

  } for lesion in the midline (vermis) of cerebellum
- – Abnormal postures of the limb (due to muscular atonia)
  - Asynergia
  - Adiadokokinesia
  - Nystagmus (on the same side of affected limb).

  } for lesion in the cerebellar hemisphere

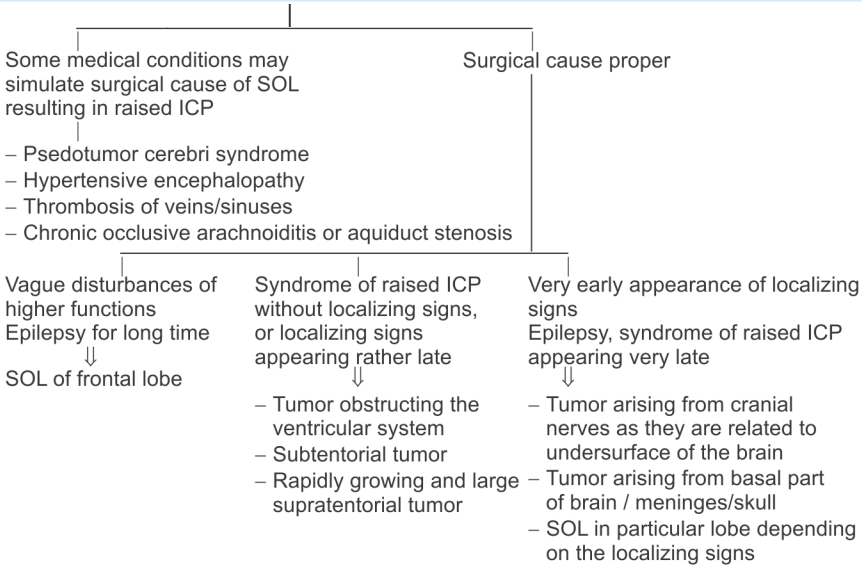
www.Ebook777.com

- **Headcahe**:
  - Duration?
  - Mode of onset?
  - Intensity?
  - H/o head injury?—When?—a few hours before the onset of headache, OR weeks to months earlier?
  - Have you noticed any particular precipitating/aggravating/relieving factors?
  - Periodicity?
  - Fever?
  - Neck pain/stiffness, deterioration of LOC?
  - Diminished vision?
  - Unilateral stuffy running nose, reddening and tearing of the eye?
  - In a suspected c/o post-traumatic chronic SDH: Alteration of LOC, changes in personality, hemiparesis?
- **Syndrome of raised ICP**
  - Vomiting:
    - Frequency?
    - Is it preceded by nausea?
    - Is it related to food?
  - Is the cerebration retarded? Drowsiness?

EXAMINE THE NERVOUS SYSTEM

(Beyond the scope of this book)

ARRIVING AT THE DIAGNOSIS



Clinical features of the **syndrome of raised intracranial pressure**

- Headache: May be bilateral (bifrontal / bioccipital / bitemporal) OR localized overlying the site of tumor
- Vomiting: Irregular. Projectile. Not associated with nausea. Not related to food. Apparently causeless. More common in posterior fossa tumors
- Retarded cerebration—Due to pressure on cerebral cortex
- Drowsiness

Free ebooks ==> www.Ebook777.com

– Bradycardia. Mechanism: Raised ICP → compression of veins → stasis in the circulation → rise in systolic BP to overcome stasis in the circulation → reflex bradycardia
– Papilledema. Mechanism: Obstruction of venous drainage from retina. (Subarachnoid space also covers the optic nerve).

Clinical features of the **syndrome of coning**

Rapid and high rise in ICP occurs due to temporal lobe herniation from supratentorial to infratentorial compartment through tentorial opening causing compression of the midbrain, and/or cerebellar tonsilar herniation through foramen magnum causing compression of medulla oblongata.

– Severe paroxysmal headache
– Drowsiness
– Bradycardia
– (Initially) unilateral dilated pupil
– Neck stiffness.

www.Ebook777.com

SECTION 4

Trauma (Essentially Soft Tissue)

56. Acute mechanical trauma —general principles
57. Head injury
58. Facial (Maxillofacial) injury
59. Injury to the neck
60. Chest injury
61. Esophageal injury
- Perforation
- Corrosive injury

62. Ingested foreign body
63. Abdominal trauma
64. Urogenital trauma
65. Injury to anorectum, perineum, buttock
66. Spinal trauma
67. Hand injury
68. Vascular injury
69. Burn injury: Thermal, chemical, electrical



Free ebooks ==> www.Ebook777.com

www.Ebook777.com

56 ACUTE MECHANICAL TRAUMA —GENERAL PRINCIPLES

Simple first-aid principles account for the saving of more lives than heroic surgery.

DEFINITIONS

Trauma is defined as a wound or injury, whether physical or psychic.

Mass casualty incident means any incident in which emergency medical service personnel and equipment are overwhelmed by the number and severity of casualties. For example, an incident where a 2-member medical team is responding to 3 severely injured people could be considered a mass casualty incident.

Multiple system trauma (*Syn.* Polytrauma) is defined as a trauma, complex in its nature or effects, which inflicts injures to more than one region/system in the body.

Wound: A traumatic break in the continuity of any tissue.

Abrasion: A superficial injury involving only the outer layers of the skin, and is caused by the tangential application of blunt force.

Scratch = A liner abrasion.

Graze = A broad abrasion.

Contusion (*Syn. Bruise)*: an infiltration of blood into a tissue following rupture of capillaries as a result of the application of blunt force. The injury results in a diffuse swelling; there is no gaping of the tissue.

Hematoma: A 'large' collection of blood into a soft tissue or a joint following rupture of small blood vessels as a result of the application of blunt force. The injury results in a swelling; there is no gaping of the tissue.

Contused lacerated wound (CLW) = a wound in which the skin and underlying soft tissue are torn, sheared or crushed as a result of the application of blunt force; these wounds are commonly seen following road traffic accidents and consequently may be frequently dirty, containing foreign bodies and devitalized tissues, and contaminated with organic matter. The wound edges are irregular, jagged and often abraded or contused, as are the surrounding tissues. When the underlying tissue is rigid or hard like a bone, the wound simulates an incised wound.

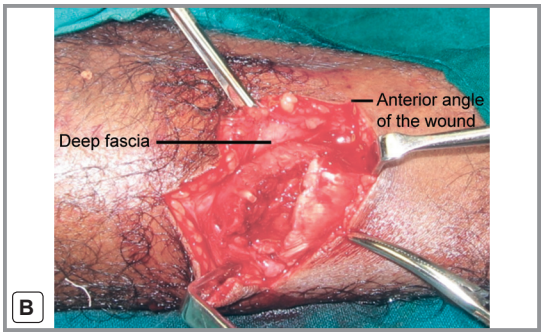


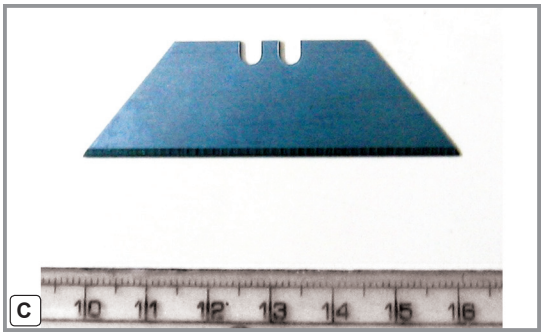
Fig 56-1 Contused lacerated wound on the scalp

Incised wound (IW) = a wound caused by a weapon having a sharp cutting edge when it is drawn across a soft tissue, commonly the skin. The edges are clean and the margin is clear. The greatest dimension of an incised wound is its length.

Free ebooks ==> www.Ebook777.com







Figs 56-2A to C (A) An incised wound; **(B)** Found to be muscle-deep; muscle layer partially cutk; **(C)** The injuring agent

Puncture wound = a wound caused by a pointed weapon when it is driven in through the skin. Depth is its greatest dimension. The entry wound is often too small to have any measurable length and breadth.

Stab wound = a wound caused by a more or less pointed weapon with sharp cutting edge(s) when it is driven in through the skin. The wound does have a length and breadth though depth is the greatest dimension.

When the wound pierces a body cavity, it is **penetrating.** The criterion of penetration is breach in the continuity of the lining of a body cavity, e.g. dura mater / parietal pleura / parietal peritoneum. A viscus may or may not have been injured.

If the injuring weapon exits partially or completely (completely like a bullet for example,) through the body wall (often but not necessarily always opposite the entry side) *after having penetrated a body cavity*, then it is called a **perforating** wound. Miraculous survivals are known after ghastly-looking perforating wounds.

Degloving wound = a wound caused by traction/avulsion causing shearing forces that separate tissue planes, rupturing their vascular interconnections leading to tissue ischemia.

A **simple injury** is defined as one which is neither extensive nor serious (severe / grievous), and which heals (rapidly) without leaving any permanent deformity or disfigurement.

A **severe** (*syn.* **dangerous**) **injury** is an injury which threatens life. It is either extensive or serious in relation to the organ or part wounded. The injury is fatal without surgical treatment. A severe injury is a variety of grievous injury.

A **potentially severe** (*syn.* **dangerous**) **injury** may be defined as an injury which potentially threatens life.

The criteria of a **grievous injury** may vary to some extent according to the penal code followed. It is advisable to refer to the penal code or authentic textbook on medical jurisprudence.

Accident may be defined as any unexpected or sudden event especially of a harmful character.

Assault is an offer of threat or attempt to apply force to the body of another individual in a hostile manner.

Homicide means the killing of one human being as a result of the conduct of the other.

www.Ebook777.com

Suicide means self murder. Attempted suicide is an unlawful act and a person is held responsible for the immediate consequences of the act.

The **caliber of a gun** refers to the internal diameter of the barrel and is usually measured in fractions of an inch (e.g. 0.38 or 0.45) or in millimeters (e.g. 9 mm). The larger the caliber, the larger the missile (projectile) that is fired.

GENERAL REMARKS

1. Severe injuries are often missed.
2. A severely injured patient can be a very gruesome sight. Do not panic. Call for help—a nurse, a medical assistant, an anesthesiologist, etc.
3. The usual standard sequence of case-taking does not apply to a severely injured patient; the sequence needs to be varied. Learn to recognize emergency situations quickly while they are still treatable, e.g. rupture of spleen/liver, an expanding EDH, etc.
4. More lives are lost from failing to care for the airway than from any other cause. Air passages of children are small and are easily blocked.

Causes of trauma

1. Mechanical or physical violence.
2. Thermal: Heat leading to scalds or burns / Cold leading to frost bite or immersion foot/hand.
3. Chemical: Acid / alkali burns.
4. Electrical: → burns / gangrene.
5. Radiation.

BLUNT TRAUMA

Examples: Road traffic accidents, falls, blunt assaults, most sports-related injuries.
The energy imparted is distributed over a wider surface area compared with penetrating mechanisms.

Road traffic accident (Motor vehicle accident), the injured being an occupant of the vehicle

Factors influencing the pattern and severity of injuries:

1. The size of the car. Injury frequency is higher in smaller cars.
2. The position of the occupant within the car.
3. The use and type of restraint.
4. The type of accident: Head-on (frontal impact) collision / lateral impact collision / rollover and ejection. Injuries can be predicted based on the force vector.

Road traffic accident (Motor vehicle accident), the injured being a pedestrian

This mechanism of injury is associated with a high degree of energy transfer to an unprotected victim. These patients should be considered to have polytrauma until proven otherwise.

Factors which modify the basic pattern of injury:

1. *Age.* Children, by virtue of being smaller, are usually struck anatomically higher than adults. Also, the child victim may fall underneath the car where almost any type of injury can occur.
2. *The type of vehicle*. Passenger cars produce mostly lower extremity and pelvic injuries, while light trucks and vans cause a higher incidence of abdominal and thoracic injuries.

FALL FROM HEIGHT

The major determinant of injury and the chance of death is directly proportional to the height fallen, the nature of the landing surface, and the body's orientation on landing. A soft yielding landing surface reduces deceleration forces and hence magnitude of injury.

Free ebooks ==> www.Ebook777.com

PENETRATING TRAUMA

Penetrating trauma is categorized by the energy level imparted by the source of energy and is usually discussed in terms of low-, medium- and high-energy trauma, and the weapons are also classified accordingly.

Hand-driven weapons such as knives, ice-picks, etc. are generally considered as low-energy weapons. Injuries can be roughly predicted based on a description of the weapon (size, shape and length) and the site of entrance of the weapon.

Firearms may be described as low-, medium- and high-energy weapons depending on the muzzle velocity of the projectile as it leaves the barrel of the gun.

Muzzle velocity	*Energy type of the firearm*
< 1100 feet/s	Low-energy
1100–2000 feet/s	Medium-energy
> 2000 feet/s	High-energy

Handguns are usually considered low- to medium-energy weapons.

Single-action revolvers require manual cocking of the hammer, which is then released when the trigger is pulled. *Double-action revolvers* are so-called because the action of pulling the trigger will both cock the hammer and then cause its release.

Semi-automatic guns will fire one bullet each time the trigger is pulled.

(Fully) Automatic guns fire continuously as long as the trigger is kept pulled.

Rifles and derivatives are high-energy weapons.

Shotguns can be highly destructive at close range, but they cause less significant injury as the range increases. In the majority of *civilian shootings*, the victims are shot at a range of less than 30′. At that distance, impact velocity is approximately the same as muzzle velocity.

Relatively elastic tissue, such as muscle, sustain less injury than denser, less elastic tissue, such as the spleen or liver. Other parts of the body, such as teeth or bone fragments, can become missiles themselves, causing damage to surrounding tissue.

BLAST INJURY

The most vulnerable organs to the blast wave are the ear drum, lungs, GIT and urinary bladder. **These organs may be injured even without any external injury to either thorax or abdomen**. Casualties may present in a dramatic form with traumatic amputation. Deafness resulting from rupture of tympanic membrane makes communication difficult. These patients must be carefully examined for many have multiple wounds.

The positive pressure phase of a blast wave lasts only a few milliseconds, but close to an explosion it may rise to over 6000 kN/m^2; such an explosion can be devastating. It will tear into pieces anything within the radius of ~ 10′ and persons may be killed even without any external sign of injury. Outside this radius victim may develop multiple bleeds mainly in the lungs, bronchi and pleural cavity, and pneumothorax.

CLINICAL FEATURES S/O LIFE-THREATENING INJURY

Immediately after trauma

– Deteriorated **level of consciousness**
  - Confusion, restlessness, agitation ⇒ hypoxia
  - Drowsiness / obtundation / stupor / unconsciousness may suggest head injury or shock
– Inadequate **airway** or/and **breathing** or/and **circulation**
– Obvious **exanguinating** wound/s
– Specific acute life-threatening regional/systemic injuries

Subsequently

Natural history of specific injury

PREHOSPITAL ASSESSMENT AND TRIAGE particularly in 'mass casualty'

Trauma patients may be divided into 3 different groups, based on the extent of their injuries.

1. Those that are potentially fatal: Examples—penetrating injuries of the chest/abdomen, hypovolemic shock, head injury, etc.
2. Those that are rapidly fatal due to devastating injuries: Expire within minutes after the incident.
3. Those that are not fatal: Injuries are minor and will not cause a threat to life if treatment is delayed.

www.Ebook777.com

The 'golden hour' after a traumatic event is the optimum period in which proper management will provide the best outcome. The period is considered to be about 4 hours from the time of sustaining injury.

Triage means sorting and classification of casualties (as mentioned above) and determination of the priority of need and proper place of treatment. The purpose is to be selective because there is a finite amount of resource available within a given medical system.

The best triage system is one that employs a combination of—nature, cause, and mode of the trauma, anatomical region/s affected, physiological data, pre-existing illnesses, and paramedic judgment.

FIRST AID

There are no hard and fast rules as to which measures constitute first aid and which measures are carried out in hospital. The measures mentioned below should be applied in practice as *appropriate*.

Transporting an unconscious patient on her/his back without proper attention to the airway is a major cause of death.

***General** first aid measures:*

- Extend the head. Thrust the jaw forwards
- Clear the airway of secretions/vomitus/blood by a gauze-on-a-finger-tip
- Remove any foreign body if present
- Insert an oropharyngeal airway, if available
- Place the patient in recovery position for transport to hospital. The recovery position allows blood and saliva to run out of the nose and mouth
- Fit a temporary cervical collar
- Control bleeding by raising a wounded limb, by applying local pressure to the wound, and by pressing on the 'pressure points'
- Ventilate with a self-inflating bag
- Close an open chest wound
- Elevate the lower limbs (as a measure to treat shock).

***Specific** first aid measures:*

Moving a patient with suspected spinal injury

4 people are required to move/turn a patient with suspected spinal injury.

Minimize the movements of the spine, especially the cervical region. Move the patient 'in one piece'.

If the patient is on the ground, carefully turn her/him on one side and then roll onto a stretcher or a stretched blanket. While the patient is in turned position (before rolling onto a stretcher), take the opportunity to examine the back and the spine.

If a cervical spinal injury is suspected, place one hand under the chin and the other under the occiput. Exert gentle traction on the neck, while the body is being turned. Do not let the head drop to one side. Holding the head is the task for the most skilled/responsible person in the team. Once the patient is finally placed onto the back, wedge the head between sandbags, or rolled up sheets or blankets, or fit in a collar.

If a thoracic/lumbar spinal injury is suspected, transport the patient in prone position with a pillow under the shoulders and pelvis to hyperextend the spine at the site of the injury, *unless there are multiple injuries or airway is in doubt*. If the airway is in doubt, transport in supine position.

Fracture pelvis. Tie 3 triangular bandages firmly round the pelvis. Put pads between the legs and tie them together.

Fracture above the knee. Put the limb in a well-padded Thomas splint.

Fracture leg. Put the limb in a well-padded Thomas splint. If a Thomas splint is not available pad a piece of wood or bamboo or even a palm branch, and this to the patient's injured leg. If nothing is available, bandage the injured leg to the normal one.

Fracture arm. Put the arm in a sling and bandage it firmly to the body. Or Tie the arm to a splint which reaches to the axilla.

Bleeding from a hand wound: Apply plenty of gauze and firm dressing. Raise the hand.

Abdominal injury. Any foreign body embedded in the trunk should not be removed as major bleeding might follow removal. Evisceration is best left undisturbed, except to apply (a sterile) dressing and protect the patient from further injury.

Free ebooks ==> www.Ebook777.com

NONVISCERAL SOFT TISSUE INJURIES

Skin and subcutaneous tissue

Abrasion, contusion, blebs (vesicles), hematoma, incised wound, contused lacerated wound, surgical emphysema, extravasation of body fluids like urine, etc; traumatic edema, loss of tissue.

Abrasion

Dirt is often ingrained in the skin. The site of abrasions may give useful clues as to the direction and magnitude of injury forces.

- A fresh abrasion is bright red in color
- The exudate of serum dries and forms a red scab in 12–24 hours
- The scab becomes reddish brown in 2–3 days
- The abrasion heals from the periphery by re-epithelialization in 4–7 days by which time the scab falls off
- Healing is complete by 10th day.

Contusion (*Syn.* ***Bruise***)

- A fresh contusion is red in color
- The color changes from red to blue in a day, bluish to black/brown in 2–4 days, green in 5–7 days, and yellow in 7–10 days
- The yellow color slowly fades and the skin regains its normal color in 2 weeks.

Hematoma

Blood within a hematoma is initially fluid, later it will clot. A clotted hematoma will not be fluctuant.

A clean open wound *(incised / contused lacerated)* ***whose edges are in apposition***

Such a wound heals by first intension, i.e. without formation of a significant granulation tissue

- The edges become red and swollen about 12 hours after infliction of the wound
- A scab may form by about 24 hours
- Re-epithelialization begins at the edges after 36 hours. Epithelialization of 'small' wounds may be complete in 4–7 days. A scar is formed in 1–2 weeks.

An open wound *(incised / contused lacerated)* ***that is contaminated or/and is gaping***

Such a wound heals by second intension.

- Acute cellulitis is apparent in 12–24 hours
- Pus may be apparent after 36 hours
- Granulation tissue will appear in due course of time
- Further healing may take a variable period depending on a number of factors and therefore it is not possible to determine the age of the wound with any degree of accuracy.

Wound with skin loss

Healing of the skin is of importance as a barrier to infection, and because the healing of deeper tissues can also take place satisfactorily in the presence of an intact skin cover. The longer any exposed surface remains raw the greater will be the scarring, deformity and disability.

Muscles and tendons

Contusion, hematoma, strain, sprain (partial rupture), complete rupture of belly of muscle or of tendon, rupture of muscle sheath producing 'hernia of muscle', traumatic myositis, displacement of tendon.

Bursa

Hematoma, traumatic bursitis, rupture.

Artery

Contusion (leading to thrombosis), rupture (leading to bleeding / (false) aneurysm / A-V fistula), compression, spasm, traumatic arteritis.

Vein

Bleeding, thrombosis.

Lymphatic vessels

Rupture, particularly of a large number of lymphatic vessels due a transverse cut across the limb may lead to lymphedema in the dependent limb.

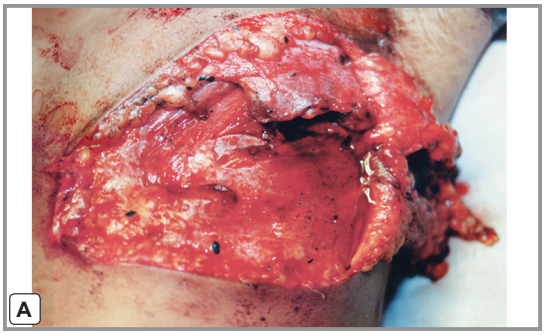
www.Ebook777.com

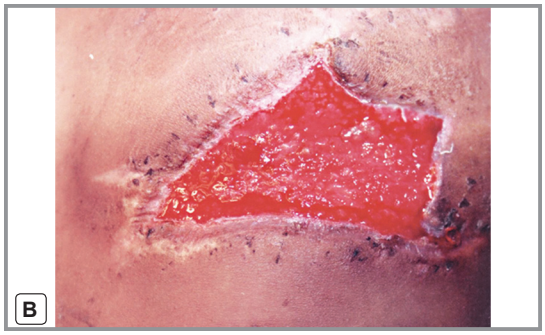
Peripheral nerves

Neurotmesis (— complete or partial (anatomical break)), axonotmesis, neuroprexia (concussion), ischemia, compression. These injuries may lead to disturbances of motor, sensory, vasomotor, nutritional and perspirational functions, and reflexes.

Degloving wound

- Can be open or closed, localized or circumferential, uniplanar or multiplanar.
- Most commonly occurs in the plane between subcutaneous fat and deep fascia.
- Multiplanar injury suggests a high-energy transfer. This has a limited potential for primary healing.
- In a closed degloving wound, the skin is merely separated from the underlying tissues by a hematoma, and the diagnosis is not so easy. *Clinically*, however, it may be possible to pinch the avulsed layers (commonly the skin and subcutaneous tissue) revealing its detachment from the normal deep anchorage.
- Skin necrosis will slowly but inevitably appear over the following few days. Even tissue that initially demonstrates venous bleeding may subsequently undergo necrosis.





Figs 56-3A and B An open multiplanar degloving injury. **(A)** At the time of presentation; **(B)** 2 weeks later. (The distal portion of the flap was necrosed.) This wound was grafted successfully

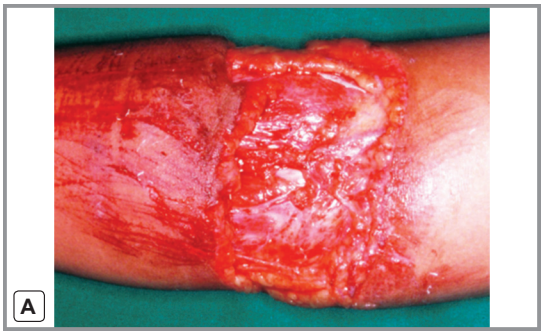






Figs 56-4A to C An open uniplanar (subcutaneous tissue deep) degloving injury. **(A)** At the time of presentation; **(B)** 4 days later, necrotic area established with demarcation; **(C)** on 5th day, the necrotic skin was excised. Eventually, the wound healed well by 2nd intension

Free ebooks ==> www.Ebook777.com



Figs 56-5A and B An open uniplanar (subcutaneous tissue deep) degloving injury with a wide base. There was no necrosis of any portion of the skin

Crush (and devitalized) wound

Industrial, severe road traffic accidents and war injuries account for the majority of these wounds. The area and depth of devitalized tissue depends upon the area and weight of the crushing force and on the duration and velocity of impact. They are often accompanied by degloving, and compartment syndrome. Severe crushing injury may have a profound general as well as local effect especially if pressure is unrelieved ('crush syndrome').



Fig 56-6 A nonsurvivable crush injury caused by a severe RTA

Difficulties in dealing with crush wounds:

1. The wound may be heavily contaminated with bacteria.
2. It may be difficult to distinguish with certainty between viable and nonviable tissue.
3. Inflammatory edema and therefore the tissue tension will, if attempts are made to reconstitute the wound by primary suturing, rise to dangerously high levels resulting in local ischemia in tissues which were previously viable. Dead muscle is the ideal environment for the development of gas gangrene.

BONE AND JOINT INJURIES

Bone

Contusion, fracture, separation of epiphysis, traumatic periostitis.

Joint

Sprain (partial rupture of ligament), subluxation/dislocation, fracture-dislocation, fracture involving the joint, traumatic synovitis and effusion, traumatic hemarthrosis, contusion of the joint, tear of intra-articular cartilage and other structures.

When a **tooth has been knocked out**, bleeding from the socket stops in about 24 hours; sometimes it may take 2–3 days. A clot is formed. The clot is obliterated by fibrous tissue in 2 weeks. The socket is completely filled with gradual new bone formation in about a year (as seen on X-ray examination).

PERTAINING CHILDREN

Young children have greater powers of regeneration than adults. This applies especially to finger-tip amputations, where pulp is able to regenerate provided that the amputation has occurred distal to the midpoint of the distal phalanx.

Difficulties in clinical assessment:

1. A child may be too small to cooperate. Examination requires patience and perseverance.
2 Assessment is also a problem in unconscious child.

www.Ebook777.com

Pitfalls in arriving at the diagnosis:

1. Neurapraxia with temporary loss of function may mimic transection.
2. Tendons may be incompletely severed but have the appearance of intact function on clinical examination.
3. Major intrathoracic injury can occur without fracture of ribs.

OUTLINE OF ASSESSMENT AND MANAGEMENT OF A SEVERELY INJURED PATIENT

1. Primary survey—ABCDE (i.e. quick initial assessment), and immediate life-saving measures.
2. Secondary survey = a comprehensive assessment directed at identification of suspected and unsuspected injuries.
   This includes:
   - A detailed history
   - A head-to-toe physical examination
   - Investigations.
3. Attempt at arriving at a clinical diagnosis.
4. Document points of medicolegal importance.
5. Commence general management.
6. Monitoring and frequent reassessment.
7. Additional specific investigations.
8. Definitive treatment.
9. Rehabilitation.

PRIMARY SURVEY—ABCDE (QUICK INITIAL ASSESSMENT) AND IMMEDIATE LIFE-SAVING MEASURES

Identify life-threatening conditions and begin management simultaneously.
Assessment takes few seconds. In practice, different regions/systems of the body are often assessed simultaneously or in a rapid succession. The priority of assessment and management goes by the acronym 'ABCDE'.

A = Airway.
B = Breathing.
C = Circulation.
D = Disability/deformity due to a musculoskeletal injury; injury of the cervical spine is of paramount importance (including during airway management).
E = Encephalic (i.e. Neurological) assessment.

Brief history from the patient if conscious, otherwise from the onlooker.

- Cause and nature of trauma?
- Difficulty in breathing?
- The site of pain, disability, deformity, swelling, open wound, bleeding?
- Amount of bleeding that occurred before first aid began?: Approximate number of teaspoonfuls / tablespoonfuls / glassfuls, OR how much area of floor or cloth (such as bed sheet, towel, etc.) was stained/ soaked with blood?

Quickly inspect the body from head to toe.

Localize the injuries from the site of pain, disability, deformity, swelling, open wound, bleeding etc. **Aim to categorize them** into one of the two—acute life-threatening, and non-life-threatening, while further assessment and management continues often simultaneously.

Pointers of severe injury

a. *Nature, causes and modes of injury*: Falls of > 10–15′, RTA (MVA) with closing speed of the vehicle being > 60 kmph or fatality at the scene, passenger ejection, prolonged extrication (> 20 min), or major intrusion of the passenger compartment, pedestrian struck by a motor vehicle with a closing speed > 30 kmph.

Free ebooks ==> www.Ebook777.com

b. *Anatomic criteria* (including but not limiting the following): Penetrating injury to the head/neck/chest/abdomen/proximal extremities, ≥ 2 proximal long bone fractures, pelvic fracture, flail chest, amputation proximal to the wrist/ankle, limb paralysis, burn injury involving > 10% total body surface area, inhalational injury.

c. *Other criteria*: Elderly patient, patient with altered vital parameters, patient with pre-existing chronic disease and patient exposed to environmental extremes.

Clinical manifestations of hypoxia

- Confusion, restlessness, agitation
- Pallor, sweating
- Tachycardia.

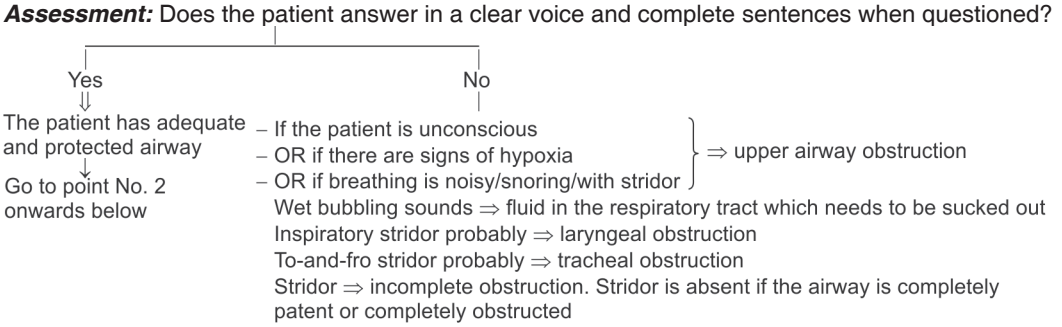
Cyanosis is uncommon and difficult to detect clinically, particularly if hypovolemia is present.

1. Airway

Injuries in which airway obstruction must be assumed/anticipated: Essentially major craniofacial trauma.

- Head injury with unconsciousness. Mechanisms of airway obstruction: 1. Loss of control over tongue and jaw. 2. Depression of cough reflex. A level of consciousness of < 9/15 on GCS usually mandates early definitive airway intervention, as the protective gag and swallow airway reflexes are likely to be absent or compromised.
- Injuries of the face, mouth, mandible
- Injury to the neck leading to expanding soft tissue hematoma or laryngeal fracture
- Burns of face or respiratory tract.

Assessment: Does the patient answer in a clear voice and complete sentences when questioned?



Airway obstruction can be completely silent.

***Management** of the obstructed airway*:

- Thrust the jaw forwards
- Extend the head minimally and just enough to maintain the airway patent. Fracture cervical spine must be assumed until proven otherwise
- Inspect the oral cavity and pharynx
  - Clear the airway (mouth, oropharynx and nasopharynx) of secretions/vomitus/blood by sweeping a gauze-on-a-finger-tip / by using a tongue depressor and suctioning
  - Remove any foreign body/lumps of food if present, with forceps under direct vision
  - Remove any intra-oral prosthesis like dental plate/dentures, etc.
- Insert an oropharyngeal airway if the patient is unconscious and will tolerate it
- Give bag-mask ventilation and high-flow O_2
- Monitor O_2 saturation continuously with pulse oxymetry.

Constantly keep reassessing the airway by looking at the respiratory excursions (rise and fall of the chest-wall), feeling for the patient's breath using the palm and listening for abnormal airway sounds.

- *If a patient has a maxillofacial injury*, tongue may have to be pulled forwards with forceps or a stitch, or pass a nasotracheal catheter, or insert a nasotracheal airway, or apply a postnasal pack. Sometimes a patient may need bronchoscopic aspiration.
- With these maneuvers, if respiratory insufficiency continues, OR if the patient remains unable to protect her/his airway, then consider endotracheal intubation or cricothyroidotomy or tracheostomy for a definitive airway control.

www.Ebook777.com

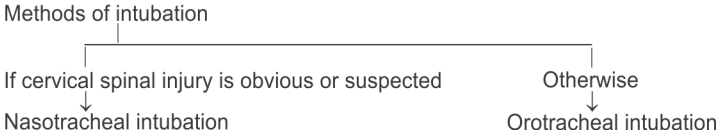
Intubation

If the patient is noisy, restless and struggling for breath, intubation may have to be done when the patient is still awake ('awake intubation'). An anesthesiologist will be able to handle such a situation more confidently than anyone else.

If the patient is unconscious, intubation can be done as if s/he were anesthetized.

Prior to intubation, preoxygenate the patient.

Methods of intubation



Contraindications for nasotracheal intubation: Apneic patient, major facial fracture.

Possibility of cervical spine fracture must be assumed and hyperflexion/hyperextension of the head must be avoided during any maneuver. Fortunately, most cervical spinal injuries are flexion injuries, and the little extension or even the neutral position required for intubation is not dangerous—provided one remembers to avoid too much extension.

Nasotracheal intubation is more difficult, and there will not be a cuff on the tube.

After intubation, check for adequate ventilation by auscultation of both lungs.

Laryngotomy / tracheostomy is indicated if intubation fails or is impractical. The choice between the two will be dictated by the availability of resources such as technical expertise, equipment, the quality of postoperative nursing care, etc.

Essential indications for *emergency tracheostomy*: 1. Severe laryngeal injury. 2. If intubation fails or is unsatisfactory, and there is no other way of maintaining the airway.

Emergency tracheostomy is performed in a desperate situation. Formal tracheostomy after intubation is a safer proposition.

A formal tracheostomy is often indicated in patients who have been intubated for 48 hours in whom, the period of intubation is anticipated to be further prolonged.

Management of airway ensures protection from aspiration, control of gas exchange and the ability to hyperventilate to assist in the management of (increasing) intracranial pressure in patients with closed head injury.

2. Ensure adequate exposure (by removing (or tearing away if necessary) the clothes) **and lighting** to allow adequate access for examination and to avoid missing an occult injury.

3. Look at the **Neck veins** and measure **Respiratory rate, Pulse rate and BP**.

4. Breathing

The absence of spontaneous breathing or the presence of at the most ineffectual gasping movements ⇒ respiratory/cardiorespiratory arrest → Commence resuscitation at once.

– Patient making great respiratory efforts, but still hungry for air
  The accessory muscles of respiration put into use
– Distension or retraction of IC spaces
– Cyanosis in the presence of an adequate airway
– Tachypnea OR Bradypnea
– Paradoxical (see-saw) movement of the chest and abdomen, indicating failure of normal diaphragmatic function.

} ⇒ *Compromised breathing*

Causes of inadequate ventilation which need immediate (emergency) action then and there without wasting time in investigating the case: A sucking chest wound (open pneumothorax) and tension pneumothorax.

A. An obviously open wound in the chest may be making a sucking noise each time patient breathes. Froth from the injured lung may be coming out of the hole (open wound) in the chest. Shortness of breath. Tachypnea. Mediastinal shift ± ⇒ **Sucking chest** wound. *Treatment of obvious sucking chest wound*: Simple occlusive dressing on the wound followed by formal insertion of a chest tube. The occlusive dressing taped on 3 sides acts like a 'flutter valve' allowing exit of air from the pleural cavity but preventing entry of atmospheric air into the pleural cavity.

   Tension pneumothorax may be associated with a sucking chest wound.

Free ebooks ==> www.Ebook777.com

B. Shortness of breath, tachypnea, hypotension, distension of IC spaces of the affected hemithorax, hyperresonant note on percussion, and reduced/absent breath sounds ⇒ **Tension pneumothorax**. Sometimes severe pain in abdomen may confuse the diagnosis.

↓

Immediately insert a needle (whichever is available at hand) in the 5th or 6th intercostal space in the anterior axillary line; followed by expeditious formal insertion of a chest tube. Until chest tube drainage is organized, attach an IV-tube to the needle and dip the other end of the tube under water in a bowl. ('Under-water seal' prevents atmospheric air getting sucked in the pleural cavity). Remove the originally inserted needle after chest tube is secured.

Other injuries which can compromise breathing:
- Massive hemothorax, (massive) visceral herniation
- Flail chest
- Pulmonary contusion
- Aspiration pneumonitis.

5. Circulation: Cardiac tamponade, severe bleeding.
- A penetrating injury in proximity to the heart, e.g. parasternal stab
- Anxious look
- Distended neck veins (unless the patient is hypotensive)
- Profound hypotension
- Pulse may be irregular, thready, and stronger on inspiration than on expiration
- Apex beat neither visible nor palpable
- Percussion note—normal
- Muffled heart sounds.

} ⇒ **Cardiac tamponade**

Investigation of choice: 2-D echocardiography.

Principle of treatment: Emergency thoracotomy. In dire situation, pericardiocentesis may play a role in diagnosis as well as a temporizing life-saving measure.

Quickly look for, and achieve immediate (temporary) hemostasis of **severely bleeding wounds**.

Methods

a. If the limb is bleeding, ***elevate*** it to a degree slightly above the level of the heart. *Caution*: Do not give a 'head low' position to the entire body by tilting the bed. Reason: 'Head low' → impairment of the movements of the diaphragm, cerebral congestion, and discomfort.
   If the wound is in the upper part of the body, make the patient sit up if possible, or propped up. Be careful; the patient may faint.
   This method will usually control venous bleeding.

b. *Direct* ***pressure***
   Press a large dressing firmly over the wound, maintain the pressure by applying a tight bandage, + manually, and wait at least 5 minutes. If a torrent of blood pours from the dressing, replace the dressing and quickly consider other methods of achieving hemostasis.

c. As an additional method, try pressure on certain proximal major arteries at specific points, although this may not be much effective.
   - Carotid artery against the transverse process of the C-6
   - Temporal artery against the skull just in front of the ear
   - Subclavian artery against the 1st rib
   - Brachial artery against the middle of the humerus
   - Femoral artery against the acetabulum at the midinguinal point.

d. ***Application of a hemostat*** ('artery forceps')
   If bleeding continues after 5 minutes of application of direct pressure, then it indicates that a 'large' vessel may have been injured, probably an artery, more likely from a tear rather than complete transection.
   Technique: Get proximal control by pressing on a pressure point first. Make sure that the vessel is clearly visible. Never jab the hemostat blindly into a pool of blood; an accompanying critical anatomical structure

www.Ebook777.com

may be damaged. When the hemostat has been successfully applied, leave it in place. Do not attempt to tie the bleeding point and remove the hemostat until measures for achieving definitive hemostasis have been organized, usually in the operating room.

e. ***Packing*** is indicated to control deep inaccessible bleeding.

f. In some situations, such as the face, ***temporary hemostatic sutures*** may be useful. Do not let them strangle the tissues.

g. ***Tourniquet***

Indication: When other methods have failed, bleeding threatens the life and the risk of loosing the limb (as possible sequela/complication of application of a tourniquet) can be accepted.

Comments: The most effective method is direct pressure. The most dangerous method is application of a tourniquet. In some desperate situations, application of a tourniquet may be the only (the last) resort, however, 3 points must be remembered in relation to the use of a tourniquet. (i) Indication must be absolute. (ii) Tourniquet must be applied effectively and (iii) It must be released intermittently (every 15 minutes) to prevent 'crush syndrome' or irreversible ischemia of the limb.

If a patient arrives with an effective tourniquet that has been in place for > 2 hours, s/he is in a serious danger of 'crush syndrome'. If irreversible ischemia of the limb seems to have been established ('late' grade 2, or grade 3), then do not remove the tourniquet.

Clinical features of hypovolemic shock:

– *Skin*—pale, cold and clammy
– *The state of veins on the dorsum of the feet in a patient lying supine in horizontal position*—flat or empty
– *Venous refilling time*—appreciably increased
– *Capillary circulation*—poor
– *Pulse*—rapid and thready
– *BP* - ↓. Recordings of BP indicate the severity of the vascular disturbance
– *Respiration*—rapid and shallow
– *JVP* - ↓.

Shock in a trauma patient initially must be assumed to be due to hypovolemia and is therefore treated with volume expansion.

Gradation of **hemorrhagic shock**, **based on initial presentation** – Refer to page 90.

Management

Administer O_2 by face mask or nasal catheter.

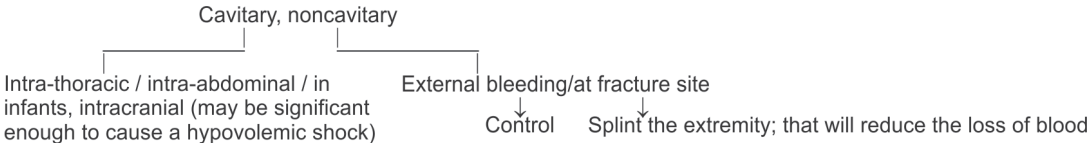
IV infusion is absolutely indicated if systolic BP is < 100.

Start at least 2 large-bore intravenous lines.

Rapid infusion of 1–2 l of Ringer's lactate in 5–10 minutes promptly re-establishes and maintains normal blood pressure in patients who do not have ongoing bleeding.

If patient has air hunger or BP < 60, infuse 2–3 l in 5–10 minutes.

Indication for BTF: If the initial infusion of 2 l of RL fails to restore blood pressure, or if blood-loss exceeds 1.5 l. Ongoing fluid requirements indicate significant bleeding and necessitates immediate measures to identify and control the source of bleeding. Sources of bleeding:



Significant cavitary bleeding should be suspected in all hypotensive patients until proved otherwise, even though many will have only a noncavitary source of blood loss.

If a knife/dagger/arrow/spear is still in the patient's body leave it there until the patient reaches the operating room. If it is removed earlier say for example when the patient is still in the accident and emergency room, the wound may bleed torrentially if the weapon has torn a major blood vessel.

Free ebooks ==> www.Ebook777.com

6. A brief primary neurological assessment and resuscitation

– LOC? An LOC of < 9 of GCS ⇒ severe head injury.
– Pupillary size and reactivity?
– Lateralizing signs?

Clinical criteria of cardiorespiratory arrest

– Unconsciousness
– Cessation of heart beat (practically, the absence of carotid artery pulsations on both sides)
– Cessation of spontaneous respiration. However, **respiratory movements need not be completely absent**; there may be ineffectual gasping movements, but the patient usually becomes cyanosed.

Commence cardiorespiratory resuscitation.

SECONDARY SURVEY

DESIRABLE SPECIFIC DETAILS IN THE EMS PERSONNELS'S REPORT

- The mechanism of injury?
- Vital parameters?
- Treatment given at the scene and during transport?

Knowledge of the mechanism of injury can help the clinician to judge those areas of the body that may have sustained the greatest energy transfer so that subsequent management can be directed appropriately.

HISTORY related to the mode and mechanism of injury

– 'Mode' means intention. It can be accidental / occupational / assault / abuse / attempted homicidal / attempted suicidal / bite
– Cause and nature constitute 'mechanism' of injury
  Examples of cause of injury: RTA, railway accident, airplane crash, fall from height, stumbling, sports-induced injury, fist/kick blows, gunshot injury, etc.
– Nature of injury may be acceleration/deceleration / blunt / penetrating / perforating.

In case of **fall while walking**

- How exactly did you fall?
- Which part of the body came in contact with the ground first? Did you fall on the outstretched hand?

In case of **occupational trauma**

- Nature and other details of the machine?
- Details of the mechanism of injury?

In case of **assault with fist/kick blows, club, stick, rod, chain, etc**.

- Site/s of injury? Ask further specific details accordingly.

In case of **attempted homicidal penetrating trauma caused by a hand-driven weapon** (knife, ice-pick, etc.)

- Was the victim asleep or intoxicated when the injury was inflicted?
- Were the injuries inflicted after restraining the victim?
- Was the assailant single, or were they many?
- What was the gender of the assailant?
- Was the assailant seemed to have been trained in hand-to-hand combat techniques? – e.g. a seasoned criminal / a policeman / a soldier / a commando / a terrorist?
- Did/Could you make attempt to flee? Did you fall down and sustain injury anywhere else during the attempt?

INTERPRETATION

– Magnitude of the injury will be logically more severe if the victim was asleep, intoxicated or restrained during infliction of injury
– More the number of assailants, more likely to be the magnitude of injury
– In general, men are able to generate more force than women. Also, men tend to hold the knife with the blade originating from the radial side of the hand and thrust upwards

www.Ebook777.com

Women tend to hold the knife with the blade originating from the ulnar side and thrust downwards
- (Military) training in hand-to-hand combat techniques teaches that to cause more harm after intracelomic penetration of the weapon, the blade should be moved from side-to-side and up-and-down
- If the victim tries to flee, additional penetrating injuries may be inflicted at other points. Also while attempting to flee if the victim falls down, s/he may sustain yet additional injuries, which should be carefully looked for during subsequent physical examination.

In case of **fall from height**

- From how much height did you fall down?
- How exactly did you fall?
- Did you land straight on the ground OR was the fall broken into more than one step by landing on different slabs/platforms/obstacles before finally landing on the ground?
- How was the landing surface—more or less uniform or spiked?
  Was the landing surface hard or was it yielding such as soft earth, snow, mud or water?
- Which part of the body took the brunt first?

INTERPRETATION
- Regardless of position on landing, for falls > 15' there is high incidence of deceleration injuries to intrathoracic and intra-abdominal structures, e.g. the root of the aorta, root of the mesenteric arteries
- A fall that is broken into steps may reduce impact velocity and consequently the magnitude of injury may be reduced
- Soft yielding landing surface reduces the magnitude of injury
- Feet-first falls involve a relatively small area of contact, but deceleration forces can be reduced by flexion at the knees and hips.

In case of **fall of a heavy object on the body**

- What was the object? Details of the object?
- From how much height did it fall?
- On which part of the body did it fall?

In case of **road traffic accident in which the victim was an occupant of the vehicle**

- The size of the vehicle?
- What was the speed?
- The position within the car at the time of impact—Was the victim occupying the driver's seat, front seat or rear seat?
  These details are related to the occupant's risk of hitting the steering wheel/column, dashboard, windscreen, windscreen side column.
- Was the restraint device applied?
- Was the victim wearing a helmet?—Applicable to a 2-wheeler.
- The type of collision/impact—Was it head-on (frontal impact) collision, lateral impact collision, or rollover and ejection?
- What happened to the other occupant(s)? Which injuries did they sustain? What is the state of their health?
- Was the victim entrapped?

INTERPRETATION
- The magnitude of the impact is inversely proportional to the size of the vehicle
- The magnitude of the impact is directly proportional to the speed of the vehicle. A closing speed > 60 kmph → severe injury
- An example of significance of the type of collision: Lateral impact collision does not produce much displacement of the brain due to the presence of falx cerebri decreasing the chances of laceration of brain
- Death of another individual in an accident greatly increases the chances of having sustained severe injury by other individuals involved in the same accident.

In case of **road traffic accident in which the victim was a pedestrian struck by a vehicle**

- The type/size of the vehicle?—Car / van / a light truck / a heavy truck.
- How fast was the vehicle moving?
- Was the victim struck from front or behind?

Free ebooks ==> www.Ebook777.com

- Was the victim intoxicated?
- If it was a run-over accident injurying more than one person, then what happened to others?

INTERPRETATION

– Cars tend to produce mostly lower extremity and pelvic injuries (unless the victim is a child)
– Vans and light trucks tend to produce abdominal and thoracic injuries
– A closing speed > 30 kmph ⇒ strong possibility of severe injury.

In case of **gunshot trauma**

- Was it accidental / attempted homicidal / attempted suicidal?
- The type of gun – handgun / rifle / shot-gun?: – Calibre?
  ↓ – Semiautomatic or automatic?
  - Magnum-load or standard load?
  - In case of a revolver: Single-action or double-action?
- From how much range was the victim fired? Was the range > 30′ by any chance?
- The type of bullet?—Was it particularly 'soft-point' / 'hollow-point'?

INTERPRETATION

– The larger the caliber, the larger the missile that is fired
– Logically, a (fully) automatic firearm can inflict much more damage as compared to a semiautomatic one
– Magnum loads have a larger amount of gunpowder than standard loads for a given caliber of gun, and consequently produce severe injury
– A double-action revolver may inflict more damage than a single-action as the assailant may get more time to fire more number of bullets with a double-action revolver
– In majority of civilian shootings, the assailant and victim are usually separated by < 30′. At that distance, impact velocity is approximately the same as initial muzzle velocity. At greater distances, however, several factors can affect the external ballistics of the bullet.

In case of **crushing (and devitalized) wound**

- What was the cause—Industrial or RTA?
- What must have been the weight of the job (the industrial equipment under which crushing occurred)?
- What was the area of the crushing force?
- Duration of impact?

HISTORY to fix the anatomical sites/regions and (pathological) effects of injury particularly those which can be life-threatening or associated with significant morbidity

- Amount of bleeding that occurred before first aid began?
  – Approximate number of teaspoonfuls / tablespoonfuls / glassfuls
  OR
  – How much area of floor or cloth (such as bed sheet, towel, etc.) was stained/soaked with blood?
- – (Complete) loss of consciousness / transient clouding of consciousness / confusion?
  – Headache?
  – Giddiness, drowsiness, mental confusion?
  } *Intracranial injury*
- – Pain in the neck? Difficulty in moving the neck?—*Cervical spinal injury*.
  – Pain, worsened by flexion of the neck?
  – Odynophagia?
  – Dysphagia?
  } *Cervical esophageal injury*
- – Hoarseness of voice, dysphagia—may be due to *blunt injury of soft tissues of the neck*.
- Hemoptysis? ⇒ *Injury to the lung (including contusion)*.
- – Pain in abdomen?—Exact site?
  – Any pain in the shoulder?
  – Nausea? Vomiting? Hematemesis? Melena? Diarrhea?
  } *Abdominal injury*

www.Ebook777.com

– Did you pass urine or not, after the injury?
– Difficulty in passing urine / increasing distension of the lower abdomen? } *Urogenital injury*
– Hematuria?—Getting better, or increasing?
– Thirst? Dyspnea? Fainting? ⇒ *Hemorrhagic shock*.

- – Pain, disability, deformity, swelling in the limb?
  ↓
  Intensity?
  – Is it severe agonizing continuous bursting pain, radiating down the course of an artery? } *Arterial injury*
  – Weakness?
  – Coldness and deadness of the limb?

ROUTINE HISTORY

- Review of systems.
- Obstetric. Gynecologic, and contraceptive history in appropriate details.
- Any long-term medication(s) on which the patient has been?
- Past history:
  – Details of previous injuries, if any.
  – Childhood illnesses.
  – Adult illnesses with dates: Medical, surgical, and details of hospital stay if any.
- Addictions—smoking, alcohol, other?
- Hypersensitivity?
- The time of the **last meal**?

PHYSICAL HEAD-TO-TOE EXAMINATION and inference therefrom

Head and face

– Smell of breath:
  - Particularly of alcohol?
– Scalp:
  - Any acutely bleeding scalp wound?
  - Bogginess in the temporal region?
– Obviously visible or/and palpable # of the vault of the skull?
– Ecchymosis in the mastoid/suboccipital region?
– Neck—tenderness, rigidity?
– Ear: Blood/CSF?
– Nose:
  - Blood/CSF?
  - Septal deflection?
  - Septal hematoma?
– Mouth:
  - Profuse bleeding? Is the blood rather watery (due to dilution with CSF) or mixed with brain matter?
  - Tongue injury?
  - Any hematoma in the lingual sulcus/buccal sulcus?
  - Any aspirated foreign body?
  - Retropharyngeal hematoma?
  - Teeth: Inspect, tap, rock.
– Derangement of dental occlusion?
  Downward and backward pull of the symphysial region?

Free ebooks ==> www.Ebook777.com

- An unstable bilateral fracture of the anterior part of the mandible?

  Immediate therapeutic measure to be taken if it is causing upper airway obstruction: Grasp the tongue with a towel clip and pull it forward gently OR insert a large suture through the dorsum of the tongue and pull it forward gently. Subsequently consider definitive measures for sustained airway control.
- A fracture of the maxilla with gross posterior displacement?—may → acute airway obstruction.
- Any other facial bone #?
- Injury to the cheek?
- LOC?
- Eyes: Ptosis, proptosis, black eye, subconjunctival bleed, pupils, visual acuity, movements of the eyeball, corneal reflex, and power of orbicularis occuli? ↓ Size, equality and reactivity to light?

Neck

While maintaining axial immobilization, remove the cervical collar gently and examine the neck.

- *In case of massive external bleeding*, attempt to achieve a temporary hemostasis by external pressure or direct digital control by insertion of a gloved finger through the wound. Do not clamp anything blindly.
- Skin abrasions on the neck associated with the use of a shoulder harness ⇒ **possibility of a blunt carotid injury**.
- Overlooked occult bleeding from seemingly innocuous penetrating wounds is a dangerous pitfall.
- Assess the penetrating injury if any, for platysma penetration **but do not probe**.
- Venous distension?
- Tracheal deviation?
- Palpate the clavicle to r/o fracture. *Clinical method*: Look for obvious deformity, tenderness, step-off. Grasp the clavicle between the fingers hooking the bone from above (in the supraclavicular fossa) and the thumb form anteroinferior aspect, and very gently push and pull from before backwards and up and down.
- Run the fingers gently down the back of the neck palpating each neural spine.
- Search for tenderness in the associated longitudinal muscles.
- Wounds at the base of the neck may produce unexpected sudden and massive bleeding into the mediastinum, pleural space or an adjacent tracheal wound.

Chest

Look for: retraction, splinting, paradoxical movements of the chest wall, deformity and wounds.
Feel for: Tenderness, deformity, crepitus.

- Paradoxical movements of a part of the chest wall ⇒ flail chest.
- Tenderness and deformity may be indicative of fracture of rib(s)/sternum.
- Marked crepitus ⇒ subcutaneous emphysema.—Causes: pulmonary parenchymal injury / bronchial injury / esophageal injury.
- Causes of reduced breath sounds (on auscultation): Pneumothorax, hemothorax, and if the patient has been intubated, then intubation of the right main bronchus.

Abdomen including pelvis

The key objective of physical examination is to identify the need of surgical intervention. The precise determination of organ injury may not be necessary.
The primary aim is to clinically determine intra-abdominal bleeding or/and peritoneal irritation.
Inspection:

- Halted, labored breathing?—may be indicative of diaphragmatic injury, upper abdominal injury
- Mark/s of external injury?

  Small areas of abrasions/contusions/ecchymoses should not be disregarded as they may represent warnings of significant intra-abdominal injury

  Posterior ecchymosis should raise the possibility of retroperitoneal injury.
- Imprint of clothing or injuring agent?
- Distension?
- Fullness in a specific region?

www.Ebook777.com

Palpation:
– Tenderness and guarding/rigidity ⇒ significant intra-abdominal injury provided fallacies are ruled out.
  Extra-abdominal causes of tenderness and guarding mimicking peritoneal irritation: Fracture ribs (only guarding, no tenderness although), fracture pelvis, and soft tissue injury of the anterolateral abdominal wall, e.g. contusion.
– Depth of penetrating wound, if any?

Percussion:
– Areas of dullness?
– Fixed / shifting dullness?
– Obliteration of liver dullness?

Auscultation:
- Try to hear for peristaltic sounds over the abdomen as well as over the chest for 2 minutes.
- Are the peristaltic sounds present and normal, or diminished/absent?
- *Examine the pelvis and external genitalia*

– # pelvis?
– Blood at the external urethral meatus? ⇒ urethral injury.
– Ecchymosis/hematoma in the perineum?
– DRE: Look for integrity of the rectal wall, position and mobility of the prostate, and any blood on the finger-stall.
  Pubic or pelvic crest tenderness, pelvic instability or perineal hematoma should raise the suspicion of a major pelvic fracture.

NGT aspirate?—Aspiration of blood through a NG tube is usually an indication for laparotomy.

Examine the spine.

Record abdominal girth.

In hemodynamically unstable patients, it is necessary to determine rapidly whether there is significant intraperitoneal bleeding.
Hemodynamically stable patients also require objective evaluation of the abdomen. Focused abdominal ultrasound scanning may be used as a screening tool to identify fluid within the peritoneum.

Limbs

Inspection:
– Asymmetry
– Attitude
– Shortening
– Deformity
– Swelling / depression
– Relation of bony points
– Skin. Wounds.

Tenderness**: General: First with flat of the fingers, very gently; then, with tip of a finger, for exact localization.**

Palpation of soft tissue:
– Swelling?
– Any gap in the soft tissue?
– Crackling-type of crepitus? ⇒ subcutaneous emphysema.

Palpation of bones

Specific objective is to look for fracture.
– Examine for tenderness overlying bones, joint-lines and points of attachments of ligaments.
  Direct methods of eliciting tenderness: Palpating with finger / by tapping.
  Indirect methods:
  - Longitudinal pressure/pull, for examples of digits, metacarpals.
  - Springing / Compression / Separation of two bones, e.g. forearm, leg, (and also in ribs, pelvis).
  - Rotation of bone.
– Irregularity.
– Deformity.
  Various types of deformities in fracture: (a) Angulation—Anterior / Posterior / Medial / Lateral. (b) Rotation —Medial / Lateral. (c) Lateral displacement—Anterior / Posterior / Medial / Lateral.

Free ebooks ==> www.Ebook777.com

- Abnormal mobility.
- Abnormal sounds:
  - Click ⇒ intra-articular cartilage injury.
  - Coarse grating ⇒ bony injury.
  - Fine grating ⇒ (nonarticular) cartilage injury.
- Measurements. Fracture usually leads to shortening; rarely to lengthening.

Specific local signs of fracture

- Excruciating localized tenderness.
- Deformity. Causes/mechanisms—Spasm / direction of original force / force of gravity / extravasation of blood.
- Abnormal mobility.
- Crepitus.
- Loss of function.

Palpation of joints

Specific objectives are to look for strain / sprain / subluxation / dislocation / fracture-dislocation.

- Swelling due to effusion.
- Deformity.
- Restricted mobility.
- Movements. Impairment of function, requiring support?
- Special tests—to demonstrate the absence of head in normal position; and the presence of head in abnormal position.

Specific local signs of dislocation

- Excruciating localized tenderness.
- Swelling (effusion).
- Deformity.
- Restricted mobility.
- Impairment of function, requiring support.

Muscles and tendons

Rupture of the belly of a muscle usually causes little disability, but rupture of tendon or the junction of tendon with muscle is usually (functionally) serious. Tendon injuries most commonly involve the hand.

Assessment of muscle power on the MRC scale

– Not even a flicker of contraction	0
– A flicker of contraction, but no movement	1
– Movement with gravity eliminated	2
– Movement against gravity	3
– Movement against added resistance	4
– Normal power	5.

Peripheral blood vessels

- Discoloration?
- (Skin) Temperature?
- Tenderness?
- Consistency?
- A diffuse swelling in the region of injury s/o a hematoma?
- Sensation?
- Pulsations?
- Function (of the muscles)?

www.Ebook777.com

– Assess the neurological status in c/o suspected carotid artery injury.
– Bruit? Systolic murmur? Continuous machinery murmur?

Clinical grade of acute ischemia?

Examine **peripheral nerves**

Hand

– Wound/s?
– Position of the hand?
– Fracture?
– Dislocation?
– Circulation?
– Tendon injury?
– Nerve injury?

Spine

Clinical features which suggest the possibility of a spinal injury: 1. Injury to the head and face. In patients suffering from head/face injury, the presence of a cervical spinal injury must be assumed unless proved otherwise. 2. Severe pain in the occipital region, shoulder or arm. 3. Weakness / numbness in the limb. Maintaining axial spine alignment, 'log-roll' the patient on the side and examine the back and spine in details. Specifically look for bruising, swelling, tenderness and step-off.

Specific points in examination of the wounds

Pain and bleeding often make examination difficult.

Have any deep structures been divided by the injury?

More complications occur simply because the clinician has failed to think about what structures might have been involved than because of any technical shortcomings in the operation.

Stab wound

Define the end of the wound track. Inability to define it is an indication for exploration of the track; in case of chest/abdomen the exploration may amount to a thoracotomy or laparotomy respectively.

Firearm injury

Examine the entry wound, exit wound if present, and tenderness in between.

- If the entry and exit wounds are small and there is not much tenderness in between, it is probably a low velocity injury.
- If the exit wound is large, the limb grossly swollen, the bone much fragmented and if the patient is shocked, s/he has been fired by a high velocity weapon.

Examine the perineum and rectum

ATTEMPT AT ARRIVING AT A CLINICAL DIAGNOSIS

PRINCIPLES AND PITFALLS

– Severe injuries may not be obvious initially.
– (Often delayed) Neurological deficit may result from initially missed carotid artery dissection.
– With blunt trauma of the abdomen, there are more diagnostic and decision-making challenges as compared to those with penetrating abdominal injury.
– Blunt trauma may lead to a significant life-threatening intra-abdominal injury.

Free ebooks ==> www.Ebook777.com

Indicators of possibly serious injury

Mecahnism of trauma / physical finding(s)	*Possible serious injury*
Rapid deceleration	Thoracic aortic disruption
Penetrating injury between nipples and costal margins	Diaphragmatic injury
Steering wheel impact to the abdomen	Duodenal and pancreatic injury
Fracture of lower ribs	Liver / Spleen injury
Injury to the back	20% chance of significant renal injury
Ecchymosis on the posterior abdominal wall	Retroperitoneal injury

Some injuries are known to occur in combination for anatomical reasons. If one such injury is obvious, look specifically for the other.

Markers of nonaccidental injury (may be abuse)

– Repeated hospital visits for minor injuries.
– Multiple visits to different hospital.
– Vague/inconsistent history from the child's parent/guardian.
– Bite marks or cigarette burns.
– Multiple fractures of different ages.
– Perineal injury.
– Intracranial injury without a h/o RTA.

Hypovolemic Shock

– Hemodynamic condition of a trauma patient particularly who is shocked, is never static. It keeps changing from moment to moment. It may get better or worse or initially better and then catastrophically worse.
– Shock usually develops slowly over hours, although it can develop rapidly.
– Symptoms may be out of proportion to the volume of blood lost. A small loss may occasionally cause severe shock and vice versa.
– A single sign may not be reliable, so look at many.
– BP recordings may be unreliable. For example, the BP of a child/young adult may not fall at all initially, until it finally falls catastrophically, when the patient has lost a third or more of blood volume.
– **A rising pulse rate is an early and more reliable sign of continuing blood loss. But even pulse may not rise until late, particularly if the patient is old.**

STEPWISE ANALYSIS OF DATA

Step 1: **Mode** (/ intention), **cause**, and **nature** of the injury?

- Accident / occupational / assault / abuse / attempted homicidal / attempted suicidal / bite?
- – Assault with fist/kick blows, club, stick, rod, chain, etc.
  – Sports-induced injury
  – Fall while walking
  } mostly cause blunt trauma.
  – Assault with a knife, razor, dagger, ice pick, axe, screw driver, glass piece, or any other sharp weapon
  – An arrow
  – A gunshot (pistol / revolver / rifle, etc. with its specifications if known)
  } → Penetrating trauma
  – Other causes: Fall from height, fall of a heavy object on the body, RTA (*syn.* MVA) the patient being an occupant of the vehicle/a pedestrian struck; railway accident, stone throw, etc. may → blunt or/and penetrating trauma.

Step 2: **Anatomical site of injury**?

- Is the injury ***(likely to be) confined to one region/system or likely to be multiple****?*
  A. - Occupational trauma
  - Assault with fist/kick blows, club, stick, rod, chain, etc.
  - Injury with a sharp cutting or sharp pointed weapon
  - Human/animal/insect bite
  - Fall while walking
  } Usually cause injury in one region of the body only

www.Ebook777.com

B. - RTA, runover accident
- Railway accident
- Airplane crash (This may also cause burn injury)
- Fall from height
- Fall of a heavy object on the body
- Bullet / missile / blast injury

} Usually cause polytrauma (multiple injury)

- ***Possible sites*** of injury:
  1. At the site of impact
  2. Opposite (often diagonally opposite) to the site of impact—'contre coup'
  3. Due to sudden vigorous muscular contraction
  4. Keep in mind possibilities of some combined injuries.

Examples of (particularly difficult) combined injuries

– Head/face injury and cervical spinal injury. The patient may be unconscious from head injury and so unable to complain of pain in the neck/shoulder/arm.
– A head injury or any other obvious severe injury or an external gruesome injury even if not life-threatening, and abdominal injury. An unconscious patient (from head injury) will not be able to complain of pain in abdomen. An obvious severe injury or a gruesome injury anywhere else may distract the attention from abdomen (particularly in blunt trauma).
– Multiple injuries and a hemothorax. A hemothorax may not be noticeable on the initial X-ray. If a patient has multiple injuries, watch for a hemothorax over a week or more.
– Chest injury and injury to the thoracic spine.
– Fracture pelvis and rupture of urethra ± rectum.
– Fracture pelvis and fracture of lumbar spine and paraplegia.
– Fracture femur and (ipsilateral) dislocation of the hip.

Step 3: Pathological effects of injury?

Local ↓
– Blocking of a normal passage
– Increased tension in closed compartments like thoracic cavity, cranial cavity, muscular compartments of limbs
– Bleeding: Internal / External / both
– Displacement / twisting / compression
– (Traumatic) Edema
– Extravasation of excretions/secretions like urine, etc.
– (Surgical) Emphysema
– Concussion of delicate tissues like brain for example
– Irritation of tissues, e.g. convulsions
– Specific effects pertaining to specific tissue injured, e.g. ischemia, spasm, etc.
– Partial/complete, temporary/permanent loss of function of the tissue/organ injured

General ↓
– Shock. Grade of hemorrhagic shock?
– General effects due to injuries to vital structures
– Embolism: Air / Fat / Pulmonary
– General effects due to specific / nonspecific infections
– Traumatic fever
– Traumatic delirium

The effects of injury are likely to be severe, if the patient has been trapped or was not on guard or was intoxicated, OR has pre-existing general ill health / bleeding disorder / aneurysm, etc.

Hemorrhagic shock

- *Injuries which in themselves do not cause hemorrhagic shock*
  – 'Minor' injuries.
  – Head injury. Exception: A significant intracranial bleeding in neonates and infants can cause hemorrhagic shock.
  Note: A patient with scalp laceration leading to significant bleeding and hemorrhagic shock may not have an associated brain injury.
  – Maxillofacial injury.

Free ebooks ==> www.Ebook777.com

- Degree of shock?
  For making the diagnosis and determining the degree of shock, *do not rely on any one sign alone; look at as many as possible*.
- Does the degree of shock match the estimated volume of blood loss? If not, i.e. if the degree of shock appears to be more than the estimated volume of blood loss, there are 2 possibilities. (a) The patient probably lost more blood externally at the scene of injury, OR (b) The patient has lost blood into the abdomen or chest.

If patient has tachycardia and hypotension with only a small wound, suspect some serious internal injury, or if a day or two has passed since traumatic event, some massive infection or gas gangrene.

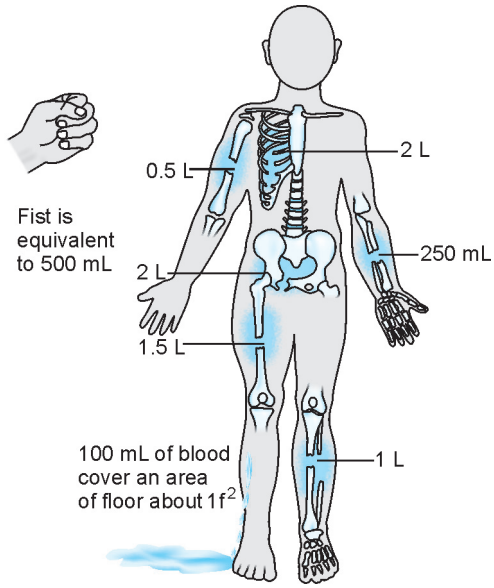


Fig 56-7 Estimating blood loss

Wounds prone for tetanus

– Deep puncture wounds, burns, injuries of the leg, thigh, buttock or axilla.
– Heavily contaminated wounds, especially crush injuries, and wounds where there is much injured muscle, especially if they occurred on cultivated land, or if dung has been applied to the wound.

Wounds prone for gas gangrene

– Wounds with extensively lacerated muscle, missile wounds
– Large wounds with necrotic tissue, e.g. battle casualties, severe comminuted compound fractures sustained in road traffic or railway injuries, agricultural tractor injuries, etc.
– Wounds that are grossly or deeply contaminated with soil.

} Especially, if they involve buttocks, thighs or axillae.

– Laceration of retroperitoneal muscles associated with an injury to the colon.
– Foreign body implanted in the depth of a puncture wound, e.g. shrapnel, etc.

Step 4: Medicolegal aspect

A. Determine (and document) whether the injury is **simple, dangerous (severe) or grievous**.
B. Remarks, if any.
   Examples—Injuries and stains on the clothing, if any?
   – Do the injuries appear to be fabricated?
   – Probable age of the injury (in the absence of any cause/s responsible for delayed healing)?—This is applicable to some nonvisceral (essentially superficial) wounds, in case if the patient presents for consultation after a delay of a few hours to a week or two from the time of infliction of the wound.

Step 5: Are there any factors responsible for delayed wound-healing?

– These are similar to factors responsible for delayed healing of chronic ulcer. Refer to chapter 42.

Step 6: Document the severity of injury using a standardized scoring system.

This job may be more conveniently done on discharge of the patient or autopsy. This documentation helps in audit of trauma patients.

Points of medicolegal importance, which must be documented

1. The patient **brought by** …….

If brought by a police personnel	If brought by a relative / friend / onlooker
↓	↓
Note down the name, identification number (Registration No; I-card No, etc.) and the police station to which the personnel belongs to	Note down the name, addresses, phone numbers and intimate same to the police station

www.Ebook777.com

2. **2 identification marks** of the injured.
3. **Appropriate sketches and diagrams** with **measurements of all wounds in 3 dimensions**.
   The 3rd dimension viz. the depth should be mentioned in tissue unit (and not in centimeters or inches), e.g. subcutaneous tissue deep / fascia deep / muscle deep / bone deep, etc. The base of the defect should be considered for the depth.
   When a wound is bone-deep, a mention must be made about whether a fracture is visible or palpable clinically or not. A crack fracture of a curved bone is usually visible or palpable if the wound is bone deep, but the same may not be appreciably visible on a X-ray film, e.g. fracture of vault of skull.
   If the depth cannot be defined, it should be clearly so mentioned.
   If possible, wounds may be photographed with attached scale.
4. The **weapon by which the wound must have been resulted**. While it may not be possible to name the weapon, it is usually possible to infer the type of weapon from an examination of the wound and clothes.
5. Record the results of clinical tests for peripheral nerves and tendons before anesthetizing the patient for surgical treatment. It will then be certain that paresis/paralysis is not the result (complication) of treatment.
6. Record important relevant negative findings.

PITFALLS IN PEDIATRIC TRAUMA

- For all practical purposes sprains do not occur in children, and if there is pain, swelling and loss of function, a fracture is nearly always present. In children, the ligaments are far stronger than the bones to which they are attached.
- Diagnosis of nerve injury is difficult in a child who is often frightened following injury and unwilling to comprehend.
- Neurapraxia with temporary loss of function may mimic transection.
- Tendons may be incompletely severed but have the appearance of intact function on clinical examination.
- The major problems with an injured hand arise from failure to diagnose the extent of the injury when the child is first seen. In the absence of a fracture, any disturbance of the normal posture of a finger in relation to the other fingers indicates a tendon injury.
- Major intrathoracic injury can occur without fracture of ribs.
- In the first 24 hours after reduction of a fracture, the patient should suffer no more than discomfort. The limb should be much more comfortable than it was before the fracture was treated.
  Causes of persistence/presence of pain beyond 24 hours after reduction of a fracture:
  1. Too tight a plaster. The fingers become swollen and blue.
  2. The plaster causing localized pressure: Local pain is present, usually over a bony prominence.
  3. Arterial ischemia.

Clinical features which should raise an index of suspicion for a battered baby/child

- Age < 3 years (i.e. before speech is sufficiently developed to constitute a deterrent).
- Variable h/o of the supposed accident.
- Denial or distorted information about previous injuries.
- Unusual modes of presentation:
  - Unexplained fractures especially when multiple
  - Multiple soft tissue injuries
  - Symmetrical burns/scald in unusual sites
  - SDH, particularly when bilateral
  - Failure to thrive, with spectacular gain in weight when admitted to the hospital for management
  - A peculiar defensive posture
  - Abnormal behavior of the child in hospital.
- An abnormal/unusual attitude in the parents to the injury', e.g. lack of affect, indifference, panic, etc.

Free ebooks ==> www.Ebook777.com

Diagnosis of battered baby/child depends on an index of suspicion. Final confirmation may come from the findings of a social worker, from an eye-witness, or ultimately from an admission by the person concerned.



Fig 56-8 Well-equipped ambulances affiliated to the trauma ward, ready to take a 'van call' 24 × 7—should prove a nostalgic photograph for the past students of L T M Medical College and General Hospital, Mumbai!

www.Ebook777.com

57 (ACUTE) HEAD INJURY

= injury to the scalp / vault of the skull / base of the skull (± cranial nerves) / the brain—in isolation or in combination(s).

Only those injuries to the head are designated as 'brain injury' in which there is a loss of consciousness or some deterioration of the level of consciousness at the least.

A brain injury may occur without (a significant) injury to the scalp/skull (e.g. cerebral concussion, cerebral laceration in a whiplash injury, etc.). Similarly, injuries of the scalp/skull/face however ghastly looking, may not be associated with brain injury.

DEFINITIONS

Simple fracture of the vault of the skull is one in which the scalp is not breached, but there may be bruising or grazing as distinct from a full thickness laceration.

Unconsciousness (Complete loss of consciousness)

= deterioration of the level of consciousness to such an extent that the patient remains unarousable even after application of painful stimuli. It implies a potentially life-threatening event.

Criteria in terms of Glasgow Coma Score: 8–3/15.

Coma = a prolonged and perhaps profound state of unconsciousness from which the patient cannot be aroused.

GENERAL REMARKS

1. Bleeding from open scalp wounds must be controlled immediately since it is profuse and seldom stops by itself. Scalp is very vascular, and torn blood vessels cannot contract and retract as their walls are fixed to fibrous tissue stroma in the subcutaneous layer of the scalp. The great vascularity of the scalp indicates that wounds heal well, and areas which initially seem necrotic may in due course prove to be viable and heal.
2. The absence of a fracture does not exclude life-threatening brain injury, particularly in young children. Plain X-ray will miss many fractures, particularly of the skull base. Poor films are useless. Even good ones are difficult to interpret. Most patients with EDH have a fracture skull, but not vice versa.

   Indeed EDH is more common in the presence of a simple liner facture than with depressed fractures.

   Fractures of base of the skull are the result of skull distortion, and indicate that a considerable force was applied to the skull at the time of impact.
3. Cranial cavity is a closed cavity where expansion is not possible.

   Either of the supratentorial compartments is usually the main area affected in injury where bleeding and edema increase the pressure. As skull and dura cannot expand, increased pressure displaces out the CSF which has minimum pressure. If the pressure increases still further veins are compressed leading to congestion in brain tissue as arteries still continue to supply the blood. Further increase in pressure compresses the capillaries and ischemia of brain occurs. While the part near the expanding contusion or bleed bears the maximum brunt of pressure, part farther away experiences a little less pressure so when the adjacent part is congested, part farther away may not be compressed. When the part immediately adjacent to the expanding contusion or bleed becomes ischemic, part farther away becomes congested, and finally both become ischemic. This is the mechanism of pupillary reaction and changes on either side.

   Congestion of a part of the brain produces symptoms of irritation of that part, i.e. if motor area is congested, convulsions will result while ischemia of a part of the brain produces symptoms of paralysis of that part so ischemia of motor area will result in paresis/plegia.

Free ebooks ==> www.Ebook777.com

When pressure increases further in the supratentorial compartment, cerebrum is pushed towards the opening in the tentorium and part of cerebrum near the tentorial opening (temporal lobe) herniates into the infratentorial compartment. The space is limited in the opening which is occupied by midbrain and III cranial nerve lying on its surface, so that the temporal lobe compresses the III nerve and first produces symptoms of irritation resulting in constriction of the pupil on the side of the lesion and then produces symptoms of paralysis leading to moderate dilatation, loss of light reflex and finally full dilatation of pupil. Late by one stage, the opposite III nerve follows the same suit. Pressure on midbrain → unconsciousness and decerebrate type of rigidity.

If pressure increases still further or if there is subtentorial bleed (posterior fossa hematoma), then pressure will increase in the infratentorial compartment leading to displacement of cerebellum, tonsil of cerebellum being displaced inside the foramen magnum. The lower part of medulla oblongata extends in foramen magnum and will get compressed. Patient becomes deeply unconscious with bilaterally dilated pupils. Vasomotor and respiratory centers in the medulla become congested due to pressure obstruction of the venous return.

Congestion leads to irritation of centers which produce deep and fast respiration with increased blood pressure. Raised BP increases the intracranial bleed which further increases the pressure on medulla leading to further congestion and eventually ischemia of the vital centers leading to death. A diagnostic lumbar puncture is likely to precipitate the herniation of cerebellar tonsil, and therefore contraindicated when a rising ICP is suspected.

4. Even an apparently mildly injured patient can deteriorate suddenly.
5. If patient has more serious injuries elsewhere, head injury can usually wait 12–18 hours (with respect to surgical treatment if indicated).
6. *If at all* surgery is indicated in a case of a simple depressed fracture of vault of the skull, it may be delayed for 2–3 days.
7. Meningitis as complication of a basal skull fracture may occurs after the first 48 or 72 hours.
8. If epilepsy occurs 'very late' (e.g. 10 years or so) after a head injury it may be wiser to investigate the patient as a case of 'late onset epilepsy' rather than ascribe it to trauma (which occurred many years ago).

After having completed preliminary management as per the 'ABCDE' protocol, and cervical spinal injury having been ruled out or stabilized, the case of head injury is further evaluated in the following way.

SPECIFIC HISTORY

Source of the history: – Patient himself/herself if conscious and 'alert enough to give a coherent account'.
– Otherwise, an onlooker / a relative.

How did the injury occur, i.e. mode, cause and nature? Exact time?

Acceleration/deceleration — Blunt — Penetrating — Perforating

- High velocity, e.g. a RTA, high velocity bullet injury, etc.
- Low velocity, e.g. stumbling while walking leading to a fall in the street, etc.

- Any injury to the scalp? – Open Or Closed?
  ↓
  How much area of floor or cloth (such as bed sheet, towel, etc.) was stained/soaked with blood?
- E/N/T bleeding?
  - Amount?
  - Is it rather watery?
- Any discharge of watery fluid (CSF) from the ear or nose?
- 'Salty taste' in the mouth (suggesting discharge of CSF in the throat)?

} These points are rather more applicable to a subacute/chronic case.
↓
- Duration?

- (Complete) loss of consciousness / transient clouding of consciousness / confusion?
  If yes, Did it occur soon after the traumatic event or did it commence later on?
  - Duration of deteriorated level of consciousness?
  - Is unconsciousness continuous since its occurrence?

  OR

  Was consciousness regained for a while in between before falling unconscious again ('Lucid interval')?
  - Duration of the lucid interval?

www.Ebook777.com

  - Had the patient consumed alcohol / any other narcotic drug?
  - Is the patient known to have been suffering from DM / uremia / any other disorder which can predispose to suddenly loosing consciousness and fall down?
- – Do you know/remember what happened next after sustaining the injury?
  – How were you transported to the hospital? – Which vehicle, the person accompanying, etc.
  } (— for post-traumatic amnesia.)
  - Do you remember the events before the injury occurred? (—for retrograde amnesia.)
- Vomiting?
  - Number of bouts?
  - Is it persistent?
- Hematemesis?
  Number of bouts?
- Convulsions?
  - Details—Unilateral OR bilateral?
    ↓
    Do they start in fingers / toes / one forearm or leg / one side of the face?
- Headache?—Persistent and progressive in intensity, OR getting better?
- Giddiness, drowsiness, mental confusion?
- Any difficulty in vision?
- Fever?—Time of onset after injury?
- Sneezing?

INTERPRETATION OF SOME OF THE SYMPTOMS

Mechanism of injury may help to take a judgment of possible pathological effect and consequently anticipate finding on imaging investigation.

Examples

- A high velocity blunt trauma may → diffuse axonal injury
- A low velocity blunt trauma may → concussion / contusion.

Amnesia

- Post-traumatic amnesia = the period of permanent amnesia occurring after brain injury.
- A retrograde amnesia for anything longer than a few minutes signifies serious brain damage. It is of less value than post-traumatic amnesia as a prognostic indicator *wrt* functional outcome, and may improve with time.

Convulsions

- Unilateral fits may be suggestive of middle meningeal hemorrahge. Fits may start in fingers / toes / one forearm or leg / one side of the face depending on the site of irritation (motor area) of the cerebral cortex.
- In bleeding from the superior sagittal sinus, fits are usually bilateral.

Persistent headache, giddiness, drowsiness, mental confusion, apathy ⇒ possibility of intracranial hematoma.

Sneezing suggests aerocele (pneumatocele). Aerocele = an air-filled cavity in the brain connecting with one of the sinuses, usually the frontal sinus. This is associated with a great danger of meningitis.

EXAMINATION

Head may have to be shaved and washed clean if adequate examination is to be carried out without missing any wound. This is particularly essential in railway accidents, RTAs.

- **Smell of breath**: - particularly of alcohol?
- **Vital parameters**

Specifically look for the following points:

- Fever in general?
- Hypothermia?
- Feel for temperature in all 4 limbs.
- Tachycardia / Bradycardia?
- Deep and fast respiration? Chyne-stokes breathing? Ataxic breathing? Stertorous breathing? Apnea?
- BP - ↓ / ↑?

Free ebooks ==> www.Ebook777.com

Temperature is usually subnormal in concussion.
Fever occurring 'early' after trauma denotes injury to the midbrain.
Temperature reaching as high as 108°F ⇒ pontine bleed.
Temperature of up to 101°F ⇒ intracranial bleeding.
Temperature is usually higher by 1 or 2 degrees in the paralyzed limb as compared to the opposite (normal) side.
Fever occurring in later stages may be due to infection.
Hypothermia may suggest pituitary failure.

Rapid and thready **pulse** and reduction in **BP** ⇒ cerebral concussion / decompensation from persistently rising ICP. Therefore, rapid and thready pulse and reduction in BP in a deeply unconscious patient heralds impending death.
Tachycardia and profound fall in BP may also suggest acute pituitary failure, provided hemorrhagic shock has been ruled out.

Cerebral concussion → slow and shallow **respiration**.
Slowing pulse rate, respiration becoming deep and fast, and rising BP may be suggestive of increasing ICP, which if not corrected will progress to coning (—herniation of cerebellar tonsil through the foramen magnum causing pressure on vital centers in the medulla).

Chyne-stokes breathing ⇒ injury on both sides of cerebral hemisphere / diancephalon / upper midbrain.

Ataxic breathing ⇒ injury at medullary level.

Stertorous breathing (lips and cheeks blowing in and out with each breath and loud snoring noise caused by obstruction to the airway by the relaxed soft palate and falling back of the tongue) ⇒ grave prognosis.

Causes of apnea ⇒ major, probably fatal brain injury / phrenic nerve palsy (due to cervical spinal injury).

- **Scalp wounds**

OR

Closed?

Contusion / Hematoma?

↓

Note: Shape, size, consistency and mobility

↓

– Is it circular and relatively small in size?
OR
Does it extend all throughout the scalp?
OR
Does the shape and size correspond to the underlying bone?
– Does the peripheral margin appear to be indurated?
– Can the indurated margin be indented by application of pressure?

Open?

IW / CLW / Avulsion?

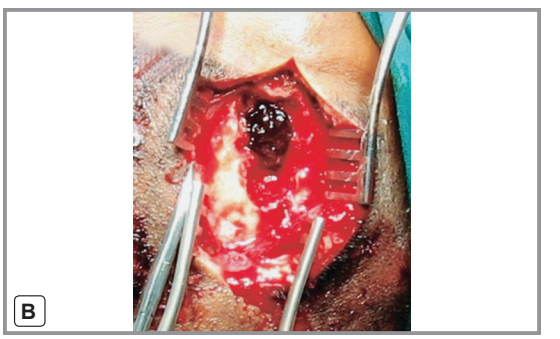
↓

CLWs often appear as IWs as tightly stretched scalp over the skull breaks in linear fashion
Hence, it is very important to document the weapon with which the injury was inflicted, for future medicolegal reference

– Is the wound gaping?
A gaping wound ⇒ division of galea aponeurotica
– Look and/or feel for a fracture of the underlying skull. **If a fracture is clearly seen and/or felt through an open scalp wound, document it**. This point is of medicolegal importance. A fracture that may be clinically diagnosed through an open scalp wound may not be visible on an X-ray

– Any **boggy swelling in the temporal region?**
This may suggest middle meningeal bleed (leading to an EDH), the blood coming out through the fracture and collecting under the temporal muscle
– Determine the possible underlying cortical area of the brain (which might have been damaged) in relation to the position of the wound





Figs 57-1A and B (A) Graze and boggy swelling in the temporal region; **(B)** The patient had an EDH

www.Ebook777.com

- **Vault of the skull**: Fracture?
  Although the site of injury to the brain usually corresponds to the site of injury of the scalp/skull, a contre-coup injury is also possible.
- **Mastoid** and **suboccipital regions**: Ecchymosis? ⇒ fracture base of skull through the posterior cranial fossa.
- **Neck**: Tenderness, rigidity?
  Neck rigidity ⇒ SAH / meningitis / contusion of cervical spine.
  A SAH denotes rupture of cerebral veins and laceration of the brain, thus contraindicating early operation in most cases.
  Rigidity in SAH results from irritation of meninges by the blood.
  Rigidity caused by SAH or meningitis *v/s* rigidity caused by contusion of the cervical spine:
  – The presence of fever ⇒ SAH/meningitis.
  – The absence of fever ⇒ cervical spinal injury.
- **ENT** (including **mouth**)
  Blood/CSF in the ear/nose ⇒ # base of the skull OR a local soft tissue injury.
  Tongue injury may have occurred due to a seizure.
  Profuse bleeding and the blood more watery (due to dilution with CSF) or mixed with brain matter ⇒ # base of the skull.
- **LOC**?
  A Glasgow Coma Scale (GCS) is widely used to measure the level of consciousness in patients with brain injury or illness. It makes no assumptions about the nature of the pathological process. It is a useful tool for measuring LOC and monitoring its trend over time.

The clinical parameter tested		*GCS*
Eye opening:	– Spontaneous	4
	– to speech	3
	– to pain	2
	– None	1
Best verbal response:	– Oriented in time (time, day, date, month, year), place and person = Normal.	5
	– Disoriented in time and place, although otherwise talks in sentences = Confused.	4
	– Utters occasional words rather than sentences.	3
	– Groans or grunts but no words.	2
	– None.	1
Best motor response:	– Obeys commands such as 'put out your tongue; lift up your hand; squeeze my finger'.	6
	– Localizes to pain (stimulus applied to supraorbital ridge/ear lobe) by lifting up one or both upper limbs above the level of the clavicles in the direction of the painful stimulus.	5
	– Withdrawal to pain (stimulus applied to finger nail bed).	4
	– Flexes the limb at the elbow to pain (stimulus applied to finger nail bed).	3
	– Extends the limb at the elbow to pain (stimulus applied to finger nail bed). This is always accompanied by spastic flexion at the wrist.	2
	– None.	1

During examination the motor response may vary. One arm may localize to pain; the other may flex. When this occurs, record the best response; this correlates best with final outcome. Leg response to pain gives less consistent results; hence, response to pain is tested in the upper limbs rather than in the lower limbs.

There are often circumstances where one or more parts of the GCS cannot be assessed, e.g. when both eyes are closed by swelling, or the patient is intubated, or in a young child, or someone with learning difficulties. However, the other components can and should still be assessed.

LOC should be observed repeatedly at appropriate intervals to establish the trend. For example, a seriously head-injured patient in the resuscitation room might need observations every 10–15 minutes. **In a deteriorating patient LOC almost always changes before focal signs develop.**

Incontinence of urine and feces ⇒ complete loss of consciouness.

Free ebooks ==> www.Ebook777.com

- **Posture**?
  - No response to pain at all, i.e. flaccid paralysis with the jaw relaxed—This is a serious situation.
  - Abnormal extensor response = Decerebrate rigidity. ⇒ lesion in the diancephalon, midbrain or pons OR Severe metabolic cause like hypoxia or hypoglycemia.

    The head is extended. The jaws are clenched. The arms are adducted and stiffly extended at the elbows. Forearms pronated. Fingers flexed. The legs are stiffly extended at the knees. Feet are plantarflexed. This posture may occur spontaneously or only as response to external stimuli such as light, or pain.
  - Abnormal flexor response = Decorticate rigidity. ⇒ destructive lesion of the corticospinal tracts within or very near the cerebral hemispheres.

    The upper arms are held tight to the sides. Elbows, wrists and fingers are flexed. The legs are extended and internally rotated. The feet are plantarflexed.
- **Nervous system** as far as the LOC permits—Particularly look for paresis/paralysis and plantar reflex.
  - Assess power in the (muscles of the) limbs if LOC permits.
  - Are there any spontaneous movements of the limbs?
  - Note the effect of painful stimuli.
  - Raise a limb and allow it to drop. The paralyzed limb falls down like a log of wood.

    Paresis/paralysis occurs usually on the opposite side of the intracranial lesion.

    Lesser degrees of impairment of movements of the limbs may be caused by spinal cord injury / nerve root injury / limb #s.
  - Plantar reflex: Scratch the outer border of the foot with a slightly sharp object like the back of the handle of a hammer. Note the movement of the great toe. Normally, the great toe is flexed (flexor response).

    In lesions of the pyramidal tract and in infants (in whom the tract is not yet myelinated) the great toe is extended (extensor response).
- **Eyes**

Ptosis?

Proptosis?

Proptosis may suggest excessive retrobulbar bleeding from fracture base of the skull.

Black eye? = hematoma of the eyelids.

By itself it is not serious, but it may be a part of fracture of base of the skull or maxillofacial injury (eye / periorbital area).

Black eye resulting from # of base of the skull	*v/s*	*Black eye resulting from local injury to the eye / periorbital area*
Ecchymosis: Develops later, usually after 24 hours.		Ecchymosis: Appears soon after injury.
Appears first in the lower lid due to gravitation and then in the upper.		
Its spread around the orbital margin is limited by the attachment of orbital fascia to the orbital margin.		May spread on to the forehead, side of the nose or cheek.
There may not be signs of periorbital injury.		Signs of periorbital injury present.

Subconjunctival bleed?

Subconjunctival bleed ⇒ fracture of base of the skull through anterior cranial fossa.

Criteria:
- Pain is absent.
- Vision is not affected.
- A homogeneous, sharply demarcated, red/bluish-purple area, and its posterior/superior limit are not visible as it is coming from the back of the orbit. The color of the bleed fades over days to yellow, and then it disappears.

Pupils – Size, equality and reactivity to light (present or absent)?—They help in assessing the cause of unconsciousness and in determining the region of the brain that is impaired.

Clinical method for reactivity to light

1. Shine torch-light in the affected eye: Response is impaired or absent in the illuminated eye.
   The contralateral pupil constricts normally (its III nerve being intact).
2. Shine torch-light in the normal eye: The pupil constricts normally.
   Response is impaired or absent in the contralateral, i.e. the affected pupil.

The light reflex tests optic (II) and occulomotor (III) nerve function. Although II nerve dysfunction after head injury is important to record, it is the III nerve function which is the most useful indicator of an expanding intracranial lesion.

www.Ebook777.com

Begin assessment of pupils by looking for equality

Bilaterally equal in size

Reactive to light ⇓
Toxic-metabolic disorder is likely to be the cause of unconsciousness

Fixed, i.e. unreactive to light ⇓
Structural damage is likely to be the cause of unconsciousness

Unequal
Usually fixed, i.e. unreactive to light ⇓
Structural damage, e.g. unilateral large and fixed pupil ⇒ herniation of the temporal lobe, causing compression of the III nerve and midbrain

For additional details on pupils, refer to chapter 11.

Test ***visual acuity***: – For severe deficit:
- Can you see the light, movement?
- Can you count fingers?

– For mild deficit: Record reading acuity with hand/wall chart.

Movements *of the eyeball*
Clinical method
Steady the patient's head and ask her/him to follow an object held at arm's length.
Observe the full range of horizontal and vertical movements. Note any malalignment or limitation of range.
Examine movements in the 6 different directions of gaze representing maximal individual muscle strength.

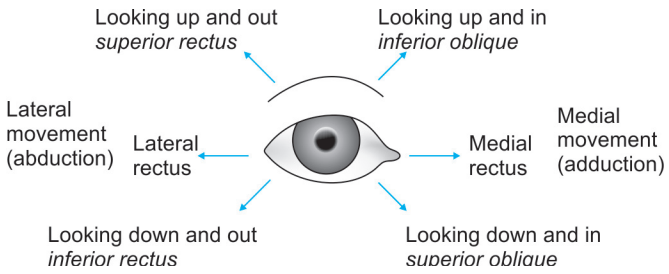


Fig 57-2

Fundi (on fundoscopic examination)
– Papilledema: Usually develops after 12–24 hours. Acute papilledema developing within 2 hours of injury ⇒ grossly elevated ICP which is usually fatal.
– Retinal bleeding ⇒ major head injury. May be seen with subarachnoid bleed/acute SDH.

Corneal reflex
Clinical method
Test corneal sensation by touching with a wisp of wet cotton wool. – A blink response should occur bilaterally. (Afferent route: Ophthalmic division of V. Efferent route: Facial nerve (VII).)
Corneal reflex will be absent, apart from due to cranial nerve injury, in a completely unconscious patient.

Test the ***power of orbicularis occuli*** (Facial nerve).
Inability by the patient to keep the eyes closed tightly while clinician attempts to open them ⇒ paralysis of orbicularis occuli.

- A quick **cranial nerve examination**
  Although the description that follows in the table below is in modest details, not all points will be applicable in a case of acute head injury for reasons such as 1. The patient may not be conscious to comprehend verbal instructions. 2. Wasting of muscles takes some time to develop.

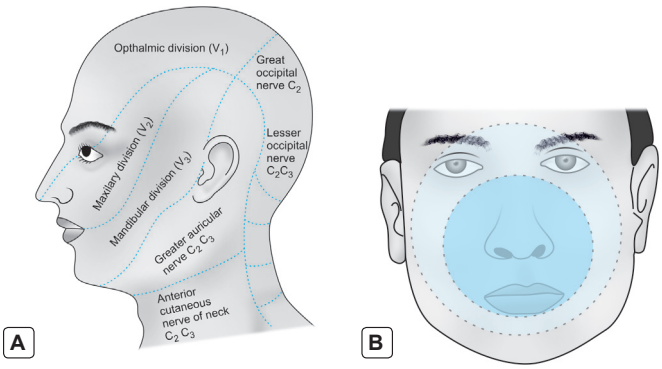
Free ebooks ==> www.Ebook777.com

Nerve	*Origin*	*Exit from the basal foramina of skull (Cranial fossa)*	*Functional assessment*
I Olfactory	Central process of olfactory bipolar neurons	Cribriform plate of ethmoid (ACF)	– *History of anosmia*? – *Test both perception and identification* using aromatic *nonirritant* materials that avoid stimulation of trigeminal nerve fibers in the nasal mucosa (e.g. soap, tobacco, etc.). One nostril is closed while the patient sniffs with the other.
II Optic	Central process of ganglion cells in retina	Optic canal (ACF)	*Test visual acuity*. For more detailed examination, e.g. testing visual fields, ophthalmoscopy, etc; refer the case to ophthalmologist.
III Occulomotor	Between crura of midbrain	Superior orbital fissure (ACF)	*Response of the pupil to light*. Eye *movements* (Look in, up and down). Palsy → – Ptosis. – Displacement of the eye downwards and outwards. – Pupil dilated and fixed.
IV Trochlear	Dorsal surface of midbrain	Superior orbital fissure (ACF)	*Eye movements* (Look down and in). Palsy → impairment of downward movement of the eye.
V Trigeminal Ophthalmic Maxillary Mandibular	Pons Trigeminal ganglion Trigeminal ganglion Trigeminal ganglion	 Superior orbital fissure (ACF) Foramen rotundum (MCF) Foramen ovale (MCF)	Sensory to face and cornea. Motor to masseter and temporalis. – 'Hollowing out' of the temporal fossa? ⇒ wasting of temporalis muscle. – Wasting overlying the masseter? – *Ask the patient to clasp jaws together, and feel* for the contraction of masseter and temporalis muscles. – *Corneal reflex*. – *Sensation on the face:* Test with pin prick and light touch over the whole face. Compare each side and map out the deficit. Does it fit in a root/division pattern or brainstem (onion-skin) pattern?

Contd...

www.Ebook777.com

Contd...



Figs 57-3A and B (A) Root-division pattern; **(B)** Onion-skin pattern

Nerve	*Origin*	*Exit from the basal foramina of skull (Cranial fossa)*	*Functional assessment*
VI Abducens	Pontomedullary junction	Superior orbital fissure (ACF)	*Eye movement* (Look outward). Palsy → inability to look outward.
VII Facial	Pontomedullary junction	Internal acoustic meatus (MCF)	Motor to muscles of facial expression. *Signs of lower motor neuron type of facial nerve paralysis* – Forehead does not wrinkle. – Eye: - Palpabral fissure becomes broad. - Tear does not fall because it fails to enter the lacrimal punctum as the lower lid is not in contact with the conjunctiva. – Lids do not close, and give rise to a blind man's appearance. – Nasolabial fold is vanished. – Ala of the nose does not move. – Mouth: - Lips do not part, pout or purse. - Angle will not rise. - Teeth will not show. - Cheeks will not puff. - Food accumulates in the mouth.
Chorda tympani			*Taste to anterior 2/3rd of the tongue*. Test by placing a small quantity of sugar/salt anteriorly on the appropriate side of the protruded tongue.

Contd...

Free ebooks ==> www.Ebook777.com

Contd...

Nerve	*Origin*	*Exit from the basal foramina of skull (Cranial fossa)*	*Functional assessment*
VIII Auditory Vestibular Cochlear	Pontomedullary junction	Internal acoustic meatus (MCF)	 *Balance.* *Hearing.* – Test by whispering numbers in to one ear while masking hearing in the other ear by occluding the external meatus. – Differentiate conductive (middle ear) deafness from perceptive (nerve) deafness by doing Weber's test and Rinne's test. In conductive deafness, bone conduction is better than air conduction. In nerve deafness, both bone and air conduction are impaired.
IX Glossopharyngeal X Vagus	Medulla oblongata Medulla oblongata	Jugular foramen (PCF) Jugular foramen (PCF)	These nerves are examined together; their actions are seldom individually impaired. – High-pitched voice ⇒ vocal cord paresis (X nerve palsy). – H/o nasal regurgitation, suffocation, coughing while attempting to swallow? ⇒ neuromuscular inco-ordination. – Ask the patient to open mouth and say 'Ah'. Note any asymmetry of palatal movements. – Gag reflex: Depress the patient's tongue and touch palate/tonsil on one side until the patient 'gags'. Compare sensitivity on each side (afferent route—IX nerve) and observe if there is any asymmetry of palatal contraction (efferent route— X nerve).
XI Accessory (spinal)	Medulla oblongata	Jugular foramen (PCF)	Test sternocliedomastoid and trapezius muscles.
XII Hypoglossal	Medulla oblongata	Hypoglossal foramen (PCF)	Palsy → the following clinical features. – Atrophy (increased folds and wasting) of the tongue. – Fasciculations (small wriggling movements) in the tongue. – On protrusion, tongue deviates to the affected side.

Key: ACF Anterior Cranial Fossa. MCF Middle Cranial Fossa. PCF Posterior Cranial Fossa.

www.Ebook777.com

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. An intracranial injury may occur either on the side of the blow to the head (*coup*) or opposite (often diagonally opposite) to the site of the blow (*contrecoup*). Provided it is clear where, and where alone, the head was struck, this knowledge can provide a useful guide to pathology when used in conjunction with the lateralizing signs of a compressing lesion.
   - Scalp injuries, skull fracture, and EDH usually occur only on the side of the blow.
   - Brain edema, contusion, laceration, SDH, and ICH can occur on both sides (*coup* and/or *contre-coup*).
2. Beware of an unimpressive scalp injury that might have been caused by a sharp pointed object (puncture wound); the brain may also have been penetrated.
3. Traumatic intracranial hematoma can occur in patients with no external evidence of injury.
4. A relatively trivial injury causing lateral impact on the head can inflict a fracture on the temporal bone and consequently cause an EDH. In the elastic skull of children the injury may occur without fracture, as it may, but less commonly, in adults.
5. Fractures of skull base are often easier to diagnose clinically than radiologically.
6. The absence of ecchymosis overlying the mastoid/suboccipital region does not exclude fracture base of the skull.
7. Loss of consciousness ⇒ trauma to the cerebrum / lower brainstem (contains reticular formation) and pressure over the midbrain and medulla.
8. Unconsciousness and decerebrate type of rigidity ⇒ pressure over the midbrain.
9. – Deep unconsciousness
– Bradycardia
– Deep and fast respiration
– Raised BP
⇒ pressure on vital centers in medulla oblongata. This may result either from an isolated increase in pressure in infratentorial compartment or increase primarily in supratentorial compartment which later passes on to the infratentorial compartment.
10. Unconsciousness occurring some time (days/weeks) after a traumatic event ⇒ increasing reactionary bleeding (extradural or subdural or both). Rarely, it may be due to ↑ in ICP caused by infection.
11. Deeper the level of unconsciousness, more the intracranial pressure.
12. A period of unconsciousness of > 6 hours ⇒ a 'significant' intracranial injury.
    However, if unconsciousness is maintained for ≥ 36 hours it denotes that intracranial injury is not severe enough to kill the patient.
13. The absence of unconsciousness does not always exclude a serious intracranial injury.
14. Increasing headache complained by a patient who had a 'lucid interval', should be taken seriously. It is the first symptom suggesting an EDH.
15. Vomiting occurring a couple of times is of no particular significance. Persistent vomiting denotes ↑ in ICP.
16. Hematemesis or blood-tinged vomiting may be due to swallowed blood from fracture of base of skull.
17. Hemiparesis/hemiplegia usually occurs in the limbs contralateral to the side of the lesion but may also occur in the ipsilateral limbs.
    Limb deficits are therefore of limited value in localizing the site of lesion.
18. A unilaterally dilated pupil denotes rise in pressure in supratentorial compartment on the same side. However, **rarely** temporal lobe herniation may push midbrain against the opposite margin of tentorial opening thus compressing the opposite III nerve earlier than that on the same side. This gives early dilatation of pupil on the opposite side of the lesion.
19. There are many reasons other than head injury why a patient might have pupils of unequal size or response to light (e.g. local trauma to the eye, previous eye disease or surgery), but after head injury the worst must be assumed and acted upon.

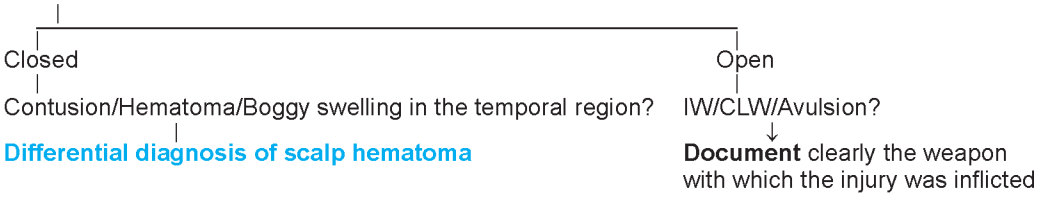
Free ebooks ==> www.Ebook777.com

20. As with the pupils, any asymmetry should be ascribed to the brain injury and appropriate action taken. Paralysis of one arm or leg ⇒ compression of particular part of motor area on the opposite side. Paraplegia ⇒ lesion in the midline over the cortex (/ spinal cord injury).
21. The risk of intracranial hematoma can be judged from 3 features: The period of retrograde amnesia, LOC, and the presence/absence of a skull fracture. Retrograde amnesia over 30 minutes, lower LOC, or the presence of a skull fracture increases the risk.
22. A chronic subdural hematoma is notorious because it is difficult to diagnose.
23. Clinical assessment may not reliably distinguish the type or even the site of intracranial hematoma, but is invaluable in indicating the need for further investigation and in providing a baseline against which any change can be compared.
24. Patients thought to be under the influence of alcohol or other drugs need very careful observation. The assumption that the drug is the cause of deterioration or failure to improve must be resisted. If the alcohol level is < 200 mg% (43.5 mmols/l), then persistent confusion and inability to obey commands warrants an urgent CT scan.

STEPWISE ANALYSIS OF DATA

I. **Mode, cause,** and **nature** of the injury?
If an unconscious patient is brought in, decide whether unconsciousness is the result of head injury or else the patient (first) became unconscious due to some other cause (alcohol, a narcotic drug, diabetic complication, uremia, etc.) **and then fell down to sustain a head injury.**

II. **Injury to the scalp**?



1. - Small, circular.
- Soft in the center, indurated at periphery
The indurated margin can be indented by application of pressure
- The whole abnormal area can be moved over the underlying bone

⇒ **Subcutaneous hematoma**

A DD: depressed fracture of skull

- An estimated amount of blood loss?
- Is the patient shocked?
- Depth of the wound?

Visible/palpable fracture of the underlying skull?

Both conditions may coexist, in which case differentiation may be clinically impossible without an X-ray

2. - Large—extending throughout
⇒ **Subaponeurotic hematoma**
A massive subaponeurotic hematoma may be produced by an injury of the superior sagittal sinus

3. - Size and shape of the hematoma—same as that of the bone over which it occurs
⇒ **Subpericranial hematoma**

www.Ebook777.com

III. Injury to the skull?

Fracture of vault
- Simple/Compound?
  - Any foreign body in the wound, particularly in the brain?
- Linear/Depressed?
  - Associated with focal neurological symptoms/signs (i.e. indicating a neuronal damage)?
  - Is the depression 'significant'? (Criterion of 'significant' depression: the degree of depression greater than the depth of the inner table of the skull)
  - How much is the inward angulation of the fragments? Any spicules of bone?
  - Is there any underlying hematoma (on a CT scan)?
  - Any complications?
  - Site?
    Does it appear to cross the groove of the middle meningeal artery or does it appear to overlie a dural venous sinus?

Fracture of base
- Anterior/middle/posterior cranial fossa?
- Associated injury to cranial nerve(s)?
- Duration of the CSF leak, if present?
- Any complications?

Depressed # in children is called ping-pong ball #. The skull being soft, virtually dents instead of fracturing.

Fracture of vault of the skull

The type of fracture whether linear or depressed can **only** be established radiologically. Unless there is a very obvious skull defect beneath an open scalp wound, attempts to differentiate between linear and depressed fractures clinically are rarely accurate.

The importance of fracture is as a predictor of intracranial injury.

Differential diagnosis of fracture of base of the skull

- – Bleeding or escape of CSF from the nose
  – Blood-tinged vomiting/hematemesis
  – Subconjunctival bleed above the cornea whose upper border is not visible
  } ⇒ # base of the skull through anterior cranial fossa.

  CSF leak from the nose rarely lasts > a few days. Leaks for > 10 days calls for repair of the dura.

  Cranial nerves likely to be injured: I, III, IV, ophthalmic division of the V, and VI. Optic nerve usually escapes.
- – Bleeding in the throat
  – Blood-tinged vomiting/hematemesis
  – Bleeding or CSF leak from the ear
  } ⇒ # base of the skull through middle cranial fossa.

  CSF leak from the ear for > 3 days probably suggests intracranial bleeding.

  Cranial nerves likely to be injured: Commonly the VII, and sometime VIII.
- Ecchymosis overlying the mastoid/suboccipital region developing within 3–4 days of injury ⇒ # base of the skull through posterior cranial fossa.

  Cranial nerves likely to be injured: IX, X, and XI. Hypoglossal nerve usually escapes being protected by a strong bony buttress.
- *Characteristics of* CSF that leaks:
  – In the early stages it may be mixed with blood and nasal catarrh (applicable to CSF rhinorrhea). Later it is present alone.
  – Pale amber appearance. Leaves a faint stain on the skin.
  – Contains glucose (can be tested with a urine test strip).
  – The detection of β-2 transferrin in the discharge is confirmatory.

Possible complications of fracture of base of the skull

1. Aerocele (pneumatocele) = an air-filled cavity in the brain connecting with one of the sinuses, usually the frontal sinus. Air may enter the subarachnoid space and ventricular system, or the substance of the frontal lobe. This occurs particularly if the patient blows his nose.

Free ebooks ==> www.Ebook777.com

2. Meningitis (may complicate a # base of the skull through the anterior fossa). When it occurs, it does so after the first 48–72 hours.
3. Pituitary failure.

IV. Injury to the brain?—**Has there been a period of unconsciousness OR some deterioration in the level of consciousness at the least?**

No ⇓ Patient unlikely to have a 'brain injury'.

Yes ⇓ **'brain injury'**

(A) *Points to note* ↓

- **Time of onset of deterioration of LOC** after sustaining the impact?
- **Duration** of the period of unconsciousness: Few minutes / hours (> 6 hours. / > 24 hours. / > 36 hours.)?
- Any **'lucid interval'**?
- (Other neurological) **Symptoms**?
- **LOC** at the time of clinical evaluation?
- Any focal **neurological signs**—present/absent? If present, do they persist for > 4 hours?
- **Recovery**: complete without any residual symptom, OR otherwise?
  Speed of recovery: Rapid, OR gradual?
  (Recovery usually occurs through these stages: Unconsciousness → semiconsciousness → confusion → recovery of intellectual impairment → complete recovery.)

(**B**) *Arriving at the diagnosis*

- Is there a **cerebral compression**? Is the **ICP rising**? Is the patient **coning**?
- Is the brain injury **focal** / **diffuse** / **both**? Often both types co-exist.
- **Localize** the injury as far as possible. The following clinical points may help:
  - Details of convulsions if any.
  - Temperature.
  - The side and site of injury of the scalp/skull.
  - Paresis/paralysis. | However, keep in mind possibilities of exception.
  - Pupils. | Refer to point numbers 17 and 18 under 'principles and pitfalls'.
- Is the injury **open** / **closed**?

With respect to the time of onset of deterioration of LOC, there may be 3 possibilities:

1. Deterioration of LOC from the outset, and the deterioration is maintained.
2. Deterioration of LOC from the outset—Lucid interval—Deterioration of LOC again, which is subsequently maintained.
3. Deterioration of LOC occurring a few hours (to a week or so) after impact: Possible causes—SDH due to a low velocity blunt assault, posterior fossa compression, dehydration/electrolyte imbalance, fat embolism.

Examples of *focal brain injury*: Contusion, laceration, hematoma, coning, infection (brain abscess).

Examples of *diffuse brain injury*: Cerebral concussion, cerebral irritation, diffuse axonal injury, cerebral edema, cerebral ischemia, infection (meningitis).

Diffuse brain injury basically represents a wide spectrum of severity. At one end of the spectrum, i.e. in mild cases the clinical syndrome of cerebral concussion is associated with little structural damage and a normal CT scan. At the other end of the spectrum is seen diffuse axonal injury.

A wound on scalp does not necessarily qualify for an open head injury. In **open head injury**, not only the scalp and bones are injured but dura is also torn. Usually the underlying brain is contused and lacerated, associated with bleeding. The brain tissue and blood may protrude to the exterior through the wound. Foreign bodies may be present. Laceration of brain may be quite deep though localized. There is no danger of raised ICP, but infection is likely to occur. Late cases may present as ***brain fungus*** which is a stinking fungating swelling on the head consisting of profuse granulation tissue on surface and protruding brain beneath it. It is a serious consequence of an infected open wound of the vault. The brain becomes infected and swells through the gap in the dura and skull.

www.Ebook777.com

In **closed head injury,** scalp may or may not be torn and skull may show no fracture or may show linear (fissured) fracture while dura is intact so that any rise in ICP is not able to get released towards the scalp and consequently compression of intradural structures will occur.

Differential diagnosis of brain injury

1. – A brief period of unconsciousness developing immediately after trauma. Duration—usually in minutes. (Clinician hardly has a chance to examine the case when the patient has fallen unconscious. Patient is usually seen in the stage of recovery; usually in a dazed condition.)
– Vomiting—a couple of times. (Vomiting may also occur later during recovery from concussion.)
– Body temperature may be subnormal.
– Pulse may be rapid and of low volume.
– Respiration may be rapid and shallow.
– Complete absence of neurological signs.
⇒ **'Cerebral concussion'**.

Natural history: Complete recovery without any residual symptom except for amnesia about the incident. Or, the patient may pass through a phase of cerebral irritation before complete recovery occurs.

2. – Period of unconsciousness—'a little longer' – may be > 6 hours.
– Recovery—gradual, although complete.
– Postconcussional symptoms: Headache, giddiness, insomnia, amnesia (retrograde and post-traumatic), impairment of memory and concentration, mental fatigue, personality changes.
⇒ **'Cerebral concussion combined with small areas of contusion/laceration of the brain'**.

3. – Unconsciousness - continuous from the start, or initially recovery of consciousness ('lucid interval') may occur f/b development of severe headache, drowsiness with further deterioration of LOC that is maintained.
Period of unconsciousness: > 6 hours; can extend beyond 24 hours.
– Stertorous breathing.
– Bradycardia.
– Focal neurological signs like:
- Convulsions.
- Muscles of the concerned area first show incoordinated contractions f/b flaccid paralysis—monoplegia / hemiplegia / paraplegia depending on the side and site of lesion.
- Temperature on the paralyzed side may be higher by ~ 1–2 degrees.
- Decerebrate rigidity.
- Pupillary changes.
– Progressive development of other manifestations suggestive of increasing ICP.
⇒ **'Cerebral compression'**.

Causes of cerebral compression:
– Severe brain edema.
– Contusion / laceration of brain (with surrounding edema)—usually multiple or/and large.
– Extradural / subdural / intracerebral / subarachnoid bleeding.
– ('Significantly') Depressed fracture of the skull.

'Lucid interval' ⇒
– Expanding extradural hematoma /subdural hematoma / both. Duration of lucid interval is usually only few hours, but it can be a week or more.
– Increasing reactionary (intracranial) bleeding. | This scenario is more applicable in an
– Rarely, raised ICP due to intracranial infection. | operated case, in the postoperative period.

Cerebral edema. The brain often reacts to any insult by swelling due to edema. Edema may be localized at least initially to the part of the brain most damaged, but it may extend throughout one or even both cerebral hemispheres, causing a severe rise in ICP.

Contusion/s. Contusions do not in themselves contribute to deterioration of LOC, but this may arise when bleeding into the contusion produces a SOL. Even small contusions can develop significant edema around them or develop into hematomas, and there is a risk of deterioration on the 3rd/4th day after injury. Despite this risk, even patients with severe contusions can make a good recovery.

Free ebooks ==> www.Ebook777.com

EDH can result due to bleeding from 3 possible sites: 1. Middle meningeal artery (the main trunk or its anterior or posterior branch), 2. Fracture alone, 3. Rarely a venous sinus (e.g. sagittal, transverse). Some patients with EDH have a lucid interval which is usually only few hours, but it can be a week or more. Others have a steadily deteriorating LOC from the outset. If a patient had a lucid interval, then the important first symptom to develop is increasing headache; confusion, irritability, giddiness, drowsiness follow later.

(*An acute*) ***SDH*** results usually from a tear of a bridging vein from the cerebral cortex to the superior sagittal sinus/inner surface of the dura, but can also result from a small tear in a cortical vessel on the brain surface. Low velocity blunt trauma (assaults) may result in a lucid interval before the LOC deteriorates due to the expanding hematoma, but high velocity blunt trauma (e.g. RTA) tends to cause continued loss of consciousness from the outset due to primary brain injury.
Morbidity/mortality is often high because of the severity of the primary brain injury.
Subdivision of subdural hematomas into acute and subacute forms has no practical relevance.

A chronic SDH often presents 1–4 months after an apparently trivial head injury, especially in the elderly.
SDH is the most common type of intracranial injury found in infants with nonaccidental injury.

ICH results from direct rupture of small blood vessels within the brain, but rupture through to the cortical surface may produce associated *SAH*. When bleeding occurs deep in the hemisphere, rupture into the ventricular system is common. Usually associated with cerebral contusion, laceration, edema and necrosis, the compressing lesion being formed by all these elements. Therefore, the benefit of removing the clot is unpredictable and often disappointing. Rarely a major intracerebral vessel may rupture as a result of trauma and produce an expanding hematoma which is the sole cause of cerebral compression. In such an event it is more likely that bleed has occurred from a pre-existing vascular lesion (an aneurysm/AVM). If time permits arteriography before surgery is advisable.

– Bradycardia
– (Initially unilateral) Dilated pupil
– Neck stiffness

} ⇒ **Coning**—*a complication of progressive cerebral compression*.

'Posterior fossa compression'

Compression of the cerebellum and medulla by posterior fossa EDH may result from fracture of base of the skull of the posterior cranial fossa crossing the lateral sinus. Presentation tends to be somewhat delayed as compared to a supratentorial EDH, usually after 48 hours.
The most important physical sign—slowing and irregularity of respiration.

4.
– Patient lying curled up (in an attitude of flexion) on one side, facing away from light.
– Keeping the eyes closed and resisting all attempts to open them.
– LOC: Stuporous / Semiconscious.
– Resentment to questioning, clothes, covering, and may become rowdy (frequently abusive and irritative) particularly when urinary bladder is full.
– Photophobia.
– Headache, giddiness, nausea, vomiting ±.
⇒ **'Cerebral irritation'**.

This condition develops about 3 days or so after trauma. Not a life-threatening condition. Results from some ill-understood biochemical change in nerve cells.
Natural history: Recovery may be apparently complete, but in some cases headache, irritability, depression and lack of concentration may persist.

5. Diffuse axonal injury

There is a loss of consciousness from the outset.
ICP is often normal.
Clinical effect depending on the severity: mild coma ↔ death.
Examination findings of pupils are variable (and unpredictable), i.e. both pupils may be normal, or they may be unequal in size or one or both may be normally reacting or unreacting to light.
DAI is common after high-speed RTAs.

6. Cerebral ischemia commonly occurs after severe brain injury and is caused by either hypoxia or impaired cerebral perfusion.

7. Intraventricular bleed: Single / multiple; Small / large. The bleed may be immediate or delayed.

8. Primary brainstem injury (injury to the pons or/and medulla)
– Signs of external injury may be absent, but the patient is unconscious, with spontaneous extensor spasm of all 4 limbs, arching of the trunk (opisthotonus). Any stimuli will tend to increase the tendency to extension.

www.Ebook777.com

- Pyrexia and sweating.
- Tachycardia.
- Rapid and often shallow and therefore inadequate respiration.
- Small pupils.

Clinical features s/o brainstem injury developing upon primarily existing unrelieved supratentorial compression (e.g. an expanding compressing hematoma) is called secondary brainstem injury.

Pyrexia (may be as high as 108° F)
Paresis/paralysis
Pinpoint and fixed pupils
} ⇒ **Pontine bleed**.

9. Pituitary failure

This may occur when basal fractures pass across the pituitary fossa causing acute pituitary damage and endocrine failure.

- Deteriorated LOC
- Pallor
- Hypothermia
- Tachycardia
- A profound fall in BP.

V. Closely associated injuries which must be looked for: Facial and cervical spinal injuries.

VI . Class of head injury *wrt* severity?

- A GC score of 15–13 = Mild injury
- A GC score of 12–9 = Moderate injury
- A penetrating injury / a GC score of 8–3 / an intracranial hematoma needing surgery = Severe injury.

Indications for hospitalization

- A patient who qualifies for severe head injury
- Circumstances of injury unknown
- Severe bleeding from scalp wound; patient in hemorrhagic shock
- E/N/T bleeding
- H/s/o discharge of CSF from ear/nose
- A h/o or actual impairment of LOC of any degree, including irritability, altered behavior, drowsiness, or mental confusion
- A h/o transient clouding of consciousness *alone* can be a matter of debate with respect to consider it as an indication for admission
- Persistent post-traumatic amnesia
- Persistent headache, giddiness
- Vomiting / hematemesis
- Convulsions
- Fever
- Sneezing
- A full-thickness scalp wound
- Skull #
- Focal neurological signs
- Anything that makes it difficult to assess the pupils or limb function
- Uncertainty about the degree of alcohol intoxication
- Other medical conditions such as bleeding disorder, patient on anticoagulant therapy, etc.
- Criminal case
- Absence of responsible relatives.

A normal CT scan (which sometimes is obtained too quickly) is not necessarily an indication for the patient to be discharged from A and E, if any one of the indications for hospitalization mentioned above is present.

Free ebooks ==> www.Ebook777.com

Purpose of admission to hospital

1. For continuing hospital care for those who have suffered severe injury and who are in obvious need of care and possibly surgery.
2. For observation, despite the patient being perfectly well at the time of presentation. Patients with mild head injury can deteriorate unexpectedly, and can become neurologically devastated if the decline in LOC is not noticed early. The duration of admission for observation is arbitrary, but since it is exceptional for acute EDH to present > 18 hours after injury, overnight admission should suffice. However, there is an exception: It is wise to observe the patient with a fracture of base of the skull through the posterior fossa for at least 3 days.

CLINICAL INDICATORS helpful in making decision to operate for relieving cerebral compression

- A penetrating injury.
- A significant impairment of GCS score associated with clear signs of raised ICP such as bradycardia, dilated and unreactive pupils or extensor motor response in the limbs.
- In a conscious patient, a very severe/protracted headache or vomiting, and restlessness.
- Boggy edematous swelling of the scalp over the site of a fracture.
- Deterioration of LOC; the rate of deterioration.
- Localizing signs. However, hemiparesis/hemiplegia *alone* is never an indication for urgent surgery.
- A rising BP and a slowing pulse rate. Like in LOC, it is the trend in the values of these parameters that is important, specially in children, rather than static values at a particular point of time.

If surgery is indicated, the patient should be transferred to a neurosurgical unit, however, the patient should be operated upon without delay if neurosurgical service is not available.

The decision taken will depend upon:
- The rate of deterioration.
- Dilatation of one *v/s* both pupils.
- The traveling time to (rather than the distance from) the neurosurgical department.
- The experience of the general surgeon.
- The availability of special surgical instruments.

Signs which do not in themselves indicate the need for urgent surgery (exploratory burr hole)

- Focal neurological signs in an alert patient.
- A simple depressed # skull.

Prognostic factors in brain injury

Favorable: Young age (< 20). Children can recover remarkably from seemingly severe trauma.

Unfavorable:
- Fixity of pupils for > 48 hours.
- Decerebrate rigidity with paralysis of all 4 limbs × 1 month.
- The absence of any improvement in the first month.

Possible causes of failure of recovery which should be kept in mind even at a late stage: SDH, communicating hydrocephalus.

Sequelae/Complications of brain injury

- Morbidity
  - Disability (delayed effects)
    1. Postconcussional symptoms: may be persistent
       - Headache, insomnia, fatigue
       - Irritability, lack of concentration, lapses of memory
       - Difficulty in coping with work, depression

       Vestibular concussion may → dizziness and vertigo
    2. Contusions and intracerebral hematoma can cause cognitive and focal deficits
  - Complications
    - Intracranial hematoma—Chronic/recurrent
    - Intracranial infection—meningitis/abscess (May develop even months/years later)
    - Epilepsy
    - Subdural cyst
    - Schizophrenia is a rare complication of even a minor injury
- Mortality
  - Usually relates to acute phase.

www.Ebook777.com

3. Postsevere head injury
   - Mental disabilities—behavioral problems (aggressive behavior, divorce), inability to concentrate, impaired intellect, impaired memory
   - Physical disabilities—hemiparesis, dysphasia.
   - 'Vegetative state':
     - ~ 1/3 of those vegetative at 1 month regain consciousness within 1 year; of those who regain consciousness, over 2/3rds either subsequently die or remain severely disabled.
     - Of those vegetative at 3 months after injury, never regain an independent existence.
4. Prognosis of brainstem injury: The ultimate intellectual deficit may be considerably less severe than following supratentorial brain damage, but often there is marked spasticity and incoordination.
5. Prognosis of cranial nerve damage is variable.
   - Recovery may be expected: I, III, IV, VI, vestibular VIII, and in delayed damage of VII.
   - Disability mostly permanent: II, V, cochlear VIII, and immediate damage of VII.
   - Damage to IX, X, XI, and XII is associated with a very severe fracture of base of the skull; patient seldom survives primary damage.
6. Cumulative brain damage: Examples—'punch-drunk' state, dementia in boxers, jockeys.

A NOTE ON CHRONIC SDH

Chronic SDH occurs predominantly in infants and elderly.
Trauma is the likely cause, although a history of this is not always obtained.
Predisposing factors: Cerebral atrophy, low CSF pressure (after a shunt/fistula), alcoholism, coagulation disorder.

Because the collection can occur slowly, there may be significant midline shift and sometimes very few symptoms and signs. Indeed, a chronic SDH can mimic just about any neurological syndrome in its presentation.

Clinical features are nonspecific.
- Dementia.
- Deterioration of LOC, occasionally with fluctuating course.
- Symptoms and signs of raised ICP.

Free ebooks ==> www.Ebook777.com

– Focal signs occasionally occur, especially limb weakness; however this may be ipsilateral to the side of hematoma.

CT scan appearances depend on the time between the injury and scanning.

GCS USED IN CHILDREN

	Infant <1 year	*Child 1–4 years*	*Age 4–Adult*
	Eyes	**Eyes**	**Eyes**
4	Open	Open	Open
3	To voice	To voice	To voice
2	To pain	To pain	To pain
1	No response	No response	No response
	Verbal	**Verbal**	**Verbal**
5	Coos, babbles	Oriented, speaks interacts, social	Oriented and alert
4	Irritable cry, consolable	Confused speeach disoriented, consolable	Disoriented
3	Cries persistently to pain	Inappropriate words, inconsolable	Nonsensical speech
2	Moans to pain	Incomprehensible, agitated	Moans, unintelligible
1	No response	No response	No response
	Motor	**Motor**	**Motor**
6	Normal spontaneous movement	Normal spontaneous movement	Follows commands
5	Withdraws to touch	Localizes pain	Localizes pain
4	Withdraws to pain	Withdraws to pain	Withdraws to pain
3	Decorticate flexion	Decorticate flexion	Decorticate flexion
2	Decerebrate extension	Decerebrate extension	Decerebrare extension
1	No response	No response	No response

Normal GCS score

Age	*Eye opening*	*Best verbal response*	*Best motor response*	*Total*
0–6 months	4	2–3	4	10–11
6–12 months	4	3	5	12
1–2 years	4	4	5	13
2–4 years	4	4-5	6	14–15
> 4 years	4	5	6	15

www.Ebook777.com

58 FACIAL (MAXILLOFACIAL) TRAUMA

The face is that part of the head that is visible in a frontal (anterior) view, i.e. all that is anterior to the external ears and between the hairline (or where it was) and the tip of the chin.

GENERAL REMARKS

1. Facial injuries do not constitute an (acute life-threatening) emergency unless they threaten the airway or are a source of profuse bleeding. Profuse bleeding can occur particularly from the tongue and lip injuries.
2. Fracture of maxilla suggests very high energy transfer.
3. Accurate radiographic diagnosis is a preliminary to good reduction of a fracture.
4. Surprisingly many maxillofacial injuries are not very painful in the early stages. Presumably, this is due to trauma of the peripheral nerve branches at the fracture site.

Modes/mechanisms of injury

a. Fall on the face: An example—'parade ground fracture'. This occurs when someone faints, as for example a soldier on parade, and falls directly on the point of chin. In the elderly, keep in mind the possibility of a medical cause responsible for such a fall, e.g. MI, CVA, etc. In the child, the elasticity of the facial bones is remarkable but fractures can occur. Injuries caused by such a fall: Laceration below the chin, fractures of the thin condylar necks of the lower jaw, and often a midline fracture.
b. Blows by fist/kick/sporting equipment such as a bat/racket, etc.
c. RTA, railway accidents.

If the patient is fully conscious and feels more comfortable sitting up, let her/him do so bending well forward, so that the tongue falls forward, and blood and saliva can dribble out of the mouth. The airway, if seemed to have been obstructed, may improve remarkably after assuming sitting position.

HISTORY

- Numbness (of the facial dermatomes)?
- Visual problems? Double vision?
- Bleeding through the nose? Unilateral or bilateral?
  - Unilateral epistaxis $\Rightarrow$ paranasal facial fracture
  - Nasal fracture $\rightarrow$ bilateral epistaxis.
- Can you occlude the teeth?
- Pain in the neck? Difficulty in moving the neck?

LOCOREGIONAL EXAMINATION

Examine the neck at the outset to rule out cervical fracture.

– Run the fingers gently down the back of the neck palpating each neural spine
– Search for tenderness in the associated longitudinal muscles

Free ebooks ==> www.Ebook777.com

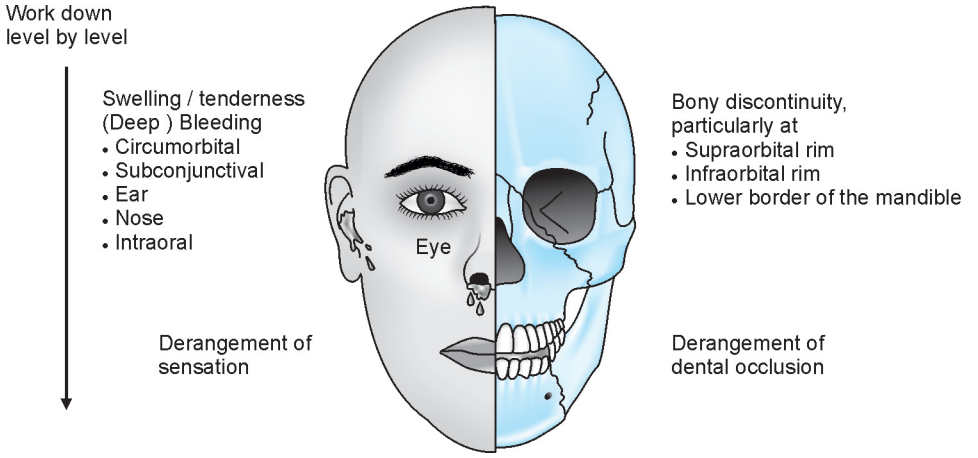


Fig 58-1

- Gently wash the patient's face with warm water to remove caked blood
- **Start examination at the top of the head and move down steadily level by level until the chin is reached**
- Look for asymmetry. Compare one side with the other throughout the examination
- (At each level) Look for swelling and tenderness, (deep) bleeding, bony discontinuity, derangement of dental occlusion, derangement of sensations, and possible cranial nerve injury.
- **Swelling and tenderness** overlie the site of injury
- These signs however do not necessarily indicate a fracture.

Deep bleeding

- **Circumorbital**—'Black eye' ('Panda eyes' = 'Racoon eyes') ⇒ fracture of any of the bones contributing to the orbit. Deep bleeding tends to be confined to the limits of the orbit by the orbital septum.
- **Subconjunctival—**unilateral or bilateral?

Bilateral bleed ⇒ bilateral injury. It may also occur in strangulation

Small localized ⇓ Local trauma to the sclera

Is the posterior/superior limit of the bleed visible (when the eye is moved to contralateral side)?

If no ⇓

Bleeding coming from within the orbit and spreading underneath the conjunctiva. It indicates a fracture somewhere within the orbit

- **Ear** bleed

  Causes:
  - Fracture base of the skull through middle cranial fossa and rupture of the tympanic membrane
  - Local injury to the ear canal. Rupture of the walls of the bony meatus can occur when the condylar head is driven forcibly back after a blow on the lower jaw
- **Nasal** bleed (Epistaxis)

  Causes:
  - Fracture base of the skull through anterior cranial fossa
  - Local injury including # nasal bone/maxilla/zygoma.

  The nose may bleed after relatively minor trauma in some individuals.
  - Is there a septal deflection?
  - Is there a septal hematoma?

www.Ebook777.com

- **Intraoral** bleed
  - Any hematoma in the lingual sulcus?
  - Any hematoma in the buccal sulcus?
  - Bleed in the region of the greater palatine foramen (Guérin's sign)?

Bony discontinuity

- A step ⇒ bony discontinuity
- Edema occurs very rapidly in the face so that after an hour or so postinjury, it may not be possible to detect lesser degrees of bony discontinuity.

Following sites are particularly important:

- **Frontal** bone
- **Supraorbital rim**. # frontal bone, # zygoma
- **Infraorbital rim**. Pyramidal fractures of the maxilla, # zygoma.
- **Lower border of the mandible**.
  - Palpate the whole of the lower border
  - Place a finger in the external auditory canal and ask the patient to open and close the mouth. Normally, the condyle should be felt to move. The absence of this normal finding may be suggestive of a #-dislocation.

Derangement of dental occlusion

Examination:

  - Open and shut the mouth. Is it possible? Is the bite normal or abnormal?
  - Move the jaw from side to side, and protrude it.
- **Open bite** may be indicative of # of the maxilla (all types) or bilateral # of the mandibular condyles.
  - Traumatic open bite may have to be differentiated from a pre-existing malocclusion
  - The persistence of small tubercles on the incisal edges (which are normally present at the time of eruption) suggests pre-existing malocclusion; this is because the front teeth have never met
- **Premature contact of the posterior molar teeth** occurs in fractures of the condyles, vertical ramus and angle region
- Fracture in the posterior body region → upward and inward pulling of the minor fragment
- **Downward and backward pull of the symphyseal region** suggests bilateral fracture of the anterior part of the mandible. There is a risk of airway obstruction
- Midline fracture of the mandible may → inward collapse of both sides.

Deranged sensations

  - Anesthesia / hypoesthesia / paresthesia / hyperesthesia?
  - Hyperesthesia is more usually seen during a phase of nerve recovery.

Eyes

- Look for: Proptosis, black eye, comparative level of both the eyes; whether either of the eyes has sunk inwards or downwards; subconjunctival bleed, pupils.
  - **A massive proptosis** may suggest excessive retrobulbar bleeding
  - **Displacement** may indicate herniation of the contents of the orbit through the floor into the maxillary sinus, or a fracture at the frontozygomatic suture line.
- Test **visual acuity**:
  - For severe deficit:
    - Can you see the light, movement?
    - Can you count fingers?
  - For mild deficit: Record reading acuity with hand/wall chart.
  - Blindness may indicate damage to the optic nerve.
  - **Causes of diplopia**: displacement of the orbit, displacement of the globe, VI nerve palsy, edema.
- **Movements** of the eyeball
  - *Clinical method*: Steady the patient's head and ask her/him to follow an object held at arm's length
  - Observe the full range of horizontal and vertical movements. Note any malalignment or limitation of range
  - Examine movements in the 6 different directions of gaze representing maximal individual muscle strength.

Free ebooks ==> www.Ebook777.com

Signs of injury to the eye:
- Black eye
- Open injury of the eyelid. A tiny laceration may be the opening of a track which penetrates the globe
- Subconjunctival bleed
- Shallow anterior chamber suggests a penetrating injury of the cornea
- Trembling of the iris when the eye moves may be suggestive of dislocation of the lens
- Grayish area in the cornea with swollen margin ⇒ perforation of the cornea. In severe cases, the anterior chamber may completely collapse, so that the iris touches the cornea
- Bulging of a black mass of tissue through the lips of a wound suggest prolapse of the iris or choroid
- Soft consistency of the eyeball may be suggestive of rupture of the globe
- Impairment of visual acuity.

Injury of the cheek
- Site and depth?
  - Look for possible damage to the muscles (of mastication and facial expression), facial nerve, parotid duct, and parotid gland.
- Examine for facial nerve injury.
  - Drop an imaginary vertical line from the lateral canthus of the patient's eye. Does the facial laceration lie anterior to this line or posterior to this line?
    - If the laceration lies anterior to the line, only the peripheral branches of the facial nerve could have been injured
    - If the laceration lies posterior to the line, major branches of the nerve could have been injured
- Possibility of parotid duct injury? Suspect if the injury lies over the middle 3rd of an imaginary line joining the tragus of the ear to the commisure of the lip
- Leakage of fluid from a wound situated on the posterior aspect of the cheek? ⇒ injury to the parotid gland.

Teeth
- Inspect. If a piece of tooth is missing, X-ray the chest; the patient may have inhaled it!
- Tap the teeth. A 'cracked cup' sound suggests fracture of the bone above
- Rock the teeth. Causes of abnormal movement of individual tooth or several adjacent teeth: Fracture, exposure of the roots, periodontal disease.

Specific signs of individual facial fractures/dislocations
Mandible

Salient features in general:
- A hematoma in the lingual sulcus related to the mandible is the pathognomonic sign of fracture of mandible
  A hematoma in the buccal sulcus does not necessarily indicate a fracture
- The absence of feeling of movements in the glenoid fossa to the finger placed in the external auditory canal suggests #-dislocation
- If the patient has teeth or wears dentures then in a displaced fracture it is likely that the dental occlusion will be altered
- Deranged mental sensation suggests damage to the inferior dental nerve.

# of condyle/s
- These are the most common mandibular fractures. They are often bilateral. They are often missed
- Pain, swelling and tenderness over the TM joint
- Bleeding from the ear ±
- When the patient tries to move the jaw, it deviates towards the side of the fracture
- Bite—normal or abnormal (anterior open).

of the ramus
- Usually, practically undisplaced
- Displaced # ⇒ extreme violence.

www.Ebook777.com

of the angle

– May be undisplaced
– Causes of displacement: 1. Extreme violence. 2. Disposition of the fracture line in such a way that different muscle-pulls can allow the displacement of the fragments to occur in opposite directions.
  Sometimes, there can be a tooth on the posterior fragment.

Recent dislocation of the jaw (TM joint)

Causes:

– Laughing/yawning with opening the mouth too widely
– Blow on the jaw while the mouth was open.

Clinical signs are less pronounced in unilateral dislocation.

Examination

1. Palpate just behind the condyle. Feeling of a small hollow ⇒ dislocation.
2. Insert the little fingers into the external ears simultaneously with pulps of the fingers facing forwards (anteriorly).
   Ask the patient to try to open and close the mouth.
   – Try to feel for movement of the condyles
   – Inability to feel movement of the condyle ⇒ dislocation (on the concerned side).

Maxilla, i.e. **fractures of the middle 3rd of the face**

There are 3 classic fracture sites of the maxilla as described by Le Fort. However, the classic Le Fort sites are not always found.

Features of # maxilla:

1. Always multiple, sometimes with 50 or more fragments.
2. Cranial nerves may be injured, especially the infraorbital and superior dental nerves.
3. Ethmoid may be fractured and the dura torn, so that CSF leaks from the nose.
4. Orbit may be fractured, sometimes with the displacements of its contents into maxillary sinus ('blow-out' fracture).
5. The circulation of the eye may be obstructed and result in blindness if the obstruction is not relieved within minutes of the trauma (the ophthalmic canal syndrome).
6. The maxillary sinuses may be filled with blood.
7. The nasolacrimal ducts may be injured and cause a flow of tears.

***Salient** features of each type*:

Le Fort I

– Epistaxis is common
– Hematoma in the buccal sulcus throughout its extent
– Bleed in the region of the greater palatine foramen (Guérin's sign) indicates fracture through the region of the greater palatine canal
– The fractured fragment may be surprisingly mobile (floating maxilla) because the fracture line separates the dentoalveolar part of the maxilla from the rest of the cranial skeleton.

Le Fort II

This is characterized by major facial edema.

Le Fort III

A very major injury. The whole of the maxilla and the zygoma is sheared off the rest of the cranium.

– This type is also characterized by major facial edema
– A hooded appearance of the upper eyelids may occur due to a characteristic structural damage.

Zygomatic complex

– Flattening of the side of the face can be seen if patient is seen soon after injury. Edema fills out the flattening within 3 hours. The flattening can be seen again after a week, i.e. only after the edema has subsided
– Unilateral epistaxis

Free ebooks ==> www.Ebook777.com

– Numbness over the cheek suggests damage to the infraorbital nerve
– Diplopia ±.

Zygomatic arch
– Depression may be visible if patient is seen soon after injury. Edema fills out the depression within 3 hours. The depression can be seen again after a week, i.e. only after the edema has subsided
– Restriction of movements of the TM joint due to restriction of movements of the coronoid process
– If the mouth was open at the time of injury, the patient will be unable to close the mouth.

Orbital 'blow-out' fracture is one in which the floor of the orbit caves into the maxillary antral cavity.

Alone → Pure 'blow-out'
Occurring in combination with other neighboring fractures → Impure 'blow-out'

A major 'blow-out' fracture may produce a progressive diplopia and enophthalmos. This may occur some weeks or even months after the actual injury.

Nose
Salient features
– Lateral blows → displacement of the nose to the contralateral side
– Anterior blow → depression of the nasal bridge and widening of the intercanthal distance
– Epistaxis is very common and will be bilateral
– Septal deflection may occur
– Septal hematoma may occur; it requires urgent drainage to prevent necrosis of the cartilage.

ARRIVING AT THE DIAGNOSIS

Step 1
– Is there an **acute life-threatening emergency**?
– An unstable bilateral fracture of the anterior part of the mandible
– A fracture of the maxilla with gross posterior displacement
} May → **acute airway obstruction**
– Maxillary or nasal fractures may occasionally → **profuse bleeding**.

Step 2
Is there an **acute organ-threatening injury**?—for example, injury to the eye, a massive proptosis.

Step 3
Is there **any injury with cosmetic concern**?—for example, injury to the nose, pinna of the ear.

Step 4
In rest of the situations (other than those mentioned above in steps 1–3)
- Exact anatomical site?—soft tissue injury / bony injury / dental injury / cranial nerve injury?
- Pathological nature?
- Complication?—superficial bleeding? Deep bleeding? CSF leak?

Step 5
Any closely associated injury to the—head / neck?

Injuries to different parts of the face, and specific points of interest in each to be looked for

Injury to the **eye**
– Blunt or penetrating?
– Visual acuity?
  In case of a suspected severe eye injury with apparently a loss of vision: Is there perception of a strong light at the least?
– Any foreign body?

www.Ebook777.com

Injuries of the **lips**
- Site?
  - Does it involve the skin-vermilion border?
  - Does it involve the labial sulcus?
- Without or with tissue loss?
  |
  How much—up to 1/4th of the length of the lip or more?

Injury of the **gum**
In case of a laceration: Is the lacerated gum retracted to expose the alveolus?

Injury of the **tongue**
- Site?
- Type?
- Is it severe and which cannot be accessed?

Injury to the **cheek**
- Injury to the muscles (of mastication/facial expression)?
- Injury to the facial nerve?—peripheral branches or major branches?
- Injury to the parotid duct?
- Injury to the parotid gland?

Injury of the **ear**
- Devitalization of the skin?
- Any loss of a part of the pinna?—if so, apparently minor or major?
- Hematoma?

Injury of the **nose**
- **Epistaxis**?
- **Septal displacement**?
- **Septal hematoma**?
- **#** of the **nasal bone** leading to deviation or depression?
- **#** of the **frontal process of the maxilla**?
- **#** of the **vomer**?
- **#** of the **ethmoids** ± **CSF leak** from the nose? Most leaks stop spontaneously, except in severely comminuted fractures.

of the zygoma
- **#** of the **zygomatic complex**: The zygomatic bone is so closely united to the neighboring bones (frontal, temporal and maxillary), that when the zygoma is fractured, the neighboring parts of these bones are also fractured. The zygomatic complex, therefore, usually fractures as a whole.
- Sometimes only the **zygomatic arch** fractures
- **Unable to open and close the mouth** (i.e. move the jaw)?
- **Diplopia**?

Dislocation of the jaw
- **Recent** or **old**?
- Unilateral or bilateral?

of the mandible
Features to be assessed in general:
- Single or multiple?
- Site/s?
- Simple or compound? #s of the ramus, condyle and coronoid process are simple. #s of the angle and body are compound

Free ebooks ==> www.Ebook777.com

- Is the airway compromised?
- Is the bite normal or abnormal?
- Is the patient dentulous or edentulous?

Features to be assessed of fractures of specific areas of the mandible:

of the **condyle**/s:
- Unilateral or bilateral?
- Bite—normal or abnormal (anterior open)?
- Is the patient dentulous or edentulous?

of the **ramus**:
- Undisplaced or displaced?

of the **angle**:
- Undisplaced or displaced?
- Bite—normal or abnormal?
- Is the patient dentulous or edentulous?

Undisplaced # of the **body of the mandible**: Nothing particular apart from general features to be assessed.

Displaced # of the **body of the mandible without tissue loss**:
- Does the fracture lie within the tooth-bearing area?
- Does the fracture lie outside the tooth-bearing area?
- Is the patient edentulous?

of the **body of the mandible with severe tissue loss** is usually associated with severe displacement as well.

Injuries to the **teeth and alveolus**

- **Fracture of the crown**. The exposed pulp is visible as a pink spot on the root surface. Could the missing piece have been inhaled?—take X-rays of the chest
- **Subluxation** of the tooth
- **Dislocation** of the tooth. The tooth becomes so loose that it can be lifted up and down in its socket
- **Complete traumatic extraction and loss** of the tooth. Could it have been inhaled?—take X-rays of the chest
- Traumatic **oroantral fistula**
- **#** of the **alveolus**
- **Missing denture**. Could it have been inhaled?—take X-rays of the chest. **Normal X-rays do not exclude inhalation** as the dentures may not be radiopaque.

Principles of conventional radiographic examination

Radiographic examination is indicated when a facial fracture is suspected.

Purposes served by radiography:
- Detection of a fracture
- Ascertaining stable and undisplaced fractures which do not require active treatment
- Detailed assessment of the injury to plan the treatment.

A word of caution: Some views (e.g. occipitomental) require the patient to be turned on her/his face. This may be unsafe and impracticable in a severely injured patient or in a patient who may have spinal injuries. Alternative views not requiring the prone position may have to be obtained in these cases, even though they may prove somewhat inferior.

Mandible
- *Classical views*:
  1. Right and left lateral obliques (with the relevant side of the face against the film).
  2. PA. PA view needs the subject to face downwards, and if this is inadvisable an AP view may be substituted.
- *OPG*, if available, is preferable to the lateral oblique views

www.Ebook777.com

- *For central region* (which is superimposed by the image of cervical vertebrae in the OPG or PA projections): True occlusal and oblique occlusal. The true occlusal film shows the outline well, and the oblique occlusal film shows bony details well.
- *For condylar areas*:
  - Towne's view, or even better is the reverse Towne's
  - In cases of difficulty, transcranial condylar X-ray may be needed
- *For examining the relationship of a fracture to individual teeth and detecting root damage*: Dental periapical film.

Maxilla

- *Occipitomental*: This shows vertical displacement of the orbital margin
- *30° occipitomental*: The inferior orbital margin can be viewed almost from below so that horizontal displacement can be judged
- *Mento-occipital*: This is an inferior substitute for occipitomental view, but may have to be obtained if occipitomental view is contraindicated when turning down of the patient to prone position is not advisable
- *Lateral facial bones*: This view shows fracture lines running through the region of the frontomaxillary suture lines or nasal septum, and also any steps in the line of pterygoid plates.

Zygoma

- *Occipitomental*
- *30° occipitomental*
- *Submentovertical* view shows the zygomatic arch outlines
- *Rotated occipitomental* view is occasionally indicated where fracture is uncertain.

Nose

- *Occipitofrontal* view shows mediolateral displacement of the nasal bones and the position of the nasal septum
- An *AP* view of the skull is a substitute for the occipitofrontal view if the patient cannot be turned on her/his face
- In *lateral nasal bones* view, do not misinterpret the normal suture between the frontal and nasal bones as a discontinuity (#).

Clinical examination is of more importance than radiographs in nasal fractures.

Primary treatment of maxillofacial injuries which present as acute life-threatening / eye-threatening emergency

A maxillofacial injury must be seen in the context of the patient's general status and other injuries.

A severe injury of the tongue which is not accessible

The dangers associated with bleeding: 1. Airway obstruction, and 2. Aspiration.
Principle of treatment:

- Emergency tracheostomy ↓ local analgesia, and pack the pharynx
- Repair the wound with deep stitches later ↓ GA with adequate preparation.

An unstable bilateral fracture of the anterior part of the mandible may → acute airway obstruction.
Immediate therapeutic measure to be taken:

- Grasp the tongue with a towel clip and pull it forward gently
  OR
- Insert a large suture through the dorsum of the tongue and pull it forward gently.

Subsequently, consider definitive measures for sustained airway control.

A fracture of the maxilla with gross posterior displacement may → acute airway obstruction.
Immediate therapeutic measure to be taken:

- Pass 2 fingers behind the palate to quickly bring the jaw (maxilla) forwards and upwards. Reduction may not be easy; considerable force may be needed. If reduction fails, an emergency tracheostomy may be needed

Free ebooks ==> www.Ebook777.com

– If reduction is successful, pass nasopharyngeal airways through both nostrils.

Subsequently, consider definitive measures for sustained airway control.

Bleeding

- *Superficial bleeding:* Place and firmly hold pressure packs for approximately 10 minutes. A persistent arterial bleeder requires clamping
- *Deep bleeding* occasionally occurs in maxillary or nasal fractures; nose bleeding may be very profuse
  - Try anterior nasal packing, but it is often necessary to pass bilateral postnasal packs. Following the insertion of the postnasal pack, also insert conventional anterior nasal packs to reinforce the effect of posterior packs. Postnasal packs should not normally be left for longer than 48 hours
  - In a very occasional case where nasal packing fails, consider ligation of external carotid artery on the affected side. This is often not as effective as might be hoped because of the crossover circulation from the opposite side, but may help in the overall control of a difficult situation.

Indications for tracheostomy

1. Inability to disimpact and reduce the fracture of the middle third of the face.
2. Inability to control severe posterior bleeding.
3. Edema of the glottis, particularly following a neck injury.
4. A severe injury with much tissue loss.

A massive proptosis suggests retrobulbar hemorrhage which may be compressing the optic nerve.

Make a small incision at the outer canthus, take a hemostat and push it into the incision; blood will squirt out. Failure to drain the hematoma may lead to permanent blindness.

Transportation of patients with maxillofacial injuries

- *If the tongue or lower jaw has fallen backwards*: Lay the patient on her/his side
- *Severe jaw injury with much tissue loss*: Lying on her/his front with the head over the end of the stretcher and the forehead supported by bandages between its handles
- *If a patient needs to be referred whose breathing is in danger*, it is safer to do so with a tracheostomy than with a suture through the tongue to pull it forward, which is the other alternative.

www.Ebook777.com

59 SOFT-TISSUE INJURIES OF THE NECK

Visceral compartment of the neck = the space between pretracheal and prevertebral layers of the deep cervical fascia. All the vital structures are located in this compartment.

GENERAL REMARKS

1. **The first priority** always is security of the airway.
   Causes of upper airway obstruction due primarily to injuries of the neck:
   – Intraluminal bleeding
   – Laryngeal #
   – Soft tissue swelling, e.g. hematoma.
   Other causes of upper airway obstruction may be due to associated major craniofacial trauma.
2. Methods of achieving immediate temporary hemostasis in case of **massive external bleeding**: Apply an external pressure, or achieve a direct digital control by inserting a gloved finger through the wound. Blind clamping should never be attempted.
3. **Overlooked occult bleeding from seemingly innocuous penetrating neck wounds** is a dangerous pitfall!
4. **Wounds at the base of the neck** may produce unexpected sudden and massive bleeding into the mediastinum, pleural space or an adjacent tracheal wound.

CLINICAL FEATURES which should be specifically looked for:

– Hoarseness of voice
– Dysphagia
– Hemoptysis / Hematemesis
– Signs of hypoxia
– If breathing is noisy / snoring / with stridor
} ⇒ upper airway obstruction
– Hypovolemic shock
– Active bleeding:
  - External, from the wound
  - from the E/N/T
– Diffuse swelling—could be due to expanding hematoma or surgical emphysema
– Distal pulse deficit, bruit
– Neurological deficits: Brachial plexus, cranial nerves VII, IX, X, XI, XII, hemiparesis
– Hemothorax / pneumothorax.

Free ebooks ==> www.Ebook777.com

PRINCIPLES OF MANAGEMENT

Following initial resuscitation (if and when required)

- Patients who are hemodynamically stable
  - ↓ Assess for the need of operative intervention*
    During this period, avoid any maneuver that may act to exacerbate or produce recurrent bleeding e.g. probing of the wound, nasogastric intubation (as it may stimulate gagging/coughing)
    - Further decision-making
      - Superficial wound (maximum platysma-deep) with complete absence of any other physical signs
        - ↓ No further evaluation
      - Shock / active external bleeding / expanding hematoma
        - ↓ Emergency operation
      - In rest of the patients*
        - ↓ Investigate to localize the injury
        - ↓ Definitive treatment accordingly
- Patient remaining hemodynamically unstable
  - ↓ Emergency exploration (if practically feasible)
    Prognosis may be bad

* *Indications for operative intervention in the management of neck injuries*:

– Vascular injuries: Hypotension/shock, active external bleeding, large/expanding hematoma, distal pulse deficit, bruit, hemispherical deficit/hypoperfusion, significant hemothorax/hemomediastinum, peripheral nerve lesion adjacent to major vascular structures

– Aerodigestive injuries: Subcutaneous crepitus, air emanating from the wound, unexplained blood in aerodigestive tract, respiratory stridor, dysphagia/hoarseness

– Neurological injuries: Cranial nerves VII, IX, X, XI, XII, impaired diaphragmatic motion (phrenic nerve), brachial plexus deficits.

Investigations in general

1. X-ray chest PA in upright position
   X-ray neck—AP and lateral
   } Basic investigations.
   – Hemothorax / pneumothorax / air in the cervical soft tissue indicate serious injury.
   – Any foreign body?
   – X-rays are also helpful in assessment of approximate gunshot trajectories.
2. Arteriography is very accurate in assessing high cervical injuries.
3. Contrast swallow for esophageal injury
   – Incidence of missed esophageal injury is as high as 50%
   – The fascial investment of cervical esophagus does not allow extravasation of contrast medium to occur as freely as against in the case of thoracic esophagus.
4. Sensitivity of esophagoscopy to demonstrate esophageal injury is also ~ 50%.

PENETRATING INJURIES OF THE NECK

- For the purpose of diagnostic and therapeutic implications (especially operative approach), the neck may be divided in 3 zones
- **High zone** (superior to the angle of mandible, i.e. level with CV_3)
- Blood vessels at risk—internal carotid, deep branches of external carotid and vertebral arteries.
- *Clinical features*—one/more of the following:
  – Bleeding from the wound/E/N/T
  – Expanding hematoma
  – Neurological injuries—CNs VII, IX, X, XI, XII.

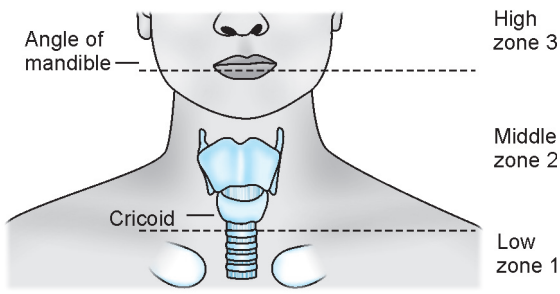


Fig 59-1

www.Ebook777.com

- *Problems in the treatment*
  1. Limited exposure of the anatomical area.
  2. Difficult to obtain distal vascular control.

  ∴ Routine diagnostic exploration is hazardous and inaccurate.
- *Arteriography* is the investigation of choice. It may be extended to therapeutic embolization.
- Arteriography is highly sensitive and specific. Negative study generally eliminates the need for further management.

Middle zone (= the zone between the high and the low zone)

Gunshot injuries	*Asymptomatic* patients	*Symptomatic patients*
↓	↓	↓
Better to explore	Observe	Appropriate investigation(s) if indicated, followed by decision-making regarding the selection of the modality of treatment. However, if any doubt exists, it may be better to explore If complete investigative evaluation cannot be performed, then diagnostic-cum-therapeutic surgical exploration is the best policy

Low zone (inferior to the cricoid cartilage i.e. level with CV_6)

- Great vessels are at risk e.g. proximal carotid, subclavian, brachiocephalic.
- *Clinical features*—one/more of the following:
  - External bleeding, shock, pulse deficit, intrathoracic (/ mediastinal) bleeding.
  - Exanguinating bleeding occurs in ~ 40% patients.

Principles of management:

- If patient responds to resuscitation (hemodynamic stability achieved), investigate to localize the injury and plan operative approach. Investigation of choice—arteriography
- In asymptomatic patients if arteriography is negative: Observe, i.e. they can be safely managed nonoperatively.

BLUNT INJURIES OF THE NECK

Causes: Direct blow, 'clothes-line' accident, acceleration-deceleration, significant flexion-extension of the cervical spine, etc.

Modes of presentation

- Severe injuries may not be obvious initially
- Airway obstruction, blood in the aerodigestive tracts, hoarseness, dysphagia, soft-tissue swelling, crepitus due to surgical emphysema, laryngeal deformities
- (Often delayed) Neurological deficit due to initially missed carotid artery dissection

Duplex scanning is the best noninvasive investigation for assessment of carotid artery. May be performed as a routine, or may be performed selectively if vascular injury is suspected on clinical grounds

Indications for arteriography: 1. Definite clinical evidence of arterial injury. 2. Positive Duplex scan.

Technical principles of surgical treatment of specific injuries

Arteries

- Repaired by lateral arteriorrhaphy / primary anastomosis / interposition vein grafting
- Ligation of external carotid/common carotid may be considered only in compromised situation demanding very short-operative time. Internal carotid artery should never be ligated
- Vertebral artery may be safely ligated.

Major veins

- Repaired if possible
- Otherwise, can be ligated without sequelae.

Free ebooks ==> www.Ebook777.com

Larynx and trachea

Immediate treatment: Try to pass a cuffed tube beyond the wound, either through the nose or mouth, or through the wound itself.

Definitive treatment: Primary repair.

– Mucosal approximation with fine absorbable sutures
– Approximation of cartilage/tracheal rings—extramucosal suturing with nonabsorbable material.

Pharynx and cervical esophagus

– Repaired in 1 or 2 layers, with drainage.

www.Ebook777.com

60 TRAUMA TO THE CHEST

GENERAL REMARKS

1. *Anatomical considerations*
The chest and its contents can be divided into 4 anatomical zones.

The zone	*Possible injuries which can occur*
a. Chest wall = bony thorax and associated musculature	Soft tissue injuries, # ribs, # sternum (with/without associated injuries)
b. Pleural space	Pneumothorax, hemothorax, collapse of lung, ventilatory compromise
c. Lung parenchyma	Hematoma, contusion, CLW, pneumatocele
d. Mediastinum = heart and aorta, great vessels, trachea and main bronchi, and esophagus—All are critical structures	Blunt myocardial injury, penetrating myocardial injury, blunt injury to the thoracic aorta, penetrating injury to the thoracic aorta, tracheo-bronchial injury, injury to thoracic esophagus. Aggressive management is required

2. *Topographically looking*, many abdominal organs lie under the ribs.
3. *Classification (for the purpose of description) of parts of the chest where injuries can occur*:
- Chest wall
- Intrathoracic viscera
- Both the above
- Thoracoabdominal.

4. A closed chest injury is one in which skin and subcutaneous tissue are intact or if they are torn, then pleural and pericardial sacs are not opened up.
5. Penetrating chest wounds should be managed according to the presumed trajectory of the missile based on symptoms and signs.
More extensive investigation is necessary if the trajectory crosses the mediastinum.
6. Patients with diaphragmatic injury should undergo prompt laparotomy for repair of the defect.
7. Chest injuries can be as deceptive as abdominal injuries—although a patient may seem to be in fair condition to begin with, s/he can deteriorate rapidly.
8. The great danger with all chest injuries: Retained secretions will cause infection, collapse of a lung, and death. Only active physiotherapy will prevent this unhappy outcome.

In **GENERAL EXAMINATION**, look specifically for the following points:
- Anxious look?
- Vital parameters?
  Is the pulse stronger on inspiration than on expiration?
- JVP?

Free ebooks ==> www.Ebook777.com

LOCAL EXAMINATION

Accomplish complete examination on all 3 sides viz. anterior, lateral and posterior (i.e. back).

Inspection

- Is the patient hungry for air?
  Are the accessory muscles of respiration in action?
- IC spaces—distended or do they show retraction during respiratory excursions?
- Cyanossis?
- Movements of the chest wall
  - Do both sides expand equally?
  - Paradoxical movements of the injured segment of the chest wall?
  - Splinting of the chest wall?

  Ask the patient to take deep breaths—is it possible, or is there a catch in breathing due to sharp pain?
  Sharp pain with a catch in the breath ⇒ chest wall injury particularly #-rib/s.
  Pitfalls
  - Paradoxical movements may be difficult to note when a patient is shocked and the respiratory movements are small; they may become apparent only later, after the patient is resuscitated
  - Indrawing of the costal margin is common in mild respiratory obstruction, especially in children.
- Deformity of the chest wall?
- Wound/s?
- Petechiae in the whole head and arms?
- Congestion of the veins of the head?

Palpation

- Tenderness?
  - If patient is too ill, gently spring the chest from front to back with the two hands or from side-to-side between the two hands
  - Severe tenderness most probably suggests fracture of some ribs.
- Deformity?
- Crepitus?
  - Tenderness and deformity ± crepitus may be indicative of # of the ribs/sternum
  - Crepitus overlying a tender rib/s ⇒ # of the rib/s
  - Marked crepitus ⇒ surgical emphysema. *Causes*—pulmonary parenchymal injury / bronchial injury / esophageal injury.

Percussion

- Hyperresonance ⇒ tension pneumothorax
- Flat/dull note ⇒ hemothorax or collapse of lung.

Auscultation

- Reduced or absent breath sounds (on one side) ⇒ pneumothorax / hemothorax / intubation of the right main bronchus / collapse of lung
- High-pitched breath sounds ⇒ tension pneumothorax
- Clicking sounds—fractured ribs
- Coarse crepitations—surgical emphysema.

Two-coin test

Purpose: Diagnosis of tension pneumothorax.

Clinical method and inference: Place a coin on the patient's chest and tap it with another coin. A bell-like note (combined with other signs) suggests tension pneumothorax.

www.Ebook777.com

CLINICAL ASSESSMENT

1. Is there an emergency life-threatening situation?
2. Site of the wound/s?
3. In c/o a stab wound—what is the depth?—can the end of the wound tract be defined?

– Patient making great respiratory efforts, but still hungry for air
The accessory muscles of respiration put into use
– Distension or retraction of IC spaces
– Cyanosis in the presence of an adequate airway
– Tachypnea OR bradypnea
– Paradoxical (see-saw) movement of the chest and abdomen, indicating failure of normal diaphragmatic function.

} ⇒ *Compromised breathing*

- **Emergency life-threatening situations demanding immediate action**: Open pneumothorax (sucking chest wound), tension pneumothorax, cardiac tamponade, and massive hemothorax
- **Other causes of compromised breathing** (acute life-threatening causes having been ruled out/dealt with): (Massive) Visceral herniation, flail chest, pulmonary contusion, aspiration pneumonitis
- **Inability to define the end of the tract of a stab-wound is an indication for exploration of the wound or thorax**.

INVESTIGATIONS

1. **ECG** and **continuous cardiac monitoring throughout resuscitation**
   - ECG changes may indicate blunt cardiac injury. Monitor such cases with telemetry for at least 24 hours. If there are any signs of cardiac failure, invasive monitoring and echocardiography are indicated.
2. **X-ray chest**
   - As far as possible, try to obtain an erect film
   - In c/o penetrating/perforating injuries, radiopaque markers should be placed on appropriate (entry/exit) wounds
   - A diffuse homogeneous opacity of smooth margin in a lower lung field obliterating the CP angle ⇒ **hemothorax**
   - A (horizontal) A-F level ⇒ **hemopneumothorax**
     On a *supine X-ray chest*, hemothorax causes diffuse haziness as blood layers posteriorly.
   - – The lung markings not reaching all the way to the edge of the thoracic cage
     – Clear-cut margin (a faint line) of lung (virtually the pleura), separated from chest wall/diaphragm
     – ↑ed radiolucency outside this margin caused by the presence of air and absence of lung markings

     } ⇒ **Pneumothorax**

     Radiological findings of pneumothorax are more obvious on film taken in expiration.
   - – Collapse of lung
     – ↑ed radiolucency caused by the presence of air and absence of lung markings
     – Mediastinal shift, and flattening/inversion of the hemidiaphragm
     – Widening of IC spaces

     } ⇒ **Tension pneumothorax**
   - Diffuse mottling with dense patches in places (signs of air-space filling) ⇒ **Pulmonary contusion**
     These findings intensify in the next few days and then clear.
   - Widening of the heart shadow ⇒ **cardiac tamponade**

Free ebooks ==> www.Ebook777.com

- – A significant mediastinal widening (mediastinum > 8 cm wide), loss of normal aortic contour
  – Apical capping of lung
  – Deviation of trachea or NG tube

  ⇒ **Thoracic aortic disruption**
  |
  Further evaluation
  ↙

  - Arch aortography—the gold standard.
  - Other (alternative) investigations which can be performed: Spiral CT, transesophageal echo
- – The presence of NG tube or bowel gas above the normal plane of the diaphragm
  – Failure of resolution of a suspected hemothorax after insertion of a chest tube

  ⇒ possibility of a **ruptured hemidiaphragm**
- Fine streaks of radiolucency within mediastinum, often extending upwards into the neck ⇒ **pneumomediastinum**

Causes of pneumomediastinum

Air tracking from the adjacent chest wall / root of the neck / retroperitoneum	Eesophageal perforation	(Spontaneous) air leak from bronchi Most commonly seen in patients with asthma

- *In a suspected c/o retained foreign body*: Obtain 2 views at right angles to each other—PA and lateral.

4. **Serial X-rays** may have to be requested on some occasions.
   – They confirm proper placement of an ET tube/ICD tube/CVP line, etc.
   – They also help to detect late onset hemo-/pneumothorax.
5. **Conray swallow**—in a suspected c/o esophageal perforation.
6. Additional investigations as required depending on the case, e.g. CT scan, bronchoscopy, etc.

DIFFERENTIAL DIAGNOSIS AND PRINCIPLES OF MANAGEMENT IN BRIEF

Fracture of ribs

Primary manifestation: Intense pain

Possible natural history: Inadequate relief of pain → splinting and poor inspiratory effort on the affected side → ineffective cough and progressive atelectasis → pneumonia

Primary aim of the *treatment*: Adequate analgesia

Objective estimate of the degree of ventilatory compromise can be obtained on PFT. Tidal volume of < 5 mL/kg / FVC of < 10 mL/kg / maximum inspiratory force of < 30 cm of water ⇒ significant ventilatory compromise.

Notes

– Fractured ribs are easier to diagnose/suspect clinically than to see on X-ray film
– Fracture of upper 2 ribs suggests very high energy transfer, which in turn suggests a possibility of associated major chest, abdominal and vascular injuries
– Fracture of lower ribs suggests possibility of associated injury to liver/spleen
– In children, major intrathoracic injury can occur without fracture of ribs
– In older patients:
  - Relatively minor force can result in a fracture (Reasons: Osteoporosis and reduced elasticity of the chest wall)
  - More likely to get complicated by atelectasis and pneumonia, especially if there is a pre-existing underlying pulmonary parenchymal disease.

Flail chest

The flail chest is caused by multiple contiguous comminuted or segmental rib fractures resulting in a free-floating section of the chest wall. If the free-floating section is pushed inwards, it is called **'stove-in chest'**. Stove-in-chest can occur with and due to fracture of ribs, or even without fracture of ribs. When it occurs without radiographically apparent fractures, it is due to disruption of the cartilaginous or ligamentous attachments of ribs.

Differentiating point: Paradoxical movement of the disrupted section of the chest wall, i.e. the disrupted section moves abnormally inwards with inspiration and outwards with expiration.

www.Ebook777.com

Chief points of focus in the assessment:
- Is breathing shallow?: If so, resuscitate with a self-inflating bag, especially during the 1st 24 hours
- Is breathing 'rattly'? and cannot the patient cough?: Suck out the pharynx. If this fails to clear the airway adequately, consider bronchoscopic suction
- Does bleeding continue for > 24 hours?: If so, a thoracotomy is indicated to find and control the bleeding point
- Is the patient very fat or muscular?: If so, it may be necessary to expose the ribs and apply traction directly to them.

Chief points of focus in the management:

1. Adequate analgesia.
2. Monitor clinical appearance, adequacy of ventilation and the degree of pulmonary dysfunction (– these criteria determine the need for intubation and mechanical ventilatory support.)
   Investigations: PFTs, ABG analysis, etc.
   - Patients without respiratory distress, and with good pulmonary function: Can be treated without intubation
   - Patients with significant arterial hypoxemia or ventilatory insufficiency: Intubate and commence mechanical ventilation.

   The duration of ventilatory support: Until pain is controlled adequately and until pulmonary parenchymal damage is resolved.
3. Indication for chest wall reconstruction and mechanical stabilization of the flail segment (unusual situation): Large flail segment having very severe chest wall deformity.

Sternal fracture

The need for additional evaluation is based on the overall circumstance (*with* the possibility of associated visceral injury)

Assessment of the fracture per se: How much is the degree of posterior displacement?

Treatment:
- Adequate analgesia
- Indications for operative fixation: Significant posterior displacement / persistent chest wall instability.

Simple pneumothorax

Modes of entry of air in the pleural cavity: Escape from the injured lung, or entry of atmospheric air through a penetrating wound

If blunt trauma is the cause of pneumothorax, then rib fracture is usually associated.

Physical signs—one or more of the following:
- Decreased respiratory expansion on the affected side
- Subcutaneous emphysema
- On percussion—hyperresonant or tympanic note overlying the pleural air
- Breath sounds—diminished or absent overlying the pleural air.

Indications for IC drainage:
- SOB
- Lowering of the apex of the lung by $\geq$ 3 cm below the top of the pleural cavity.

Hemothorax

The bleeding may range from minimal to massive life-threatening.

Differentiating physical signs
- On percussion—dull-to-flat note overlying the accumulated blood
- Breath sounds—diminished or absent.

Sometimes even a large hemothorax causes hardly any physical sign

A hemothorax may not be easy to diagnose radiologiclly; so rely on physical findings

A hemothorax may take some days to a week or so to develop.

Cause of hemothorax in the majority: Pulmonary laceration involving injury to low pressure vessels of pulmonary circulation. Bleeding usually stops spontaneously. Only ICD suffices. Thoracotomy is not required.

Free ebooks ==> www.Ebook777.com

Causes of massive hemothorax: Injury to the systemic arteries, e.g. intercostal, internal thoracic, etc; especially if they are partially transected.

The size of an ICD tube used for draining a hemothorax should be at least 36 F (in adults).

A guideline regarding indications for thoracotomy to achieve hemostasis:

1. Initial evacuation of ~ 1–1.5 l of blood.
2. Continued bleeding in excess of a glassful (~ 200 ml) per hour over a period of time / in excess of 500 ml in the second hour.

Possible causes of persistent (large) air leak in patients with ICD—i.e. air continues to bubble out of the underwater seal after 5 days:

1. Patient on positive pressure ventilation.
2. Significant bronchial injury.
3. Pulmonary/bronchial disease.
4. A hitherto undiagnosed esophageal perforation.

Pulmonary contusion

Morbidity will depend on the size of contusion, and associated injuries, pre-existing underlying lung disease and age of the patient

Pathophysiology in major contusion eventually → rapidly progressing ventilatory failure.

The basis of diagnosis:

1. The mechanism of injury. Blunt injury is more likely to cause contusion.
2. X-ray chest finding: An ill-defined air-space filling shadow usually apparent within 1–2 hours after injury.
3. ABG analysis: Hypoxemia.

Principles of treatment:

- Adequate analgesia for the associated chest-wall injury
- Minor contusions can be managed with general supportive measures
- Patients who manifest signs of respiratory failure: Intubate and commence mechanical ventilatory support
- Duration of ventilation—variable—from 1 day to several weeks
- Fluid overload is detrimental; ∴ Resuscitation should be done judiciously and titrated to physiological end points.

Pulmonary hematoma

The mechanism of injury and presenting symptoms are similar to those of pulmonary contusion

In a pure hematoma, alveolar dysfunction may be minimal (– this is in contrast with contusion), and the clinical manifestations less severe than the X-ray chest might suggest

X-ray chest finding: An air-space filling shadow. Differentiating features from contusion: 1. Hematoma has sharper margin, and a more spherical shape. 2. Resolution of hematoma is generally slower (occurs over ~ 2–3 weeks) than that of contusion.

Treatment: Essentially conservative. Mechanical ventilatory support may be considered for patients showing respiratory compromise.

Traumatic pneumatocele

= a (air-filled) cavity within the pulmonary substance.

Mechanisms/causes: 1. During resolution of a hematoma. 2. Injury to a small bronchus without significant bleeding

As an isolated injury, it is well-tolerated and is relatively asymptomatic

X-ray chest finding: An air-filled cavity ± a fluid level

A potential complication: Infection, if at all

Resolution occurs slowly over many weeks

Treatment: Per se, only observation. Usually no specific intervention required.

www.Ebook777.com

Myocardial contusion (Result of severe blunt chest trauma)

There are no classic *symptoms/signs*

There are no definite criteria on *investigation*

- ECG changes are nonspecific
- 2-D Echo is the best investigation. However, the significance of echocardiographic abnormalities in the otherwise asymptomatic patient is unclear. Subtle motion abnormalities are commonly seen, but do not correlate with likelihood of complications, especially dysrhythmia.

Treatment: Essentially conservative (monitoring) + treatment of complications if they arise during the course of monitoring, viz. dysrhythmia and pump dysfunction.

Cardiac rupture resulting from severe blunt chest trauma is almost uniformly fatal.

Modes of death: 1. If pericardium is also torn, then exanguination. 2. If pericardium is intact, then tamponade.

Penetrating myocardial injury

Chances of survival can be good in the case of single chamber injury with a small pericardial defect resulting in tamponade rather than with a larger pericardial defect resulting in rapid exanguination

The keys to maximizing survival are recognition, resuscitation and rapid operative intervention

An indicator of suspicion of a cardiac injury: Penetrating injury in proximity to the heart, e.g. a parasternal stab.

Cardiac tamponade usually follows a penetrating chest injury, but occasionally can result from a blunt one.

- Initially, patients often remain hemodynamically relatively stable. Once pressure within the pericardial sac reaches a critical level, cardiac output falls precipitously leading to profound shock and death if aggressive treatment is not instituted immediately.
- *Clinical features*: Refer to page 834.
- *Investigation of choice*: 2-D echocardiography
- *Principle of treatment*: Emergency thoracotomy. In dire situation, pericardiocentesis may play a role in diagnosis as well as a temporizing life-saving measure
- *Technical principles of thoracotomy*: The pericardial sac is opened. After tamponade is relieved, bleeding usually can be controlled by digital pressure while preparations for definitive repair are made. Small lacerations can be repaired using pledgetted sutures on the beating heart. Large or complex wounds are repaired on cardiopulmonary bypass.

Blunt injury to the thoracic aorta (Common with RTA)

Causes: Deceleration injury causing horizontal shear, compression injury (anteroposterior/lateral).

There may not be an external sign of a significant injury.

The patient may fit into one of the 2 broad categories:

1. Hemodynamically unstable: Evidence of leak is present. Emergency surgical intervention is necessary.
2. Small and stable pseudoaneurysm: Relatively low-risk of acute rupture.

Basis of diagnosis:

- A high index of suspicion
- Significant respiratory distress due to massive hemothorax
- Abnormal pulses in the extremities
- Neurological compromise due to spinal cord ischemia
- Associated other significant thoracic injuries.

Radiological features s/o aortic injury on plain X-ray chest:

- Widening of superior mediastinum by > 8 cm
- The loss of normal contour of the aortic knob—a very sensitive feature
- Massive left hemothorax especially in the absence of fracture of ribs

} All these findings can be very subtle or may be entirely absent

Plain X-ray chest can offer information that may heighten suspicion, but cannot be relied upon to exclude the injury.

- *Indication for arch aortography*: Suspicion of aortic injury on the basis of mechanism of injury, clinical manifestations, or plain X-ray chest findings.

Free ebooks ==> www.Ebook777.com

- *Transesophageal echocardiography* which can be performed at the bedside may be considered in a patient who cannot be transported to imaging department for aortography or CT.

Penetrating injury to the thoracic aorta

The patient may fit into one of the 2 broad categories:

1. Presenting with shock, with a penetrating wound in proximity to the great vessels.
   – Require an urgent (? emergency) thoracotomy.
2. Hemodynamically stable, with a penetrating wound in proximity to the great vessels:
   – Angiography.
   – False –ve results are possible. A highly suspicious injury should be explored despite a –ve angiogram.

Specific points to be kept in mind regarding treatment during the preoperative period:

– Avoid ↑ in BP with judicious use of β-blockers
– Adequate analgesia
– Anxiolysis.

Tracheobronchial injury

- Blunt trauma tends to cause generally a circumferential injury within 2.5 cm of carina
- Penetrating injury may occur at any level
- *Presentation* depends on whether the injury opens into the pleural space or into the mediastinum
- Rupture into pleural space → hemoptysis, pneumothorax (and a significant persistent air leak through ICD)
- Rupture into mediastinum → hemoptysis, subcutaneous emphysema in the neck, and mediastinal emphysema (on X-ray)
- *Investigation of choice*: Bronchioscopy.

Principles of treatment:

– Injury involving < 1/3 of the circumference (of the trachea/major bronchus): Expectant line of treatment
– Injury involving > 1/3 of the circumference (of the trachea/major bronchus): Primary reconstitution.

Surgical emphysema involving the face, neck and chest

- *Causes*: Pulmonary parenchymal injury, bronchial injury, esophageal injury.
- In spite of the alarming appearance, emphysema itself is benign. Sometimes, the patient may swell from pelvis to forehead.
  Principles of treatment:
  – Small quantities of air may be removed by massaging air into pockets and then aspirating these pockets
  – If the emphysema spreads to threaten life: Tracheostomy. Tracheostomy abolishes coughing and the large rises in intrathoracic pressure it causes
- Failure of a whole lobe of a lung to expand after having inserted an ICD ⇒ **Pulmonary collapse**
- This is common and can complicate any severe chest injury. Collapse occurs partly due to retained secretions.

Traumatic asphyxia

- This is caused by violent compression of the chest, which forces blood into the veins of the head, neck and arms. Small blood vessels burst and → petechiae. Conjunctival and retinal hemorrhages ±. Patient may fall unconscious
- Provided the other injuries do not prove fatal, traumatic asphyxia is not in itself serious
- **Congestion of the veins of the head** may suggest pressure on the veins at the base of the neck from air escaping into the mediastinum and pleura due to tears in the trachea/esophagus/bronchi.

Injury to thoracic esophagus: Refer to chapter 61.

Basic principles of intercostal drainage (ICD)

The IC space recommendation:

– On the right side, 5th
– On the left side, 7th/8th

} In the midaxillary line

www.Ebook777.com

Chest tube size recommendation in adults:
- For drainage of air, 28 F may suffice
- For drainage of hemothorax, at least 36 F.

Weight in kg	*Chest tube size recommendation in children*	
	For air/transudate	*For blood/exudate*
< 3	8–10 F	10–12 F
3–8	10–12 F	12–16 F
9–15	12–16 F	16–20 F
16–40	16–20 F	20–28 F

After opening the pleural space, a finger should be inserted and swept in all directions to breakdown any fibrinous septa, and also to palpate the lung, pericardium and diaphragm
Postprocedure X-ray chest (preferably in upright posture) should be obtained for confirmation of correct placement of the tube, and also ensuring the desired therapeutic effect.

Free ebooks ==> www.Ebook777.com

61 ESOPHAGEAL INJURY

ESOPHAGEAL PERFORATION

GENERAL REMARKS

1. Clinical manifestations, radiological findings, treatment and prognosis of cervical esophageal perforation differ from those of thoracic and abdominal perforation.
2. The symptoms of mediastinitis may be as meager as a temperature blip or as fulminant as shock.
3. The critical thing in avoiding delay in the diagnosis is to think about the esophagus early and hard. Diagnosis precedes treratment, but suspicion precedes diagnosis.
4. 60% of cervical esophageal perforations occur due to complication of endoscopy. Remaining 40% occur due to penetrating injury and foreign body ingestion.
5. The zone more likely perforated by ingested foreign body: Esophageal narrowing in the region of aortic arch/left main bronchus.
6. Severe perforations can be caused by attempted removal of foreign bodies.

SUSPECT

1. On development of pain, odynophagia, dysphagia following UGI endoscopy: Suspect **cervical** esophageal perforation.
   Risk of perforation increases in case of the following:
   - Failure to obtain an esophagogram prior to esophagoscopy. This risks perforation in areas of unsuspected pathology
   - Inadequate sedation/anesthesia
   - Failure to deflate the cuff of an ET tube, if one has been in place
   - Failure to use a rubber-tipped lumen finder and thus becoming lost in the pyriform fossa
   - Failure to elevate the scope anteriorly away from the spine.

2. On development of substernal/epigastric pain following dilatation of stricture / achalasia: Suspect **thoracic** esophageal perforation.

3. On development of epigastric pain often referred to the back/shoulder, epigastric tenderness and guarding—following esophageal instrumentation or periesophageal surgery: Suspect **abdominal** esophageal perforation.

4. Sudden onset of tearing substernal / epigastric pain, which may be radiating to the back / shoulder, and which may be accentuated by swallowing / bodily movement following repetitive vomiting / bouts of vomiting following overindulgence of food and drink / bouts of vomiting following sedation / GA / thoracic / abdominal blunt trauma / excessive straining during defecation / childbirth / sports event / epileptic seizure: Suspect **Boerhaave syndrome**.

www.Ebook777.com

CERVICAL ESOPHAGEAL PERFORATION

Pathology

– Basically, following endoscopy / following sharp foreign body ingestion,

OR

Perforation may occur as an immediate complete breach of the esophageal wall	Perforation may begin as only a mucosal laceration, which in turn may progress to intramural abscess. Subsequent bursting of the abscess → mural rupture In this situation, the development of clinical manifestations of overt perforation may be delayed for many hours after endoscopy

– Blunt foreign bodies may → pressure necrosis and delayed perforation
– Spread of infection

In case of posterior perforation	In case of anterior/lateral perforation
↓	↓
Retrovisceral space → posterior mediastinum	Pretracheal space → retrosternal space

Clinical features

– Pain, worsened by flexion of the neck
– Odynophagia
– Dysphagia
– Fever
– Tenderness in the neck.
A clinical test: Hold the thyroid cartilage between the thumb and fingers, and move from side-to-side—Severe tenderness is suggestive.
– Crepitus.

Investigations with possible findings:

- **FBC**. Leukocytosis
- **X-ray neck** lateral view
  – Loss of normal lordosis
  – Widening of retrovisceral space
  – Streaks of air in the soft tissue planes
- **X-ray chest**
  – Pneumomediastinum
  – Mediastinal pleural fluid collection
  – Pleural effusion, usually right sided; develops after 24 hours
- **Esophagogram with water-soluble** contrast medium: Extravasation

  ↓

  If negative (in ~ 20% cases)

  ↓

  Barium swallow using thin barium

  If clinical features and plain X-ray findings are strongly suggestive of perforation, a negative barium swallow should not prevent surgical treatment.
- **Role of esophagoscopy**: Occasionally indicated in cases of perforations caused by foreign body / penetrating trauma.

THORACIC AND ABDOMINAL ESOPHAGEAL PERFORATION

- In contrast to cervical esophageal perforations, thoracic esophageal perforations have a multiplicity of causes. Their **management and outcome depend upon the etiology, associated pre-existing esophageal pathology and the condition of the patient** at the time of presentation and management

Free ebooks ==> www.Ebook777.com

- Dilatation of strictures and achalasia together → 45% of perforations
- Dilatation of strictures over a guide-wire under fluoroscopic control reduces the incidence of perforations
- In some institutions, it is a routine practice to take esophagogram with a water-soluble contrast medium f/b thin barium if necessary, after pneumatic dilatation.

THORACIC esophageal perforation

Clinical features

– Substernal/epigastric pain
– Cervical subcutaneous emphysema ± (present in only 20% cases).

Investigations

- **X-ray chest.**
  – Pneumomediastinum
  – Mediastinal pleural fluid collection
  – Pleural effusion.
- **Esophagogram with water-soluble** contrast medium

↓

If negative

↓

Barium swallow using thin barium

ABDOMINAL esophageal perforation

Clinical features

– Epigastric pain, often referred to back/shoulder
– Epigastric tenderness and guarding

} In a patient who has undergone esophageal instrumentation or periesophageal surgery

Investigation

Esophagogram with water-soluble contrast medium: Extravasation

↓

If negative

↓

Barium swallow using thin barium

BOERHAAVE SYNDROME

= postemetic rupture of the normal esophagus.

- It results from a sudden increase in intra-abdominal pressure leading to esophageal barotraumatic rupture
- The term 'spontaneous rupture' is apparently inappropriate, because the rupture invariably follows barotrauma
- Despite the dramatic clinical manifestations, diagnosis is frequently missed because of the rarity of the condition.

Pathology

- In most of the cases, the rent is longitudinal with well-defined edges 0.5–20 cm long, located on the left posterolateral wall, ~ 5 cm above the diaphragm. However, unusually, a rent can be transverse. A rent can occur in the posterior wall or right posterolateral wall
- Flooding of mediastinum with gastric contents and mixed oral flora → a fulminant mediastinitis with hemorrhagic necrosis. Mediastinal pleura soon ruptures leading to massive pleural effusion leading in turn to ventilatory insufficiency, hypovolemia and shock.

Mallory-Weiss syndrome = massive hematemesis ± melena following persistent retching/vomiting. Single/multiple mucosal tears occur at the esophagogastric junction. t

Boerhaave syndrome	*Mallory-Weiss syndrome*
– Results from failure of relaxation of the upper esophageal sphincter	Results from failure of relaxation of the lower esophageal sphincter
– Transmural tear	Mucosal tear(s)

www.Ebook777.com

Clinical features of Boerhaave syndrome
- Most common in males; however otherwise it can occur in anybody
- Sudden onset of tearing substernal/epigastric pain, which may be radiating to the back/shoulder, and which may be accentuated by swallowing/bodily movement following any of the precipitating event.
- Possible precipitating event: Repetitive vomiting / bouts of vomiting following overindulgence of food and drink / bouts of vomiting following sedation / GA / thoracic / abdominal blunt trauma / excessive straining during defecation / childbirth / sports event / epileptic seizure.
- Haematemesis is rare
- Ventilatory insufficiency. Tachypnea. Cyanosis
- Shock
- Cervical subcutaneous emphysema ±
- Guarding in the epigastric region.

One set of differential diagnosis includes acute cardiovascular emergency like MI, PE, dissecting aneurysm. – Patient is admitted then to ICCU. If clinical picture remains unrecognized for ≥ 24 hours, an appalling array of complications are virtually certain.

Investigations
- **X-ray chest**.
  - Pleural effusion/hydropneumothorax on anyone or both sides. Pneumothorax alone is rare
  - Cervical/mediastinal emphysema
  - Mediastinal widening
  - Subdiaphragmatic air ± (rare).
- **Pleural tapping**

In the initial period, before rupture of mediastinal pleura → Serous fluid

Gastric contents, food particles in the tapped fluid suggests that rupture of mediastinal pleura has occurred

- **Esophagogram with water-soluble** contrast medium: Extravasation

If negative ↓

Barium swallow using thin barium
- **In late/complicated cases**: **CT scan (with oral contrast)**
- **Indication for esophagoscopy**: Only occasionally, if all other investigations are negative or equivocal.

CORROSIVE INJURY

= injury sustained following ingestion of strong acids/alkalies.

GENERAL REMARKS

Corrosive **injury in**

Children
- Usually accidental
- Injury is sustained only on the 1st swallow. Corrosive material is so noxious that the child will not ingest more than a mouthful as a rule

∴ Injury tends to be confined only to the esophagus

Adults

Often suicidal ↓

∴ Tendency to ingest larger quantities ↓

∴ Injury may extend to involve stomach or even duodenum

Free ebooks ==> www.Ebook777.com

Corrosives

– Batteries: Sulfuric and hydrochloric acids; may contain strong alkalies also
– Oven cleaners, drain cleaners, toilet bowl cleaners, farm equipment cleaners: Na- and K-hydroxide
– Household cleaners: Ammonium solutions
– Household bleach: Hypochlorite solution

} These are weak bases. They are less caustic. They cause only mucosal injury. No deeper penetration. No long-term consequences

Mechanism of producing injury

– Acids → coagulative necrosis
– Bases → liquifactive necrosis

Both strong acids and bases are extremely hygroscopic; they penetrate deep into the tissues by attaching water of the tissue to their molecule.

HISTORY

- Has **anyone witnessed** actual ingestion?
- **Tearing substernal/epigastric pain**, which may be radiating to the back/shoulder, and which may be accentuated by swallowing/bodily movement?
- Pain in abdomen?
- Vomiting, hematemesis?
  – H/O ACTUAL INGESTION MAY OR MAY NOT BE RELIABLE.
    Particularly in children, there may be extensive corrosive injury in children whose parents may give h/o ingestion of hardly any amount of corrosive. The reverse may also be true.
  – Tearing substernal/epigastric pain suggests esophageal (frank) perforation.

PHYSICAL EXAMINATION

- Is there any **compromise of the airway**?
  Compromise of the airway ⇒ possibility of laryngeal/tracheal injury. Laryngoscopy/bronchoscopy will be necessary in the subsequent work-up.
- Drooling of saliva?
- Examine the **lips and oral cavity**. Any **perioral/intraoral burns**?
  Associated perioral/intraoral burns usually suggests strong possibility of esophageal injury too. However, the absence of perioral/intraoral burns DOES NOT rule out esophageal injury necessarily.
- Palpate the **neck** for subcutaneous emphysema.
  Tachypnea/cyanosis, shock, cervical subcutaneous emphysema ⇒ (frank) esophageal perforation.
- Examine the **abdomen**.
  Peritonitis ⇒ perforation of the stomach/duodenum.

IDENTIFY THE NATURE OF THE INGESTED MATERIAL

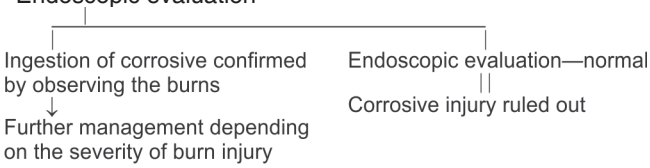
– From label on the container/batteries if available
– From chemical analysis in the laboratory, if necessary and possible.

PRINCIPLES OF MANAGEMENT

– Hospitalize even on suspicion of ingestion
– NBM. IV fluids. Broad-spectrum antibiotic. Analgesic. Steroids have no role
– Acute airway obstruction if present, suggests laryngeal edema caused by aspiration. → Tracheostomy.
  Long-term complication of laryngeal/tracheal burn: Laryngeal/tracheal stricture
– Urgent gastric lavage *only if* the patient is seen *within 6 hours*
– Add the antidote to the lavage fluid:
  - Magnesia/chalk/washing soda for corrosive acid
  - Vinegar or juice of 5 lemons for corrosive alkali

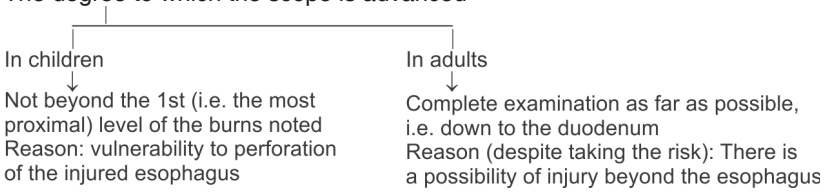
www.Ebook777.com

- Lavage fluid: Tepid water (preferably with added antidote). 500 ml in an adult, 250 ml in a child at a time
- Total amount of fluid to be used for lavage—5 L. Send the first washouts to the laboratory for analysis
- Maintain the balance of the fluid instilled and the fluid drained. A marked discrepancy suggest perforation of the stomach; in that case, discontinue lavage.
- Endoscopic evaluation



Principles of initial endoscopic evaluation

- Instrument used: Flexible or rigid. However, practically almost always a flexible scope is used
- Size of the scope:
  - In children—the smallest size available
  - In adults—it is safest to use a pediatric scope
- Minimum air insufflation
- The degree to which the scope is advanced*



*In case of difficulty or if the lumen is not easily visible, IT IS SAFER TO TERMINATE the procedure.

Simple classification of the degree of injury based on endoscopic finding

1°: Erythema and edema of the mucosa.
2°: Ulceration (floor made of white/dark colored slough), involving less than the full circumference.
3°: Ulceration involving the full circumference of the esophagus.

Criteria of 'severity' of the injury

- Ingestion of industrial-strength corrosive
- Ingestion of a 'large' quantity

} In isolation, they may be considered as predictors of severe burns

- Third-degree burns on endoscopic evaluation—this is the exclusive criterion.

Free ebooks ==> www.Ebook777.com

62 INGESTED FOREIGN BODY

GENERAL REMARKS

*Foreign body ingestion in **children***	v/s	*Foreign body ingestion in **adults***
– Types of foreign bodies—wide variety. Common: Toys and coins		Common: Bone splinters, fish bones
– Prolonged incarceration of a foreign body is possible		Prolonged incarceration is exceptional. A rare situation: a foreign body was incarcerated in childhood and diagnosed in adulthood

- **High-risk group** in general: Children, people with dentures, psychotic patients, prisoners, alcoholics.
- *Ways in which denture-wearers become predisposed*: 1. Palatal sensation is diminished with maxillary dentures, 2. A loose-fitting denture itself may be swallowed accidentally.

Sites of impaction

- A foreign body can stick in the tonsils, vallecula, pyriform fossa, or postcricoid region
- Most fish bones stick in accessible regions, usually the back of the tongue or tonsils
- Foreign body seldom gets stuck in the larynx itself. If however it does, the person starts gasping all of a sudden and collapses.

Sites of impaction in the esophagus

- of a foreign body
  - The commonest level of impaction in a healthy esophagus is cervical (just below the posterior lip of cricopharyngeal muscle)
  - Mid-thoracic or lower esophagus
    - A pre-existing esophageal disease must be suspected
- of a bolus of food
  - Food-impaction is always related to a pre-existing esophageal disease (organic/motor)
  - Levels of impaction in the descending order of frequency: Lower > cervical > mid-thoracic

A foreign body that passes harmlessly through the upper esophageal sphincter and cervical esophagus usually reaches the stomach without difficulty.

Objects likely to cause perforation or impaction

– Long / sharp / pointed
– Objects with a configuration likely to prevent passage through the pylorus or D_2

Natural history of a swallowed foreign body

- Most reach the stomach without difficulty
- May reach the stomach in spite of difficult esophageal passage
  - If the foreign body is sharp it may cause mucosal laceration (usually superficial)
- Get incarcerated (i.e. impacted) in the esophagus
  - If untreated
    - **Complication—esophageal perforation** (leading to mediastinitis / empyema / esophago-aortic fistula / T-E fistula.)
    - Rarely, remains undiagnosed for a long time
  - On endoscopic removal
    - In most cases, no mucosal damage
    - In ~ 5% cases → Deep laceration
    - In another 5% cases → Esophageal perforation is evident

www.Ebook777.com

Late gastric/duodenal perforations complicating toothpick / chicken / fishbone ingestion are insidious and associated with much morbidity; they are rarely identified preoperatively.

The complications of small intestinal foreign bodies requiring surgical treatment: Obstruction, perforation, bleeding. In the absence of a history of unusual ingestion or of the finding of a radiopaque foreign body, the specific cause is rarely identified preoperatively. The clinical presentation and the indications for surgery in patients with complications of foreign body ingestion are indistinguishable from other causes of obstruction, perforation, bleeding.

The site of obstruction is most commonly in the area of narrowing:

– Normal areas of narrowing—the distal ileum, IC junction
– Abnormal areas of narrowing—inflammatory stricture, tumor, diverticula, etc.

Foreign bodies in the small intestine: True foreign bodies (metallic/wooden/plastic, etc.), bezoars, food bolus, enteroliths, gallstones, concretions, intestinal parasites, and narcotic packets/any other objects swallowed for the purpose of smuggling.

Foreign bodies are **frequently missed on stool examinations**.

HISTORY

- Ingestion of a foreign body—**definite or probable**?
- **Exact time** of swallowing of the object?
- **Details of the foreign body**:
  – Which object?
  – Size, shape, smooth or having sharp edges, pointed or otherwise?

  Reasons for knowing the exact nature of the foreign body: Certain types of foreign bodies need active management e.g. **1**. Foreign body of any nature, proved or suspected to have been stuck in the esophagus. **2**. A rigid object longer than 6 cm (even if successful in reaching the stomach, will not negotiate the DJ flexure). **3**. Sharp/pointed objects may perforate, especially the terminal ileum. **4**. Batteries, however small in size may be hazardous as they may leak strong alkali leading to necrosis and perforation of the intestine.
- Any **problem during swallowing**?:
  – Was the episode of swallowing difficult?
  – Was the (cricopharyngeal) passage painful?
  – Was it associated with coughing/choking?
- **Vomiting of blood**?—Severity?

  Possible causes of hematemesis
  – Laceration of the esophageal mucosa by sharp edge of the object
  – Excessive retching leading to M-W tear
  – Erosion of a major artery, especially in a long-standing case
  – Severe hematemesis could suggest aortic puncture (provided the foreign body is sharp and the impaction is at the level of aortic arch).
- **Pain**, worsened by flexion of the neck?
Odynophagia? } ⇒ Perforation of the cervical esophagus
- Substernal pain? ⇒ Perforation of the thoracic esophagus
- Epigastric pain? ⇒ Perforation of the thoracic or lower esophagus
- **Painful sensation** aggravated by swallowing, **behind the sternum**?—may suggest incarceration. The site of discomfort/pain is often higher up than the actual level of impaction
- Pain in the **back**?—may suggest mediastinitis.
- **Dysphagia**? ⇒ Perforation or incarceration of the esophagus
- **Drooling of saliva**?
- **Distress** behind the sternum?
- **Gag**?
- **Paroxysmal cough**?
- **Refusal to feed**?

} ⇒ Incarceration of the esophagus

Free ebooks ==> www.Ebook777.com

- **Have you been suffering from an esophageal disease**?
  Symptoms s/o esophageal disorder: Dysphagia, excessive belching, difficult/unable to belch, odynophagia (= painful swallowing), heartburn, vanishing heartburn, regurgitation, water-brash, pain in the chest
- **SOB**?
- **Spiking fever**? } ⇒ Mediastinitis
- **Similar h/o foreign body ingestion in the past**?
  Possible causes of recurrent such episodes: Pre-existing esophageal disease, or the patient may be psychotic or a prisoner.

EXAMINATION

– Tachypnea, cyanosis, shock ⇒ esophageal perforation
– Oral cavity
– IDL: Look for retention of saliva, hypopharyngeal foreign body, edema of arytenoid region
– Neck: Tenderness, emphysema?
– PA/E: Perforative peritonitis?

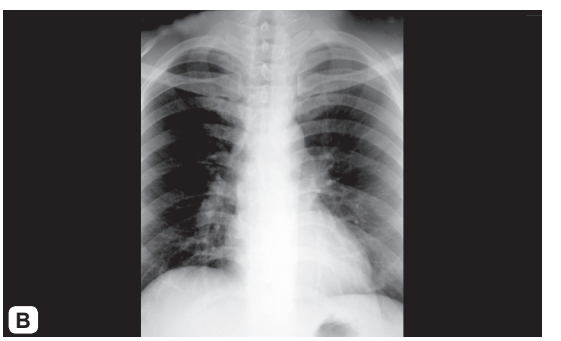
OBTAIN A DUPLICATE OF THE FOREIGN BODY

Whenever possible, obtain a duplicate of the foreign body in order to select accurately the proper endoscope and forceps to use. The duplicate can also afford an opportunity to study possible presentations of the object in the esophageal lumen.

PITFALLS IN ARRIVING AT THE DIAGNOSIS

1. The patient may have almost no symptoms.
2. The child may present merely with 'failure to thrive'.
3. If the patient is very young or mentally incompetent, there may not be any history available of having swallowed a foreign body.
4. Symptoms following ingestion of a foreign body may have lasted for hours or years!
5. The diagnosis of foreign body ingestion is usually obvious, but a missed diagnosis can cause persistent dysphagia and loss of weight.
6. Gastric outlet obstruction due to a foreign body is not encountered as frequently as is small intestinal obstruction. Gastric retention of foreign bodies may be related to pre-existing gastric outlet obstruction.
7. **A foreign body under the age of 8 months** (i.e. too young to crawl or grasp efficiently), **or multiple foreign bodies, especially if sharp/pointed, should raise a strong suspicion of child abuse**.





Figs 62-1A and B Size and shape of the object: **(A)** Could not have been imagined only by evaluating the X-ray; **(B)** Look carefully at the level of T3

www.Ebook777.com

INVESTIGATIONS

Select from the following list as applicable in the given case.

1. **X-ray** (of soft tissues) of the **neck—AP** and **lateral** views.
   A clue which has anatomical basis, to differentiate a foreign body in the trachea from one in the esophagus: The plane of lodgment of the object revealed in a X-ray provides a useful information; for the maximum dimension of the trachea is in the sagittal plane, and of the esophagus in the coronal plane.
   In addition, emphysema if present, in the retrovisceral space in front of C6 ⇒ basically foreign body in the esophagus.
   Emphysema in the retrovisceral space is the earliest sign of esophageal perforation.
2. **X-ray chest—AP** and **lateral** in **erect** position.
   – A radioopaque foreign body with its details viz. level in the esophagus, size, shape, smooth or having sharp edges, pointed or otherwise?
   – Signs s/o esophageal perforation: Pleural effusion / hydropneumothorax, cervical / mediastinal emphysema, mediastinal widening.
3. **X-ray abdomen—AP** and **lateral** in **erect** position.
4. **If a foreign body is thought to be radiolucent**: Thin barium—swallow.
5. **If a fish bone is suspected**: Ask the patient to swallow a barium-dipped cotton wool ball or marshmallow. Repeat X-rays after some time.
6. Investigate for esophageal perforation, if suspected.

Guideline statements of diagnosis of some selected cases

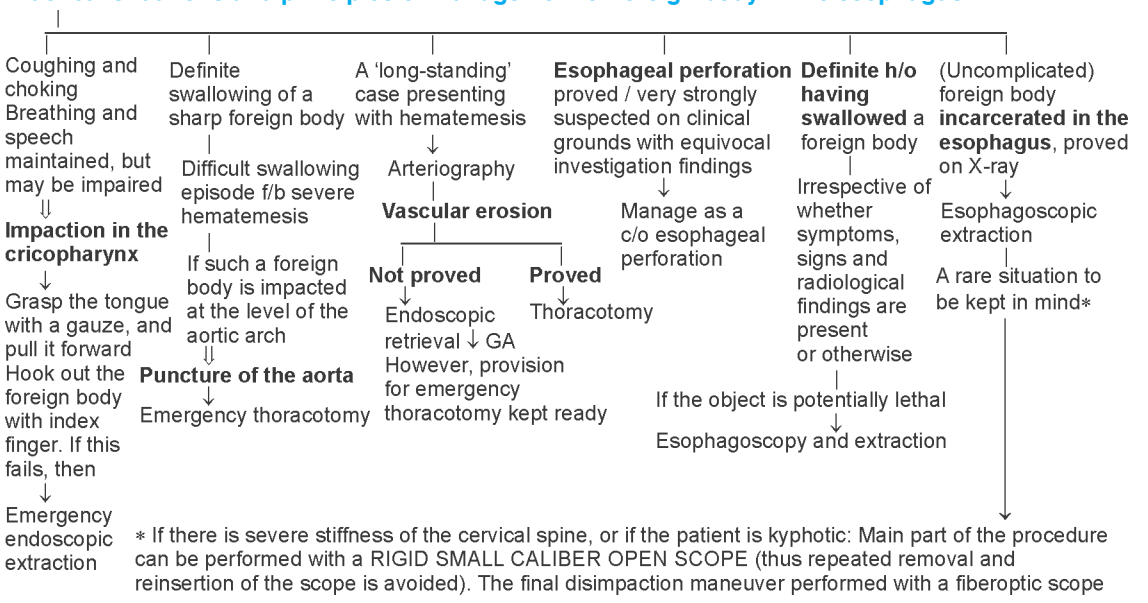
- A foreign body **impacted in the cricopharynx**.
  Details of the object: The object; its size, shape, edges, pointed/otherwise, if pointed, is the point facing cranially or caudally, any other details (? ferromagnetic, etc.)
- A foreign body in the esophagus **at the level of the aortic arch**, **most probably leading to puncture of the aorta**.
  Details of the object: The object; its size, shape, edges, pointed/otherwise, if pointed, is the point facing cranially or caudally, any other details (? ferromagnetic, etc.)
- A foreign body in the esophagus, (incarcerated) since ….., complicated by **esophageal perforation**.
  Details of the object: The object; its size, shape, edges, pointed/otherwise, if pointed, is the point facing cranially or caudally, any other details (? ferromagnetic, etc.)
- **Suspicion** of swallowing a foreign body (on ….. at ~ …..), **without symptoms**, **without positive physical findings** and **without positive radiological findings**
- **Suspicion** of swallowing a foreign body on ….. at ~ ….., **most probably incarcerated in the esophagus**, **without positive radiological findings**
- **Definite h**/o swallowing a foreign body **(…..)** on ….. at ~ ….., **without symptoms**, **without positive physical findings** and **without positive radiological findings**
- **Definite h**/o swallowing a foreign body on ….. at ~ ….., **most probably incarcerated in the esophagus**, **without positive radiological findings**
- A foreign body **in the esophagus, (incarcerated) since** ….
  Details of the object: The object; its size, shape, edges, pointed/otherwise, if pointed, is the point facing cranially or caudally, any other details (? ferromagnetic, etc.)
- A foreign body **in the stomach, without complication as yet**
  – Details of the object: The object; its size, shape, edges, pointed/otherwise, if pointed, is the point facing cranially or caudally, any other details (? ferromagnetic, etc.)
  – Duration: …….

Indications for hospitalization of the patient

1. Foreign bodies known to have potential for causing trouble.
2. If outpatient observation is not reliable.

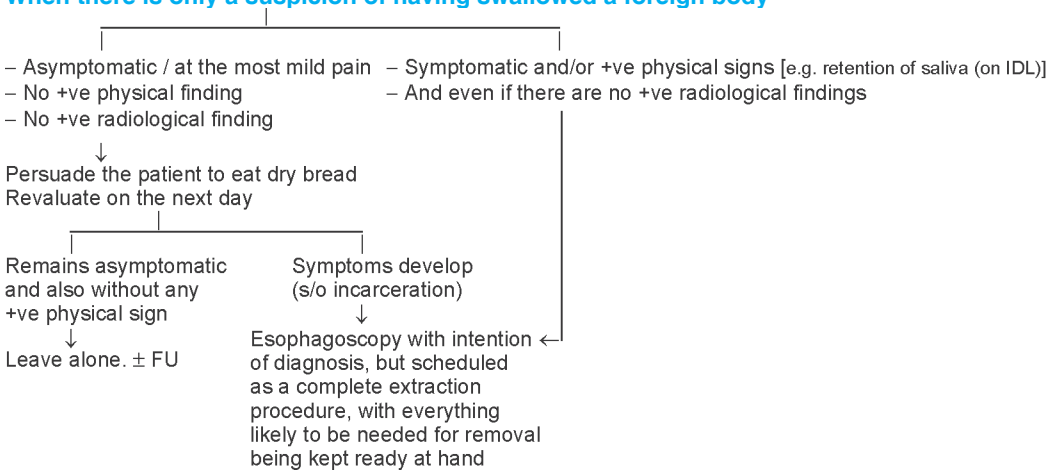
Free ebooks ==> www.Ebook777.com

Practical situations and principles of management of foreign body in the esophagus



* If there is severe stiffness of the cervical spine, or if the patient is kyphotic: Main part of the procedure can be performed with a RIGID SMALL CALIBER OPEN SCOPE (thus repeated removal and reinsertion of the scope is avoided). The final disimpaction maneuver performed with a fiberoptic scope

When there is only a suspicion of having swallowed a foreign body



If a **smooth** foreign body is **lost in the stomach**, leave it to pass naturally

Exceptions: 1. A caustic foreign body e.g. alkaline battery. 2. The presence of a pre-existing gastric outlet obstruction

A **smooth** object **which seems to have been caught in the lower esophagus due to sphincter spasm, which should otherwise pass down the rest of the GIT with ease**: Try injection probanthine ± diazepam—to relax the lower esophageal sphincter, and observe if the object passes on into the stomach.

Indications for removal of a foreign body from the stomach (/duodenum), in the absence of complication

- Object that is long and sharp / pointed
- Object larger than the duodenal loop
- Object retained in for > 4 weeks
- The presence of pre-existing gastric outlet obstruction.

www.Ebook777.com

Indications for operative removal of gastric foreign bodies

- Perforation, bleeding
- Accumulation of multiple foreign bodies
- Failure of, or contraindication for endoscopic extraction.

Principles of treatment of foreign bodies in the small intestine, in the absence of complication

- The treatment is essentially dictated by the nature of the foreign body.
  A foreign body which can negotiate the loop ('C') of the duodenum will usually proceed through the small intestine and colon unimpeded and will pass out naturally without untoward effects
- *Surgery may be indicated* if a foreign body fails to pass as evidenced by lack of progress on serial X-rays. In children, the average time for passage of an object through the small intestine is 5 days. If their parents are reliable, children can be managed as outpatients to await spontaneous passage confirmed by observation of stools or X-ray if required
- Certain categories of foreign bodies have special features that may dictate surgical removal
- A proportion of pointed objects (toothpicks, chicken bones, fish bones, splinters, etc.) may cause perforation.

Disk batteries

Alkaline batteries can be harmful if their caustic contents leak in the stomach. Otherwise they are innocuous unless there is radiographic evidence of disruption of the battery case.
Mercury, lithium, silver batteries are innocuous.
Once such round smooth batteries reach the small intestine, virtually all will pass.

Objects containing lead

Lead poisoning rarely occurs with a single solid lead object until it has been in the GI tract for at least 4 weeks.

Narcotic packets

- Packets of narcotic powders, e.g. heroin, cocaine, etc. covered with latex are swallowed for the purpose of smuggling
- *Packets are frequently visible on plain X-rays*
- *Complications*: 1. Obstruction, 2. Rupture of the packets leading to acute narcotic toxicity and even death
  The fatal oral dose of cocaine = 1–3 g
- On confirmed diagnosis (definite history or/and detection on X-ray), or if toxicity is present, surgical removal is indicated
- In the case of **narcotic package ingestion for the purpose of smuggling**, endoscopic extraction is contraindicated as intragastric breakage of the package may prove fatal
- If there is no risk of rupture of packet (due to unbreakable covering), mild cathartics may be tried and patients may be discharged when proved free of packets by radiological and serial stool examination.

DON'Ts

1. Do not attempt extraction by a flexible endoscope, ↓ LA, WITHOUT protecting the airway (by ET intubation ↓ GA).
   Note: Flexible endoscopy may be employed only as an investigative modality, if X-rays failed to confirm the presence of a foreign body. Flexible endoscopy ↓ LA should never be employed to retrieve the foreign body unless it is very small and can be firmly grasped; **the best policy however, is to avoid the temptation**. Reason—if unfortunately the object is dropped, it may enter the airway.
2. Do not attempt at digestion of impacted vegetable / meat foreign bodies using 'tenderizer'. Reason—it can cause erosion and perforation of esophagus / stomach.

Life-saving measures for a foreign body stuck in the larynx causing asphyxia

Carry out the following measures one after the other in succession, if the previous one fails.

1. Grasp the tongue with gauze, pull it forward. Hook out the foreign body with a (index) finger.
2. Heimlich maneuver

Free ebooks ==> www.Ebook777.com

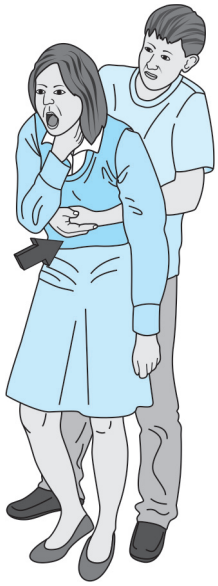


Fig 62-2 Heimlich maneuver

- Stand behind the patient
- Grasp the arms firmly over the epigastrium and lower chest from behind the patient. In other words, embrace the patient from behind and put the fist under the xiphoid
- Give short firm squeezes to the upper abdomen and chest every 10 seconds for half a minute. This maneuver rapidly compresses the lungs, and may push out the foreign body, particularly food.

3. Insert a wide-bore needle (or a blade) through the cricothyroid membrane to create an airway to save life (by bypassing the obstruction caused by the foreign body).

www.Ebook777.com

63 ABDOMINAL TRAUMA
(Including Pelvic Trauma)

Abdomen (including pelvis) = a circumferential region between the diaphragm and the infragluteal fold.

GENERAL REMARKS

1. The recognition of two major groups, blunt and penetrating, is of greatest importance since it has direct implications for the diagnostic work-up and treatment.

2. *Anatomical considerations*

For the purpose of evaluation, abdomen may be divided into 4 areas / zones—intrathoracic, true, pelvic and retroperitoneal. Assessment on physical examination is relatively easier only in true abdomen; it is difficult in rest of the zones.

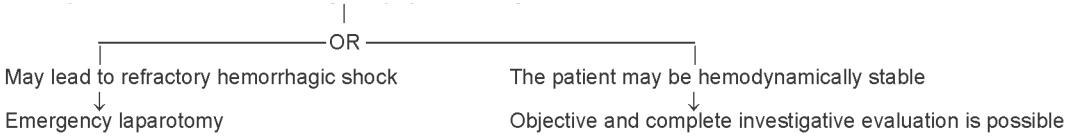
Zone	*Contents*	*Remarks*
• Intrathoracic abdomen	Diaphragm, liver, spleen and stomach	Specific investigations which play an important role in evaluation: Plain X-ray, DPL
• True abdomen	Small and large intestine, bladder when distended, and uterus when gravid	Specific investigations which play an important role in evaluation: Plain X-ray, DPL
• Pelvic abdomen	Small intestine, rectum, urinary bladder, urethra and internal genital organs	Mechanisms which can injure this area of abdomen: # pelvis, penetrating injuries of the buttocks Adjunctive procedures helpful in making the diagnosis: Bladder catheterization, urethrocystography, sigmoidoscopy
• Retroperitoneal abdomen	Aorta, vena cava, pancreas, D_1 and D_2, kidneys and ureters, and ascending and descending colon	Investigations which may play important role in evaluation: Salivary amylase, IVP, CT, angiography

3. *A note on causes* of abdominal trauma

- Road-traffic accidents are the most common cause of blunt trauma
- Iatrogenic causes: Barium enema, endoscopy, laparoscopy, PTC, liver biopsy, paracentesis, peritoneal dialysis, suprapubic cystostomy, external cardiac massage
- Penetrating wounds of the thorax often cause significant intra-abdominal injury.

Sudden application of pressure to the abdomen is more likely to rupture a solid organ than a hollow viscus.

Primary manifestation of a solid organ injury = bleeding



Free ebooks ==> www.Ebook777.com

4. Clinical manifestations following abdominal trauma may be minimal/vague. Investigation is essential to confirm the diagnosis.
5. In any patient with unexplained abdominal symptoms and signs, inquire carefully about a h/o trauma, perhaps mild and forgotten during the previous week.
6. More elastic tissues of the young tolerate trauma better, leading to a less severe intra-abdominal injury in children as compared to adults.
7. Few patients survive if peritonitis has been developing for 16 hours, but most will survive if they are operated upon in the 1st 6 hours.

HISTORY

- **Cause** of trauma. Details. **Time** of injury?

Pain.

- Exact site?
  |
  Does the site of pain seem to be elsewhere than the site of impact?
- Any pain in the **shoulder**?
- Is the pain **getting better or worse**?
- **Nausea**?
- **Vomiting**?
- **Hematemesis**?
- **Melena**?
- **Diarrhea**?
- Did you **pass urine** or not, after the injury?
- **Difficulty in passing urine / increasing distension of the lower abdomen**?
- **Hematuria**?—getting better, or increasing?
- **Thirst**?
- **Dyspnea**?
- **Fainting**?
- How much time before the trauma had you taken meal?
- How much time before the trauma had you passed urine?

} This is to know whether the stomach/bladder was distended at the time of impact to the abdomen

An anesthesiologist is more concerned about the distended stomach.

In **GENERAL EXAMINATION**

- Prostration?
- Is breathing halted, labored?
- Does the patient appear to be restless, or does s/he lie quietly on the bed?

EXAMINATION OF THE ABDOMEN

The key objective is to determine intra-abdominal bleeding or/and peritoneal irritation and consequently identify the need of surgery. The precise anatomical localization of organ-injury may not be necessary.

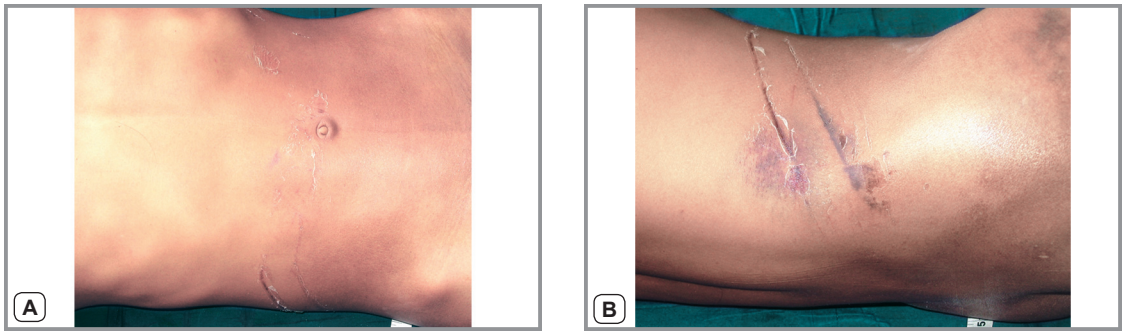
INSPECTION

- Mark/s of **external injury**?
- **Imprint** of clothing or injuring agent?
- **Distension**?
- **Fullness** in a specific region?

PALPATION

- **Tenderness**?
  - Site?
  - Degree?

www.Ebook777.com



Figs 63-1A and B Marks of external injury and ecchymosis (Both photos belong to the same patient)

- **Rigidity**?
  - Site; localized or diffuse?
  - Degree?
- **Depth of penetrating wound**, if any?
  - Objective: to determine whether the peritoneum is torn or it is intact. If the peritoneum is torn, there is likelihood of a serious intra-abdominal injury
  - If the length and breadth of the wound are small and / or if the patient contracts the abdominal wall muscles, it may not be possible to examine the peritoneum without exploring the wound.

PERCUSSION

- Areas of dullness?
- Fixed / shifting dullness?
- Obliteration of liver dullness?

AUSCULTATION

- Try to hear for peristaltic sounds over the abdomen **as well as over the chest** for 2 minutes.
- Are the peristaltic sounds present and normal, or diminished/absent?

EXAMINATION OF THE PELVIS AND EXTERNAL GENITALIA

- **Tests for # pelvis**
  - Press over the pubic symphysis: Tenderness over the sacroiliac joints suggests # pelvis
  - A marked suprapubic tenderness suggests possibility of urogenital injury
  - Feeling a gap at the pubic symphysis suggests a hinge fracture
  - Palpate for tenderness, irregularity and crepitus over the pubic bone in the genitocrural recess, the obturator foramina, and the iliac crests
  - Grasp the pelvis firmly over its anterior superior iliac spines on both sides, and compress from side to side
  - Press the pelvis firmly backwards onto the bed
  - Try to pull the two sides apart (by holding the anterior superior iliac spines)

  } Severe tenderness / abnormal mobility / crepitus ⇒ # pelvis
- Blood at the **external urethral meatus**?
- Ecchymosis / hematoma in the **perineum**?
- On **DRE**, look specifically for:
  - Integrity of the rectal wall
  - Fullness/tenderness in the rectovaginal / rectovesical pouch
  - Position and mobility of the prostate
  - Blood on the finger-stall.

Free ebooks ==> www.Ebook777.com

EXAMINE THE LOWER LIMBS FOR # ACETABULUM WITH/WITHOUT DISLOCATION OF THE HIP

- Is there any apparent shortening?
  However, normal limb length does not exclude dislocation. Shortening occurs only if the displacement is gross.
- Attitude of the limb?
- Ask the patient to lift the foot off the bed.—is it possible?

EXAMINE THE SPINE

Examination of the patient completes at this juncture.

ABDOMINAL GIRTH

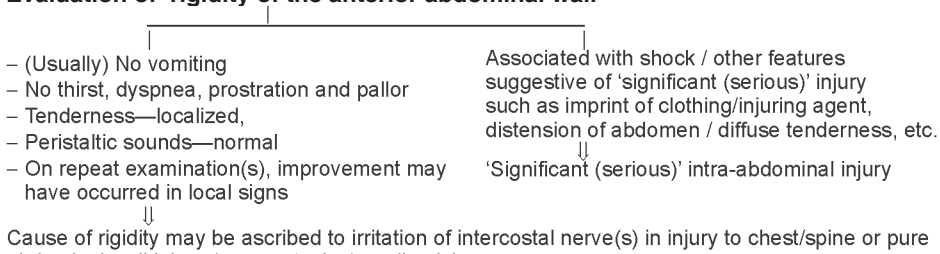
Take a baseline measurement at the level of the umbilicus.
The baseline value will be useful if serial measurements need to be taken subsequently.

INFERENCE FROM SYMPTOMS AND SIGNS

Pain away from the site of blunt impact ⇒ very high probability of a serious intra-abdominal injury; and a laparotomy is strongly indicated
Pain in the shoulder may suggest referred pain due to irritation of undersurface of the diaphragm by intra-abdominal bleed
Pain getting worse probably means continued bleeding, or a leaking gut
Hematuria suggests injury to the urinary tract Injury to the urinary tract is usually not associated with intraperitoneal injuries (by virtue of the nature of trauma)
An injury to the distended urinary bladder may result in intraperitoneal rupture with peritonitis
Causes of **halted**, **labored breathing**: Diaphragmatic irritation / upper abdominal injury
The site of the mark of external injury suggests the organ beneath that is likely to have been injured
Imprint of clothing or injuring agent suggests a severe compression force leading to severe internal injuries usually requiring emergency operation
Small areas of **abrasions/ecchymoses** may represent warnings of significant intra-abdominal injury underneath
Posterior ecchymosis should raise the possibility of retroperitoneal injury
Distension occurring rapidly after trauma suggests intraperitoneal injury
However, retroperitoneal injury may lead to distension after 24 hours
Tenderness, and **guarding/rigidity** ⇒ significant intra-abdominal injury provided fallacies are ruled out
Tenderness away from the site of blunt impact ⇒ very high probability of serious intra-abdominal injury; and a laparotomy is strongly indicated.

The site of tenderness suggests the possible organ injured underneath.
- Diffuse tenderness suggests perforative peritonitis or hemoperitoneum
- Severe tenderness ⇒ (septic) peritonitis
- Tenderness that is less marked ⇒ a hemoperitoneum
- Increasing tenderness (over a period of time) usually requires a laparotomy.

Evaluation of 'rigidity of the anterior abdominal wall'



www.Ebook777.com

- **# ribs** → guarding, but usually no tenderness of the abdominal wall
- Upper abdominal rigidity may be present due to fracture of lower ribs (irritation of intercostal nerves)
- Localized rigidity may be present at the site of contusion of abdominal wall
- 'Board-like' rigidity (i.e. 'severe degree' of rigidity) ⇒ perforative peritonitis (due to perforation of GIT, biliary tract)
- 'Lesser degree' of rigidity ⇒ internal (intraperitoneal) bleeding / 'urinary peritonitis' (intraperitoneal rupture of urinary bladder).

Convexity and dullness over a fixed area ⇒ fluid hematoma or clotted blood underneath, which can suggest possible organ injured. Example: Fullness and fixed dullness in the loin may be due to retroperitoneal hematoma, which could be due to renal injury

The absence of peristaltic sounds ⇒ internal bleeding (intraperitoneal or retroperitoneal) OR perforation of a viscus (perforative peritonitis)

Peristaltic sounds heard in the chest (practically left side) ⇒ rupture of diaphragm

Blood at the external urethral meatus ⇒ urethral injury. Catheterization should be deferred until urethrography rules out a rupture

Fullness/tenderness in the rectovaginal/rectovesical pouch may indicate hemoperitoneum

Aspiration of blood through NGT ⇒ injury to the stomach; and usually is an indication for laparotomy in penetrating trauma

In a c/o blunt abdominal trauma
- Suprapubic/lateral pelvic wall tenderness
- Blood at the external urethral meatus
- Ecchymosis/hematoma in the perineum

} ⇒ **# pelvis**.

Fracture of pelvis
- Without signs in the peritoneal cavity, and the presence of normal peristaltic sounds ⇒ no associated intraperitoneal injury
- Associated with peritoneal irritation may suggest intraperitoneal rupture of bladder
- Associated with difficulty in passing urine, hematuria, increasing fullness in the lower abdomen ⇒ extraperitoneal rupture of bladder / intrapelvic rupture of urethra.

Special clinical methods applicable in only doubtful/difficult cases

1. Look for **orthostatic hypotension**: This test may be useful if a patient does not have a significant hypotension and has no other obvious cause of blood loss.
2. Serial measurements of **abdominal girth** at the same baseline level viz. the umbilicus.
   - An increasing girth ⇒ paralytic obstruction / free fluid/gas in the peritoneal cavity
   - Avoid fallacy by making sure that a NG tube is in place. Without a NG tube, swallowed air can cause a false positive ↑ in girth.

PRINCIPLES AND PITFALLS IN ARRIVING AT THE DIAGNOSIS

1. Blunt or penetrating injury to the back also may result in significant intra-abdominal injury.
2. Wounds in the lower part of the chest, and occasionally wounds in the groin, gluteal region or perineum may penetrate inside the abdominal cavity.
3. In multiple system trauma, the associated injury(ies) to the CNS/chest/musculocutaneous system may obscure the abdominal injury.
4. *Blunt trauma is particularly difficult because*:
   a. A patient may give no clear history that s/he has had an abdominal injury, especially if she/he is a frightened child.
   b. For the first few hours after trauma, the abdomen may be deceptively normal, especially in children.

Free ebooks ==> www.Ebook777.com

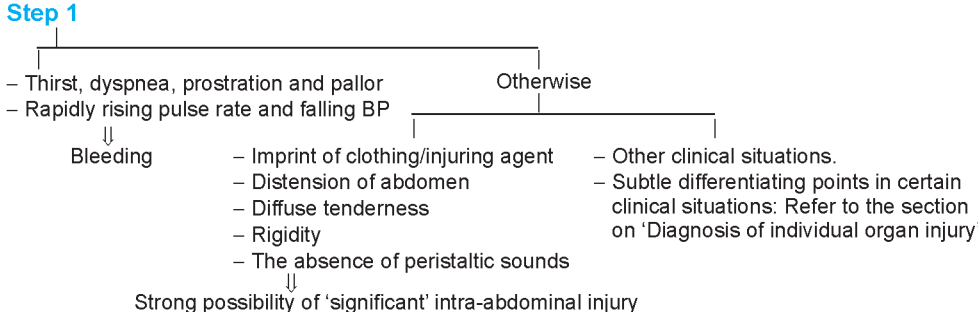
c. Distinguishing between muscle pain and peritoneal irritation can be very difficult.
d. Some injuries may not show themselves for several days, especially a subcapsular hematoma of the spleen, or a retroperitoneal injury of the pancreas or duodenum.
e. Other more obvious injuries may distract the attention of focus away from the abdomen. Moreover, if such a patient is undergoing anesthesia for a prolonged operative procedure for (a non-abdominal) injury, neither the patient will be able to complain of (increasing) pain in abdomen during and for some time afterwards, nor will s/he be available for frequent evaluation.
f. A patient who is drunk or is unconscious from brain injury will not be able to tell the symptoms.

5. Patient is quiet in peritonitis but restless in bleeding.
6. Determining the absence of peristaltic sounds may be helpful to assess significant peritoneal irritation from blood or intestinal contents, but it often takes significant time to develop.
7. With suspicious abdominal injury, constant monitoring and repeated revaluation are essential.
8. Intraperitoneal injury can occur in blood vessels, solid organs or hollow organs. Development of physical findings is often a function of the amount of time that each of the types of organ requires to produce the clinical effects of injury.
   - Mesenteric artery laceration may → only hypovolemic shock without any +ve abdominal findings
   - Gastric/colonic perforation → immediate peritoneal irritation
   - Small intestinal perforation may not produce any significant abdominal findings for up to 24 hours.

   Due to such varied timing of development of abdominal physical findings, frequent revaluation (half hourly) **is essential**.
9. Small areas of abrasions/ecchymoses should not be disregarded as they may represent warnings of significant intra-abdominal injury. As a matter of fact, blunt trauma may lead to an acute life-threatening intra-abdominal injury sometimes without even a scratch mark on the abdominal wall.
10. *Open v/s closed injury*: Closed injury = Skin and subcutaneous tissue are intact or if they are torn, then the peritoneal cavity is not opened up (i.e. peritoneum is intact).
11. Upper abdominal injuries are associated with internal bleeding due to rupture of liver/spleen.
12. Lower abdominal injuries are associated with rupture of the intestine.
13. Bleeding and perforation may both coexist.
14. '**Does the patient seem to have lost more blood than can be accounted for by the known injuries**?' – This is a good evidence of abdominal or thoracic bleeding.
15. Assume that all patients with a fractured pelvis have complications until proved otherwise.
16. **A particularly prominent sign of occult infection** is (post-traumatic) pulmonary insufficiency.

ARRIVING AT THE DIAGNOSIS in a case of BLUNT ABDOMINAL TRAUMA

Step 1



www.Ebook777.com

Step 2: Possible site of injury and pathological effect?

Mechanism/nature/site of trauma	*Possible organ/s injured*
– Penetrating injury between the nipples and costal margin	Diaphragm
– Penetrating injury in the epigastrium	Stomach, duodenum, pancreas, retroperitoneal vessels
– Sudden application of pressure to the abdomen	Solid organ
– Blunt/penetrating injury in the inferior aspect of the chest (with/without # lower ribs) or upper quadrant of abdomen	Liver / spleen
– Fist/kick/handlebar blow in the epigastrium / steering wheel impact / seat-belt injury	Duodenum, pancreas.
– Trauma to the back	~ 20% chance of a significant renal injury

Classification of parts of the abdomen where injury can occur

- ***Abdominal wall***
- ***Intraperitoneal***
  a. Internal bleeding:
     – Immediate or delayed
     – Due to rupture of
       - Solid organs (liver / spleen)
       - Mesenteric or other blood vessels
       - Rarely gravid uterus.
  b. Peritonitis due to rupture of a hollow viscus like stomach/duodenum/intestine, etc; OR intraperitoneal rupture of urinary bladder.
- ***Retroperitoneal***
- ***Pelvic***: Bladder, urethra, rectum, blood vessels
- ***More than one or all of the above***
- ***Thoracoabdominal***.

Differentiation between intraperitoneal and (extensive) retroperitoneal bleeding:

- Note the pace of deterioration, and the presence or absence of shifting dullness
- More rapid deterioration of the condition and the presence of shifting dullness suggest intraperitoneal bleeding.

ARRIVING AT THE DIAGNOSIS in a case of PENETRATING ABDOMINAL TRAUMA

1. Is the patient in **hemorrhagic shock**?
   |
   If yes, an emergency laparotomy is almost invariably indicated (after making the fullest attempts at resuscitation in the shortest possible time).
2. **Mechanism of injury**?—hand-driven weapon (knife, dagger, etc.) OR firearm?
3. Is there an **obvious evidence of one/more of the following**?
   - 'Open abdominal wound', i.e. peritoneum has been opened suggests that visceral injury could be present
   - Protrusion of omentum / other viscus
   - Internal bleeding
   - Peritoneal irritation (peritonitis).
4. In the absence of any obvious finding/sign as above in step 3
   ↓
   What is the depth of the wound?—is the peritoneum intact or torn?
5. **Which organ/s** possibly could have been damaged? Is the **damage serious enough to warrant a laparotomy**?

Free ebooks ==> www.Ebook777.com

Points which may give a clue

a. Mechanism of injury:
   - Wounds from a hand-driven weapon usually but not always necessarily tend to follow a predictable path
   - Bullets may follow an unpredictable path, and cause widespread damage.

b. The site of injury: For example, penetrating injury between the nipples and costal margin.
   – Think of possibility of diaphragmatic injury.

Some DO's and DON'T's

– **Any foreign body embedded should not be removed until** arrangement for a major exploration is ready and the patient shifted to the operation theater as major bleeding might follow removal of the embedded foreign body
– If injury to the lower urinary tract is suspected because of an associated pelvic fracture, catheterization should be deferred until urethrography is performed to rule out injury to the urethra.

INDICATIONS FOR REFERRAL/ADMISSION TO HOSPITAL (for active management or for observation)

1. Shock. Cold nose could be the only subtle sign of shock initially.
2. Obvious or probable breach of peritoneum.
   – Usually applicable to a penetrating injury.
3. Any abdominal symptom following trauma. Even the mildest nausea, diarrhea, or pain in abdomen after abdominal trauma, however trivial the trauma may appear to have been, should be admitted to hospital. **There should be no exception to this rule**.
   – This situation is more usually applicable to a case of blunt trauma.
4. GI symptoms such as gaseous eructation, epigastric distress, postprandial discomfort and unexplained attacks of vomiting developing after abdominal trauma, should be fully investigated; they may be due to post-traumatic diaphragmatic hernia.
5. Hematemesis / melena.
6. Hematuria.
7. Difficulty in passing urine / increasing distension of the lower abdomen.
8. Examination findings:
   – Fracture of lower ribs/pelvis
   – Distension
   – Tenderness
   – Rigidity
   – Diminished OR absent peristaltic sounds
   – Peristaltic sounds heard in the chest
   – Blood at the external urethral meatus
   – Vaginal/digital rectal examination suggesting a pelvic injury: Loss of integrity of the rectal wall, fullness/tenderness in rectovaginal / rectovesical pouch, high / floating prostate, blood on fingerstall on vaginal and / or rectal examination.

KEY INVESTIGATIONS

Urine—Routine and microscopic

- > 30 RBCs/hpf is an indication for radiographic evaluation of bladder/kidneys.

Full blood count

- A leukocytosis is suggestive
- A leukocytosis of $\geq$ 15000 is common with a hemoperitoneum.
- A leukocytosis is more useful than a low Hb or hematocrit.

US scanning in a c/o blunt abdominal trauma

- *Specific objective* is to detect free fluid in the peritoneal cavity (and also in the pleural cavities and pericardium) rather than specific injuries
- *Limitations*: 1. Obesity, gas interposition, subcutaneous emphysema. 2. Small amounts of free fluid are difficult to identify

www.Ebook777.com

- *In a hemodynamically unstable patient, a negative US scan* indicates that the bleeding source is other than the abdomen
- *In a hemodynamically stable patient, a positive US scan* does not, per se, indicate the need for surgical exploration
- In this situation, CT may help to identify the source of bleeding and additionally help in decision-making about the treatment—operative *v/s* nonoperative.

Role of abdominal paracentesis ('4 Quadrant Tap')

- *Indication*: Suspected hemoperitoneum
- Free aspiration of essentially nonclotting blood = *positive result*
- On positive result, mobilization of the case to operation theater can be expedited.

Pitfalls

– Although the blood from a hemoperitoneum is usually defibrinated and does not clot, there is always a chance that it may not do so
– Negative result has no diagnostic significance. In that case, either diagnostic tap may be repeated in an hour or two, or DPL may be performed.

The test can also be useful in the diagnosis of other conditions such as:

– Ruptured urinary bladder (aspirate—urine)
– Perforated GIT in general (aspirate—cloudy/frankly purulent)
– Perforated upper GIT/gallbladder (aspirate—bile-stained fluid/cloudy/frankly purulent).

Diagnostic peritoneal lavage (DPL)

Indications:

A. Equivocal physical findings in a patient with suspected abdominal injury in following situations:
   1. Deteriorated LOC due to brain injury/intoxication.
   2. High energy transfer.
   3. Multiple injuries and unexplained shock.
   4. Spinal cord injury.
B. Possible candidate for intra-abdominal injury with equivocal physical findings, who will be undergoing prolonged general anesthesia and surgery for other (i.e. non-abdominal) injuries and thus unavailable for continued evaluation.

Contraindications: Obvious surgical abdomen with clear-cut indication for laparotomy, pregnancy, previous abdominal operation, morbid obesity.

Comment: A negative result does not exclude an abdominal injury.

Culdocentesis

– One of the most useful and accurate ways of confirming intraperitoneal bleeding in a woman
– Aspiration of > 1–2 mL of essentially non-clotting blood suggests hemoperitoneum.

Role of plain X-ray of chest with diaphragm, and abdomen including pelvis, in blunt trauma

– Fractures with potential for associated visceral injury
– Free gas ↓ the diaphragm
– Raised hemidiaphragm
– Loss of psoas shadow indicates retroperitoneal bleeding
– Indentation on, or displacement of the shadow of the stomach/splenic flexure of colon
– Ground-glass appearance between loops of small intestine may be the first sign of hemoperitoneum
– Herniation of the viscera in the pleural cavity suggests rupture of the diaphragm
– Hemothorax/hemopneumothorax.

Role of CT scanning

– Best for retroperitoneal injury, e.g. suspected pancreatic, duodenal injuries
– Another indication: Positive US scan in a hemodynamically stable patient.

Role of diagnostic laparoscopy

– In anterior or flank stabs—to determine peritoneal violation
– In blunt trauma it is not useful as it is not superior to other investigative modalities.

Free ebooks ==> www.Ebook777.com

Role of arteriography
- Therapeutic: Embolization
- Diagnostic: Renal artery thrombosis.

Monitoring
Revaluate half hourly.
Watch for:
- Restlessness
- Rising pulse rate, falling BP
- An increase in abdominal girth
- Development of diffuse tenderness, rigidity, paralytic obstruction.

Patient may be stable for a long-time and then deteriorate rapidly.

Indication for local exploration of a stab wound ↓ anesthesia (usually local anesthesia)
- When it is not possible clinically to ascertain whether the peritoneum is intact or torn, in the absence of a clear indication for laparotomy on the basis of other criteria.

GENERAL INDICATIONS FOR LAPAROTOMY

In general, decision-making is based on hemodynamic status, mechanism of injury, physical findings, transfusion-requirements, and metabolic derangement.

Obvious indications:
- Hemorrhagic shock thought to be due to abdominal injury
- Firearm wounds (Most of the patients will have intra-abdominal injury)
- The presence of pain/tenderness away from the site of a blunt impact
- GI bleeding (Hematemesis / rectal bleeding)
- Evisceration (Laparotomy is indicated even if there is only a tag of omentum protruding)
- Peritonitis
- A penetrating injury involving the peritoneum (i.e. peritoneum torn)
- Penetrating anal/vaginal injury
- Herniation of abdominal viscus into the thorax through a diaphragmatic injury
- +ve DPL
- Deterioration (restlessness, tachycardia, hypotension), increasing distension/tenderness during the course of monitoring.

Stab wounds can be managed using a policy of selective exploration.
If there is a doubt as to whether to operate or not, it may be safer to operate. “It is better to open and see rather than to wait and see”.

Establishment of priorities
Examples:
- Two teams may simultaneously operate on two different system-injuries, e.g. intracranial and intra-abdominal
- Consider ICD in a c/o fracture of ribs who also requires GA with ET intubation for laparotomy. This is to prevent tension pneumothorax
- Combined orthopedic and vascular injuries: If operative treatment is indicated for both, then reduction and fixation of a fracture before repair of vascular injury (particularly interposition grafting) may be more appropriate.

BRIEF NOTES ON **DIAGNOSIS OF INDIVIDUAL ORGAN INJURIES**

Diaphragm
Suspect: Penetrating injury between the nipples and costal margin.

Presentation
- Visceral herniation detected on X-ray chest upright
- Respiratory distress and radiological e/o pleural effusion not getting relieved after IC drainage.

www.Ebook777.com

Investigations other than X-ray chest
- In patients with hemo-/pneumothorax: DPL / thoracoscopy
- In patients with normal X-ray chest: Laparoscopy.

During exploratory laparotomy for traumatic abdomen, diaphragm MUST be examined.
A possible delayed complication due to missed injury: Complicated enterocele.

Spleen
Suspect
- Blunt/penetrating injury in the inferior aspect of the left side of chest or LUQ of abdomen.
- Fracture of left lower ribs.

Presentation
- Clinical manifestations may be surprisingly few.
- Hemorrhagic shock, locoregional pain, tenderness and guarding, pain in the left shoulder.
- Presentation can be delayed for a few days/weeks, due to delayed bursting of a sealed hematoma.

Types of injury: Contusion, capsular tear, laceration, fragmentation.

Specific investigation
US scan. If free fluid is detected, and if the patient is hemodynamically stable, then → CT.

Liver
Suspect
- Blunt/penetrating injury in the inferior aspect of the right side of chest or RUQ of abdomen.
- Fracture of right lower ribs.

Presentation
- Clinical manifestations may be surprisingly few
- Hemorrhagic shock, pain, tenderness and guarding in RUQ, pain in the right shoulder.

Types of injury: Subcapsular hematoma, capsular tear, laceration, fragmentation, lobar destruction, hepatic artery/venous injury.

Stomach
Stomach is injured more commonly with penetrating rather than with blunt trauma
Bloody NG tube aspirate is highly suggestive
During laparotomy, posterior wall of the stomach should also be completely examined.

Duodenum
Basically, duodenal injury could be intraperitoneal or retroperitoneal. Signs are delayed in retroperitoneal injury
The cause/mechanism of high morbidity/mortality associated with missed injury is the development of lesser sac abscess with consequent sepsis.

Suspect
- Penetrating injury between xiphoid and umbilicus
- Blunt trauma in epigastrium.

Specific investigations
1. S. Amylase by itself is neither specific nor diagnostic. It plays a supporting role.
2. Plain X-ray:
   - Obliteration of psoas shadow, air in the retroperitoneum outlining the kidney
   - Spastic lumbar lordosis in association with fracture of transverse process suggests major injury in the area and therefore, a likelihood of duodenal injury.
3. Contrast duodenography (or CT with oral contrast)
   - Extravasation of contrast—obviously an absolute indication for laparotomy
   - Distortion of duodenum suggests a significant injury—a relative indication for laparotomy.
   - Intramural hematoma—should be left alone. However, if it causes obstruction that fails to resolve, then consider laparotomy.

Free ebooks ==> www.Ebook777.com

Pancreas

Suspect

– Penetrating injury in epigastrium
– Blunt trauma in epigastrium.

Clinical manifestations may be insignificant.

Specific investigations

1. Serum amylase by itself is neither specific nor diagnostic. It plays a supporting role.
2. Plain X-ray:
   – Obliteration of psoas shadow
   – Left pleural effusion.
3. Contrast duodenography, if done for suspicion of duodenal injury, may show widening of the C-loop of the duodenum.
4. CT.

Patients seldom undergo laparotomy because of pancreatic injury alone. Instead, they are operated upon for associated injury(ies) leading to more evident/clear clinical manifestations forming stronger indication for laparotomy.

Colon and rectum

– Most common cause—penetrating trauma
– Fracture of pelvis, penetrating injury to buttock can → rectal injury
– Cause of morbidity/mortality associated with rectal injury is mainly delayed diagnosis and sepsis
– Colon and rectum are relatively refractory to blunt trauma. Blunt trauma → only 5% of colonic injuries. However, when it occurs, the consequences can be devastating. Colonic injury is usually a part of multisystem/multiorgan injury. Alteration in the LOC as well as the presence of associated injury present problems for diagnosis and therapeutic priorities
– A high index of suspicion is required to diagnose the colonic injury
– Perforation may be discovered 7–10 days following trauma and may be indicated by findings s/o sepsis.

A particularly prominent sign of occult infection is (post-traumatic) pulmonary insufficiency.

Clinical manifestations and investigations in a colorectal injury

1. In c/o intraperitoneal injury
   – Peritonitis
   – On DRE: Blood on the finger-stall
   – Proctoscopy / sigmoidoscopy
   – Plain X-ray may show free gas ↓ the diaphragm
   – DPL.
2. Extraperitoneal injury of the colon and rectum is extremely difficult to diagnose.
   – On DRE: Blood on the fingerstall
   – Proctoscopy / sigmoidoscopy.

Fracture of pelvis

Suspect

– Crush injury
– Contusion / local tenderness in the pubic area / groin / perineum / posteriorly over the sacroiliac joints.

Investigations

– Urine: Routine and microscopic
– X-ray pelvis and hip—AP and lateral views.

Classification

– **Minor**: A piece of the pelvis breaks off; the ring itself remains intact. Apart from the associated soft tissue / bony injuries (e.g. # acetabulum) if any, these fractures are of little importance in themselves and need no treatment
– **Major** fractures open the pelvic ring
– 'Compound pelvic fractures' = fractures associated with deep perineal lacerations / rectal / vaginal lacerations.

www.Ebook777.com

The main concerns about pelvic fractures: 1. Associated injuries to the pelvic organs, e.g. urethra, rectum, femoral artery, etc; and 2. Massive bleeding
The principle cause of mortality in fracture pelvis is excessive bleeding. A patient may loose > 20 units of blood. Control of ongoing pelvic retroperitoneal bleeding is the greatest challenge.

Fracture of acetabulum with or without dislocation of hip

Suspect
- Head-on-collision of motor vehicles—dashboard injury
- Fall of heavy weight on a stooping person

} And when such a patient is unable to walk

Clinical features of dislocation
Basically, the patient is unable to walk, and there may be apparent shortening.

Differentiating points
- Thigh—flexed, adducted and medially rotated
- Fullness of femoral head palpable in the gluteal region
- Femoral pulse may be weak

} ⇒ Posterior dislocation

- Thigh—flexed, abducted and laterally rotated
- Fullness of femoral head palpable on DRE

} ⇒ Central dislocation

- Head of the femur felt over obturator foramen. ⇒ Anterior dislocation.

Notes
- The outlook for a patient with an acetabular fracture depends mostly on whether or not the head of the femur has destroyed the upper part of the acetabulum. This is the part which bears the weight, and if enough of it remains unbroken, the outlook is good
- # of the posterior rim, and # of the floor with central dislocation are often missed.

If a patient with abdominal trauma is brought in late (> 18 hours after the trauma)

If the patient feels well, looks well, is afebrile, has no signs of peritonitis or abscess formation, and if the site of the wound is only minimally tender: None of the viscera may have been perforated or the perforation may have got sealed off
A laparotomy may not be necessary. Observe such a patient. Operate if s/he deteriorates.

ABDOMINAL COMPARTMENT SYNDROME FOLLOWING TRAUMA

= Development of sudden increase in intra-abdominal pressure
Patients who are most susceptible: Those with severe intra-abdominal injuries presenting in profound shock and requiring large amounts of intravenous fluids
Criteria: Abdominal distension, oliguria, hypoxia and increased pulmonary inspiratory pressure.

Free ebooks ==> www.Ebook777.com

64 UROGENITAL TRAUMA

GENERAL REMARKS

The female lower urinary tract is vulnerable to obstetric disaster, but seldom to accidental trauma.
The prostatic urethra is protected by the prostate and is seldom injured.

Suspect urinary tract injury in the following situations:

1. In blunt trauma, if the victim has # lower ribs (10–12)/# lumbar vertebrae/hematuria/contusion or ecchymosis in the flank.
2. Penetrating injury to the back/flank/upper abdomen/lower chest/anywhere along the course of the urinary tract.
3. Decelerating injury in a RTA/fall from height—may cause renal injury like disruption of PUJ/renal artery thrombosis.

Any significant **clinical manifestations** suggestive of urinary tract injury may be totally absent, including hematuria.

The key clinical points in a c/o lower urinary tract injury:

- Can the patient void?
- Urethral bleeding? Even a single drop at the external urethral meatus is significant.
- Is the bladder palpably distended?

Interpretation of the above mentioned key clinical points:

– 'Inability to void' is common in both bladder and urethral injuries.
– Urethral bleeding and palpably distended bladder ⇒ site of injury distal to the bladder neck, and only occasionally proximal to the bladder neck. A pitfall: Bladder may not distend if patient's hypovolemic shock has not been corrected.
– Hematuria if at all patient can void, and impalpable bladder ⇒ bladder injury.

By and large, physical examination is not reliable in the diagnosis of urinary tract injuries.
Urinary tract injuries are amenable to radiological evaluation.

INVESTIGATIONS

Urine R and M

Hematuria ⇒ injury to the urinary tract. Hematuria is present in many cases of renal/ureteral injury, and in > 95% cases of bladder injury. However, hematuria may be absent. The absence of hematuria does not exclude a possibility of urinary tract injury.

IVU

Practical indications:

1. Penetrating injury in the vicinity of the urinary tract regardless of the presence of hematuria.
2. > 50 RBCs/hpf in urine.

All said and done, many patients with blunt trauma and hematuria have a normal IVU. A normal IVU does not necessarily mean a normal kidney.

Retrograde urethrography (RGU) (essentially for visualization of a male urethra, especially the anterior urethra). RGU may be performed in suspected urethral injury. For indications and policy, refer to the section on 'Urethral injury' below.

www.Ebook777.com

Posterior urethra is either incompletely opacified or not opacified at all, depending upon pressure applied and resistance offered by the external urethral sphincter.

Retrograde cystography (RGC)
Indication: Suspected bladder rupture in a case of hematuria with lower abdominal (pelvic) trauma.

Findings and interpretation

a. – Unclear outline, collapse of bladder
   – Extravasation of medium into the peritoneal cavity outlining loops of intestine with opacification of paracolic gutters, subhepatic spaces
   – Rapid absorption of contrast medium and excretion by kidneys

   } ⇒ intraperitoneal rupture of bladder.

b. – 'Teardrop' shaped bladder (due to compression by a large pelvic hematoma)
   – Irregular, stellate-shaped opacification
   – Flame-like distribution of contrast medium confined to the pelvis
   – Very slow absorption of extravasated contrast medium.

   } ⇒ extraperitoneal rupture of bladder.

Fallacy: Cystogram may fail to disclose extravasation if a clot occludes the perforation.

CT
Indication/utility: Preoperative staging of renal injury. CT-IVP interpretation of renal injury is indefinite.

RENAL INJURY

Remarks

1. Hematuria out of proportion to the h/o trauma or magnitude of physical finding ⇒ probable presence of a pre-existing disease (of the kidney) like vascular malformation/tumor/hydronephrosis/cystic disease, etc.
2. Most cases of blunt renal trauma are related to a direct blow to the loin.
   Blunt renal trauma (especially due to a RTA) may be associated with other major intra-abdominal injuries.
3. Renal artery thrombosis is rare but is classically associated with an acceleration/deceleration trauma, such as a fall from height onto the feet.
4. Causes of iatrogenic trauma: PCNL, renal biopsy. These cases may either present with primary bleeding, or secondary bleeding due to a false aneurysm.
5. Blood may clot in the ureter and initially present hematuria may stop, even though bleeding from the injured kidney may be continuing. Stoppage of hematuria therefore does not necessarily mean that all is well.
6. Gross hematuria *may be* associated with more severe injury as compared to microscopic hematuria; however, the degree of hematuria *does not* correlate with the severity of the injury.
7. Secondary bleeding and severe hematuria can occur up to 3 weeks later, so it is essential to observe even apparently minor kidney injuries carefully.
8. In c/o a blunt trauma, if adequate blood is transfused, the patient will probably recover, even if there has been severe hematuria, a falling BP, and a large mass in the loin.

Classification of renal injuries:
Minor: – Contusion, subcapsular hematoma, superficial (cortical) laceration.

Major: – Deep (corticomedullary) laceration with extravasation and large perinephric hematoma.
– Renal pelvic injury: Disruption of PUJ (i.e. avulsion of the pelvis from the upper ureter), penetrating / perforating injury of the pelvis which may not be associated with renal parenchymal injury.
– Pedicle injury: Partial/complete transection with free bleeding, arterial/venous thrombosis, avulsion of the pedicle.
– 'Shattered' kidney.

Suspect
Mechanism of trauma:
– Blunt trauma in the loin/upper abdomen/lower chest.
– Penetrating trauma in the loin/upper abdomen/lower chest.

Free ebooks ==> www.Ebook777.com

– Decelerating injury in a RTA/fall from height landing onto feet (—may cause renal injury like disruption of PUJ/renal artery thrombosis).

Symptoms and signs:

– Hematuria.

  Over a period of time, the color of red urine changing to brown ⇒ the injured kidney is healing. If it becomes red again, then it means that further bleeding has started.

– Contusion or ecchymosis in the flank.
– A renal mass (suggests a major injury associated with a perinephric hematoma or urinoma). However, a renal mass may not be felt easily in early stages, because of tenderness and guarding of the overlying abdominal wall.
– A penetrating wound behind the anterior axillary line.
– A bruit, after a penetrating trauma (suggests a traumatic AV fistula).
– # lower ribs (10–12)/# lumbar vertebrae (L1/2).

Modes of clinical presentation

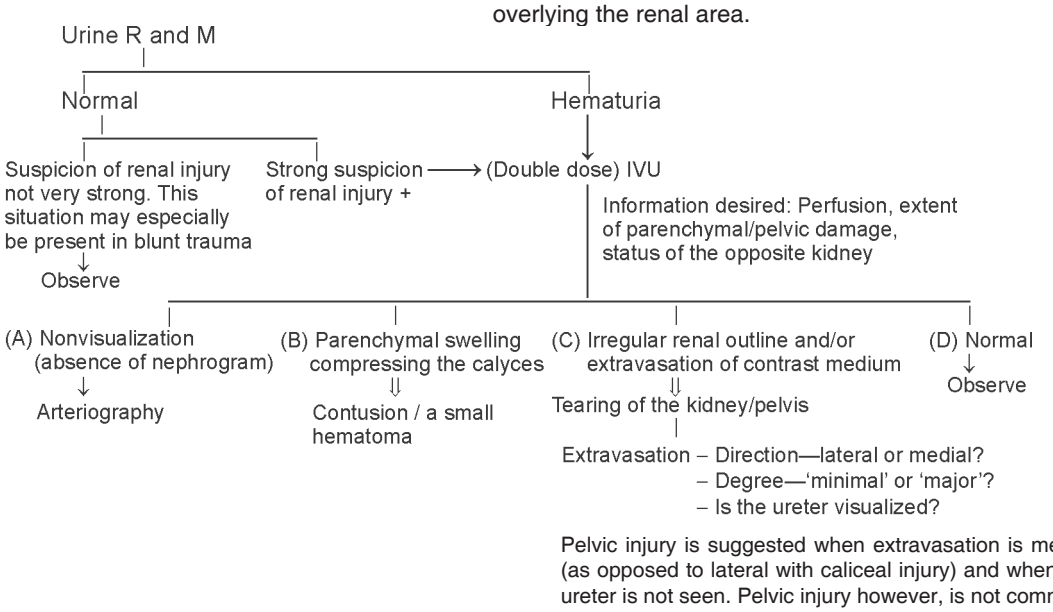
1. Gross hematuria.
2. Microscopic hematuria in a c/o blunt/penetrating trauma in the loin/upper abdomen/lower chest.

Investigative evaluation

Principles:

– All patients with suspected renal injury and hematuria (gross or microscopic), *must be* investigated radiologically.
– A following exception may be made in *an adult* patient with *blunt trauma having microscopic* hematuria: If there is no h/o rapid deceleration, no shock, no abnormal physical finding in the loin, and no e/o intra-abdominal injury, IVU may be deferred. In such patients the incidence of major renal injury is very low. Some minor renal injuries will be missed but they would in any case be treated conservatively and have a low complication rate.
– This exception *does not apply to pediatric patients*. Any degree of hematuria in a child following blunt trauma must be investigated fully.

Findings suggestive of renal injury on a plain X-ray: # lower ribs (10–12), # lumbar vertebrae (L1/2), scoliosis, obliteration of psoas shadow, ground–glass density overlying the renal area.



Pelvic injury is suggested when extravasation is medial (as opposed to lateral with caliceal injury) and when the ureter is not seen. Pelvic injury however, is not common

www.Ebook777.com

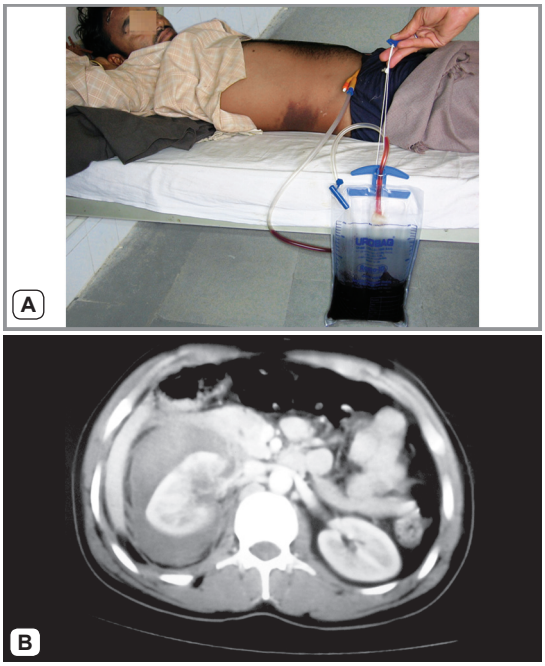
In c/o (A): If facility for arteriography is not available, then US scan may help in decision-making. On US scan if there is a presence of kidney, but which is not visualized on IVU obviously logically ⇒ a pedicle injury viz. arterial tear or acute thrombosis.

In c/o (B) and (C): A CT should be requested.

Role of (indications for) **renal angiography**
- Nonvisualization of the kidney (the absence of a nephrogram) on IVU.
- In secondary bleeding, to demonstrate a false aneurysm.
- Also useful in interval exploration for a major laceration, where partial nephrectomy is contemplated.

Role of US scanning

US scan shows parenchymal lacerations and perinephric collections well and is a useful complement to IVU if CT is unavailable.



Figs 64-1A and B (A) A case of blunt trauma. Note ecchymosis in the flank and gross hematuria; **(B)** CECT shows deep laceration of the kidney and retroperitoneal hematoma

Complications of renal trauma

1. Bleeding:
   - Persistent primary bleeding.
   - Secondary (delayed) bleeding 10–14 days post-injury.
2. Urinary extravasation:
   - Urinary fistula.
   - Urinoma/pseudocyst.
   - Hydronephrosis secondary to PUJ obstruction due to fibrosis or to pressure from a urinoma.
3. Infection:
   - Pyelonephritis.
   - Infected perinephric hematoma/urinoma.
   - Septicemia.
4. Infarction—segmental or total.
5. Hypertension—following infarction or scarring.

URETERAL INJURY

Causes
1. Ureteral injury is most commonly caused by penetrating trauma.
2. Blunt trauma causing ureteral injury occurs more commonly in children. – Severe flexion hyperextension injury leading to PU junction disruption.
3. Iatrogenic:
   - During urological procedures—endourological procedures, bladder neck suspension operation, etc.
   - During nonurological procedures—hysterectomy, colectomy, aortoiliac bypass, lumbar laminectomy, any other difficult retroperitoneal/pelvic surgery.

***Penetrating** injury*

Mostly patients will have associated nonurological intra-abdominal injury for which most patients will have a laparotomy.

1. *Ureteral injury suspected preoperatively*:
   - A penetrating wound behind the anterior axillary line.
   - Hematuria (usually microscopic) ±.
   - IVU will confirm the diagnosis. In a small number of cases IVU may fail to confirm the injury; in these cases retrograde ureterography will demonstrate the tear.

Free ebooks ==> www.Ebook777.com

2. *Factors which lead to suspect ureteral injury intraoperatively*:
   - From trajectory of the penetrating wound (hand-driven weapon/bullet).
   - Accumulation of excessive clear fluid in the abdomen, the clear fluid being thought to be urine.

   Method of demonstration and localization of the site of injury in those patients who were forced to be taken up for emergency laparotomy without preoperative evaluation by IVU: Chromo-ureterography – 5 mL of methylene blue or indigocarmine are injected intravenously. Extravasation of dye (blue-tinged urine) in the operative filed confirms injury and localizes the site.

Iatrogenic** ureteral injury **associated with (nonurological) abdominal surgery

The ureter may have been crushed/divided/ligated/included in a suture.

Most injuries involve the distal ureter, thus the ureter can be exposed proximally and followed distally to the suspect area. If in doubt, catheterize the ureter(s) via open cystotomy or cystoscopy. Proximal ureterotomy with distal passage of a ureteric catheter should be avoided (as it may cause a stricture, whereas open cystotomy has negligible morbidity).

***Ureteroscopic** ureteral injury*

→ usually perforation.

Modes of presentation of missed ureteral injuries

- Paralytic obstruction with raised S. Creatinine (due to reabsorption).
- Intra-abdominal (often retroperitoneal) sepsis. Pain ± lump in the flank, and fever.
- A urinary fistula through the tract of penetrating wound or drain site or an internal organ, e.g. vagina.
- Ureteric stricture with its consequences.
- Occasionally patient may remain asymptomatic, and the case may be diagnosed months/years later with a nonfunctioning kidney.

Imaging investigations in general. Selection needs to be individualized.

- US scan.
- IVU.
- Retrograde ureteropyelography if IVU fails to demonstrate/localize the tear.
- (Antegrade) Nephrostography (if a percutaneous nephrostomy has already been inserted) will give the same information as retrograde ureteropyelography.
- CT scan.
- Radioisotope scan.

Points to be taken into account while stating the diagnosis of a ureteral injury (These points have therapeutic and prognostic implications.)

1. **Acute** OR **Delayed**?
   - The earlier the diagnosis, the better the results. Late diagnosis is associated with a much higher nephrectomy rate.
   - In c/o an acute injury, a primary definitive repair may be possible.
   - In a delayed case, there is intense tissue inflammation and extravasation with associated sepsis. Immediate treatment recommended—Drainage and proximal diversion. A definitive repair should be done only later.
2. **Isolated** OR **As a part of multi-system trauma**?

   Operative technique, rate of healing and possibility of complication depends upon which additional organs are injured.
3. **Level**?
   - This determines the incision used for open repair.
   - Chances of healing are better for upper 3rd and lower 3rd ureteral injuries as they have a better blood supply. Repairs of the middle 3rd ureter are vulnerable with respect to breakdown due to relatively poor blood supply.
4. **Cause**?
   - Iatrogenic injuries occur most commonly at the pelvic brim.
   - Low-velocity penetrating wounds require minimal debridement before anastomosis. High-velocity penetrating wounds need extensive debridement and often 're-look surgery' at 48–72 hours to check viability.

www.Ebook777.com

BLADDER INJURY

Empty bladder at the time of trauma is more prone to extraperitoneal rupture, whereas a full bladder at the time of trauma is more prone to intraperitoneal rupture.

pelvis may rarely → combined intra- and extraperitoneal bladder rupture.

Types of rupture

1. Extraperitoneal: Almost always associated with # pelvis.
2. Intraperitoneal: The commonest site of rupture is the dome.
3. Combined extra- and intraperitoneal. – High morbidity and mortality.

Clinical manifestations suggestive of **acute traumatic bladder injury**

Differentiating points

– Inability to void without the sign of urethral injury (i.e. no urethral bleeding), and the bladder is impalpable.
– On catheterization—gross hematuria.

Supporting points

– Referred pain in the shoulder (—may suggest intraperitoneal rupture).
– Mark of blunt/penetrating injury in the suprapubic region, e.g. contusion/entry wound.
– Suprapubic tenderness.
– # pelvis.

(Make sure to look for injury to other pelvic organs viz. rectum, vagina).

A pitfall: Features of peritonitis usually suggest associated intra-abdominal injury (rather than an intraperitoneal bladder rupture) as the presence of uninfected urine in the peritoneal cavity elicits a minimal inflammatory response.

Clinical points to differentiate between intraperitoneal and extraperitoneal bladder rupture

1. *Mechanism of injury*
   – A blow to the abdomen suggests intraperitoneal rupture.
   – # pelvis suggests extraperitoneal rupture.
2. *The distribution of tenderness*
   – More diffuse tenderness over the lower abdomen suggests intraperitoneal rupture.
   – Narrowly localized suprapubic tenderness suggests extraperitoneal rupture.

It is wiser to wait a few hours to confirm the diagnosis, rather than to operate hurriedly unnecessarily and find only a hematoma which bleeds profusely or even disastrously if it is opened.

Delayed (~ 2–3 days after traumatic incident) **presentation of intraperitoneal traumatic bladder injury**

Diagnosis can be very difficult. The bladder tear may have been 'plugged' by omentum and the patient may be voiding urethrally, often with clear urine.

Clinical effects associated with delayed presentation:

– Ascites.
– Paralytic obstruction.
– Features of uremia (—due to reabsorption).

Iatrogenic bladder injury at open surgery or endoscopy is usually evident.

A missed iatrogenic bladder injury will present with complication.

Specific investigations

– Urine R and M, if patient voids.
– US scan.
– RGC.

Free ebooks ==> www.Ebook777.com

Complications

1. Pelvic abscess (a complication of extraperitoneal rupture).
2. Urinary ascites (a complication of an undiagnosed intraperitoneal rupture). Sequelae – peritonitis/intra-abdominal abscess/respiratory difficulty.
3. Urinary fistula (a complication of particularly missed iatrogenic injury, e.g. VVF following missed bladder injury at hysterectomy).

The female urethra, because of its short length and protected position under the symphysis pubis, is very rarely injured.

MALE URETHRAL INJURY

Causes

A. *Blunt trauma*
   - # pelvis (→ membranous urethral injury).
   - Fall astride or blow to perineum (→ bulbar urethral injury).
   - Sudden downward flexion of an erect penis (→ anterior urethral injury).

B. *Penetrating trauma*

C. *Iatrogenic trauma*
   - Traumatic catheterization/inadvertent inflation of balloon in urethra (instead of in the bladder).
   - Pulling out of a catheter without the balloon having been deflated.
   - Endoscope, especially resectoscope.
   - Open surgery: Rectal operations, anorectal operations, perineal debridement for necrotizing fasciitis.

Classification

Mucosal tear

- Urethra is intact.
- The clinical picture of a urethral injury following blunt trauma, with a normal urethrogram ⇒ a mucosal tear.

Partial rupture

- Laceration involving only a part of the circumference; so, continuity of the urethra still maintained.
- Urethrography picture: Extravasation and passage of contrast medium in the urethra beyond the area of injury.

Complete rupture

- Laceration involving the entire circumference with loss of continuity leading to distraction of the severed ends.
- Distraction may be minor or major. A major distraction is seen in posterior urethral injury, where the bladder and prostate are displaced upwards, and the ends are separated by a hematoma.

Types of posterior urethral injury

I = the mildest form: Stretching-elongation without rupture.
II = partial/complete rupture; extravasation is confined below the urogenital diaphragm.
III = the most severe form. Partial/complete rupture (of prostatomembranous urethra) and rupture of urogenital diaphragm and bulbar urethra. Extravasation in the perineum and pelvis.

Suspect

- Blunt/penetrating trauma to the lower abdomen/perineum/external genitalia. This is particular relevant if patient has a # pelvis.
- Urethral bleeding following catheterization or diagnostic/therapeutic endourethral procedure.

Classical clinical presentation irrespective of the site (i.e. injury anywhere along the length of the urethra)

- Urethral bleeding. *Even a drop* at the external urethral meatus is significant.
- Inability to void, although occasionally patients with a partial injury may void small volumes; and in that case, there will be hematuria.
- Acute urinary retention with a palpable bladder.

www.Ebook777.com

– Contusion / hematoma / diffuse vague swelling due to fluid collection in the scrotum/perineum/upper thigh.
– If necessary, milk the urethra to demonstrate blood at the tip.

Specific clinical features in posterior urethral injury
On DRE:
– Prostate in the normal position ⇒ a partial rupture, or a complete rupture with only minor distraction.
– Boggy fluid collection in the otherwise normal position of prostate and the prostate being impalpable ⇒ a complete rupture with major distraction.

Specific clinical features in anterior urethral injury
– Urethral rupture contained by Buck's fascia → dissection by urine and blood along the penile shaft, giving an appearance of sleeve on the penis.
– Rupture of urethra and corpora cavernosa through Buck's fascia → extravasation of urine and blood corresponding to the attachments of Colles' fascia.

Radiological investigations
A. *Policy for retrograde urethrography on suspecting a urethral injury clinically*: There are 2 schools of thought.
Policy 1: RGU in all cases.
Policy 2 = Selective policy (for certain reasons). An example of an indication for RGU in the acute phase under the 'selective policy': Penetrating trauma with a suspected urethral injury, where immediate exploration is contemplated (urethrogram helps to plan surgery).
B. An *IVU* is useful in patients with # pelvis and suspected posterior urethral injury.
   – Bladder located in the pelvis ⇒ a partial rupture, or a complete rupture with only minor distraction.
   – Bladder displaced out of pelvis and having a relatively normal shape ⇒ a complete rupture with major distraction.
C. If a 'trocar puncture' suprapubic cystostomy is done for a suspected posterior urethral injury, and urine is found to be bloodstained, a cystography must be done to exclude a bladder rupture (particularly extraperitoneal).

Complications of urethral injury
1. *Periurethral complications*: Extravasation of urine, periurethral abscess, necrotizing fasciitis, pelvic abscess.
2. *Stricture* – The most important complication.
   Partial rupture heals either without stricture or with a short stricture easily managed endoscopically.
   Complete rupture always forms a stricture.
3. *Fistula*:
   – Urethrorectal – due to original injury or attempted endoscopy of an impassable stricture.
   – Urethrocutaneous.
4. *Incontinence*.
5. *Erectile dysfunction*.

PENILE INJURY

This is discussed below with respect to causes, types of injury, clinical features, and principles of treatment.

Penetrating trauma
Low-velocity *gunshot wound/stab wound*
The clinical picture will depend on the site and the trajectory of the injuring agent.
Urethral bleeding would suggest a urethral injury. – managed on its own merits.
Penile injury will require repairing of defects in corpora cavernosa.

High-velocity *gunshot wounds and '****blow-out'*** *injuries* may cause enormous damage.
Rectal injury needs to be carefully looked for.

Fracture of penis without or with urethral injury
This is *caused by* sudden downward or upward bending of an erect penis during masturbation or intercourse.

Free ebooks ==> www.Ebook777.com

Clinical picture:
- Hearing of a snap/cracking sound and Painful tearing sensation at the base of penis.
- Minimal or no hematoma ⇒ tearing of the suspensory ligament. – Should be repaired to provide penile stability during intercourse.
- Tearing of the corpora cavernosa → hematoma at the site of injury if Buck's fascia is intact. Extravasation of urine and blood corresponding to the attachments of Colles' fascia ⇒ tearing of Buck's fascia. The defect itself may be palpable.
- Urethral bleeding ⇒ urethral injury.

Principle of treatment: Urgent surgical exploration and appropriate repair.

The organ **caught in machinery** may → a **degloving injury**.

Amputation
- Causes: Self-mutilation by a psychotic individual, assault.
- Bleeding from the stump should be controlled by a tourniquet.
- The amputated part should be cleaned and packed in ice in a sterile saline solution, and the patient transferred within 8 hours to a hospital with microsurgical facilities.

Complication of circumcision
Excision of too much of skin, glandular injury (with possible meatal stenosis), necrosis of a part or the entire penis.
Initial treatment: Antibiotics and topical dressings.
Skin grafting and urethroplasty may be needed later.

Human/animal bite
- High incidence of sepsis.
- Wound should never be closed primarily. Liberal debridement should be done under antibiotic cover.

Burns
Genital burns should be managed in the same way as burns elsewhere but a urethral catheter may be needed to facilitate dressings.

TESTICULAR INJURY

Types: Rupture, amputation.
Disruption of the tunica albuginea and extrusion of seminiferous tubules qualify for rupture.

Physical findings: Scrotal swelling, hematoma and tenderness make the testis and epididymis indistinct.

Principle of management: Urgent exploration. Decision to operate must be based on clinical judgment rather than on US scan report (irrespective of whether normal or abnormal).

UROLOGIC TRAUMA IN PREGNANCY

Some principles
1. The mother's life should take precedence over that of the fetus in case if life of both cannot be secured simultaneously.
2. Repeated physical examination and serial US scanning. IVU and CT scanning should be avoided as much as possible, and definitely so in the 1st trimester.
3. Indications for repair basically are the same as in a nonpregnant patient.
4. Options for major urinary extravasation: Double J stent, and PCN for temporary diversion of urine.

www.Ebook777.com

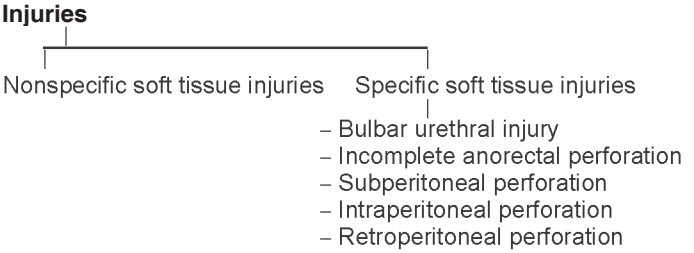
65 ANORECTAL AND PERINEAL TRAUMA

GENERAL REMARKS

1. Do not underestimate the possible gravity of the injury.
2. Usually, the diagnosis is not difficult to establish, because the h/o trauma is self-evident. Occasionally, a high index of suspicion must be maintained.
3. The failure to recognize an extraperitoneal perforation is **as potentially lethal as** failure to identify an intraperitoneal rupture of the rectum.

Causes of anorectal/perineal trauma
- Fall astride on a projecting object, impalement.
- Blows to perineum.
- Bull-gore injury.
- Pneumatic injury: Vacuum toilet, sexual assault, autoeroticism.
- Foreign bodies.
- Suicidal attempt by rectal administration of corrosive.
- RTA.
- Surgical procedures (obstetric, gynecologic and urologic).
- Endoscopic procedures.
- Insertion of enema nozzle, thermometer.

Injuries



Symptoms
- Rectal pain.—May be delayed for several weeks.
- Abdominal pain is an ominous sign because it implies peritonitis.
- Bleeding PR following a h/o trauma suggests a mucosal tear, at the minimum.

Examination
- Pelvic examination.
- Proctosigmoidoscopy. Take care to avoid extension of injury. Rectal irrigation may facilitate visualization.

Key investigations
- X-ray abdomen.
- Urine R and M.
- A water-soluble contrast enema.

Free ebooks ==> www.Ebook777.com

Principles of treatment

- Immediate commencement of broad-spectrum antibiotic.
- The choice of treatment is dictated by the anatomic location, and etiopatholoy.
- Minor to moderate injuries of the anus below the level of levator ani may be treated by debridement, suture, drainage and close observation; and IV fluids and restriction of oral intake.
- Injuries above the levators require a colostomy at the minimum.

COMPLEX PERINEAL INJURIES usually result from high-speed road traffic accidents. There is often extensive soft tissue injury.

(PARTICULARY GUNSHOT) WOUNDS TO THE BUTTOCKS

The most important question that needs to be answered: Is there associated visceral (colorectal) injury? Clinical examination can usually answer this question. However, If doubt remains, get sigmoidoscopy, CT scan.

www.Ebook777.com

66 SPINAL TRAUMA

Causes

- Fall from height in standing/sitting position
- Fall of a heavy weight across the shoulder while the patient was in stooping position

} → # body of vertebra.

- Fall of very heavy weight/roof, in flexed position
- Road traffic accident especially pedestrian struck in standing position

} may → # dislocation.

- Violent muscular contraction – may → # spinous processes and transverse processes of C_7/T_1.

Various injuries

1. Strains/Sprains/Nonspecific soft tissue injuries.
2. Fracture without dislocation—usually without neurological involvement.
3. Fracture and dislocation—may be associated with neurological involvement.
4. Associated possible injuries, e.g. fracture of calcanei.

EXAMINATION FOR SPINAL CORD INJURY

This should be carried out before examining the vertebral column since in the presence of cord injury, the patient should be disturbed as little as possible.

Attitude of the upper extremities?

- Immobile against the trunk and completely paralyzed ⇒ the level of cord injury at the 5th segment because all severe injuries above this level cause paralysis of the phrenic nerve and sudden death.
- Patient lying helplessly on the back with the arm abducted and externally rotated and the forearm flexed and supinated ⇒ injury of the 5th cervical segment.
- Patient lying helplessly on the back with the arm partially abducted and internally rotated with the forearm flexed but pronated ⇒ injury of the 6th cervical segment.

Nature of respiration?

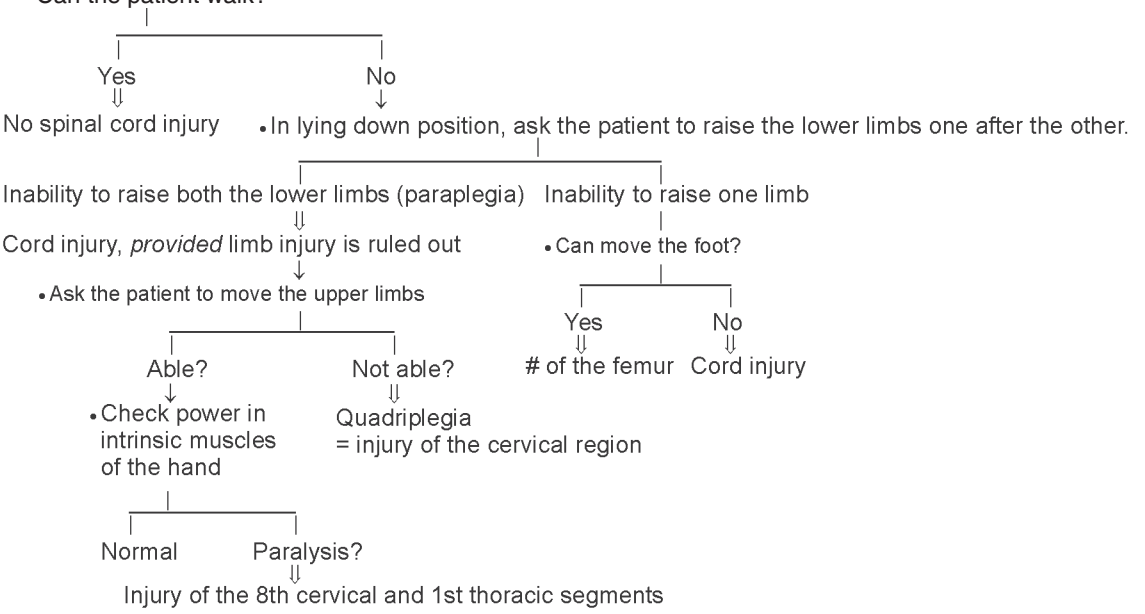
Abdominal respiration ⇒ injury of the cervicothoracic region, owing to paralysis of the intercostal muscles.

Retention of urine, overflow incontinence and **priapism**? ⇒ injury to the spinal cord.

Free ebooks ==> www.Ebook777.com

Muscle power

- Can the patient walk?



Normal muscle power in the entire upper limb ⇒ injury inferior to the 1st thoracic segment.

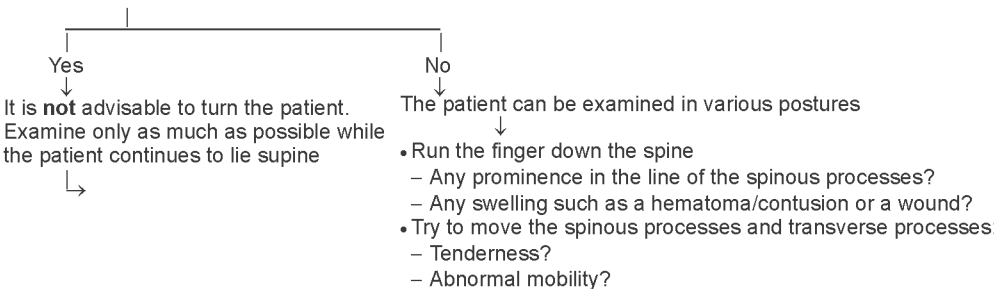
Sensations

Loss of all forms of sensations below a certain level ⇒ complete contusion of the cord.

Reflexes are absent immediately after the injury and during the period of shock, which may take as long as 3 weeks to pass off. Reflexes not returning after this period ⇒ a serious damage, and possibly a complete transverse section of the cord has taken place.

EXAMINATION OF THE VERTEBRAL COLUMN

Is paralysis present?



X-RAY Spine – AP and Lateral

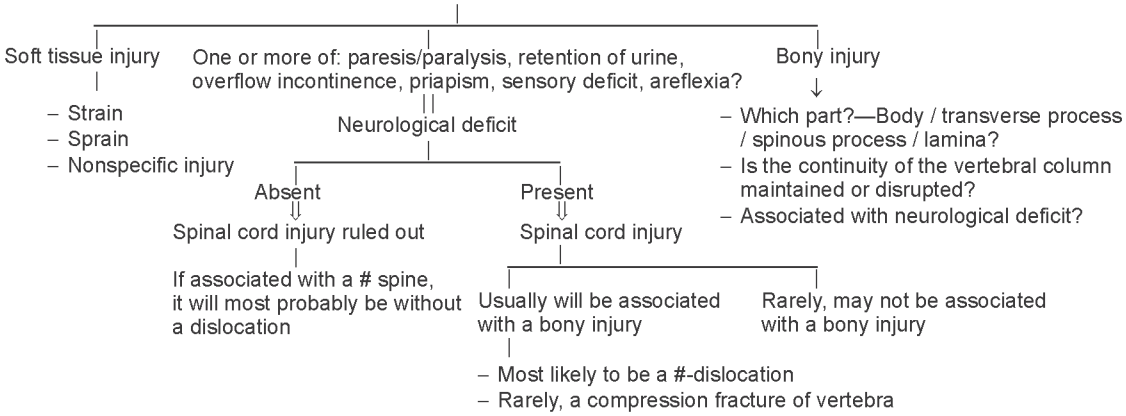
In lateral view

- Slight diminution in the depth of a vertebra is the only finding in the wedge (compression) fracture—may be easily overlooked. Although there is a compression fracture, yet there is no narrowing of the intervertebral space.
- Carefully look at the line all along the posterior surfaces of the vertebrae. Any encroachment on the spinal canal ⇒ dislocation.

In AP view, look for fracture of transverse process.

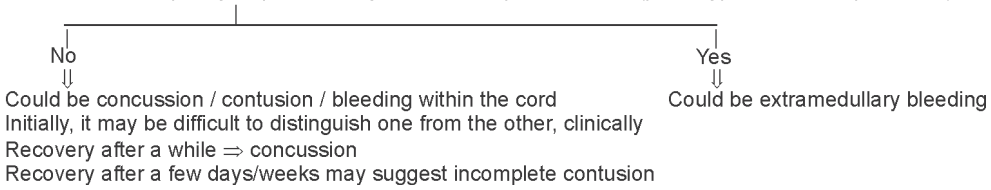
www.Ebook777.com

ARRIVING AT A PROVISIONAL CLINICAL IMPRESSION



Differential diagnosis of cord injury

Was the onset of paralysis preceded by features of spinal irritation (pain, hyperesthesia, spasm, etc.)?



Concussion = physiological paralysis of function.
Initially indistinguishable from a contusion. Diagnosis is made on finding the patient to recover after a while.

Spinal cord contusion (*Syn.* Transverse section)—May be complete or incomplete.

Complete contusion

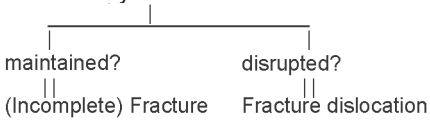
- Below the level of the injury, there is flaccid paralysis of all muscles, complete anesthesia with a hyperesthetic zone at the upper level, and total loss of reflexes—superficial, deep and visceral.
- Retention of urine; later, automatic bladder.
- Recovery is impossible.

Incomplete contusion

The early features are practically the same as complete contusion.
The condition is recognized only after a few days/weeks when some degree of motor power, sensation or spasticity of muscles reappears.

Differential diagnosis of # spine

Is continuity of the vertebral column



Types of Fracture of the vertebral bodies

1. Wedge (Compression) #: Occurs due to vertical compression in which > 1 vertebra may be involved.
2. Comminuted #: Occurs due to acute flexion of the spine; the anterior part of 1 vertebral body is crushed.
3. Fracture dislocation: Occurs due to flexion combined with a violent transverse force. May be associated with cord injury.

Free ebooks ==> www.Ebook777.com

67 HAND INJURY

GENERAL REMARKS

Although a hand injury may look minor, it is never trivial.
Assume that all structures underneath an external injury have been injured unless proved otherwise.
Five questions need to be answered in a case of hand injury:

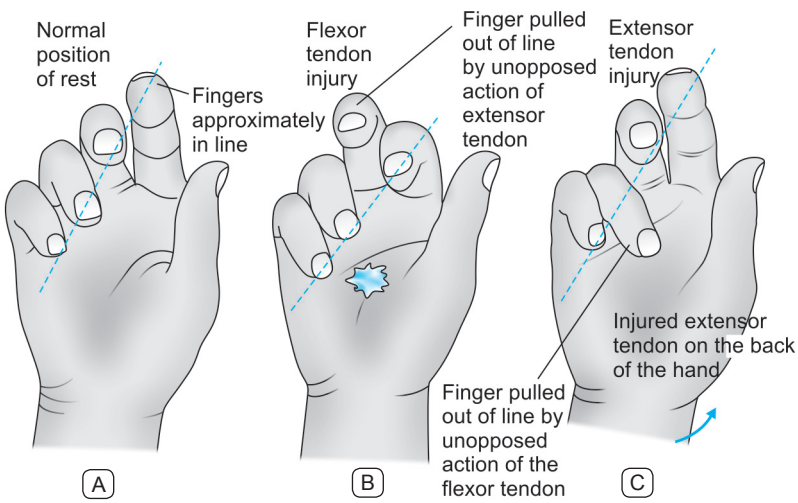
1. Is there actual or impending skin loss?
2. Is the skeleton stable?
3. Is the viability of the hand/a finger in doubt?
4. Have the tendons been injured?
5. Have the nerves to the hand been injured?

HISTORY

- How did the injury occur?
- What is your job?
  Which functions of the hand are essential for you particularly in your job?
- Which is your dominant hand?

EXAMINATION

- Any **wound/s**? – Details?
  Any loss of skin?
- What is the **position of the hand**?
  If the fingers are not in the normal position of rest ⇒ skeletal injury, i.e. #/dislocation/tendon injury (deformity caused by unopposed action of normal tendon).



Figs 67-1A to C

www.Ebook777.com

- Deformity? Tenderness? Abnormal mobility? Crepitus? Loss of function? (– for **fracture**).
  Specific local signs of fracture: Excruciating localized tenderness, deformity, abnormal mobility, crepitus, loss of function.
- Examination for **dislocation** (collateral ligament injury)
  Clinical features suggestive of dislocation: Swelling, tenderness and abnormal side-to-side mobility.
  Clinical method for abnormal side-to-side mobility:
  – MP joints must be flexed and IP joints extended.
  – Hold and stabilize the proximal bone and move the distal bone from side-to-side.
  – 'Giving way' ⇒ collateral ligament injury.
- Examination for **circulation**
  – *Color of the nails*: Pink or pale?
  ↓ ⇓
  ischemia
  Apply pressure till the nail becomes blanched. Release the pressure. Measure the time taken for refilling. Immediate refilling within few seconds ⇒ intact circulation.
  – *Sensation* in the finger tips: Normal/reduced/absent?
  – *Patency of the digital arteries*: Raise the hand. Compress the digital arteries at the base of the finger until it becomes pale. Lower the hand maintaining the compression. Release one artery (at a time) and observe the color of the finger. If it becomes pink then it suggests that the artery is patent.
- Examination for **tendon injury**
  – *FDP*: Hold the proximal IP joint in extension. Ask the patient to flex the distal phalanx. – If this is possible even by 15° then it suggests that the FDP is intact.
  – *FDS*: Hold all fingers in extension except for the one that is being tested. Ask the patient to flex the finger being tested at the proximal IP joint. – Ability to do so suggests that FDS is intact.
  – *Flexors of the wrist*: Ask the patient to flex the hand against resistance. Feel for tightening of the tendons. – Feeling of tightening tendons suggests intact tendons.
  – *Extensor tendons*: Ask the patient to extend fingers against resistance. – Ability to do so suggest that the tendons are intact.
- Clinical tests for **nerve injury**
  – *Median* nerve:
    - Opposition between thumb and index finger.
    - Sensation in the tip of the index finger.
  – *Ulnar* nerve:
    - Rest the palm on the table. Ask the patient to abduct the index finger (i.e. move towards the thumb) (This test tests deep branch of ulnar nerve.)
    - Sensation in the tip of the little finger.
  – *Radial* nerve:
    - Ask the patient to dorsiflex the hand and flex the fingers at MP joints and extend the fingers at IP joints. Maintaining this position, now ask the patient to extend the fingers at the MP joints. —Inability to do so ⇒ affection of motor function ⇒ injury in the arm.
    - Sensation over the dorsum of the MP joint of the thumb. – The absence of sensation ⇒ affection of sensory function ⇒ injury distal to the elbow.

Severe hand injuries:
- Multiple fractures
- Human/animal bite
- Industrial grease gun/pump injury.

Free ebooks ==> www.Ebook777.com

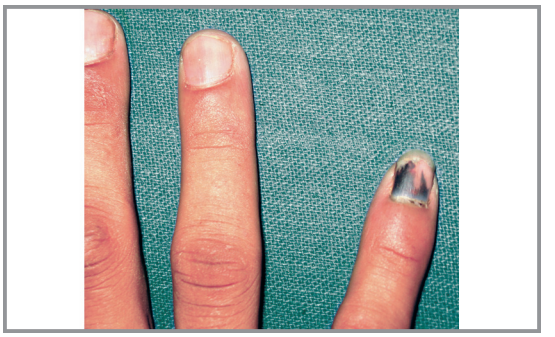


Fig 67-2 Subungual hematoma



Fig 67-3 Bursting injury of fingers



Fig 67-4 Crush injury

www.Ebook777.com

68 VASCULAR TRAUMA
(PARTICULARLY IN THE LIMBS AND NECK)

Suspect
- Penetrating injury in the vicinity of a blood vessel.
- An injury to the nerve(s) that are anatomically closely related to a blood vessel.
- Orthopedic injury (#/dislocation) of the bone/joint anatomically closely related to a blood vessel, e.g. # clavicle, # 1st rib, dislocation of shoulder, supracondylar # of humerus, posterior dislocation of hip, # of the distal femur, posterior dislocation of knee, # proximal tibia, # cervical spine.

Causes of vascular trauma:
- Direct penetrating injury.
- Blunt injury.
- Iatrogenic—sustained during intravascular diagnostic/interventional procedures.

Pathophysiology
- The spectrum of blood vessel injury: Laceration, transection, contusion, spasm.
- Any arterial injury can result in bleeding/hematoma, thrombosis, pseudoaneurysm, or formation of an AV fistula.
- Complete transection of an artery often induces retraction of the intima and media, which prevents exanguinating bleeding. An exception—scalp arteries.
  Partial transection precludes this mechanism and results in more significant blood loss.
- Intimal disruption can lead to thrombosis in the absence of external signs of blood loss or significant hematoma suggestive of a vascular injury.
- Traumatic arterial spasm: This is an arteriographic/operative diagnosis of exclusion. **"Spelling of spasm = C L O T!"**. Spasm as a possible cause of acute traumatic ischemia should never be made as a clinical diagnosis. It may be considered as a retrospective diagnosis if all organic possibilities are excluded on surgical exploration.

HISTORY

- **Amount of blood loss**?
  - Approximate number of teaspoonfuls / tablespoonfuls / glassfuls?
    OR
  - How much area of floor or cloth (such as bed sheet, towel, etc.) was stained/soaked with blood?
- **Severe agonizing continuous bursting pain**, radiating down the course of an artery?
  A patient with a significant associated orthopedic injury also will have severe pain.
- **Paresis** / **Paralysis**?
- **Coldness and deadness** of the limb?

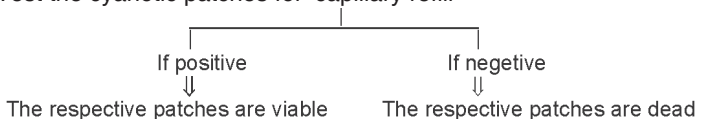
LOCOREGIONAL EXAMINATION

INSPECTION

- **Discoloration**—Serial colorchanges occur with successively advancing degree of ischemia.
  1. Marked pallor.

Free ebooks ==> www.Ebook777.com

2. Mottled (= marbled) appearance.
   Buerger's test is very useful in these patients, and may be performed if practically feasible.
   Step I: Elevate the limb and then—(a) Inspect in general. Blanching ⇒ capillary integrity is still maintained.
   (b) Test the cyanotic patches for 'capillary refill'



   Step II: Ask the patient to sit and hang the limb down.
   If most of the skin becomes bright red, then it is viable.
   Changes in color of the skin can be appreciated only in fair-skinned patients.
3. Congested and purple-blue cyanosed appearance ⇒ pregangrenous stage.
4. Black discoloration ⇒ frank gangrene.

PALPATION

- (Skin) **Temperature**
- **Tenderness**
- **Consistency**
- A diffuse swelling in the region of injury s/o a **hematoma**?
  - Size?
  - Is it pulsatile?
- **Sensation**
- **Pulsations**
- **Function**
- Test the **nerve(s)** which could have been possibly damaged.
- Assess the **neurological status in c/o suspected carotid artery injury**.

AUSCULTATION

- Bruit may be present beyond the site of occlusion.
- Systolic murmur ⇒ (false) aneurysm.
- Continuous machinery murmur ⇒ AV fistula.

Clinical grading of the severity of acute ischemia: Refer to chapter 46.

ARRIVING AT THE DIAGNOSIS

PRINCIPLES AND PITFALLS

1. Localization of the source of bleeding in major blunt trauma can be perplexing; for example in a case of combined thoracic and abdominal trauma.
2. The presence of **one or more 'hard signs'** suggests vascular injury.
3. **The presence of pulses does not exclude major vascular injury**. Distal pulses may be present in few cases of proximal arterial injury. This is a consequence of pulse wave propagation through soft clot or an intimal flap, or by way of collateral blood flow around a thrombosed vessel.
4. Skeletal muscle can withstand total ischemia for up to 6 hours before irreversible muscle damage will occur; the time commences from the point of start of pathology, **and not from the time of admission** to hospital.

www.Ebook777.com

STEP-WISE ANALYSIS OF DATA

1. **Nature of injury**?
   - A single penetrating injury?
   - Multiple penetrating injuries?
   - Blunt trauma? Associated with orthopedic injury?
2. Is/Are the '**hard sign(s)**' of vascular injury present?
   - Massive/persistent external bleeding. Hemorrhagic shock.
   - A large, expanding/pulsatile hematoma.
   - Signs of acute arterial insufficiency in the portion of the limb distal to the site of injury, including diminished/absent pulses.

   Clinical grade of ischemia?
3. **Acute comp artment syndrome**?
4. **Any injury to anatomically closely related structure(s)**?

   For example – Injury to nerve(s).
   – Neurological status in c/o carotid artery injury/vertebral artery injury.
5. **Category** of the vascular injury (as per Brink's classification)?
   - **I** – Patient in shock from ongoing bleeding.
   - **II** – Hemodynamically stable but having potential for significant morbidity.
   - **III** – A clinically suspected injury in a stable patient.

BASIC PRINCIPLES OF MANAGEMENT

- 'Arterial pressure index' (arterial pressure in the injured limb divided by the pressure in an uninvolved arm) of < 0.9 is a significant predictor of arterial injury.
- Category I patients (as per Brink's classification): Immediate operative intervention.
- Category II and III patients (as per Brink's classification): Thorough and rapid investigative evaluation.
- Single penetrating wound to the extremity presenting with hard signs of arterial injury: Explore.
- Multiple penetrating wounds in one extremity or Blunt trauma with associated orthopedic injuries presenting with hard signs of arterial injury: Angiography is recommended before surgical exploration. Angiography helps to determine the surgical approach.

Free ebooks ==> www.Ebook777.com

69 BURN INJURY— THERMAL, CHEMICAL, ELECTRICAL

THERMAL BURNS

GENERAL REMARKS

1. Human skin can tolerate temperatures up to 40° C for brief periods. Temperatures above this level result in progressively increased tissue destruction that is worsened by increased time of exposure.
2. Destruction of the skin by heat → local ± *systemic* physiological alterations.
   ↓
   Derangements in the hemodynamic, metabolic, nutritional, immunological and psychological homeostatic mechanisms.
3. Burn shock develops slowly. Half an hour after a major burn a patient may look surprisingly well, but 4 hours later shock establishes rapidly.
4. **Evaluation** of a burned victim:
   - 'ABCDE' and appropriate management.
   - Determine the severity of the burn injury – minor/moderate/major.
   - Decide the level of expertise needed to care for the patient.
5. If the sum of an adult patient's age and the percentage area of full-thickness burn come to > 80, death is probable.

(Specific) CLINICAL EVALUATION

Age of the patient?

Burns in patients under the age of 3 years and over the age of 60 years tend to be more severe.

Cause of the burn?

Scalding due to spillage/scalding because of immersion / exposure to flames/contact with hot object / chemical/ electrical?

↓

- Was it caused by a domestic electrical appliance OR high-tension line?

Details of the event?

- Did the event occur in a closed space with the presence of heavy smoke?
- Temperature of the injuring agent and the duration of exposure?
  In c/o scalding: Was the liquid (often water) boiling, or otherwise?
- How alert was the patient at the time of the burn?
  The burns are more likely to be deep if the patient was asleep/entrapped/drunk/drugged/during an attack of epileptic fit.

Pain?

- Full thickness burns are often relatively painless.
- Partial thickness injuries can be excruciating.

Restlessness?

Causes: Hypoxia in c/o major burns, extreme pain, psychological.

www.Ebook777.com

Site of the burn wound?

- Burns to the face and neck, hands, feet, perineum and genitalia carry specific problems in their treatment, reconstruction and eventual rehabilitation.
  Significant burns of these areas are qualified as major burn injury by virtue of location, regardless of the total body extent and depth of the injury.
- Texture of the skin burnt—thin or thick?
  Thin skin is more likely to be deeply burnt, e.g. in small children, in elderly, the skin on the back of the hand than the thick skin of the palm, the skin on the medial aspect of the upper limb as compared to thick skin on the back, etc.

Extent of burn?

Observe carefully and record graphically in terms of percentage of body surface involved.—This is important not only for diagnosis and treatment but also for prognosis and statistics.

Only areas of partial- and full thickness burn are included in this assessment, since 1st degree burns do not have appreciable morbidity.

'**The rule of 9s**' can be used for adults, but in children, modification of this rule is required because the child's head is proportionally larger, and the legs smaller compared to the adult.

For quick although slightly less accurate estimation, '**the rule of 7s**' can be used however, in children: Whole of the HFN—28%. Whole of the trunk—28%. Whole of each upper limb—7%. Whole of each lower limb—14%. Perineum—2%.

Yet another simple clinical tip helpful to quickly calculate the extent of patchy areas of burns: The patient's own hand and fingers (touching each other) together constitute ~ 1% of body surface area.

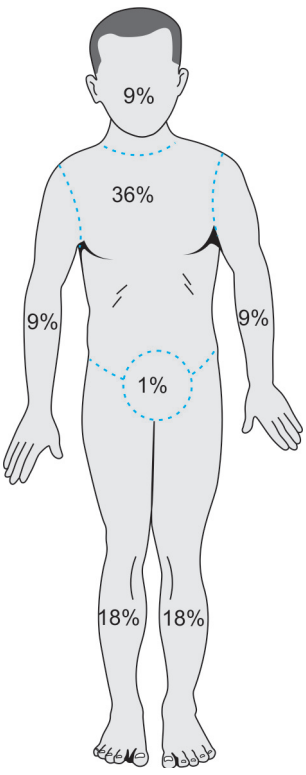


Fig 69-1 Rule of 9s in adults

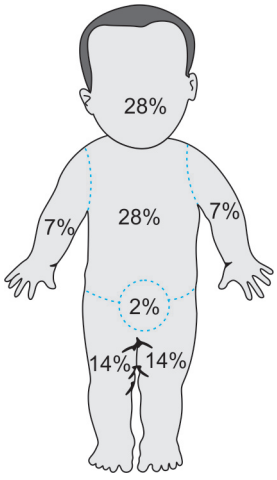


Fig 69-2 Rule of 7s in children

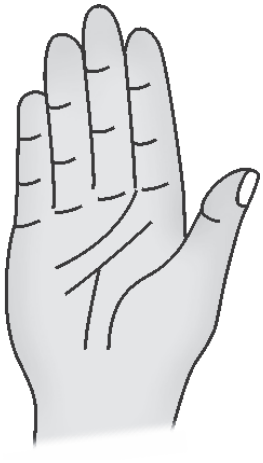


Fig 69-3 Rule of 1

Free ebooks ==> www.Ebook777.com

Depth of burn?

Since the volume of tissue destroyed is ultimately important, it is essential to assess the depth of the burn wound in addition to its extent.

Reasons for assessment of depth

1. Metabolic upset is greater with deeper burns.
2. Healing occurs more slowly with increasing depth of burns.
3. Deep burns require SSG (unless the extent is small).
4. Circumferential deep burns involving digits/limbs/chest wall may → vascular compromise or restriction of ventilation, and necessitate urgent decompression with escharotomy.

During the 1st assessment, critical distinction into different classes of burns is neither possible nor essential. Estimate TBSA of burns for calculation of immediate fluid replacement. In this estimation, ignore simple erythema (without blisters).

By the end of 24 hours, it is essential to differentiate between superficial and deep burns as blood transfusion may have to be considered on 2nd day onwards.

The full extent of the injury will reveal itself between 4 and 14 days. Usually the depth can be assessed most reliably at ~ the 7th day. The main concern at this and subsequent period of time is essentially to decide exactly how much area of burns would need excision and grafting.

Pitfalls

- The appearance of the burnt area may be deceptive. The depth may be difficult to assess accurately.
- The depth may not be uniform throughout the burn's extent.
- The depth may progress with time. **Burns are dynamic** injuries, so that the distinction between superficial and deep burns is not permanent. The deep epidermal structures which survived after a partial thickness burn can easily be destroyed later by infection.
- Testing the tactile sensation may be unreliable. Even full thickness burns have pressure sensation.
- Pin prick testing is also not a good predictor of full thickness injury. Pin prick is absent in deep burns, and it can also be absent in burns of moderate depth.

Clinical guidelines:

- **Level** of the burn wound with respect to the surrounding skin—Is it shrunk below the level of the surrounding skin?
- Does the burnt tissue appear **wet** OR **dry**?
- **Color**: Erythematous/pale/white/grayish brown/black?
- **Blisters**: Thin watery OR thick walled?
- **Blanching** on application of pressure: Does the burnt tissue blanch OR It does not blanch?
- **Consistency** of the eschar—Relatively softer OR Leathery?
- **Pin prick testi**ng with a sterile hypodermic needle

  Clinical method: Practice first on the normal skin, and ask the patient if she/he can feel sharp or blunt end. Then test the burn wound. Blunt sensation reduces the significance of the test.
- **Cause** of the burn**, other details of the event,** and the **site** of the burn, particularly *wrt* the texture of the skin?

Inference

- Scalds from liquids below boiling point usually → partial thickness burn, whereas scalds from boiling water → full thickness burn.
- Scalding due to spillage tends to cause superficial burns.
- Scalding due to immersion, burns caused by exposure to flames/contact with hot object, and chemical and electrical burns tend to be deeper.
- High tension electricity can cause devastating necrosis of muscles and other deep tissues.
- Erythema, thin watery blisters, and blanching of the tissues are associated with superficial burns.
- Pale looking tissue, thick-walled blisters, poorly or nonblanching tissue, dry leathery eschar are associated with deeper burns.

www.Ebook777.com

Classification of depth of burns

- Erythema and discomfort only; no blisters = **1st degree** burns.
  Does not cause metabolic derangement. Heals without Sequelae. An example—Sunburns.
- Thin walled blisters (form within 24 hours).
  Breaking of blisters results in weeping pink surface.
  Blanching on pressure (⇒ intact dermal capillaries).
  Sensitive to pin prick ('Sharp' sensation).
  = **Superficial partial thickness** burns.—Involves epidermis ± superficial dermis.
  Usually heal within 3 weeks in the absence of infection.
- Pale looking.
  Thick walled blisters. Many areas of marginally viable tissue.
  May be sensitive to pin prick ('Blunt' sensation).
  = **Deep partial thickness** burns (*syn.* **Deep-dermal** burns).
  – Involves epidermis and much of the dermis; so that the epidermis of at least the bottoms of hair follicles/sweat glands is preserved.
  Skin will only survive under the most optimal conditions and healing will take longer time (6 weeks) and produce hypertrophic scarring (Poor quality in cosmetic appearance, function, and also more prone to develop epitheliomas later).

= **2nd degree** burns.

Superficial burns.
(Deep partial-thickness burns may go as deep burns.)

- White/grayish brown/black.
  Red coagulated fat may be visible (Subcutaneous fat stained red by Hb released from destroyed RBCs).
  Thrombosed veins visible through translucent subcutaneous fat.
  Burn wound shrunk below the level of the surrounding skin.
  Non blanching. Dry leathery eschar. Hair can be easily pulled out.
  Insensitive to pin prick.
  = **Full thickness** burns = **3rd degree** burns.
  – Involves epidermis and dermis including epidermal appendages.
  – The wound cannot heal itself by first intention.
- Injury/death of subcutaneous tissue such as fat/fascia/muscle/bone = **4th degree** burns.

Deep burns.

Retrospective criteria which may define the depth, on revaluations between 4 and 14 days:

– If the crust separates to leave clean new skin underneath ⇒ the burn was superficial.
– Speckling at 2 weeks ⇒ (deep) partial thickness burn.
  Speckling = appearance of fine dots (skin regenerating from the bottom of sweat glands); they are gray in a black skin and red in a white one.
– If no epidermis appears to cover a wet granulating surface, or if a slough/eschar remains firmly stuck ⇒ deep burns. Eschar begins to lift after 2–3 weeks.



Fig 69-4 Superficial partial thickness burns

Free ebooks ==> www.Ebook777.com

Inhalational injury?
= injury to airways and lung parenchyma due to hot gases/poisonous toxic smoke.

Criteria which strongly suggest possibility of inhalational injury:
- Burns in a closed space with the presence of heavy smoke.
- Stridor, hoarseness of voice.
- Bronchorrhea, carbonaceous sputum, coarse crepitations on auscultation.
- Conjunctivitis, pharyngeal edema.
- The presence of soot particles around the nostrils, in the mouth.
- Burns of the nasal hair.

Early onset of respiratory symptoms ⇒ severe injury to the respiratory tract.

Associated trauma?

Comorbid ailments?
- Existing disease state prior to the thermal event?
- Seizure disorder/alcoholism/drug misuse?

Was the treatment of shock commenced before admission to the hospital?

Does the victim **appear able to care for herself/himself**?

Guidelines for **Categorization** – as per American Burn Association:

	Major burn	*Moderate burn*	*Minor burn*
– Partial-thickness size	> 25% in adults	10–25% in adults	< 15% in adults
	> 20% in children	10–20% in children	< 10% in children
– Full thickness size	> 10%	2–10%	< 2%
– Significant burns to the face and neck, hands, feet, perineum, genitalia	Yes	No	No
– Inhalational injury	Yes	No	No
– Associated trauma	Yes	No	No
– Comorbid factors	Poor risk patient	Relatively good risk patient	Not present
Treatment facility recommended	Admission to specialized burn-care center	Admission to general hospital with designated team	OPD

Clinical systemic monitoring in moderate and major burns essentially during the acute phase (initial 48 hours)
- Cerebral dysfunction.
- Vital parameters.
- JVP (if possible).
- Distal capillary refill to assess for the need for release (escharotomy), or to judge the adequacy of escharotomy if one has been performed.
- NGT output—qualitative and quantitative.
- U/O.
- Evaluation of the upper airway (pharynx, epiglottis, glottis) by IDL (bronchoscope if available) for the development of edema.

www.Ebook777.com

CHEMICAL BURNS

The principle difference between thermal burns and chemical burns: With chemical burns, tissue destruction continues as long as contact is maintained. This means that chemical burns are usually deeper than they initially appear, and progress with time.

The initial treatment

- Wash away the chemical with continuous running water or by prolonged hydrotherapy in large volume tanks for as long as several hours. *Caution*: avoid hypothermia.
- **Do not use neutralizing agents** since they may cause exothermic reactions.

ELECTRICAL BURNS

GENERAL REMARKS

The effect of passage of an electric current through the body depends on the type of current, the voltage of the current, the resistance offered by the tissue, the amperage of current flowing through the body and the duration of contact.

Skin (in addition to tendons, fat and bone) being a poor conductor, is less affected than nerves, vessels and muscles, thereby belying the gravity of injury to the deeper structures.

Types of injury classified on the basis of voltage of the current

< 1000 V	> 1000 V
Low-voltage injury, e.g. electrocution caused by domestic electrical appliances	High-voltage injury, e.g. electrocution by high-tension overhead lines
– The AC current → tetany, and ∴ patients often describe how they were unable to release the device until the power was turned off	– Damage not only at the entry and exit points, but extensive subcutaneous damage occurs between these 2 points
– Small deep burns occur at the entry and exit sites may be with damage to the nerve/tendon/muscle underneath, but with little damage between	– Damage to the underlying muscles in the affected limb can cause rapid onset of compartment syndrome
	– *Mechanisms of damage*

1. *Arc* (*syn*. flash) injury: A localized injury. Often occurs on flexor surfaces of joints where current exits and re-enters skin, attempting to find the shortest pathway. The extremely rapid heating of the air causes an explosion that often propels the victim backwards.
2. *Flame* burn can occur if the heat generated from the electricity in an arc burn is high enough to cause ignition of the clothing.
3. *Current* injury: → vessel thrombosis. The full extent of this type of injury is insidious and not immediately appreciated.

Causes of immediate death: Anoxia and ventricular fibrillation.



Fig 69-5 An electrical burn

Free ebooks ==> www.Ebook777.com

Effects of electrical burns on various systems

1. Cardiac: Cardiac arrhythmias (early/late).
   Vascular: Thrombosis. The burn wounds tend to deepen over days and delayed rupture of major vessels can occur.
2. Renal: Myoglobinuria (from severe muscle injury) (/ hemoglobinuria) → ARF.
3. GIT: Perforation of intestine → peritonitis.
4. Bones: Fractures due to tetanic muscle contractions.
5. Spinal cord and peripheral nerve damage can occur late due to a delayed demyelinating effect of the current.
6. Cataract formation may be delayed for days to months.

Investigations

- ECG. (Immediately on admission).
- Urine – R, M, and specifically for myoglobin and hemoglobin.
- FBC.
- BUN, S. Cr.
- Blood grouping.
- BS-R.

Monitoring

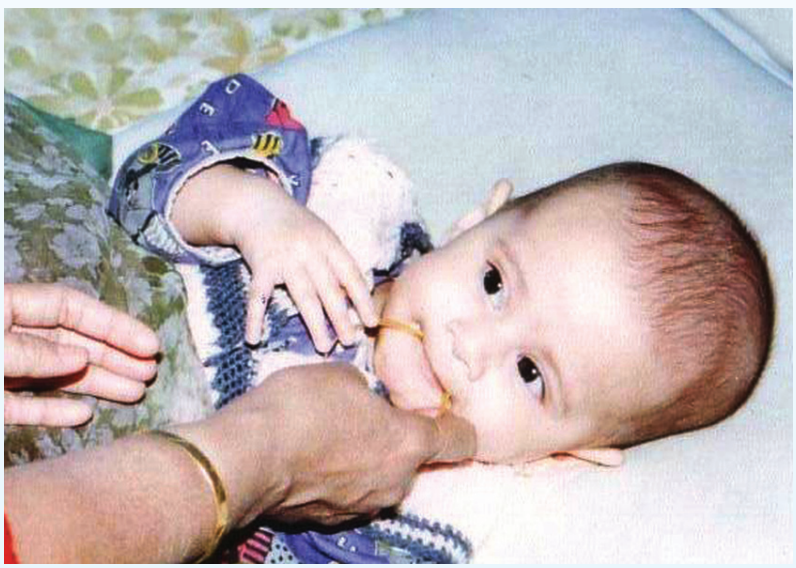
- Vital parameters hourly. Look for the development of **shock**.
- U/O hourly. Look for the development of **ARF**.
- Cardiac monitoring, if indicated (abnormal ECG on admission). Look for the development of **cardiac arrhythmia**.
- **Depth of the burn wounds**.
- Look for the development of **compartment syndrome**.

www.Ebook777.com

SECTION 5

Pediatric Surgery

70. History-taking in a child
71. Surgical examination of the child
72. Respiratory distress in the newborn
73. Acute abdominal disorders
74. Colic (Colicky pain in abdomen)
75. Neonatal intestinal obstruction
76. Recurrent pain in abdomen
77. Vomiting in the neonate
78. Non-bile-stained vomiting in infancy
79. GI bleeding
80. Jaundice / cholelithiasis / portal hypertension
81. Discharge from the umbilicus
82. Anorectal malformation
83. Urinary tract infection (UTI) / urinary stones
84. Disorders of the lower urinary tract
85. 'Acute scrotum'
Impalpable testicle / empty scrotum: Refer to chapter 26
Chronic swelling in the scrotum: Refer to chapter 25
86. Disorders of the external genitalia
87. Acute pain in the limb
88. Disorders of the mouth
89. Swellings/lesions in the face
90. Swelling in the neck / notes on selected swellings
91. Torticollis (Wry neck)
92. Disorders of the breast
93. A big head / hydrocephalus / assessing a VP shunt
94. Spinal dysraphism—spina bifida
95. Foreign bodies



Free ebooks ==> www.Ebook777.com

www.Ebook777.com

70 HISTORY-TAKING IN A CHILD

GENERAL REMARKS

1. Crying and restlessness are common non-specific symptoms in babies; they are the infantile equivalent of pain and hunger.
2. Refusal to feed, objection to movements/handling are important observations by parents, s/o '*something wrong in the abdomen*' which may be significant.
3. Vomiting, sweating, screaming, pallor and drawing up of the legs suggest moderate-to-severe colicky pain in abdomen.

COMPONENTS OF HISTORY-TAKING

1. Basic information
2. Narrative history
3. Interrogative history
4. Background information:
   - *Immunization*
   - *Birth history*
   - *Nutritional history*
   - *Developmental history*
   - *Dental milestones*
   - *Family history*: Age, health and cause of death (if applicable) of siblings, parents and grandparents?
   - *Personal and social history*:
     - Educational level
     - Family of origin
     - Personal interests, lifestyle issues, home safety, etc.

Points relevant to pediatric history-taking, particularly with reference to surgical problem only are discussed below.

Definitions of Age-groups

Up to 1 year = infancy.
1–14 years = childhood.
14–16 years = puberty.
16–22 years = adolescence.

Immunization

- See the immunization record if available.
- If not available, ask whether proper immunization schedule is followed or not.
- If appropriate, enquire reasons of failure: Contraindicated / Nonavailability / Ignorance / Religious taboos?

Birth History

- About pregnancy, delivery and perinatal events.
- Birth weight?
- Gestational age?

Free ebooks ==> www.Ebook777.com

- Length of stay in the nursery, complication in the nursery?
- Prenatal assessments—amniocentesis (if previous mishap)?
- H/o miscarriages, terminations, still births, neonatal deaths wherever applicable?

Nutritional History

- Duration of breast feeding?
- Time of weaning?
- Age of introduction of solids with quantity and quality of solid food?
- What the child currently consumes in terms of proteins and calories (whether adequate or inadequate)?
- Appetite?

Family History

- Economic background?
- Primary care taker?
- Number of family members?
- Age, health and cause of death (if applicable) of siblings, parents and grandparents?
- Hereditary diseases, congenital anomalies, developmental delay?
- Asthma, cardiovascular disease, sickle cell disease, thalassemia, mental retardation, seizure disorder, atopy?
- Communicable diseases, e.g. TB, leprosy, scabies etc?
- Non-communicable diseases like food poisoning, peptic ulcer, etc?
- If the child is suffering from a chronic disease:
  - Impact of the child's disease on other members of the family?
  - Impact of family's economic situation on treatment?

www.Ebook777.com

71 SURGICAL EXAMINATION OF THE CHILD

This chapter describes those aspects of clinical assessment which are commonly relevant with a surgical condition. It does not include full description of the medical (including neurological) examination.
Observe the entire baby, but avoid excessive handling of the sick infant. **Much information can be obtained by careful observation with minimal disturbance to the infant**.
Uncomfortable procedures such as throat or rectal examination, should be performed last.

- If one major anomaly is present, look for others.
- If multiple major and minor anomalies are found, there may be an identifiable syndrome.
- The most accurate indication of gestation is the mother's dates.

GENERAL EXAMINATION

- Any **obvious major anomaly**, which may require immediate measure to avoid morbidity?
  For example, wrap a child with gastroschisis in a plastic food-wrap to prevent evaporative heat loss.
- **Posture** and **activity**? – Does the appearance match the gestation?
- **Vital parameters**?

Normal parameters by age	*Pulse (beats)/min*	*Respiratory rate*	*BP*
– Infant	80–160	40	Formula (applicable to children)
– 4 years	80–120	30	- Mean systolic BP = 90 + age in years.
– 6–12 years	70–110	20	- Mean diastolic BP = 55 + age in years.

- – **SOB**? Does the baby seem to be '**working hard**' **for breathing**?
  – **Sternal retraction**?
  – **Tachypnea**?
  – **Protrusion of upper abdomen**?
  – **Cyanosis**?

  } ⇒ Respiratory distress
- **Color of the skin, sclera, mucous membrane**? (– to look for pallor, jaundice, and cyanosis.)
- **Head**:
  – Feel the fontanelle and sutures.
  – Measure the circumference.
  – Eyes. Is the distance between the eyes normal?
  – Nostrils – Patent?
  – Mouth: Excessive salivation, tongue, cleft-palate?
- **Face**:
  – Ears
    - Are they fully developed?
    - Examine the external auditory meatus.
    - In the neonate, look for the features for gestational age.

Free ebooks ==> www.Ebook777.com

- Lower jaw – Normal or underdeveloped?
- **Neck**: The neck of the neonate is relatively short, but should be examined.
  - Cystic hygroma?
  - SCM tumor?
- **Chest**:
  - Chest wall.
  - Breast – In the neonate, look for the features for gestational age.
  - Chest expansion – Symmetrical?
  - Dullness other than normal liver dullness on percussion? ⇒ Fluid.
  - Air entry? Breath sounds?
  - Location of the heart? Heart sounds?
- **Abdomen**:
  - Distended / Scaphoid / Flat?
  - Abnormal mass?
    - Organs normally palpable: Liver, kidneys, bladder, and less commonly, spleen.
    - A fixed tender mass may suggest a localized perforation.
  - Evidence of peritonitis?: Redness and edema of the skin of the anterior abdominal wall, tenderness, increasing distension.
  - Umbilicus.
  - Groins: Any hernia?
  - Scrotum:
    - In the neonate, look for the features for gestational age.
    - Testicles—Is the descent normal?
  - Penis:
    - Is the prepuce normal?
    - Is the external urethral meatus located at normal position?
  - Labia and clitoris—In the neonate, look for the features for gestational age.
- **Perineum and anus**:
  - Is the anus located at normal position?
  - Is it patent, particularly when it is anteriorly placed?
- If the anus is absent in a boy: Carefully inspect the median raphe of the perineum, scrotum and ventral surface of the penis for a fistulous opening which may be stained black with meconium.
- If the anus is absent in a girl: Part the labia to inspect the vestibule to look for an (anal) opening.
- **Limbs**:
  - Count the digits.
  - Any orthopedic deformity?
  - Any vascular malformation?
  - Sole creases—In the neonate, look for the features for gestational age.
  - Some newborns have edema over their hands, feet, lower legs, pubis, and sacrum. It disappears within a few days.

Finally, turn the baby over to examine the back.

- **Back**:
  - Any swelling/lesion in the midline?—It may be overlying a spina bifida.
  - Run fingers down the spinous processes and confirm the presence of the sacral segments and coccyx.
- While the infant is held prone in the hand, test for **muscle tone**.

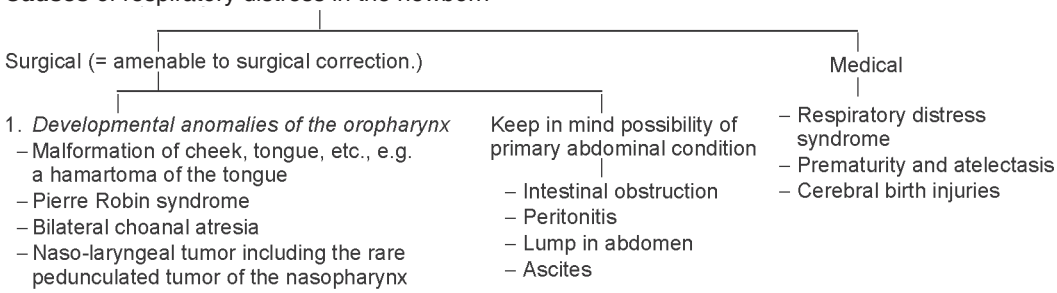
www.Ebook777.com

72 RESPIRATORY DISTRESS IN THE NEWBORN

GENERAL REMARKS AND PITFALLS

1. The signs of respiratory distress itself are not specific for the cause.
2. The degree of distress may be slight. Such babies must be observed carefully for distress may progress to become severe culminating in irreversible respiratory failure.
3. Intestinal obstruction and peritonitis can also cause abdominal distension and respiratory distress.
4. Many of the causes have a wide clinical spectrum, e.g. a CDH at one end of the spectrum may produce an acute life-threatening situation, while another case of identical size CDH may cause no distress in the neonatal period, at the other end of the spectrum.
5. In only a few cases can a firm diagnosis be made solely on clinical grounds, and X-rays of the thorax and abdomen should be obtained as soon as possible.
6. Pertaining pneumothorax: In the neonatal age-group, the severity of the symptoms is frequently out of proportion to the size of the pneumothorax, the cause of which is usually parenchymal lung disease; IC drainage is rarely required in such cases. However, if assisted respiration is necessary, then IC drainage is advisable.

Causes of respiratory distress in the newborn



2. *Laryngeal and tracheal malformations*: Microlarynx, laryngeal stenosis, laryngeal web, hemangioma of the larynx, tracheal stenosis, tracheomalacia.
3. *Massive swellings of the neck/floor of the mouth*: Cystic hygroma, hemangioma, inflammatory swelling.
4. *Pulmonary dysplasia*: Congenital lobar emphysema, congenital cystic disease of the lung.
5. *Pleural conditions*: Empyema, hemothorax.
6. *Mediastinal conditions*: Pneumomediastinum, mediastinal teratoma, duplication cyst.
7. *CDH*.
8. *Esophageal atresia*.

Free ebooks ==> www.Ebook777.com

CLINICAL FEATURES in the descending order of occurrence and severity

1. **Restlessness**—may be overlooked at first.
2. **SOB**. '**Working hard for breathing**'.
3. **Tachypnea**.
4. **Tachycardia/Bradycardia**: Tachycardia is almost invariably present, but if the rate is > 200/minute the situation is particularly serious. Bradycardia is a dangerous sign and often heralds imminent cardiorespiratory failure.
5. **Protrusion of upper abdomen**—due to increase in diaphragmatic movements in an effort to achieve effective respiration.
6. **Sternal retraction during inspiration**. *Cause*: Pliability of ribs (which support the sternum) allowing the effect similar to that in 'flail chest'.
7. **Cyanosis** f/b respiratory failure f/b cardiovascular collapse.

Immediate General Measures

- If abdominal distension due to massive pneumoperitoneum seems to be the cause, insert a (preferably large bore) needle into the peritoneal cavity (→ a hissing sound with disappearance of distension and dramatic improvement).
- If abdominal distension due to massive ascites seems to be the cause, insert a (preferably large bore) needle into the peritoneal cavity to drain some fluid as a temporizing measure.
- O_2.
- Insert a gastric tube and suck the stomach.
- If severe respiratory failure is already present, ET intubation is required possibly with assisted ventilation.

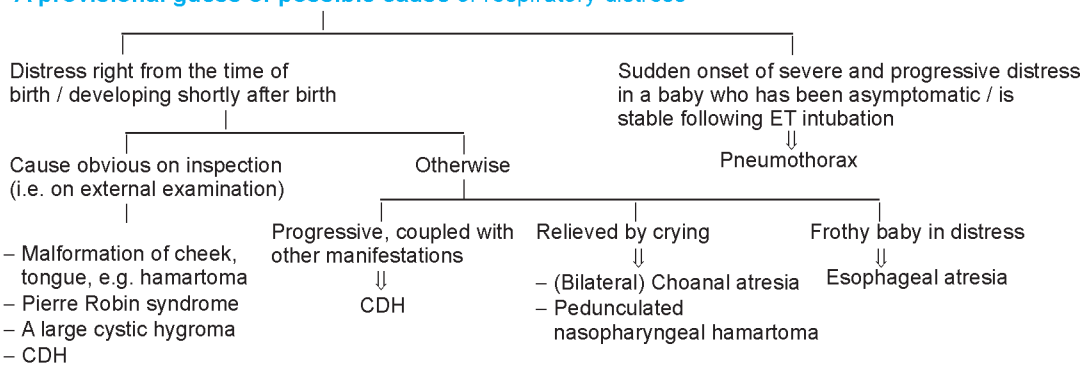
|

Difficulty in intubation may be indicative of a laryngeal pathology, e.g. stenosis

↓

Tracheostomy.

A provisional guess of possible cause of respiratory distress



Causes Obvious on External Examination

1. **A large cystic hygroma involving** the floor of the mouth/the pharynx—may be extending into the mediastinum. May demand early tracheostomy.
2. **Pierre Robin syndrome** = Micrognathia and cleft palate. The tongue is pushed backwards and upwards leading to airway obstruction.
   Early treatment: Nurse in prone position.—If not effective, then—nasopharyngeal airway / tracheostomy.
3. Barrel-shaped chest and scaphoid abdomen suggest **CDH** (Congenital Diaphragmatic Hernia)

↓

There are 2 primary features of the pathology: 1. Occupation of the thoracic cavity (most commonly left, uncommonly right) by abdominal contents. 2. Hypoplasia of lung.

The degree of pulmonary hypoplasia is the ultimate determinant of survival.

www.Ebook777.com



Fig 72-1 Congenital diafragmatic hernia

The age of presentation and severity of the condition depend on both the primary pathological features

In typical 'moderately severe' cases	In the 'most severe' cases	In 'less severe' cases
– Rapid development of respiratory distress with cyanosis shortly after birth	– Cardiovascular collapse soon after birth	– Less dramatic presentation
– Barrel-shaped chest and scaphoid abdomen. However, once air is swallowed, this sign becomes less obvious	– Low Apgar score	– Few children may remain asymptomatic for even months
	– Without immediate resuscitation, the babies die quickly	

– On auscultation:
  - Diminished air entry
  - Apparent dextrocardia in c/o left-sided CDH
  - Peristaltic sounds may be audible in the affected side of the chest. However, THIS SIGN IS NOT RELIABLE
– X-ray chest and abdomen:
  - Loops of intestine visible in the chest
  - Mediastinal shift
  - Abnormal distribution of intestinal gas (paucity of intestinal gas) within the abdomen

Radiological DD: Examples—cystic lung disease, emphysema, staphylococcal pneumonia with pneumatocele
However, these conditions rarely lead to respiratory distress shortly after birth

Respiratory Distress Relieved by Crying

Principle and mechanism: Newborn is an obligate nose-breather with little ability to breathe through mouth, even if there is complete obstruction of the nasal passage.

Clinical features

– Progressive respiratory distress and finally cyanosis. With crying, the features are reversed and the baby rapidly turns pink.
– Respiratory distress recurs after crying stops.

} Basically nasal (/ nasopharyngeal) obstruction.

Immediate temporary measure to relieve obstruction: Insert an oropharyngeal airway.

Differential diagnosis

– Sudden improvement on crying, otherwise respiratory distress ⇒ (bilateral) choanal atresia.
– Intermittent distress according to the posture of the baby. A swelling may be seen at the back of the mouth as it prolapses into the oropharynx ⇒ pedunculated hamartoma.

Clinical confirmation of bilateral choanal atresia: Inability to pass a small but firm nasogastric tube through either nasal passage. Care to be taken to avoid wrong technique of insertion of a tube: The tube should be directed posteriorly from the nostril in a horizontal plane BENEATH the turbinates, AND NOT VERTICALLY UPWARDS along the line of the nose.
A unilateral choanal atresia does not cause respiratory distress in the newborn. It is detected later because of persistent nasal discharge from the obstructed nostril.

Free ebooks ==> www.Ebook777.com

A Frothy Baby in Respiratory Distress

- Prematurity/Polyhydramnios ±.
- Saliva bubbling and dribbling from the mouth/fine frothy white bubbles of mucus appearing in the nostrils or on the lips—hence 'frothy baby'. *Cause and mechanism of respiratory distress*: TEF → preferential passage of air from trachea to stomach → gastric distension → diaphragmatic elevation → 'respiratory distress'.
  Thus, 'frothy baby and respiratory distress'.
- Cyanosis in severe cases.

This clinical picture ⇒ esophageal atresia with distal-end TEF.

Confirmation of the diagnosis:

1. Gently introduce a 10 F stiff catheter through the mouth. Arrest of its progress between 9 and 13 cm from the anterior gums confirms the diagnosis.
2. Abdominal distension and resonant note on percussion ⇒ distal-end TEF.

If the diagnosis has not been made at birth, then the following clinical features will be evident at the time of the 1st feed: Gagging, choking, coughing, spluttering, cyanotic respiratory distress (due to overflow of milk into the airway).

Important aspects of initial management:

- Frequent suctioning of pharynx.
- Contrast X-ray examination is not required as there is a high risk of aspiration.
- X-ray chest + abdomen—AP and lateral:
  - Dilated upper pouch.
  - Air in the GIT
  - Lower esophagus filled with air from the trachea

  } ⇒ distal-end TEF.

Cause(s) which may make the neonate unfit for surgery: Prematurity, coexistent developmental anomalies, pneumonia, poor general condition—either alone or in combination. The same causes may be responsible for death.

Remote post-op complications/sequelae

1. A brassy cough may persist for 12–18 months after which it usually disappears spontaneously.
2. Esophageal stricture of some degree occurs in half of the cases; most yield to bouginage.

Sudden onset of severe and progressive respiratory distress in a baby who has been asymptomatic or is stable after ET intubation

+ tachypnea, marked sternal retraction, increased resonance on percussion, reduced air entry on auscultation ± mediastinal shift
⇒ Pneumothorax. *Possible predisposing causes*: Cystic lung disease, emphysema, pneumatocele.

If condition is worsening rapidly with cyanosis and cardiovascular collapse
↓
Insert a needle in the 5th IC space in anterior axillary line immediately as an emergency life-saving measure, without wasting time in obtaining any X-ray

Otherwise
↓
X-ray chest to confirm the diagnosis
↓
ICD

Differential diagnosis of some other conditions

Congenital Lobar Emphysema

Cardinal symptom: Tachypnea amounting to dyspnea, most noticeable when the baby is being fed.

Other clinical features which may be present:

- Dry cough.
- Stridor.
- Cyanosis—is an indication for urgent treatment.
- Chest wall on the affected side—prominent and relatively immobile.
- On the affected side,—breath sounds are diminished and the percussion note is hyperresonant.

www.Ebook777.com

Congenital Cystic Lung

The clinical features are similar to those of lobar emphysema, but the condition is usually more urgent and severe.

Pneumomediastinum

Pneumomediastinum in the newborn rarely produces subcutaneous emphysema of the neck.
Pneumomediastinum is a radiological diagnosis: Loculated gas shadows in the mediastinum.
In most cases, pneumomediastinum is a consequence of interstitial pulmonary emphysema. The cause of distress lies in the lungs; in most cases treatment is directed solely to the pulmonary condition and decompression of the mediastinum is seldom, if ever, necessary.

Free ebooks ==> www.Ebook777.com

73 ACUTE ABDOMINAL DISORDERS

Three cardinal symptoms which suggest acute pain in abdomen:
1. Pain lasting > 4 hours. In infants however, abdominal colic of even shorter duration can signal the presence of an intussusception.
2. Persistent vomiting, even in the absence of pain.
3. Diarrhea lasting > 24 hours.

Refusal to feed and objection to movements/handling are important observations by parents, s/o '*something wrong in the abdomen*'.

GENERAL REMARKS

1. The presence or persistence of pain is often underestimated by parents and professionals alike. The expectation that pain must be of sudden mode of onset and dramatic progress before it can have any surgical significance is unrealistic.

2. The 'book-picture' clinical manifestations of *the fully established* case are an unreliable guide in routine practice, particularly at the general outpatient level. Early symptoms and signs (during evolution of pathology) when diagnosis may be difficult or even impossible is the usual challenging scenario.

3. **Causes** of acute abdomen:

a. *In the* ***neonate***
- NEC (Necrotizing EnteroColitis)
- Hirschprung's disease
- Meconium ileus
- Meconium plug syndrome
- Anorectal malformation
- Organic small intestinal obstruction—atresias/stenoses (e.g. CHPS (Congenital Hypertrophic Pyloric Stenosis)), malrotation without/with volvulus (volvulus neonatorum).

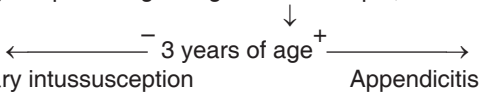
b. *In* ***infants***
- 'Colic'
- Intestinal obstruction:
  - Congenital anomalies like kinking in malrotation of gut, annular pancreas, etc.
  - Intussusception
  - Volvulus, etc.
- Inflammatory conditions: Appendicits, Meckel's diverticulitis, UTI.

c. *In* ***children beyond 1 year of age***
- More common:
  - 'Colic'
  - Inflammatory conditions: Acute appendicitis, ANIML, GE, UTI.
- Less common:
  - Intestinal obstruction due to intussusception/complicated groin hernia/other causes
  - Urinary tract obstruction
  - Primary peritonitis
  - Lower lobe pneumonia.

www.Ebook777.com

- Other:
  - Roundworms. Mechanisms – (i) Intense spasmodic contraction of intestinal wall on the bolus of worm induced by worm-toxins. (ii) Actual mechanical obstruction caused by the bolus of worms/volvulus/matting of loops loaded with worms/rarely intussusception. (iii) Cholangitis. (iv) Hepatic abscesses. (v) Pancreatitis
  - Metabolic
  - Hematological
  - Neurological
  - Psychogenic
  - Causes of recurrent episodes of pain: Emotional instability, IBS, abdominal epilepsy, intermittent porphyrinuria.

Disorders are confined largely to specific age ranges. For example,



4. Children are usually uncooperative, anxious. Patience is needed to assess them. Try to distract the child's attention with colorful toys / torch, etc.
5. In the vast majority of children the symptoms resolve spontaneously over several hours.
6. Infant's abdomen has a shallower pelvis than that of the older child/adult. Therefore, a full bladder extends up to the umbilicus.
7. Attachment of Scarpa's fascia to fascia lata → the inguinal skin crease – It is 1 cm below the inguinal ligament, especially in 6–18 months age-range.
8. In infants, processus vaginalis is often patent (and yet might not have drawn parent's attention due to the absence of an obvious hernia/hydrocele). In such a situation, an abnormal collection within the peritoneal cavity (meconium/blood/pus) may track down and lead to discoloration of scrotum.
9. Pelvic parietal peritonitis → NO abdominal wall rigidity.

HISTORY

- – Is this the **first episode** of such pain, **OR** Is it **recurrent**?

  If recurrent

  - How long does each episode last?
  - How much is the pain-free interval?
  - During the so-called pain-free intervals, is the child completely asymptomatic, OR does s/he continue to have some discomfort/dyspepsia?
  - Are the durations of pain-episodes and pain-free intervals more-or-less constant, OR are they variable?
  - Does the site of pain remain constant during each episode, OR does it vary?
- – **Duration** (of this episode)?
- – Exact **site**?
- – **Progress**?
- – **Is it paining at present**? Has the child been given any **analgesic**?
  In c/o very young children:
  - Have you observed that the child is preferring to remain unnaturally immobile?
  - Have you observed sudden spasms, may be even during sleep?
- – **Nature** of pain? (Try to obtain this information if practically possible.)
- – Does the child **cry out with drawing up of the legs**?
- – Does the child prefer to remain still? Does the child **object to movements/handling**?
- – Can the child **walk swiftly with the body straight-upright, OR** does she/he have to bend forward, with hands clasping the abdomen while being able to walk only slowly?

Free ebooks ==> www.Ebook777.com

- Is the pain **exacerbated during micturition/defecation**?
  - Is the pain experienced at the end, or is it experienced throughout the act of micturition?
  - Is it referred to the tip of penis/labia majora/perineum/suprapubic region?
  - Is it scalding?
  - Does the pain disappear after passing urine?
- Is the **pain relieved by**
  - assuming sitting position
    OR
  - passing flatus/stools/taking enema
    OR
  - applying heat?

- Does the child accept the feed or **refuse the feed**?
- **Vomiting**?
  - Did vomiting begin after, coinciding with, or before the onset of pain?
  - Frequency?
  - Does it seem to be persisting apparently even in the absence of pain (in case if pain has disappeared)?
  - Nature?
    - Green-colored (bile-stained)?
    - Yellowish (gastric) f/b greenish?
    - Yellowish f/b greenish f/b dark brown with foul odor (feculent)?
    - Non-bile-stained?
    - Bloody/coffee-grounds?
- **Diarrhea**?
  - Frequency?
  - Consistency—watery or semisolid?
  - Amount?
  - Does it contain mucus/pus/fat/blood?
  - Foul-smelling?
- **Constipation**?

INTERPRETATION OF SYMPTOMS

Pain

- *Duration and progress*
  - (Severe) Pain in abdomen lasting > 4 hours should be regarded as e/o a potential abdominal emergency until proven otherwise. In infants however, abdominal colic of even shorter duration can signal the presence of an intussusception
  - Very rapid progress could suggest volvulus
  - Progress over a few hours (18–24 or so) could suggest appendicitis
  - Pain × a week without peritoneal irritation is unlikely to be due to appendicitis
- *Crying out with drawing up of legs* may suggest severe colicky pain
- *Remaining still, disliked being disturbed/handled* ⇒ peritonitis
- Pained facies, bent forward, clasping the abdomen with hands, able to walk only slowly = '*Appendix shuffle*' – suggests localized peritonitis, which is most commonly due to acute appendicitis. Patient with diffuse peritonitis will usually not walk
- *Exacerbation of pain during micturition/defecation* ⇒ pelvic peritonitis.

Pitfalls

- Children provide many exceptions to the classic descriptions of pain; for example, pain arising in the kidney/ureter may be referred to the midline, or even to the opposite side
- Children often give contradictory/misleading answers to the 'standard' questions concerning the details of pain.

Vomiting can occur as a response to any inflammation/severe pain due to autonomic reflex.

1. Persistent vomiting, even though painless, should raise the possibility of a small intestinal obstruction, for a proximal level obstruction, e.g. duodenal/jejunal, is often painless and not accompanied by distension.

www.Ebook777.com

2. *Relationship with the onset of pain*
   - Vomiting commencing a few hours after the onset of pain—commonly seen with appendicitis. However, in very small children, the time of onset of pain may be difficult to ascertain.
   - Vomiting coinciding with pain, both being sudden and dramatic ⇒ colic.
   - In intestinal obstruction, the time of onset of vomiting depends on the level of obstruction.
3. *Frequency*
   - In proximal level intestinal obstruction, vomiting is frequent and forceful.
   - In distal intestinal obstruction, frequency of vomiting may not be impressive and may belie the seriousness of underlying pathology, e.g. intussusception.
4. *Nature of vomitus* — *Cause(s)*
   - Bile-stained — Duodenal obstruction (beyond the 2nd part) due to malrotation with volvulus.
   - Gastric, f/b bile-stained — GE.
   - Gastric f/b bile-stained f/b feculent — Intestinal obstruction at the level of mid-ileum onwards.
   - Non-bile-stained — Gastric outlet obstruction.
   - Bloody/coffee grounds — Esophageal varices, gastritis, GE reflux.

Bowel

Nature	*Cause*	*Mechanism*
– Frequent, watery, large volume	GE	Primary effect on absorption.
– Relatively less frequent, small mucous stools	Pelvic peritonitis, e.g. pelvic appendicitis, pelvic abscess	Secondary irritation of the rectum
– Initial evacuation f/b blood ± mucus	Intussusception	Initial reflex emptying. Later, vascular compression leading to venous congestion

Diarrhea lasting > 24 hours should suggest the possibility of a pelvic/retroileal appendicitis. **Even in proven cases of gastro-enteritis,** the possibility of appendicitis or intussusception (the conditions which may supervene GE) should not be ruled out.

Salient features in **PHYSICAL EXAMINATION**

Practical tips in a child < 3 years (not likely to be cooperative at all)

1. Distract the attention by a colorful toy/torch, etc.
2. Allow the mother to cuddle, allow the child to settle down and then examine from behind (such that the child cannot see the clinician).
3. Re-examine when the child is asleep.

However, with a continually crying child, adequate assessment is impossible and sedation is then essential.

Inspection

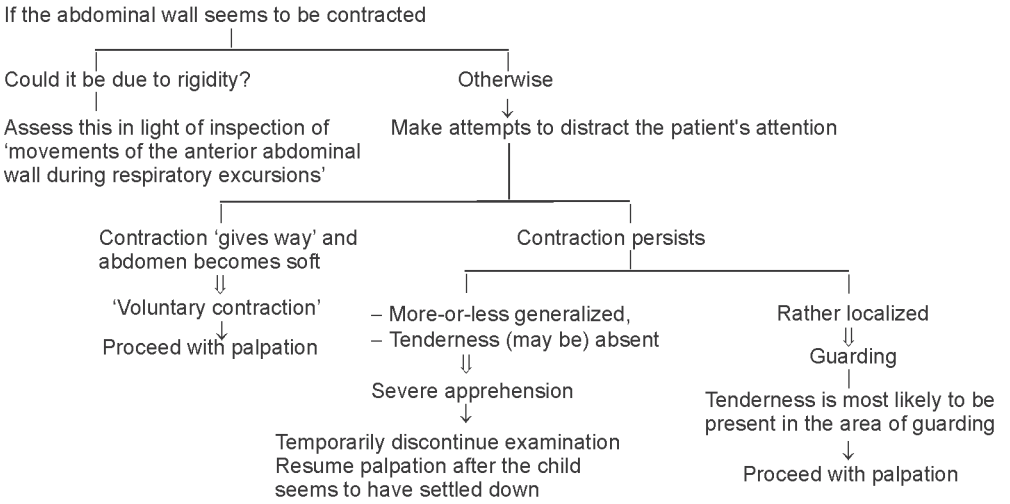
- **Movements** of the anterior abdominal wall associated with (normal) respiratory excursions
  - Normal/diminished/absent?
  - If diminished or absent—confined to a region, OR all over the abdomen?
- **Contour**: Scaphoid OR Distended?
  ↓
  - Does distension involve a particular area?
  - Is it 'severe'?
- **Skin**: Bluish discoloration in flanks/erythema/bruising?
- **Umbilicus:** Transversely stretched/pigmented/periumbilical discoloration?
- **Fullness (bulging) of intercostals spaces**?

Palpation

- Any **hyperaesthesia in** the shoulder/Boas's area/Sherren's triangle/lumbar area?
- Abdomen—**Soft, OR Contracted**?

Clinical method in addition to the usual one: Use tips of fingers to gently test the tension in the muscles. This is often useful to assess muscle tone on both sides simultaneously.

Free ebooks ==> www.Ebook777.com



- **Tenderness—**present or absent? If present—in which area/quadrant?
  The presence of tenderness should be concluded only if the patient winces the face and flinches the body. If any one of these two minimum criteria is absent, then think twice before concluding the presence of tenderness *even if the patient otherwise overreacts*!
  Clinical methods to elicit tenderness:
  1. By direct palpation.
  2. By gently shaking the child: (a) Grasp the child firmly on each side of the chest by clasping with fingers and palm, and shake the child from side-to-side. (b) Grasp the pelvis below the iliac crest in the similar way and shake.
  3. If the child is older enough to obey commands,—look for 'cough tenderness'.
  4. Look for Rovsing's sign in c/o pain in RLQ.
- **Edema**?
- **Lump**?
  Causes of lump:
  - The commonest: feces in the colon. **Scybalae in a long loop of sigmoid colon are quite often palpable in the RLQ**
  - Appendicular abscess
  - A characteristic sausage-shaped lump suggests intussusception
  - A tense loop of intestine will occasionally be felt as a less well-defined lump in intestinal obstruction due to volvulus or bands
  - A duplication cyst, causing intestinal obstruction (probably incomplete)
  - Otherwise asymptomatic lumps in the loin (Refer to chapter 35.)
- (Gross) **Organomegaly**?

Measurement

- Abdominal girth at a fixed level (such as umbilical), which can be repeated and then compared with previous readings.

Percussion

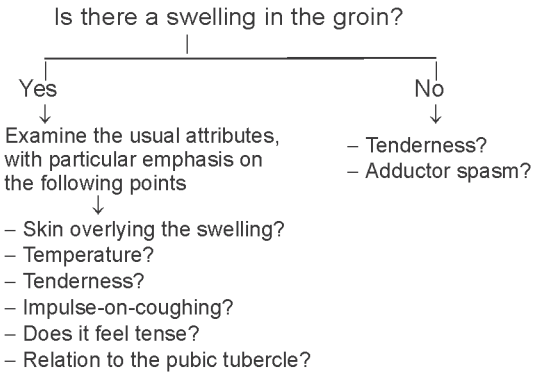
- **Obliteration of liver dullness** in midaxillary line?
- **Shifting dullness**?
- Is the **urinary bladder distended**?

Auscultation

www.Ebook777.com

Examine the groins and external genitalia

- **Pulsations of external iliac and femoral arteries**—absent/weak on both sides?
- **Hernial sites**: Epigastric, umbilical and paraumbilical, LAPAROSCOPOIC PORT SITES, inguinal, FEMORAL, OBTURATOR, GLUTEAL, SCIATIC.



- **Scrotum**

Digital Rectal Examination (DRE)

Although the lateral position is traditional, more information and more accurate orientation can be obtained by performing a DRE in dorsal position especially in infants.

– Tenderness?
– Any mass palpable in the lumen?
– Any mass palpable through the ano-rectal wall?
– Blood on the fingerstall?

The significance of rectal tenderness has the same limitations as that of abdominal tenderness. Even extreme rectal tenderness, by itself, is insufficient evidence for the diagnosis of pelvic appendicitis. DRE does not play a significant role if the diagnosis of appendicitis is otherwise clear-cut. Nevertheless, DRE is valuable in other ways and should be performed in every case of acute abdomen. Occasionally, the apex of an intussusception can be felt. At other times, there may be unexpected explosive passage of wind during/after the examination with prompt relief of all symptoms and signs!

Palpation in Sitting Position (with Hands Locked and Resting up on the Head)

- Gall-bladder tenderness?
- Localized tenderness in the rest of the abdomen?
- Any lump palpable now?
- If guarding/a lump/organomegaly was noticed earlier, are there any changes in the physical findings now?
  In pneumonia, some degree of splinting of abdominal wall muscles may occur—It can be overcome by making the child lean forward while in sitting position.
- – Mark the renal angles on both sides
  – After making sure that the patient's trunk is not twisted, inspect the renal angle for fullness, and palpate for a 'mass'
  – Fullness or a mass lateral to the renal angle?

The role of **enema**

When symptoms and signs are equivocal, a small glycerin or olive-oil enema is justified, and the enema may result in a dramatic improvement.

The administration of an enema is often helpful in reaching or ruling out a diagnosis (intestinal obstruction) and beneficial even in children with a genuine surgical condition (The first bowel action after operation is facilitated, and the discomfort of residual constipation is alleviated).

There is no evidence that a small enema complicates an acute GI inflammatory condition.

Free ebooks ==> www.Ebook777.com

PRINCIPLES AND PITFALLS in arriving at the diagnosis

- An unwell and irritable child with abdominal pain must be taken seriously
- Analyze pain, vomiting and diarrhea carefully. GE may progress to intussusception or to appendicitis, so that even a well-established and undoubted diagnosis of GE should be subject to review
- *Symptoms s/o significant intra-abdominal pathology*:
  1. Pain persisting for > 4 hours.
  2. Pain increasing in intensity.
  3. Persistent vomiting even in the absence of pain.
- Pain in abdomen/testicle/inner thigh could be 'referred'.

Site of pain	*Probable actual site of pathology from where the pain originates*
– Shoulder tip	Blood/pus irritating the undersurface of the diaphragm
– in the back, below the inferior angle of the scapula (right side)	Biliary tract
– Lumbosacrum	Rectum/uterus
– Testicle	Kidney
– Inner aspect of the thigh	Ovary

- Infections elsewhere in the body may be accompanied by pain in the abdomen and vomiting and may simulate an acute abdomen, e.g. otitis media, tonsillitis, pneumonia, a wide range of viral infections (by causing mesenteric lymphadenitis), septic arthritis of the hip. Acute appendicitis can coexist with other conditions, so that the finding of tonsillitis, pneumonia etc. should not divert attention from any abdominal signs which may also be present
- Metabolic (diabetic acidosis), hematological (hemophilia, anaphylactoid purpura, etc.), neurological (abdominal tabes, etc.), and psychogenic conditions may present with acute pain in abdomen. In these conditions surgical intervention is contraindicated except for in anaphylactoid purpura, in which initially absent intestinal obstruction may suddenly supervene
- Analyze tenderness (both per abdomen and on DRE) carefully. This finding alone is an inadequate basis for operation, as localized tenderness is found in wide variety of conditions other than those requiring surgical treatment (e.g. excess flatus, an overloaded colon, hyperplastic lymph nodes, etc.). Tenderness due to non-surgical conditions can generally be expected to subside, whereas tenderness due to 'surgical conditions' will soon be supported by development of additional clinical features such as guarding/rigidity for example
- The commonest lump in acute abdomen is feces (indentable on palpation). The sigmoid colon (which contains feces) is often palpable in RLQ
- Abdominal distension and signs of septicemia are late signs of peritonitis. Peritonitis is an indication for surgery in virtually every situation.

Practical solution when the diagnosis is in doubt: Observation and repeated re-examination for the development of (new) signs. Which specific signs to look for will vary according to the clinical suspicion.

DIFFERENTIAL DIAGNOSIS

Peritonitis

Differentiating points:

- Reluctance to move. The child prefers to lie in bed motionless; protests at being handled
- While walking if at all, will bend/stoop forward, clasping the abdomen with hands, being able to walk only slowly
- (Cough) Tenderness
- Guarding/Rigidity.

Supporting points:

The anterior abdominal wall may become erythematous and edematous, and may show dilatation of veins.

Notes:

- The presence of peristaltic sounds does not rule out peritonitis.
- Late manifestation of peritonitis—Septic shock (- tachypnea, abdominal distension, 'silent abdomen' on auscultation).

www.Ebook777.com

Appendicitis

'Anterior' appendicitis	*'Posterior' appendicitis*	*Pelvic appendicitis*
= anterior to the cecum	= posterior to the cecum / ascending colon/terminal ileum	
– Classical ('book picture') signs are usually present since the anterior parietal peritoneum gets inflamed early	– Vague deep tenderness – Guarding ± – Limp ± (due to psoas spasm) – May develop mass or perforation before diagnosis	– Pain during micturition ± – Diarrhea ± (due to irritation of the rectum) – Vague suprapubic tenderness – No guarding – On DRE: Tenderness, mass

Appendicitis under the age of 5 years is not common. The child cannot communicate symptoms, and also cannot localize the site of tenderness well. Therefore, diagnosis is often made (as peritonitis) when peritonitis has set in.

Once generalized peritonitis has developed, there are few clinical clues specifically to implicate the appendix. The diagnosis of appendicitis should be considered however as it is the commonest pathology; exceptions: 1. babies < 3 years, and 2. otherwise also, if an alternative diagnosis is being considered due to specific features (clinical ± on investigation).

Final common judgment to arrive at the diagnosis *of anterior appendicitis*: A history consistent with appendicitis, coupled with marked localized tenderness and guarding/rigidity in the RLQ.

When there is only tenderness, without guarding/rigidity or a strongly suggestive clinical history, operation for suspected appendicitis will often turn out to be negative.

All said and done, if the diagnosis is in doubt, the best policy is to operate rather than to wait. However, in practice, mortality remains high especially in infants, mainly due to delay in arriving at the diagnosis.

Conditions which may closely mimic acute appendicitis due to the presence of localized tenderness and guarding/rigidity:

- (Severe) ANIML
- Meckel's diverticulitis
- Primary peritonitis
- Deep iliac suppurative lymphadenitis
- Torsion of the omentum with infarction
- Torsion of an ovarian cyst/ovary
- Ruptured luteal cyst

} The precise diagnosis is often made only at operation.

There are no pathognomonic symptoms/signs of acute appendicitis itself, but when signs of peritonitis are localized to the RLQ, the statistical probabilities of this being due to appendicitis far outweigh any other possibility.

Acute appendicitis is not only the *commonest abdominal emergency*, but also the *greatest imitator*, and it ***may appear in many guises***.

ANIML

Refer to page 474.

GE

- The 1st symptom is diarrhea or vomiting; NOT the pain
- Pain—cramping and diffuse
- Usually comes under control within 24–72 hours.

Constipation

- H/o infrequent passage of hard stools
- Pain—colicky, and relieved by bowel action
- On PA/E: Fecal masses palpable
- On DRE: Capacious rectum full of fecal matter.

Free ebooks ==> www.Ebook777.com

UTI and/or UT obstruction
- High-grade fever
- Fewer localizing signs than are caused by appendicitis. Otherwise, differentiation may be difficult
- High index of suspicion is required
- Urine routine and microscopic examination should be requested.

Peritonitis

Classical severe 'board-like' rigidity is not always present even when there is advanced generalized peritonitis. As peritonitis progresses to become advanced, the abdomen gets more distended but paradoxically, tenderness gets poorly localized and the degree of rigidity seems to become less. *In this stage, abdominal distension, irrespective of the degree and location, is a sign of great significance*.

Common causes of generalized peritonitis in children:
- Perforative appendicitis
- Perforation/strangulation following intestinal obstruction
- Primary peritonitis
- Perforative Meckel's diverticulitis.

Intestinal obstruction: Refer to chapter 75.

Intussusception: Refer to chapter 74.

Pediatric Gynecologic Emergencies
- Midcycle pain, rupture of a small luteal cyst
- Relatively less common causes—salpingitis, primary peritonitis, tubal menstruation, torsion of the ovary—These conditions are rarely diagnosed unless they come to operation.

Differentiating points
- On bimanual palpation in dorsal position, variability in the size and degree of tenderness of the tube and ovary on each side
- Extreme tenderness in the pelvis contrasting with the relative absence of abdominal rigidity.

A rule of thumb

In all cases with painful pelvic lump or/and abdominal rigidity, exploration is necessary and should be undertaken through a lower abdominal incision rather than a McBurney's incision.

DD of a (Subacute) Mass in the RLQ
- Appendicular mass/abscess, ovarian cyst without/with complication (torsion)
- In a child < 3 years: Intussusception, provided other clinical features support the diagnosis
- In older children: Lymphoma, provided other clinical features support the diagnosis (—loss of weight, fever, lymphadenopathy, hepatosplenomegaly, etc.)
- Rare causes: Meckel's diverticulitis, ileocecal TB, actinomycosis, duplication cyst, lymphangioma, Yersinia lymphadenitis, cystic fibrosis (→ a mass from ileal obstruction with viscous contents).

Abdominal Pain of Uncertain Origin

This is a substantial group.

Probable causes:
1. Minor disturbances of bowel function—'Indigestion', wind pain, constipation, 'gripes' etc. Pain often resolves spontaneously.
2. ANIML.

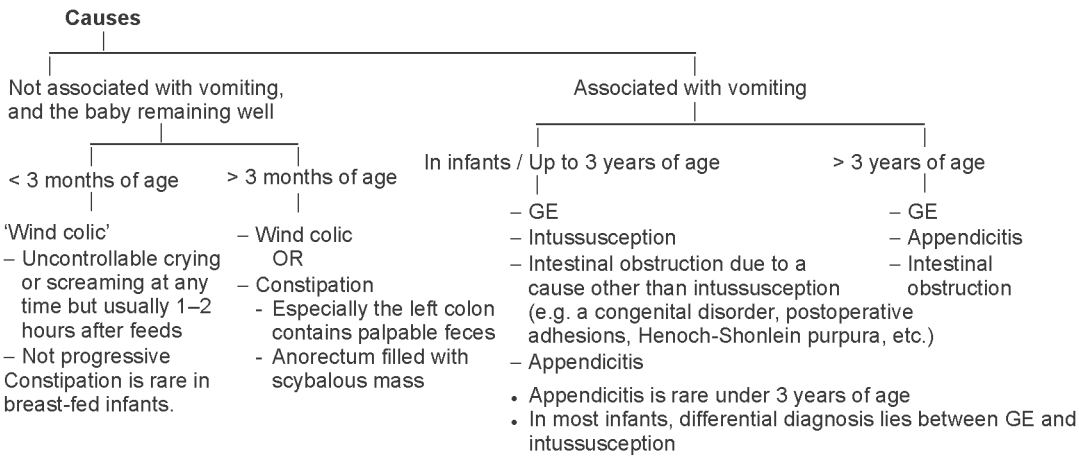
Abdominal Emergencies in Mentally Retarded Children

Think of the following two causes first.
- Acute appendicitis
- Impaction/ulceration/perforation of the GI tract by an ingested foreign body.

Paralytic ileus has usually set in by the time attention is first drawn to the abdomen.
Vomiting is more significant than in normal children, although its absence is a very unreliable guide.
The presence/absence of fever is also unreliable.
Pulse is of considerable value as an indication of spreading infection, and provides one of the most useful means of determining the course of intra-abdominal inflammatory disease.

www.Ebook777.com

74 COLIC
(Colicky Pain in Abdomen)



Intussusception

Causes *(related to lead point) with increasing age*:
- Up to 3 months: Duplication cyst.
- 3 months to 2 years: Idiopathic (Probably enlarged Peyer's patches).
- After 2 years: Inverted Meckel's diverticulum, polyp, H-S purpura.

Most common age of occurrence of idiopathic intussusception: 3–9 months.

Clinical Presentation

- **Moderate-to-severe colicky pain**—*Recognized by its reflex autonomic effects* viz. **vomiting, sweating, screaming, pallor and drawing up of the legs**.
  - Each colic spans over a minute or 2, followed by a pain-free interval of about 15 minutes or so.
  - If pain is very severe, child may not even scream!
- **Lethargy**.
- **Vomiting**—has a bimodal distribution in time.
  - The early vomiting that is non-bile-stained is reflex vomiting due to severe pain, until the stomach gets empty.
  - After a variable interval, usually of a few hours, there is resurgence of vomiting typically s/o mechanical intestinal obstruction.
- **Loose motions**
  - *Differentiating features from GE*: Brisk. Early evacuation of colon; motions cease when colon gets empty.
  - Initially, loose motions may still be confused with GE as several stools may be passed in quick succession.
  - Dysentery is a relatively late sign (Mechanism: Squeezing out of blood and mucus from congested mucosa of intussusceptum).

Free ebooks ==> www.Ebook777.com

- **Findings on abdominal examination**
  Abdominal examination should be carried out between episodes of colics.
  Rule out constipation.—Palpable fecal mass in the sigmoid and left colon, and scybala on DRE suggest constipation.
  - **Palpable mass close to the umbilicus**—i.e. within the line of the normal surface markings of the colon (having taken for granted the ileo-colic type of intussusception which is the commonest type—the prototype). The mass cannot be palpated if abdomen is tender/guarded/distended.
  - **DRE**: Lead point may be palpable. Blood on finger-stall ±.
    DD in case if intussusceptum protrudes from the anus: Prolapse of rectum. Differentiating point between the two—The presence of fornix suggests prolapse of rectum.
- **Late signs**: Established intestinal obstruction, peritonitis, shock.
  Restless movements during episodes of colic suggest that peritonitis has not set in.
- **Atypical features**
  - Few patients have no obvious colic.
  - Vomiting may be absent or may only appear after 6–12 hours.

Intestinal obstruction (due to causes other than intussusception)

Common causes

In children who have undergone a laparotomy previously
- Postoperative adhesions/bands

Otherwise
- Obstructed inguinal hernia
- Volvulus
- Meckel's band/diverticulum
- Duplication
- Internal hernia

Two situations in which obstruction is less obvious and more likely to be overlooked:

1. In the early post-op period when pain is difficult to interpret and delay in recovery from paralytic ileus may mask the presence of an early obstruction due to fibrinous adhesions.
2. When vomiting alone is the presenting feature of a proximal small intestinal obstruction in which pain and abdominal distension may be absent.

PRINCIPLES AND PITFALLS in various clinical settings

- '**Intermittent pallor without screaming':** Intermittent screaming is easily recognized as colic, but intermittent pallor without screaming may also represent colic.
- '**Severe abdominal colic persisting for > 2 hours in an infant**': Consider intussusception.
- '**Colic and vomiting (persisting over 24 hours) in an infant *without diarrhea***': It is hazardous to diagnose gastritis.
- 'Infant with **vomiting and lethargy**': Dehydration and shock (leading to lethargy) may simulate meningitis.
- The mass of intussusception may be difficult to feel as it is posterior to the rectus abdominis.

www.Ebook777.com

75 NEONATAL INTESTINAL OBSTRUCTION

Causes in the descending order of frequency

On the background of perinatal stress like prematurity, obstetric misadventure, sepsis and metabolic disturbances
- Paralytic ileus
- Necrotizing enterocolitis

Otherwise
- Hirschprung's disease
- Meconium ileus
- Meconium plug syndrome
- Anorectal malformation
- Organic small intestinal obstruction—atresia / stenosis/ malrotation without/with volvulus (volvulus neonatorum)
- A small intraluminal duplication cyst
- Meconium peritonitis (Prenatal perforation)

SYMPTOMS

The **cardinal** ones:
- Vomiting
- Abdominal distension ±
- Failure to pass / delay in the passage of meconium.

- What is the **timing of onset of symptoms related to the time of birth**?

At birth / Within the 1st 48 hours ⇓ Anatomical obstruction

After the 1st 48 hours ⇓ Functional obstruction—Hirschprung's disease/NEC

- Are the symptoms **persistent or episodic** (recurring after intervals of several days/weeks)?
  Episodic obstruction ⇒ Hirschprung's disease / Volvulus neonatorum

- Particular details of **vomiting**
  Was it bile-stained from the beginning, OR Did it initially contain milk and subsequently become bile-stained?

Inference

Bile-stained to begin-with ⇓ Proximal small intestinal obstruction
- Usually the duodenal—*Taken for granted unless proved otherwise.*

Initially of milk, subsequently becoming bile-stained ⇓ Level of obstruction: Mid-ileum onwards.

In duodenal obstruction, blood may be present in vomitus due to acute gastritis (accumulation of acid in high-level obstruction)

Free ebooks ==> www.Ebook777.com





Figs 75-1A and B Examples of distended abdomen

- **Abdominal distension**

Present

⇓

The level of obstruction: Mid-ileum onwards

↓

Distension

Present at the time of birth

⇓

– Organic small intestinal obstruction (due to atresia)
– Meconium ileus

Developing after birth

⇓

– Hirschprung's disease
– Anorectal malformation

Absent

⇓

Proximal small intestinal obstruction, usually the duodenal, e.g. duodenal atresia/stenosis / malrotation leading to duodenal obstruction by Ladd's bands, without volvulus

Exceptions: 1. Malrotation with volvulus (Volvulus → colonic obstruction, i.e. 'closed loop' obstruction)
2. Esophageal atresia put on ventilator

In general, early and extreme distension with failure to pass meconium ⇒ complete long-standing obstruction. Mild-to-moderate distension appearing some time after birth ⇒ functional / incomplete anatomical obstruction. Some non-surgical disorders can also present with similar symptoms; e.g. sepsis (local/general), congenital heart disease; inborn errors of metabolism may present with vomiting and abdominal distension. *Differentiation of non-surgical disorders from neonatal intestinal obstruction*: The feature not present in the early stages of mechanical intestinal obstruction, but present with non-surgical causes—The baby appears generally unwell or even severely toxic.

- **Dysentery**?
- H/s/o **obstetric problem / perinatal stress**?
  - Obstetric problems including APH, preeclamptic toxemia. These predispose to fetal hypoxia → secondary gut perforation
  - Polyhydramnios ⇒ the level of obstruction anywhere from proximal jejunum to esophagus
- Premature delivery?—This may be followed by the development of hyaline membrane disease or pulmonary atelectasis associated with respiratory distress and abdominal distension, and NEC
- Premature rupture of the membranes?—Predisposes to septicemia
- Precipitate or traumatic delivery?—This is often followed by cerebral birth injury
- Prolonged obstructed labor?—This may cause fetal distress, which in turn → the passage of meconium *in utero* → asphyxia and meconium aspiration pneumonia
- **Family history** may be contributory: Hirschprung's disease, cystic fibrosis (meconium ileus).

PHYSICAL FINDINGS

- In the early period of presentation, a baby appearing unwell or severely toxic ⇒ likelihood of one of the non-surgical causes responsible for the symptoms, e.g. sepsis (local/general), congenital heart disease, inborn error of metabolism
- Respiratory embarrassment from splinting of the diaphragm (due to abdominal distension)
- Shock?
- Epigastric fullness with a scaphoid lower abdomen may suggest duodenal obstruction

www.Ebook777.com

- Distension of only the colon/Distension of colon for several days preceding generalized distension ⇒ Hirschprung's disease
  Distension of abdomen can result from non-GIT cause, e.g. Hydronephrosis, ovarian cyst, etc.
- Examine the perineum for anorectal malformation
- DRE with a thermometer/little finger: A sudden gush of air and meconium in a child suspected of having Hirschprung's disease, is highly suggestive of the diagnosis of Hirschprung's disease. The anus and rectum may feel tight
- In a case suspected of having esophageal atresia: Gently introduce a 10 F stiff catheter through the mouth. Its arrest at ~ 10 cm from the anterior gums strongly suggests the diagnosis of esophageal atresia
- The absence of liver dullness in the midaxillary line ⇒ perforative peritonitis
- *Signs of gangrene*: Blood-stained stools, sunken fontanelle (due to dehydration), mottled periphery, shiny erythema and edema of the anterior abdominal wall (as the abdominal wall is thin in neonates).

X-ray Abdomen

Various findings which should be looked for: Number of air-fluid levels, free gas under the diaphragm, intramural gas, intraperitoneal calcification (⇒ prenatal perforation).
Small and large intestines cannot be readily distinguished on an X-ray of a neonate.

CLINICAL PEARLS

- Failure to pass meconium in the 1st 24 hours ⇒ Hirschprung's disease. However, initial passage of meconium *does not* rule out Hirschprung's disease
- Green vomitus should be regarded as potential emergency
- Likely causes of obstruction occurring after the evacuation of normal meconium: Hirschprung's disease, NEC, malrotation with volvulus
- Obstruction of the duodenum should be treated as malrotation with volvulus, unless proved otherwise.

DIFFERENTIAL DIAGNOSIS

Paralytic Ileus

Predisposing factors: Cerebral birth trauma, asphyxia, respiratory distress, septicemia.

Clinical features: Similar to intrinsic intestinal obstruction, i.e. failure to pass meconium, bile-stained vomiting, abdominal distension.

X-ray abdomen in upright position: Uniformly dilated loops of gas-filled intestine but usually without definite fluid levels.
The signs of obstruction settle after successful treatment of the underlying condition.

Necrotizing Enterocolitis

Perinatal stressful condition → hypoxia → mesenteric vasoconstriction → mucosal ischemia → bacterial colonization of gut wall → intramural gas f/b gangrene f/b perforative peritonitis and septic shock.

Clinical features:
- Time of onset—a few days after birth
- Refusal to feed. Fever
- Bile-stained vomiting, abdominal distension
- Dysentery
- Initially, the abdomen is non-tender and soft. Later, features of peritonitis may develop
- A mass may be palpable if a localized intraperitoneal abscess has formed.

X-ray findings:
- Dilated loops of intestine with intramural bubbles of gas
- Separation of adjacent loops of intestine ⇒ appreciable amount of intraperitoneal exudate, an indication of peritonitis.

Free ebooks ==> www.Ebook777.com

Hirschprung's Disease

- Most commonly involved segment with aganglionosis: Anus to rectosigmoid junction
- The 1st passage of meconium after birth assures the anatomical and functional continuity of the GIT
- One of the cardinal symptoms of Hirschprung's disease = failure to pass meconium within the 1st 24 hours of birth. Most of the normal children pass meconium within 12 hours. Almost all the normal children pass meconium within 24 hours
- In premature/very sick neonates, passage of meconium may be delayed, but even in these, the diagnosis of Hirschprung's disease should be considered.

The degree of obstruction may be variable: 1. Complete obstruction within 24 hours of birth, 2. Episodes of partial obstruction for several weeks before the diagnosis is made. 3. Asymptomatic during the neonatal period.

Meconium ileus and the 'meconium plug' syndrome may present with symptoms somewhat similar to Hirschprung's disease. These two conditions are much less commonly seen.

Meconium Ileus

- It is a manifestation of cystic fibrosis
- Meconium is viscous (i.e. tenacious—sticky). Impaction and dehydration of the meconium in terminal ileum → formation of meconium pelletes → obstruction—may lead to massive dilatation of the obstructed intestine, followed by its volvulus.

The 'Meconium Plug' Syndrome

There is an inspissated plug of **mucus** at the head of the column of meconium, usually in the colon.

Clinical features – same as those of Hirschprung's disease.

DRE may be normal, and it may be curative: The plug is passed, followed by a large meconium stool and gas.

Duodenal Obstruction

Causes of extrinsic obstruction: peritoneal bands (in malrotation), annular pancreas.
Causes of intrinsic obstruction: atresia / stenosis (due to a septum or a membrane with a small hole in it).
Site of occurrence of atresia: D_2, with abnormalities of the opening of pancreatic and bile ducts.
A complete obstruction will present during the neonatal period, whereas presentation of incomplete obstruction may be delayed for 2–3 years of age.

Plain X-ray in upright position demonstrates a typical 'double bubble' appearance.

Volvulus Neonatorum

- There may be no manifestation of intestinal obstruction in the first day or two after birth, meconium also having been passed normally; then obstruction develops suddenly
- The signs vary depending on the tightness of the volvulus.

Plain X-ray in upright position:

- A typical 'double bubble' appearance s/o duodenal obstruction
- Multiple air-fluid levels ⇒ obstruction at the mid-gut level.

Intestinal Atresia

- The most frequent site: distal ileum
- The least common site: colon
- There is usually only one atresia, though there may be several close together or widely scattered.

Duplication Cyst

1. Short closed cystic segments: A small intraluminal cyst may cause obstruction in the neonatal period. Larger cysts less intimately connected with the common wall usually cause progressive obstruction later in infancy or in early childhood. Occasionally a large tense cyst is palpable as a mobile lump.
2. Long tubular communicating duplications are much less common and present with GI bleeding.

Anorectal Malformations

- The least severe form = just a simple membrane covering over an otherwise normal anal canal
  The most severe form = complete agenesis of anorectum.

www.Ebook777.com

- In most patients, there is a fistulous connection between the bowel and perineal skin/urogenital tract
- *Clinical problem involves identification of* **1**. The lowest level of the normal bowel in relation to the pelvic floor and anal sphincter. **2**. Whether fistula is present or absent, and the details of the fistula when present. **3**. The presence or absence of loco-regional (sacral agenesis, UT anomalies) and general anomalies.

Prenatal Perforation

- Possible causes of perforation: Meconium ileus, ischemia of gut-wall secondary to vascular occlusion/hypoxia; intussusception with gangrene
- A helpful diagnostic clue: X-ray abdomen—Irregular calcifications in the peritoneal cavity suggest meconium peritonitis.

The main causes of death in a c/o neonatal intestinal obstruction

- Coexistent congenital heart disease
- Prematurity
- Septicemia. The early signs of spread of infection are usually non-specific.—Lethargy, abdominal distension, jaundice and hypothermia.

Free ebooks ==> www.Ebook777.com

76 RECURRENT PAIN IN ABDOMEN

This is one of the commonest and difficult problem. An objective diagnosis can be made in only few cases. In most of these children there are no symptoms/signs specifically pointing to any particular organ/system. The pain is most often vaguely located in the umbilical area, and is frequently colicky. The pain disappears after half an hour or so.

Specific points to be asked in **HISTORY**

- Dietary indiscretions?
- Pica?
- Passage of worms?
- A family h/o migraine?
- Approaching menarche?
- Emotional instability?

POINTERS OF ORGANIC, AND POSSIBLY A SURGICAL CAUSE

- Duration of pain > an hour.
- Pain consistently localized to a site away from the umbilicus.
- Pain wakes the child from sleep or/and causes the child to roll around.

www.Ebook777.com

77 VOMITING IN THE NEONATE

GENERAL REMARKS

Vomiting is one of the most common symptoms and the evaluation of its significance is particularly difficult yet most important. It is a symptom, not a diagnosis, and the cause of vomiting must be established. If the vomiting seems 'significant', investigation is indicated.

Vomiting in the neonate is **'significant', when it is**
- persistent
- bile-stained (with an exception though)
- blood-stained (including 'coffee-grounds, flecked with altered blood)
- projectile
- accompanied by loss of weight or failure to gain weight.

Bilious vomiting should be considered to be due to intestinal obstruction (mechanical or paralytic) until proved otherwise. *An exception* to this is complete duodenal obstruction proximal to the ampulla of Vater.

THE KEY CLINICAL POINTS to be evaluated

- Is the baby **premature**?
- Details of vomiting:
  - Duration?
  - Frequency?
  - Color:
    - Is it green/greenish (i.e. bile-stained)?
    - Is it blood-stained? Does it contain 'coffee-grounds', flecked with altered blood?
  - Is it projectile?
- Within how many hours after birth was the **meconium** passed?
  Did it pass normally, or was it stimulated by rectal washouts?
  Color and odor?—Was it brown-colored or Was it malodorous?
- **Distension** of abdomen?
- Does the child refuse **feeds** OR Does the child feed well?
- Loss of **weight** or failure to gain weight?

INTERPRETATION

Criteria of retained/abnormal meconium: One/more of the following:
- Failure to pass meconium within the 1st 24 hours of birth.
- Meconium produced by rectal washouts.
- Brown-colored or malodorous.

Exception: Prematurity or alimentary dyskinesia can delay the appearance of meconium, which is then passed spontaneously and the delay is seen to have been inconsequential.

Distension of abdomen ⇒ (Distal) intestinal obstruction / colonic obstruction / gastric outlet obstruction. Abdominal distension is minimal or absent in duodenal/jejunal obstruction.

Free ebooks ==> www.Ebook777.com

In general,
- Vomiting
- Retained/abnormal meconium
- Abdominal distension (Perhaps the least reliable symptom)

} ⇒ intestinal obstruction.

Causes of vomiting

Surgical

1. Intestinal obstruction
2. Sphincteric disorders at the either end of the stomach:
   - CHPS
   - GE reflux

Medical

Medical causes of vomiting are more common. The causes are numerous, but only two are mentioned here which are likely to be particularly confusing

1. Neonatal infections, e.g. meningitis, septicemia. They are associated with a wide variety of clinical features: diarrhea, twitching/convulsions, pallor, cyanosis, hyperthermia
2. Adrenogenital syndrome

www.Ebook777.com

78 NON-BILE-STAINED VOMITING IN INFANCY

THE KEY CLINICAL POINTS to be evaluated

- **Age**? – 1st few weeks of life OR between 3 months and 3 years?
- **Feeding**: Does the child refuse feeds OR feed well?

  ↓

  – **Vomiting**: Characteristically forceful and projectile, OR Nondescript?
  – Does the child look **emaciated or otherwise healthy**?
  – Any **lump** (specifically hypertrophied pylorus) palpable in the abdomen?

ARRIVING AT THE DIAGNOSIS

Age of the child

- 1st few weeks of life ⇓
  - – Feeding problems
  - – GE reflux
  - – CHPS: 3 weeks to 3 months
- 3 months to 3 years ⇓
  - Intussusception
  - Non-bile-stained vomiting represents the early reflex vomiting due to severe pain until the stomach gets empty. (After a few hours, there is resurgence of vomiting typically s/o mechanical intestinal obstruction.)

Feeding

- Refusal of feeds. Child sick/unwell, lethargic ⇓
  - *Systemic infection*, e.g. meningitis, UTI, etc.
- Feeds well
  - Characteristically forceful and projectile vomiting
    Occurs after every feed
    Child hungry; feeds with enthusiasm
    When duration of symptoms is longer (> a week), vomitus may contain 'coffee grounds' (due to gastritis) ⇓
    CHPS.—the diagnosis can be clinically ascertained when coupled with many other characteristic clinical features
  - Vomiting—nondescript
    Child 'greedy', and otherwise looks healthy ⇓
    Feeding problem
    Overfed baby, inexperienced mother
  - Vomiting—nondescript and variable
    Effortless vomiting of variable volumes before, during or after meals
    Commences at birth, becomes less pronounced with age
    Minor intercurrent illness → exacerbation of symptoms
    (Hematemesis ± (if severe esophagitis))
    Otherwise, the child is healthy ⇓
    (Uncomplicated) *GE reflux*
    – No pathognomonic clinical signs
    – Improvement in symptoms with empirical treatment confirms the diagnosis
    Empirical treatment: Food thickening, proper posturing

Both problems (i.e. feeding problem and GE reflux) may coexist

Free ebooks ==> www.Ebook777.com

DIFFERENTIAL DIAGNOSIS

Systemic infections like meningitis, UTI, etc.
Commonly there are no localizing signs in young infants.

Investigations:
- Urine—Routine and microscopic.
- LP, if meningitis is suspected.

GE Reflux
No pathognomonic signs if reflux disease is uncomplicated.

Manifestations due to complications of GE reflux:
1. Severe esophagitis → pallor from anemia or hematemesis.
2. Esophageal stricture →
   - Retrosternal discomfort, dysphagia, excessive salivation, choking.
   - Failure to thrive, weight loss.
   - Recurrent chest infection (aspiration pneumonia).

Although the diagnosis of GE reflux can be frequently made on clinical grounds, a Ba swallow is advisable to look for hiatal hernia, peptic stricture.

Three groups of patients can be recognized:
1. GE reflux without a hiatal hernia.
2. GE reflux with a hiatal hernia of variable size (usually 'sliding' variety).
3. Peptic stricture.

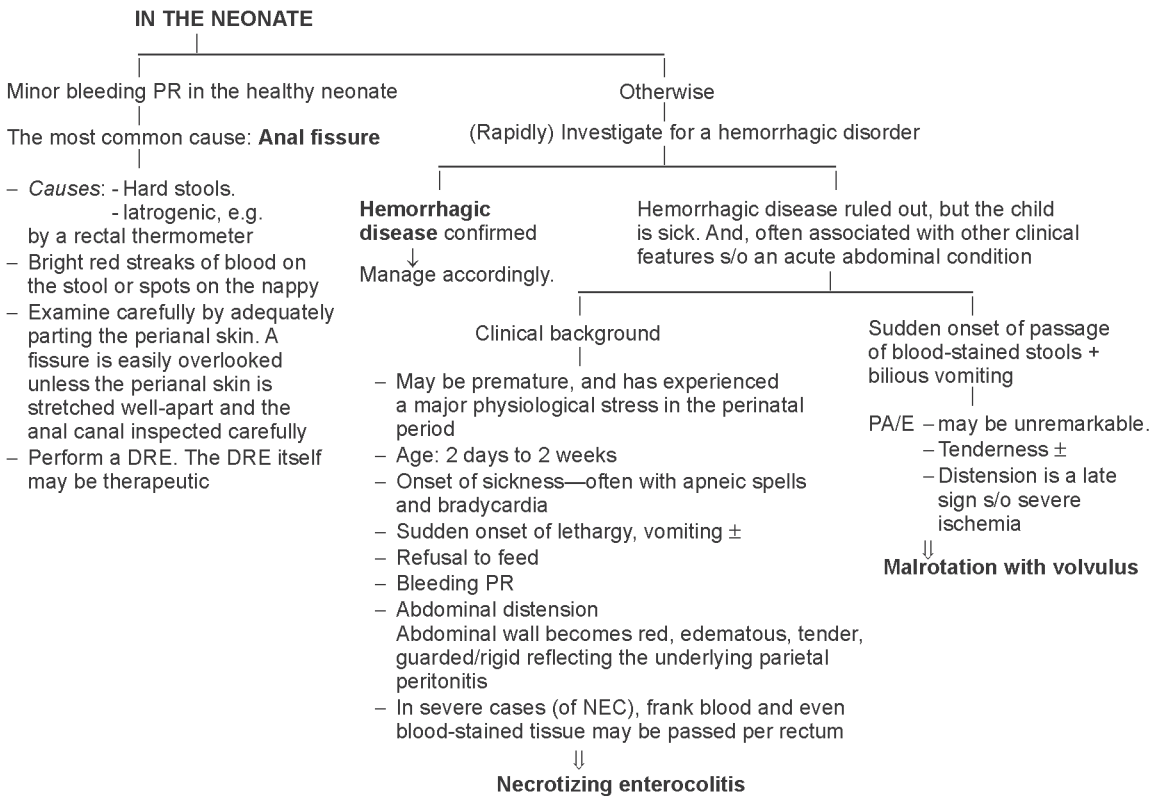
CHPS

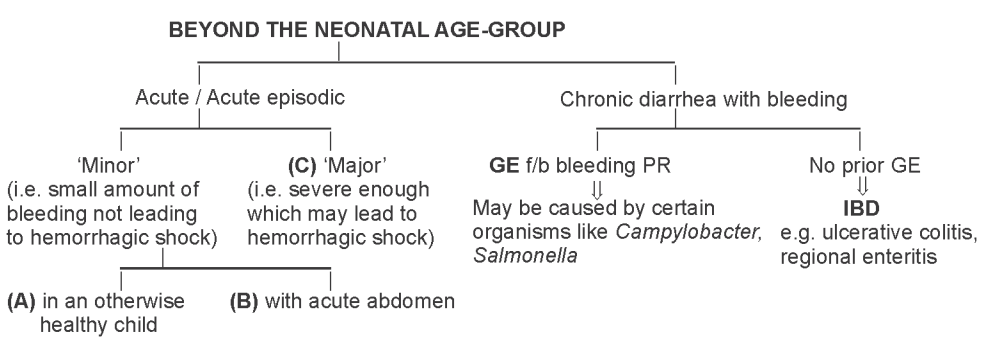
- The presence of 'coffee grounds' (hematemesis) suggests unusually severe gastritis, or/and that vomiting has been occurring for some time. Implication of this in the postoperative period: Gastritis may continue to cause some vomiting after surgery.
- Dehydration: Anterior fontanelle depressed. Eyes sunken. Skin turgor decreased. Mouth dry. Nappies dry.
- Scrawny-looking thin infant due to loss of subcutaneous fat—due to malnutrition.
- Epigastric fullness.
- Visible gastric peristaltic waves traveling from left to right costal margin.
  Duration of each wave—5–10 s. Frequency—every ~ half a minute.
  Easier to see after a feed. If not easily visible, throw a light obliquely from the right side, and then observe.
- Hypertrophied mobile pylorus is palpable against the anterolateral aspect of vertebral bodies in the RUQ: Traditionally described as 'feeling like an olive'. If difficult to feel, then **1**. Examine the baby when it is being fed (during which the baby's mind is relaxed. Feeding should not allow overdistension to occur however). **2**. Examine after the baby has fallen asleep.
- If the 1st attempt at palpation of the hypertrophied pylorus fails, make further attempts at varying intervals.
- If the diagnosis still remains uncertain, a small contrast meal and fluoroscopy will show a characteristic string sign.

Clinical pearls:
1. The pathognomonic sign of CHPS = palpation of hypertrophied pylorus.
2. Surgical treatment for CHPS should be carried out only after correction of fluid, electrolyte and acid-base imbalance.

www.Ebook777.com

79 GASTROINTESTINAL (GI) BLEEDING





Free ebooks ==> www.Ebook777.com

A. **Small amount of rectal bleeding in an otherwise healthy child**
1. Constipation is probably the commonest cause.
2. In majority of cases no cause can be found.
3. May follow an acute episode of diarrhea.
4. Anal fissure.
5. Rectal prolapse.

B. **Small amount of rectal bleeding in a sick child (usually an acute abdomen)**
1. Strangulating intestinal obstruction.
2. H-S purpura: Rare.

C. **Major GI bleeding**

Per rectum
Usually brick-red
↓
1. – Painless
± Cramping (colicky) periumbilical pain
± Several similar previous episodes
⇓
Bleeding from a **Meckel's diverticulum.**
Confirmation of diagnosis: ^{99m}Tc Na pertechnetate scan

(Usually) Melena + hematemesis
Associated with manifestations of portal HT + (chronic) liver disease
⇓
Esophagogastric varices

Otherwise
⇓
Peptic ulcer
Rare in childhood

2.– Painless bleeding per rectum, usually in children below the age of 10 years
– SCOPA ±; the child's mother might have noticed it.
– O/E: Usually pedunculated bright red/cherry red smooth swelling which often tends to slip away from the examining finger
⇓
Juvenile hamartomatous polyp/s

3. Tubular duplication

GENERAL REMARKS

- A rough guide for assessment of blood loss: A volume of blood lost up to 20 mL is considered as a 'minor' bleed, and > 200 mL is considered as a 'major' bleed.
- Anal fissures in children generally heal quickly in contrast to those in adults.
- Investigation is generally warranted only if bleeding is persistent/severe.

PRINCIPLES AND PITFALLS

- Bleeding may have stopped by the time a child presents.
- Major bleeding per rectum can occur from the upper GIT (esopohagus/stomach/duodenum) *without* hematemesis.
- Make sure that the newborn baby has received injection Vitamin K.
- Do not wait for appearance of rectal bleeding before making a diagnosis of strangulated intestinal obstruction.

www.Ebook777.com

80 JAUNDICE / CHOLELITHIASIS / PORTAL HYPERTENSION

JAUNDICE

An unconjugated bilirubin level > 300 μmol/L may damage the brain—kernicterus.
Dark urine (due to urobilinogen), pale gray-colored stools, and essentially direct hyperbilirubinemia suggest cholestatic jaundice.

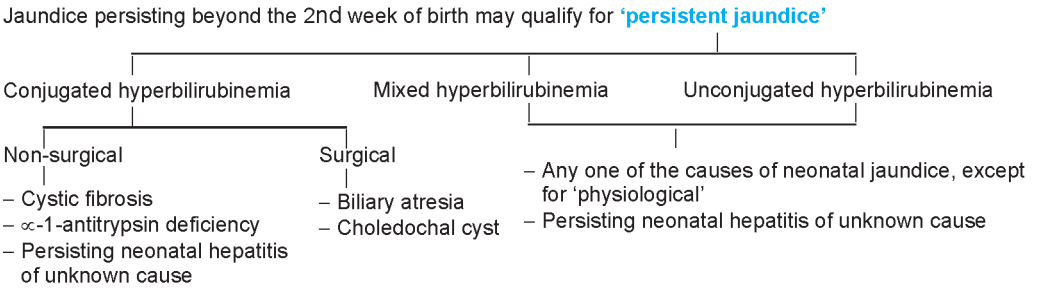
Causes of cholestatic jaundice

- **Surgical**
  - Persistent jaundice ⇒ biliary atresia
  - Intermittent jaundice and Pain ⇒ choledochal cyst
- **Non-surgical** (Severe hepatic damage associated with cholestasis)
  - **Conjugated hyperbilirubinemia**
    - Cystic fibrosis
    - ∝-1-antitrypsin deficiency
    - Persisting neonatal hepatitis because of unknown cause
  - **Mixed hyperbilirubinemia**
    - Septicemia
    - Viral infections
    - Inborn errors of metabolism, e.g. galactosemia
    - Persisting neonatal hepatitis because of unknown cause

Causes of neonatal jaundice

Day of onset	*Possible causes*	*Clinical remarks*
1	– Hemolysis – Congenital infection	- -
2–3	– Immature enzymes (Physiological) – Sepsis	Premature but well baby. Sick baby
7–8	– Hypothyroidism – Galactosemia – Breast milk jaundice – ∝-1-antitrypsin deficiency – Cystic fibrosis – Biliary atresia – Persisting hepatitis	- - Breastfed otherwise healthy baby - Delayed meconium Well baby Well baby

Free ebooks ==> www.Ebook777.com



- It may be difficult to differentiate between biliary atresia and persisting neonatal hepatitis. Doubt may persist even following investigation
- **Biliary atresia** must be excluded if jaundice persists beyond 1 month after birth. Operative correction of biliary atresia delayed beyond 10 weeks, worsens the prognosis.

Choledochal Cyst

Clinical features:

– Pain, vomiting
– 'Persisting' cholestatic jaundice
– A mass in the RUQ—This may become palpable or accentuated and tenderer during the episodes of ascending cholangitis.

CHOLELITHIASIS

- Uncommon in childhood.
- **Causes** *of cholelithiasis in early childhood*: Almost always because of hemolytic disorder, e.g. spherocytosis, thalassemia, etc.
- Cholelithiasis in *late childhood* is considered as an earlier presentation similar to that of adulthood cholelithiasis.

PORTAL HYPERTENSION

Refer to chapter 38.

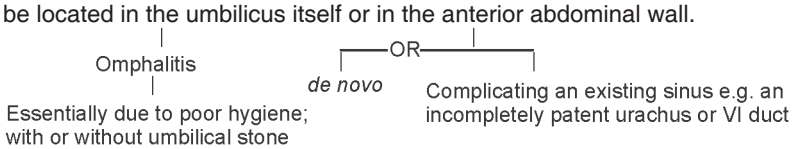
A clinical point applicable in children: Systemic signs of liver failure are uncommon in children with hepatic cirrhosis, although minor abnormalities like spider nevi may be seen.

www.Ebook777.com

81 DISCHARGE FROM THE UMBILICUS

GENERAL REMARKS

1. *Causes of discharge from the umbilicus*
   - Infection of the stump of the umbilical cord.
   - Omphalitis = infection of the umbilicus.
   - Umbilical bud (*synonyons* polyp, adenoma, enteroteratoma, raspberry tumor).
   - Umbilical granuloma.
   - Fecal fistula, urinary fistula.
2. *Acute-on-chronic sepsis* may be located in the umbilicus itself or in the anterior abdominal wall.



HISTORY

Mode of Onset

- Sudden OR Insidious?
  If sudden, is there a h/s/o acute pyogenic abscess preceding the discharge?
  Possible causes of discharge with sudden mode of onset:
  - Umbilical sepsis *per se (omphalitis)*.
  - Anterior abdominal wall sepsis.
  - Secondary infection in a sinus like an incompletely patent urachus/VI duct. (A deep tender swelling in the midline inferior to the umbilicus suggests abscess in the extraperitoneal fat—usually due to an infected urachal remnant.)
- **Progress, Regress**?
- **Smell of the discharge**?
  - General foul-smelling ⇒ sepsis.
  - Fecal ⇒ fecal fistula.
  - Urinary ⇒ urinary fistula.
- **(Additional) symptoms s/o aetiology**
  - H/s/o bladder outflow obstruction?
  - Recent past h/o any operation upon the GUT?
- **Treatment taken** (medications/any other), and response to it?
- **Past history**
  Is this the first episode or recurrent episode? Details of previous episode(s), if any.
- Hypersensitivity?

LOCOREGINAL EXAMINATION

INSPECTION

- **Bud**
- **Granuloma**

Free ebooks ==> www.Ebook777.com

- Inspect the **skin surrounding the umbilicus**
  - Sodden, because of serous / seropurulant discharge.
  - Excoriated ⇒ fecal fistula.
- Inspect the **abdomen in general, as usual.**

PALPATION

- **Temperature**.
- **Tenderness**.
- **Pressure on the surrounding area to reproduce discharge**.

a. Smell of the discharge

b. Nature of the discharge:
  - Mucus ⇒ could be due to patent VI duct.
  - Biliary ⇒ biliary fistula.
  - Serous—in umbilical granuloma.
  - Serosanguinous—in umbilical granuloma / umbilical stone.

- **Part the edges to look for** hair / stone/s (often black colored).
- Palpate the **tract**.
- Palpate the **rest of the abdomen**.

  Subumbilical (depth wise) lump may be due to an enterocyst or urachal cyst.
- **Gently probe the tract**.

DIFFERENTIAL DIAGNOSIS

Infection of the stump of the umbilical cord

Treatment:

- Systemic antibiotic therapy.
- Warm moist dressings, which help to separate the crusts, giving exit to pus.
- Local application of a silver nitrate stick to exuberant granulations, if any.

Umbilical sepsis (Omphalitis)

Infection in the granulations beneath the stump of the umbilical cord in the neonatal period.

Clinical features of minor infection: Cellulitis of the umbilicus. Seropurulent discharge. Good response to local and systemic antibiotics.

Quite often there is little local cellulitis even when infection is established and has spread farther as septicemia.

Preventive measures: Keep the stump dry and dress with an antiseptic.

Complications of infection of the stump of the umbilical cord or omphalitis

1. *Abscess of the abdominal wall*.

   On exerting pressure above and below the umbilicus, the appearance of a bead of pus at the umbilicus ⇒ a deep abscess associated with one of the defunct umbilical vessels.
2. *Extensive necrotizing cellulitis of the abdominal wall*.
3. *Septicemia*.
4. *Jaundice*—because of stenosing intrahepatic cholangiolitis. Jaundice appears some 3–6 weeks after birth.
5. *Portal vein thrombosis* and subsequent portal HT. Clinically overt infections in the portal vein and the IVC are very rare; it is the retrospective event to be considered in an established c/o portal HT.
6. *Peritonitis*.
7. *Umbilical hernia*.

www.Ebook777.com

Umbilical bud (*Synonyms* Umbilical **adenoma/polyp/raspberry tumor/enteroteratoma**)

- This is seen in infants. Only occasionally occurs later in life.
- Situated in the depths of the umbilical cicatrix, and may be covered with crusts.
- ~ 1 × 1 cm.
- Pinkish or bright red, shiny, spherical, sessile or with a small pedicle.
- Soft. moist with mucus (mucus discharge), and tends to bleed on touch.



Fig 81-1 Umbilical bud

Pathologic anatomy

Pouting intestinal mucosa of partially (occasionally completely) patent vitellointestinal duct. Mucosa prolapsing through the umbilicus → a raspberry-like swelling.

Umbilical granuloma

This occurs because of chronic infection of umbilical cicatrix.

Chronic inflammation → exuberant proliferating granulation tissue (a rounded swelling with cherry-red granulating raw surface), which in turn may lead to serous, seropurulent or sanguinous discharge.

Sometimes, differentiation between an umbilical bud and an umbilical granuloma may not be possible. Usually, an umbilical granuloma can be destroyed by one application of a silver nitrate stick followed by dry dressings. Recurrence after 'adequate (maybe at the most 2 or 3 applications) treatment for a granuloma' means it is polyp.

Umbilical fecal fistula in newborn and infants

- This occurs due to completely patent VI duct.
- 'Small' amount of discharge ⇒ a narrow fistula—most likely to close spontaneously.
- 'Larger' amount of discharge ⇒ a wide fistula / a fistula which may be associated with atresia of the intestine distally.

Urachal fistula

Suspect this when there is urinary discharge from the umbilicus in a patient with e/o bladder outflow obstruction. Refer to chapter 43.

Umbilical stone

Often black in color. Composed of desquamated epithelium which becomes inspissated and collects in the deep recess of the umbilicus.

Eventually → infection-cellulitis, and a blood-stained discharge.

Dermatitis of and around the umbilicus

Fungal and parasitic infestations are more difficult to eradicate from the umbilicus than from the skin of the abdomen.

Sometimes, the dermatitis is consequent upon a discharge from the umbilicus.

In fat patients, intertrigo occurs.

PLANNING OF MANAGEMENT—Refer to chapter 44.

Free ebooks ==> www.Ebook777.com

82 ANORECTAL MALFORMATION

GENERAL REMARKS

- Most malformations of the anus/rectum communicate by a fistulous track with either the urinary or genital system, or actually open onto the skin of the perineum.
- The identification of a fistulous opening in the perineum indicates that there is an anal anomaly, and the prognosis is good.
- Meconium in the urine indicates the need for a preliminary colostomy, for all rectourinary communications are supralevator.
- Noncommunicating anomalies in girls are so rare, that a fistula should be expected and a thorough search made.
- The basis of classification of anorectal malformation is in the relation of the end of the bowel (the proximal end of the arrested development) to the chief muscle of continence viz. the puborectalis component of levator ani.

Classification

Arrested development at/above the puborectalis sling	Arrested development below the puborectalis sling
Supralevator anomaly—Associated with rectal deformities	Translevator anomaly—Associated with anal deformities
Arrested development at the puborectalis sling	Arrested development above the puborectalis sling
'Intermediate' supralevator anomaly	'High' supralevator anomaly

The exact nature of a noncommunicating anomaly cannot be determined by clinical examination alone. X-rays are essential, and their interpretation depends upon the presence of gas in the bowel.
The differences between the supralevator anomaly (rectal deformity) and translevator anomaly (anal deformity) are vital from many points of view.

MODES OF PRESENTATION

- Intestinal obstruction.
- In girls, passage of meconium/feces through the genital orifice.
- In boys, appearance of meconium in the urine.

www.Ebook777.com

PHYSICAL EXAMINATION AND ARRIVING AT THE DIAGNOSIS

In boys, examine the perineum to find an orifice

Present ⇓
The following possibilities
1. Normal anus at normal position
2. Otherwise normal anus but placed in an anterior position: *Anterior perineal anus*
3. A fistula opening onto the perineal skin anterior to the anus: *Anocutaneous fistula*

Absent ↓
Examine urine for the presence of meconium

Meconium absent ⇓
Noncommunicating supralevator anomaly ↓
Obtain an invertogram

Meconium present ⇓
Supralevator rectourinary fistula ↓
Obtain an invertogram and MCU

A preliminary colostomy is indicated anyway

4. A small tight orifice at the normal site: Stenosis of the anal membrane / anal stenosis due to covered anus / anorectal stenosis

In girls: Is some meconium passed?

Yes ↓
Examine the perineum and orifices
Examine the vagina carefully for additional (fistulous) orifice
Conclusion particularly with relation to the total number of orifices found (inclusive of normal and abnormal (i.e. fistulous))
- 1 orifice ⇒ rectocloacal fistula } A preliminary colostomy is indicated
- 2 orifices ⇒ rectovaginal fistula }
- 3 orifices ⇒ anovestibular fistula / rectovestibular fistula / anterior anus (perineal or vulvar) / a variety of covered anus (anocutaneous or anovulvar fistula)

No ⇓
Noncommunicating (most probably supralevator) anomaly OR a supralevator rectourinary fistula (The incidence of both is very rare) ↓
Obtain an invertogram
A preliminary colostomy is indicated anyway

Examine the nerve supply to the bladder, bowel and perineal muscles, especially when X-ray shows an abnormal sacrum.
- Is the perineal sensation present or absent?
- Is the bladder expressible?

When perineal sensation is absent and when the bladder is expressible, fecal incontinence is certain, even if rectoplasty is performed, for the levator ani is paralyzed.

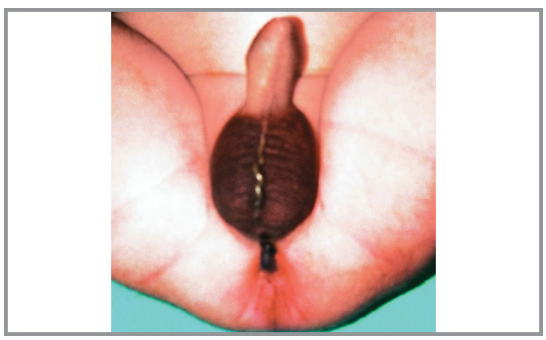


Fig 82-1 Anocutaneous fistula



Fig 82-2 Three orifices

Free ebooks ==> www.Ebook777.com

83 URINARY TRACT INFECTION (UTI) / URINARY STONES

URINARY TRACT INFECTION

Clinical features of UTI are often minimal/misleading due to which, UTI may remain undiagnosed until renal failure occurs in early life.
Frequency, scalding and wetting are common symptoms in children. Clinicians frequently tend to misdiagnose these symptoms for UTI, especially when supported by the presence of pus and bacteria in urine report. When lower urinary tract symptoms (particularly irritative symptoms) are marked and prominent, real infection is documented in only few, most turning out to be examples of 'sham syndrome' (symptoms possibly of neural origin, e.g. frequency, urgency, urgency incontinence, scalding, hesitancy).

Modes of presentation in infants and toddlers are nonspecific:
Malaise, irritability, fever, poor feeding, vomiting, diarrhea, failure to thrive, unexplained anemia.

Modes of presentation in older children:
Nonspecific manifestations (stated above) still predominate, especially fever. However, dysuria and frequency may occasionally occur.
Since the symptoms do not clarify the diagnosis of UTI and often confuse it, **it is imperative to examine the reliable, uncontaminated specimens of urine**.

Causes of UTI

1. Functional stasis:
   - Reversal of flow, e.g. vesicouretric reflux—The most common cause in childhood.
   - Inadequate emptying, e.g. neurogenic bladder, or a megaureter with poor peristalsis.
2. Structural (organic) obstruction causing stasis due to wide variety of causes.

In girls, infection often occurs without any demonstrable underlying abnormality, and is almost always confined to the bladder (cystitis), though occasionally it is found in kidney/s (pyelonephritis).

Indication for investigation
A complete urological investigation should be carried out on all children with a proven UTI regardless of age and gender.

URINARY STONES

In half of the cases of urinary stones, some or the other underlying abnormalities can be identified, but in the remaining, no recognizable cause can be demonstrated.

Modes of clinical presentation

- UTI
- (Increased) Frequency, urgency
  - Pains
  - Strangury
  - Passage of sludge / gravel / stone per urethra
  - Hematuria
  - Retention of urine.

www.Ebook777.com

Causes of the formation of stones:

1. *Stasis and infection*
   Strangely, in the commonest cause of urinary stasis in children viz. vesicoureteric reflux, stones are rarely formed.
2. *Metabolic* causes
   Cystinuria, xanthinuria, hyperparathyroidism, hyperchloremic acidosis (renal acidosis), idiopathic hypercalciuria. As well as discrete stones, calcium may be deposited in renal papillae (nephrocalcinosis).
3. *Idiopathic*
4. *Foreign bodies*
5. *Endemic*
   Endemic stones may be dietary rather than racial.

Free ebooks ==> www.Ebook777.com

84 DISORDERS OF THE LOWER URINARY TRACT

GENERAL REMARKS

Abnormalities of the lower urinary tract result primarily in obstruction to the flow of and/or stasis of urine. Congenital abnormalities in the lower urinary tract are the most common, but they may present after months or years.

CLINICAL FEATURES

Micturition

– A stream that is weak, fine and prolonged
– Intermittent or continuous dribbling

} Lower urinary tract obstruction.

Sometimes accompanied by obvious straining and discomfort
The normal stream is strong, wide and of short duration.

Wetting

Does it appear to be normal intermittent wetting, or abnormal continuous wetting? A distinction between the two is important.

Palpation of the bladder

In *acute retention* the bladder is small but overdistended, painful and tender, and difficult to feel because of guarding.
In *chronic retention* the bladder is readily palpable, nontender and firm.
Manual compression may produce a free flow of urine (expressible bladder)—most likely because of a neural defect.

Digital rectum examination (DRE) will determine whether there is an overloaded rectum or a tumor or cyst in the pelvis, any of which may press upon and obstruct the urethra.

Acute retention of urine

Causes:

1. Painful micturition
   The child withholds the urine as long as possible because of pain. An example—meatal ulcer.
2. Acute organic obstruction
   Example—a stone or a pedunculated vesical polyp impacting in the bladder neck / urethra.

Chronic retention of urine

Causes

1. Meatal stenosis.
2. Posterior urethral valves.
3. Neural defects, e.g. meningomyelocele.
4. Unusual causes—membranous stricture, diverticula, ureterocele, tumor, etc.

www.Ebook777.com

85 ACUTE SCROTUM

Acute scrotum is **defined** as the scrotum with acute pain, and swelling or other signs of acute inflammation.

GENERAL REMARKS

1. Symptoms of an acute scrotum are similar regardless of the Etiology.
2. **Causes** of acute scrotum in the descending order of frequency of their occurrence: torsion of a testicular/epididymal appendage, torsion of the testis, idiopathic or allergic scrotal edema, epididymitis/epididymo-orchitis.
3. *Torsion of the testis* is the most important cause, but is less common than torsion of testicular appendage.
   - Torsion of a testicle is a surgical emergency; if the blood supply is not restored within 12 hours, the testis infarcts and must then be excised.
   - In case of torsion of an incompletely descended testicle, signs will be present in the groin, and then it needs to be differentiated from strangulated hernia.
4. Mumps virus has little affinity for the testis until after sexual maturation.
5. An accurate clinical diagnosis should always be attempted at, as urgent surgical exploration is essential in testicular torsion whereas unnecessary surgery can be avoided in unindicated cases (definite clinical diagnosis of torsion of testicular / epididymal appendage, acute scrotal edema and epididymitis / epididymo-orchitis).

SYMPTOMS AND THEIR INTERPRETATION

IN INFANTS

Symptoms may be nonspecific, e.g. excessive crying and vomiting. Fever ±.
Mother notices a red swollen scrotum.

IN TODDLERS

The first symptom may be—Refusal to walk or an abnormal gait with the thighs abducted and the feet far apart.

IN OLDER BOYS, PARTICULARLY ADOLESCENTS

- Of late, did you suffer from **a few attacks of self-resolving pain** in the testicle / groin / lower abdomen / loin?
- Was the swelling preceded immediately by **straining,** e.g. at stools / lifting heavy weight / sexual activity, etc? There may be **vague pain in the lower quadrant** of the abdomen **or in the loin**—it is the referred pain.

IN GENERAL

- Acute pain in the scrotum may cause **vomiting**.
- **A significant reduction in pain which was excruciating in the beginning**, especially when associated with continued presence of significant signs may be indicative of established infarction of the testis.
- **Where did the edematous swelling begin**?
  If the edematous swelling begins in the foreskin of the penis, and then rapidly extends on to the scrotum, perineum and thigh, then it could be because of allergic or idiopathic acute scrotal edema.
- If there is a definite h/o **a bite/sting by an insect**, then most probably that may be the cause of (allergic) acute scrotal edema.
- **There may be a h/o injury** to the scrotum, but characteristically this is minor and vague. It represents an attempt to relate the onset of symptoms to the traumatic event, when in reality the minor injury merely draws attention to the already abnormal scrotum.
- Is the patient **known to have a significant urinary tract malformation**?

Free ebooks ==> www.Ebook777.com

- **Any procedure upon the urinary tract,** e.g. dilatation of stricture of urethra / I and D of a periurethral abscess, etc?
- **Recent episode of UTI**?:
  - Increased frequency of micturition.
  - Pain while passing urine.
  - Passage of red / brown-colored or smoky urine.
- Did you recently undergo a **urethral instrumentation,** e.g. catheterization / dilatation of a stricture, etc?
- H/s/o **poor flow**? Poor flow suggests bladder outflow obstruction.
  - Intermittency.
  - Reduced caliber of stream.
  - Inability to pass urine without having to strain (by contracting abdominal muscles).

SIGNS

- The scrotal skin is usually *edematous and pink* rather than deep red.
- Tenderness also is present although its degree varies according to the cause.

ARRIVING AT THE DIAGNOSIS

Principles and Pitfalls

- Possibility of torsion of testis should not be ruled out even if pain is mild / moderate rather than severe and mode of onset relatively less sudden rather than 'explosively sudden' as typically described / expected.
- Scrotal edema, acute hydrocele, and exquisite tenderness if present, often make precise palpation difficult or impossible.
- Unless there is a clear-cut h/s/o a predisposing cause, the diagnosis of epididymitis/epididymo-orchitis, is always debatable.
- As a general rule, a confident clinical diagnosis can only be made in cases of allergic / idiopathic scrotal edema.

Two factors to be considered for arriving at the diagnosis: 1. Age, and 2. Whether the signs of acute inflammation are present or absent in the scrotum.

Age	*A preliminary guess of a probable diagnosis*
Neonate	Torsion of the testis (Extravaginal torsion).
Infant	Epididymitis/epididymo-orchitis provided a specific predisposing circumstance is present.
Puberty	Torsion of the testis (Intravaginal torsion).

When edema and acute cellulitis in the scrotum are minimal or absent, look for signs in the groin

Signs present in the groin?
⇓
– ? Strangulated groin hernia
– ? Torsion of an incompletely descended testis.

Signs absent in the groin?
⇓
Possibility of torsion of a testicular / epididymal appendage

Visible blue-black spot near the upper pole of testis seen through the scrotal skin
⇓
Torsion of testicular appendage, the torsion having produced bleeding in the pedunculated cyst. This is the pathognomonic sign of twisted testicular appendage
|
The presence of exquisite tenderness in the blue-black swelling of the size of a pea with the rest of the testis not being tender, additionally confirms the diagnosis in favor of torsion of testicular appendage

In the absence of the pathognomonic sign of twisted testicular appendage
↓
Gently support the testis by one hand and make an attempt to palpate a tender swelling at the upper pole of the testis
|
The presence of an exquisitely tender swelling of the size of a pea, when the rest of the testis is not tender
⇓
Torsion of testicular / epididymal appendage (without bleeding)*

* When torsion of a testicular/epididymal appendage occurs leading to its (i.e. that of the appendix) infarction without bleeding in it, the diagnosis may be missed. Even at surgical exploration, a pale infarcted cyst looks trivial, and many cases of apparent epididymo-orchitis are probably examples of bloodless infarction of testicular / epididymal appendage.

www.Ebook777.com

When signs of acute inflammatory swelling or edema are present in the scrotum, there are following five possibilities.

1. **The testicle lying at a higher level** than the opposite (normal) side, and also may be lying obliquely, and exquisitely tender ⇒ torsion of the testicle. Testis lies high in the scrotum due to shortening of the cord.
   A pitfall: Edema of the skin of the lower part of the scrotum can occur in epididymo-orchitis or pyocele, and that may make the testicle appear at a higher level than the opposite (normal) side, but in these cases, the testicle touches the bottom of the scrotum, while in torsion of the testicle, there may be a gap between the lower pole of the testis and the bottom of the scrotum.
   And, if the contralateral testis is oriented in a loose hanging horizontal position like a bell-clapper, it gives an additional supportive evidence in favor of torsion of the testis*.
2. – Constitutional symptoms.
   – Associated funiculitis.
   – **The presence of a specific predisposing circumstance,** e.g. the presence of a known urinary tract anomaly / a proven UTI / recent urethral instrumentation.

   } ⇒ epididymitis or epididymo-orchitis.

   Epididymitis alone is more common.
   True acute orchitis is not common, but may occur in mumps or septicemia. The testis is larger and harder than in epididymo-orchitis.
3. **Extent** of the swelling—all over the scrotum, penis (including the foreskin), perineum, thigh and the groin in continuity. May extend even on to the opposite side.
   – Edema and cellulitis (pink/red) are superficial, having the appearance of allergic wheal.
   – Testis—not so tender, and is normal in size.
   – The testicle can be displaced gently out of the scrotum into superficial inguinal pouch, where its normal size and consistency can be determined. This maneuver is relatively nontender.

   } ⇒ allergic or idiopathic acute scrotal edema.
4. – A swathe of slightly reddened skin and subcutaneous tissue which extends beside or across one half of the scrotum.
   – An inconspicuous (streptococcal) pustule in the perineum.
   – Acute inguinal lymphadenitis.

   } ⇒ perineal lymphangitis.
5. Development of the following features in a toddler who sits suddenly on a small toy with a sharp edge may → a small breach in the posterior urethra leading to superficial extravasation of urine
   – Pain on micturition.
   – Progressive edema of the perineum, scrotum and suprapubic region.

   } ⇒ (latent) rupture of the urethra.

*The predisposing deformity like a 'bell-clapper deformity' is often bilateral. This fact has 2 implications – (a) The diagnostic implication: In c/o a unilateral acute scrotum, it helps as an important supportive evidence to make the diagnosis of torsion of testis on the affected side. And (b) The side unaffected by torsion must be opened for prophylactic orchidopexy at the same time as urgent surgical exploration for the affected side.

Free ebooks ==> www.Ebook777.com

86 DISORDERS OF THE EXTERNAL GENITALIA

FEMALE GENITALIA

Developmental anomalies are rare in girls.

Labial Adhesions

Quite a common condition.

Labia minora are adherent. The delicate midline adhesion which partially closes the introitus, covers the opening of the urethra, and may extend as far anteriorly as the clitoris. This may lead to discomfort on micturition, but is more often discovered on routine examination.



Fig 86-1 Labial adhesions

Imperforate hymen (Mucocolpos)

In newly born girls the mucocolpos bulges outwards between the labia minora.

Vaginal discharge

Chief complaint: Vulval irritation / discharge.

A very profuse offensive or blood-stained discharge suggests the presence of a foreign body.

Common causes of nonoffensive yellowish discharge: maternal hormonal influence (self-limiting, physiological), obesity, vaginal infections, foreign material (woolen underpants) / frictional dermatitis.

MALE GENITALIA

Common problems in children: Phimosis, paraphimosis, balanitis, meatal stenosis, hypospadias, epispadias.

Refer to chapter 54.

www.Ebook777.com

87 ACUTE PAIN IN THE LIMB (Acute Limb)

Anatomical plane	**Causes:** *Etiopathology*	*Clue/s / remarks*
1. Epifascial soft tissue	– Post-traumatic, e.g. contusion, hematoma	Obvious h/o trauma usually available
	– Snake bite, bites by other land / sea animals / insects	Obvious history may be available
	– Infective cellulitis	Tenderness in the soft tissues rather than overlying the bone
	– Superficial thrombophlebitis – Acute lymphangitis	May or may not be associated with edema
2. Deep fascia	Fasciitis	Rare
3. Subfascial soft tissue	– Myositis – Pyomyositis	Tenderness in the soft tissues rather than overlying the bone
	– Acute DVT	
	– Compartment syndrome	
	– Acute ischemia	
	– Poliomyelitis	No swelling. No bony tenderness
4. Bone	– Acute osteomyelitis	Signs of inflammation hidden in early stage
	– Spiral # of tibia (often hairline)	In toddlers, it is often unrecognized as h/o trauma may not be available No signs of systemic infection. Local tenderness. Callus on an X-ray
	– Malignant tumor	Much swelling, but not much fever
	– Acute subperiosteal hematoma due to scurvy	– Bleeding swollen gums – Marked anemia
	– Infarction of bone in sickle-cell disease	– Multiple bones involved – Unusual bones involved like skull, small bones of hands / feet
5. Joint	– Septic arthritis	– Signs of inflammation hidden in early stage – Maximal tenderness overlying a joint, not overlying the adjacent bone – All the movements of the joint are exquisitely tender
	– Generalized arthritis	– Multiple joints involved – Various causes can be distinguished by associated features – Viral arthritis, scurvy, rheumatic fever, juvenile rheumatoid arthritis, H-S purpura
	– Rheumatic fever	– Pain flits from joint to joint – Tenderness at the joint line and may extend both above and below the joint

Free ebooks ==> www.Ebook777.com

DIFFERENTIAL DIAGNOSIS

Acute pyomyositis: Refer to chapter 15.
Acute DVT: Refer to chapter 50.
Acute compartment syndrome: Refer to chapter 50.
Acute ischemia: Refer to chapter 46.

Acute osteomyelitis
For detailed differential diagnosis, refer to chapter 15.

Principles and pitfalls *in arriving at the diagnosis of acute osteomyelitis*:
- If neighboring joints have effusion and restriction of movements, especially in c/o shoulder and hip, it may be difficult to decide the primary pathology - ? arthritis/? osteomyelitis.
- X-rays may be normal × 1st 10 days, because the pathological changes that are seen on X-ray take at least 10 days to develop.
- Bone scan is positive at an early stage.
- Diagnosis in neonates and infants may be delayed due to the following problems:

Problem	*Explanation*
1. Fever—variable	Temperature is more labile in neonates
2. Loco-regional symptoms and signs—less marked	Periosteum is less tightly fixed to the bone - therefore, more easily elevated by edema / pus → therefore, less pain
3. Edema—greater and more diffuse	Relatively loose periosteum does not restrict the spread of inflammation

Neonatal osteomyelitis has 2 clinical forms:
1. A life-threatening septicemia. Multiple sites are often involved.
2. The more common, a misleadingly benign form due to virtual absence of constitutional features and minimal local signs. A typical example is a baby who cries when moved, e.g. when the napkins are changed. On careful examination, tender hip ± a mass due to an underlying abscess is detected. X-rays commonly show extensive bone damage.

Diagnosis in older children (over 10) also may be delayed for the following reasons:
1. Systemic manifestations may be less obvious, as infection is localized more effectively.
2. May be misdiagnosed initially as sprain.

Unusual Infections:
1. *Pseudomonas pyocyaneus* → infections in children who have been treated with antibiotics for long periods (for various unrelated diseases), and can be very troublesome.
2. *Salmonella* infections. Resolution is usually complete but may be slow.

Acute septic arthritis
Refer to chapter 15.

www.Ebook777.com

88 DISORDERS OF THE MOUTH

Tongue-tie

In neonates the lingual frenum often appears short and attached to the very tip of the tongue. The appearance changes to complete normalcy in the course of a couple of years. Such a frenum does not interfere with suckling. Anxious mothers need to be reassured.

Clinical method in an older child and inference: Ask the child to protrude the tongue.

If it is possible to protrude the tongue over the lower incisor × 1–2 cm	If protrusion is not possible; instead the tongue becomes deeply indented in the midline and elevated at its lateral margins
⇓	⇓
The diagnosis of tongue-tie is refuted	The diagnosis of tongue-tie confirmed, and treatment is indicated



Fig 88-1 Tongue-tie

Lingual frenal ulcer

Predisposing causes:

- Protracted bouts of coughing as for example in pertussis.
- Bottle- or breastfed children with premature eruption of lower incisors.

A red-colored hemispherical firm swelling ~ 5 mm in diameter, with a tiny transverse ulcer across its equator. Heals completely when the underlying cause is taken care of.

Hypertrophic labial frenum

A large thick frenum extending between the upper central incisors, which stand apart.

Clinical method: Pull the upper lip upwards and forwards and observe the interdental papilla.

If the interdental papilla does not blanch	If the interdental papilla blanches
⇓	⇓
The condition may correct itself in the course of time	The condition is not likely to correct itself. Excision of the offending segment of the frenum will be required

Free ebooks ==> www.Ebook777.com

Dental (virtually periodontal) abscess

There are 2 forms

A localized superficial swelling on the alveolar ridge around a carious deciduous tooth

- Relatively painless
- Usually not accompanied by constitutional symptoms
- Often points and drains spontaneously

A deep spreading infection arising in a carious permanent tooth—an alveolar abscess

In children over 6 years of age

- Severe constitutional symptoms
- Locally, there is a brawny cellulitis extending into the soft tissues around the jaw

For differential diagnosis of other infections in the mouth and pharynx, refer to chapter 15.

www.Ebook777.com

89 SPECIFIC REGIONAL SWELLINGS/LESIONS IN THE FACE

The region	**Acute**	**Chronic**
Orbit	Cellulitis	Congenital sequestration dermoid cyst (external angular/internal angular/midline, i.e. median nasal) Meningocele, encephalocele Mucocele of the frontal sinus / lacrimal sac Lacrimal gland swelling
Ear	Cellulitis / abscess because of infection arising at the blind end of a pre-auricular sinus	Preauricular sinus—at the anterior end of the outer helix Skin tag/Accessory pinna—anterior to the tragus - *Skin tag*—a floppy tag with a narrow stalk without any cartilage within; therefore, can be readily removed by ligation of the base - *Accessory pinna*—a wide-based skin tag with cartilage within. It is very important to identify the cartilage. An accessory pinna needs a more formal surgical excision
Parotid	Acute swelling in the parotid	Lymphangioma/Cystic hygroma Tumor

Unilateral

⇓

Intraparotid lymphadenitis (bacterial/viral)

Discrete swelling in the parotid region *cf* a 'J'-shaped enlargement, which is because of enlargement of the entire gland itself

Unilateral, and often recurrent

– Pain and swelling maximum when production of saliva is maximum, i.e. even with a thought of (tasty) food or actually during eating
– Side of the face becomes swollen
A 'J'-shaped swelling around antero-inferior part of the pinna; covering the angle of the mandible
– Acute inflammatory signs not conspicuous due to low-grade infection
– Massaging the gland → a discharge of pustular fluid from opening of the duct of parotid gland

⇓

Sialoadenitis—sialectasis. Also refer to chapter 90

Bilateral

+Symptoms and signs s/o viremia
±Contact with a c/o mumps in the previous 3 weeks

⇓

Mumps
Orchitis / pancreatitis occurs rarely in prepubertal children

CLINICAL PEARLS

- Infections of the face must be diagnosed speedily and treated aggressively to prevent cavernous sinus thrombophlebitis.
- A fluctuant angular dermoid may have an intracranial extension.
- Beware of a 'cyst' over the glabella—It may be an encephalocele!
- In c/o an abscess in front of the helix of the ear, search for a punctum (the blocked opening of a pre-auricular sinus) (May be hidden by hair).
- Unilateral / recurrent parotid swelling is more likely to be due to sialectasis than atypical mumps.
- Mouth must always be examined.

Free ebooks ==> www.Ebook777.com

90 SWELLING IN THE NECK

Nonspecific reactive hyperplasia of lymph nodes
Nonspecific hyperplasia of the cervical nodes is common. Nodes often enlarge during viral infections (affecting the nose and throat) and subside over weeks-months.
A lymph node up to 3 cm in longest diameter is 'normal' in children under 7 years of age.
Differentiating points: Non-tender, discrete, firm ('shotty'), mobile, and 0.5–1 cm in diameter. The jugulo-diagastric node is usually the largest.

Acute lymphadenitis
A brawny edema obscures the underlying enlarged node, and there is *often nothing remarkable* in the oropharynx. Most of such nodes respond to antibiotics. Development of fluctuation is an indication for I and D.

Thyroid swelling/disorder
Thyrotoxicosis is uncommon in childhood. Irritability, loss of weight and tremor are more common presenting features than a swelling in the neck.
Adenoma and carcinoma are rare, but these possibilities should be kept in mind when a patient presents with a solitary / dominant thyroid nodule.

The submandibular salivary gland
The most common cause of enlargement of the gland is a stone, often a small one in the submandibular duct.

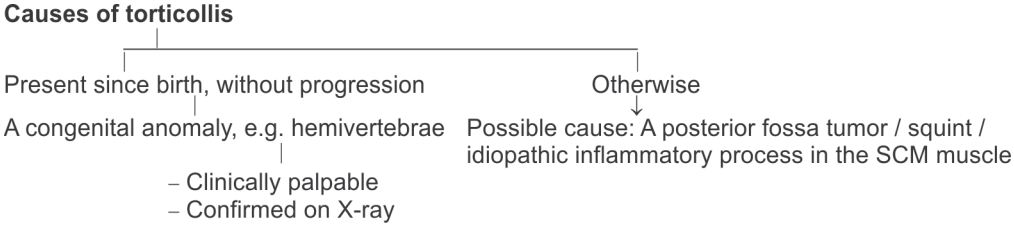
Recurrent subacute parotitis of childhood (associated with sialectasis)
- 3 months—10 years.
- Pain and constitutional symptoms are mild.
- H/o one or more previous episodes affecting either gland, each lasting 1–2 weeks ±.
- The first attack may have been mistaken for mumps.
- Purulent fluid issues from the orifice on compressing the duct.

Sialogram shows no obstruction, but a snowstorm of sacculations 2–4 mm in diameter along the radicles of the gland. Spontaneous remission occurs by 10–15 years of age.

www.Ebook777.com

91 TORTICOLLIS (WRYNECK)

Causes of torticollis



Posterior fossa tumor
– Occasionally, acute torticollis is the 1st presentation.
– Neck is 'frozen' and difficult to move.

The tumor compresses the brainstem at the foramen magnum leading to acute stiffness of the neck.

Squint

Squint may not be obvious at first. Passive straightening of the head may make the squint appear.

Sternocleidomastoid (SCM) torticollis

It results from tightness of one SCM muscle following an idiopathic inflammatory process in the perinatal period. The inflammatory process in the muscle may lead to a swelling though without obvious signs of inflammation, or may not lead to a swelling.

Two components must be carefully examined: The primary deformity and the secondary deformities.

Findings on clinical examination of the primary deformity

On inspection

- Obvious rotation of the head to contralateral side and lateral flexion (tilting) on the ipsilateral side. In a tiny baby bundled up in a rug, tilting of the neck may be less obvious.
- A fusiform swelling in lateral side of the neck without obvious signs of inflammation.
- In older children, anterior border of the SCM stands out as a tight band; it cannot be readily seen in infants as the neck is short.

On palpation

- In a neonate/infant, a fusiform swelling in the SCM, involving the whole or a part of the muscle may be palpable.
- In older children, when the swelling has disappeared, the anterior border of the SCM standing out as a tight band may be palpable or an indurated area of fibrosis (the aftermath of inflammation) may be palpable.

Clinical method to assess limitation of rotational movement of the head due to shortened SCM

Stand behind the child. Turn (Rotate) the head to each shoulder.
Normal range of rotation: 90^{O} each side.
Limited degree of rotation suggests affection of the ipsilateral SCM.

Secondary deformities

1. of the **head**: Flattening of contralateral occiput and ipsilateral forehead.
2. of the **face**: Hemihypoplasia of the face of the affected side.
3. of the **neck**: Secondary deformity of the neck is evident when the child becomes older enough to walk,—That is the time when a need arises to keep the eyes horizontal. The deformity consists of ipsilateral elevation of the shoulder to compensate for the shortened SCM. Further compensation may be obtained through cervical and thoracic scoliosis.

Free ebooks ==> www.Ebook777.com

92 DISORDERS OF THE BREAST

Abnormalities of the breast

- Congenital
  - Absence of breast
  - Absence of nipple
  - Accessory nipples
  - Accessory breast.
- Breast enlargement in the neonate
  - Neonatal hypertrophy (Neonatal mastitis)
  - Bacterial mastitis/Pyogenic abscess.
- Breast enlargement in small girls
  - Benign premature enlargement
  - Pathological precocious puberty
  - Constitutional precocious puberty (Idiopathic precocious puberty).
- Breast enlargement in boys
  - Pubertal mastitis
  - Gynecomastia.

Complete absence of one breast and nipple

- Rare.
- Usually associated with deficient development of the underlying pectoral muscles, the chest wall and ipsilateral upper limb: Poland's syndrome.

Accessory nipple ± breast

Accessory nipple may occur anywhere along the 'milk-line' which runs from the anterior axillary fold, through the normal nipple position, to the groin (Fig 30-9). Accessory breast and nipple remain unnoticed until puberty.

Neonatal hypertrophy

- This can occur in both sexes. The basis of occurrence is hormonal (maternal estrogen crossing the placenta).
- Enlargement is most obvious in the 1st week, and subsides within the 1st month.
- About 1–2 cm in diameter, i.e. same in diameter as that of the areola. Firm consistency. May secrete milk ('witch's milk').
- *A complication*: Mastitis; may proceed to form abscess. Portal of entry of organisms—through the nipple. The complication particularly occurs with repeated massage and attempts at expression of milk.

Neonatal breast abscess must be diagnosed early and treated early and adequately to prevent damage to the breast.

Breast infection is rare in the neonate. Neonatal breast is susceptible to infection only when it is secreting milk.

Benign premature enlargement in girls

- This may occur in girls between 5 and 10 years of age.
- Unilateral. The contralateral breast may be affected after a variable period (a few days to a year or so).

www.Ebook777.com

- A well-defined enlargement—2 cm in diameter. Posterior to and attached to the nipple. Firm. Usually non-tender. May remain static (until puberty commences) or may regress.
- NOT associated with any sign s/o the onset of puberty (growth spurt, menses, axillary / pubic hair growth, etc.).
- Completely harmless condition.
- **Biopsy is absolutely contraindicated**.

Bilateral hyperplasia because of pathological precocious puberty

- May occur in girls as early as 1 year of age.
- Bilateral hyperplasia of breasts is associated with other features of puberty including menstruation.
- Investigation is directed to detect an underlying cause, e.g. ovarian tumor, intracranial tumor, etc.

Pubertal mastitis in boys

- Minor degree of breast enlargement in boys at puberty.
- Well-defined enlargement beneath the areola. Firm. Pain/tenderness $\pm$.
- One side may be affected before or more than the other.
- Duration – up to 2 years.

DD in obese boys: Apparent breast enlargement because of fat in the region of the breast.

Swelling because of pubertal mastitis	*vs*	*Swelling because of obesity*
Localized to the subareolar region Well-defined and firm Can be 'lifted off' the underlying subcutaneous fat		Diffuse and in continuity with generalized obesity (Striae overlying the lateral aspect of thighs and hips. Apparently small penis because of considerable prepubic pad of fat.)

Free ebooks ==> www.Ebook777.com

93 A BIG HEAD / HYDROCEPHALUS / ASSESSING A VP SHUNT

Record the maximum fronto-occipital circumference. Plot the result on a head circumference chart.
There are 2 patterns suggesting an abnormally big head.

1. A large circumference since birth, which remains relatively bigger than normal or may even increase with age ⇒ Congenital / inherited abnormality.
2. Normal circumference to begin with, later enlarging faster than normal ⇒ Acquired abnormality.

'Relative' size—size of the head compared with the rest of the body and for the given age. **It is the relative size that is more important than absolute size**.

Causes of a big head

- *Common*:
  - Hydrocephalus
  - Cerebral bleeding in the premature baby
  - Spina bifida.
- *Uncommon*: Large brain (macrocephaly), thick skull bones (osteofibromatosis), SDH, cyst, cystic tumor.
- *Other*: Constitutional, benign enlargement of subarachnoid space.

Some pitfalls to be remembered in case-taking and assessment

1. In a c/o raised ICP

If a previous shunt surgery has been performed	If there is a past h/s/o meningitis	None of the previous two
⇓	⇓	⇓
Blocked shunt must be the most probable cause	Strong possibility of post-meningitis hydrocephalus	Brain tumor could be responsible

2. Depression of the LOC is often a late feature.
3. A normal LOC does not necessarily indicate that all is well.
4. In childhood, the vital parameters may remain normal until the ICP is so high that the patient is unconscious and close to death.

HYDROCEPHALUS

Definition: Increased volume of CSF within the ventricles, which are enlarged, or over the surface of the brain. Hydrocephalus represents a diverse group of conditions that result from impaired circulation and/or absorption of CSF or rarely because of increased production of CSF by choroid plexus papilloma.
Hydrocephalus → raised ICP → damage to the brain, loss of white matter.

Natural h/o untreated hydrocephalus:

- Unsightly enlargement of the head, and mental abnormality
- In the most advanced cases, the brain becomes very thin, and there is profound mental retardation.

Hydrocephalus can be **classified** in various ways

A. Obstructive/Communicating, depending on whether CSF can flow from ventricles to the subarachnoid space or not.

www.Ebook777.com

- Hydrocephalus because of obstruction to the flow within the ventricular system leading to dilatation of the ventricles, is termed obstructive (*synonyms* internal, noncommunicating)
- In communicating (*synonym* external) hydrocephalus, the ventricular system is patent but there is reduced flow through basal cisterns or absorption of CSF by the arachnoid villi. The ventricular CSF 'communicates' with the subarachnoid space.

Causes of obstructive hydrocephalus	*Causes of communicating hydrocephalus*
Congenital: Aqueduct stenosis, Dandy-Walker syndrome (atresia of foramina of Magendie and Luschka), Chiari malformation, Vein of Galen aneurysm *Acquired*: – Acquired aqueduct stenosis (adhesion following infection or bleeding) – Supratentorial masses causing tentorial herniation – Intraventricular hematoma – Tumor—ventricular/pineal region/posterior fossa – Abscess/granuloma – Arachnoid cysts	– Thickening of the leptomeninges and/or involvement of the arachnoid granulations because of - infection (pyogenic, TB, fungal) - subarachnoid bleeding - carcinomatous meningitis – Sagittal sinus thrombosis – Increased CSF viscosity, e.g. high protein content – Excessive CSF production from choroid plexus papilloma

B. *Congenital/Acquired/'Compensatory'*: Congenital hydrocephalus usually presents at birth or in early infancy. Compensatory hydrocephalus—Increased volume of CSF secondary to loss of brain tissue, e.g. after head injury, dementia.

C. *Arrested/Progressive*: Arrested hydrocephalus—a steady state of balance achieved between production and absorption of CSF. The child can have a big head, but serial plotting of head circumference on a chart shows that the head now grows on a new centile line. Ventricles may remain large, but ICP remains normal and brain develops relatively normally.

Presentation of hydrocephalus

Before sutures have closed
↓
Cranium expands rapidly
Pressure rise is minor and slow
↓
No signs of raised ICP, but the head becomes big

Associated with obstructive type of hydrocephalus

After sutures have closed
↓
Cranium cannot expand
Pressure rise is rapid and dramatic
↓
Head remains normal sized, but ICP ↑↑
↓
Headache, vomiting, papilledema

Associated with acquired causes of obstructive hydrocephalus, or communicating type of hydrocephalus

The so-called classical manifestations of hydrocephalus occur only in infants, before closure of the sutures.

Age of closure of sutures in different settings:

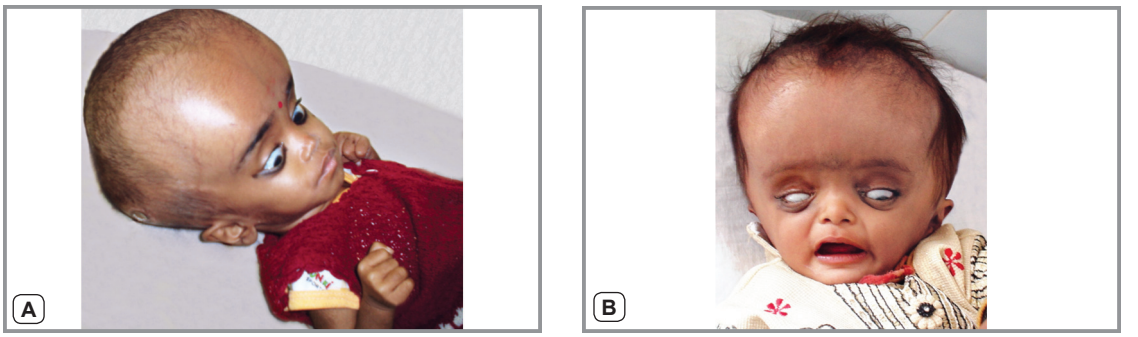
1. Normal, from 1 year onwards.
2. In a child who suffered from hydrocephalus during infancy, who was not treated in time: 4 years.
3. In a child who suffered from hydrocephalus during infancy, who was treated in time: Before 4 years.

The so-called **classical manifestations of hydrocephalus during infancy and young childhood**

Initially, i.e. during the period when increased ICP is being compensated
↓
Symptoms: – Big head
– Mental retardation
– Failure to thrive

Late i.e. with decompensation of raised ICP
↓
– Drowsiness, bradycardia, neck stiffness
– Papilledema

Free ebooks ==> www.Ebook777.com



Figs 93-1A and B Hydrocephalus during infancy / young childhood (Different cases)

Inspection findings:

In general:
- Small face and large head
- Congested superficial veins under an attenuated tight scalp.

From front: 'Setting Sun' appearance. Explanation is not completely understood.

From side:
- Small face
- Forehead overhanging the relatively smaller face
- Frontal 'bossing' because of pushed out dilated anterior horns of lateral ventricles.

Palpation findings:
- Anterior fontanelle—larger than normal and bulging because of raised ICP
- Wide sutures
- Palpable metopic and lambdoid sutures that are not commonly open
- If a shunt has been inserted, then palpate for a shunt superoposterior to the ear, and test for its function.

Transillumination findings with a big bright torch in a dark room:
- In a c/o hydrocephalus, the entire vault is translucent.
- Focal areas of translucency ⇒ subdural hygroma/localized cyst.

Percussion finding over the skull:
Change of resonant note to a dull note over the wide gap in the sutures: the 'cracked pot' sign.

Clinical features of juvenile / adult type hydrocephalus

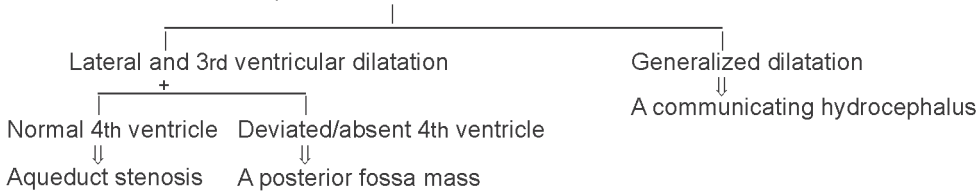
In c/o sudden mode of onset	In c/o insidious mode of onset
↓	↓
– Symptoms and signs of raised ICP – Impaired upward gaze	Dementia, gait ataxia, incontinence This triad of symptoms may occur despite an apparently 'normal' CSF pressure

Key investigations in a c/o hydrocephalus

1. US scan of the brain: Possible when fontanels are still open / sutures are sprung.
   Information obtained: (a) Confirmation of dilatation of ventricles. (b) Possible etiology, e.g. intraventricular bleeding of prematurity, 3rd ventricular cyst, tumor of the 4th ventricle, etc. (c) Thickness of the brain (cortex and white matter overlying the ventricle): More than 2 cm or less than 2 cm?
2. X-ray skull
   - Sutures in babies and young children are split
   - 'Copper beaten' appearance of the skull—because of chronic pressure from the brain on the inside of the skull
   - Erosion of dorsum sellae
   - Calcification in a tumor / tuberculoma.

www.Ebook777.com

3. CT scan/MRI scan:
   - Degree of hydrocephalus?
   - Thickness of the brain: More than 2 cm or less than 2 cm?
   - The pattern of ventricular enlargement—This may help to determine the possible cause.



4. Developmental assessment and psychometric analysis—Detect impaired cerebral function and provide a baseline for future comparison.

Details, which should be mentioned in the diagnosis of hydrocephalus:
- Congenital / acquired
- Obstructive / communicating
- Arrested / progressive. Advanced or othewise
- ICP—rising / rising to the point of decompensation / apparently normal
- Mental age.

Monitoring of progress of hydrocephalus:
- Clinically ± CT scanning/MRI scanning occasionally
- In c/o infants and young children before closure of sutures: Clinically and US scanning of the brain weekly.

Complications of hydrocephalus: Paralytic squint, optic atrophy with blindness.

Principle of treatment: Relieve pressure by bypassing the block to CSF drainage.
In some cases of aqueduct stenosis, this can be done in a minimally invasive way by endoscopic 3rd ventriculostomy. In most cases however, a ventriculoperitoneal shunt will have to be inserted.
An arrested hydrocephalus does not need specific treatment.

ASSESSING A VENTRICULOPERITONEAL SHUNT

There are 3 components of a common shunt:
- Valve (i.e. pump—reservoir)
- Proximal (ventricular) catheter
- Distal (peritoneal) catheter.

Minor obstruction / valve dysfunction may be overcome by manual pumping; this is useful when the child is unwell from a raised ICP.
Shunt malfunction may occur, if either catheter becomes blocked.
- On compression, inability of the pump to empty ⇒ distal obstruction
- Inability of refilling of the pump that has been emptied on compression ⇒ proximal obstruction.

Causes of distal obstruction: Low-grade infection of the catheter → inflammatory exudate to accumulate in the tubing → stimulation of the omentum to wrap around its lower end (May → a pseudocyst formation around the peritoneal end of the catheter).

Causes of proximal obstruction: Occlusion of the holes by inflammatory debris / clots / choroid / brain; less likely because of infection.
In any case, obstruction of the shunt → ↑ in ICP.
Proximal leakage of CSF: Escape of CSF from the ventricle, around the ventricular catheter, especially if this is blocked → formation of a cystic swelling covering the reservoir.

Distal leakage of CSF is more common. Disconnection of the catheter may occur due to traction through movement and growth. Disconnection → partial obstruction to drainage of CSF; but the pump (i.e. the reservoir) feels normal.

Investigation: X-ray skull and abdomen—to look for disconnection / malposition of the shunt.

Free ebooks ==> www.Ebook777.com

94 SPINAL DYSRAPHISM—SPINA BIFIDA

Spinal dysraphism is a developmental defect of the spinal column with failure of fusion between the arches of the vertebrae, with or without protrusion and dysplasia of the spinal cord and its membranes.

GENERAL REMARKS

Types

Occult spina bifida
- A bony deficit – present in 5–10% of the population and not clinically significant
- The overlying skin may be normal or may have a lesion like a tuft of hair, dimple, sinus, vascular malformation or lipoma
- Those who have a lumbosacral cutaneous lesion, have a high incidence of related underlying defects: Split spinal cord syndrome / lipoma / dermoid cyst

Meningocele
- Cystic CSF-filled cavity lined with meninges but devoid of neural tissue
- The cavity communicates with the spinal canal through the bone defect (usually lumbosacral)

Meningomyelocele
- The spinal cord and roots protrude through the bony defect and lie within a cystic cavity, lined with meninges and/or skin
- In most patients, the meningeal covering ruptures and the spinal cord and roots lie exposed to the air

Occult spina bifida is the least severe form; it may not present until adolescence.
Meningomyelocele uncovered by skin is the most severe form.
Problems associated with spina bifida which should be searched for:
1. Neurological deficit involving motor loss, sensory loss, and bladder and bowel function.
2. Hydrocephalus.
3. Orthopedic deformities of the spine, hips and feet.
4. Other coexisting deformities in general.

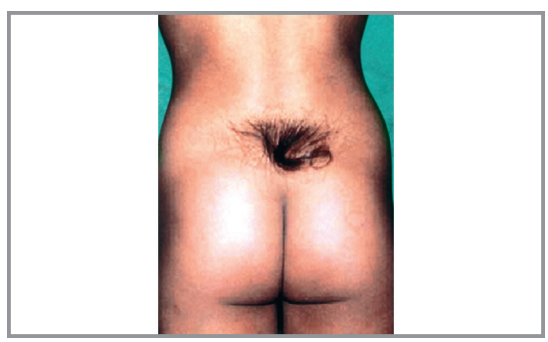


Fig 94-1 Occult spina bifida

PHYSICAL EXAMINATION

What is the level of the defect?
Lumbosacral lesions are the most common.
The degree of neurological deficit depends on the level of the spinal cord involved.

Is the defect **covered by skin, or is it open**?
- *When covered by the skin*,
  - Is the skin **completely normal**?
    OR
  - Is there **some lesion like** a tuft of hair / dimple / sinus (communicating with subarachnoid space) / vascular malformation / lipoma?

www.Ebook777.com

- *When open*,
  - **Leak of CSF**?
  - **Tissue fluid from exposed neuroepithelium**?
  - **Transudate from granulation tissue** (produced by stimulation caused by physical abrasion and bacterial contamination)?
  - **Purulent discharge** from infection?

Any **sinus**? The sinus may link with an intraspinal dermoid cyst. Infection of the sinus may → infection of the cyst – intraspinal abscess → destruction of the adjacent spinal cord.

Transillumination determines whether it contains spinal cord.

Cross fluctuation: A quick compression of the swelling leading to bulging of the anterior fontanelle ⇒ communication with subarachnoid space.
Impulse-on-crying: Crying leading to increase in ICP causes the (cystic) swelling to become tense.

Palpate the bony defect for the level and size. Size of the bony defect is related to the degree of neural tube abnormality, and consequently the severity of the neurological defect.

Assess motor deficit, sensory deficit, and bowel and bladder function. Clinical methods are beyond the scope of this book.

Orthopedic deformities?
- Deformity of feet ⇒ L_5 S_1 lesion.
- Secondary congenital dislocation of the hip—Results from imbalance of muscles around the hip joint. Positive *Ortolani's sign* is the earliest pathognomonic sign of a congenital dislocation of the hip.
  Clinical method for Ortolani's sign
  - Patient in supine position.
  - Fix the pelvis by holding the knees and hips in flexed position.
  - Gradually abduct the thigh to be tested, and at the same time exert pressure over the greater trochanter so as to push the head of the femur into the acetabulum. A 'click of entrance' will be felt as the femoral head slips into the acetabulum, and a 'click of exit' as it quits the acetabulum when the pressure is released.
- Secondary kyphoscoliosis: Result of paralysis and the absence of dorsal arches of spinal canal over a large number of segments leading to destabilization of the spine.
- Hemivertebrae suggests poor prognosis.
- Partial / complete sacral agenesis.

Examine for hydrocephalus.

OCCULT SPINA BIFIDA

– Gap in the dorsal arches of $L_{4,5}$, S_1.

Various presentations of spina bifida

1. **Asymptomatic**. Incidental detection on an X-ray taken for some other problem.
2. **Suggestive lesion on the overlying skin,** e.g. a tuft of hair / dimple / sinus (communicating with subarachnoid space) / vascular malformation / lipoma.
3. Development of **neurological deficit later, during the period of growth spurt**. Mechanism: Normal change in the relative position of spinal cord to vertebrae may → stretching of the spinal cord, if the filum terminale/nerve roots are abnormally tethered in the region of the bony defect.
4. **Gradual onset of neurogenic bladder**.
   With normal limb movements, neurogenic bladder and perianal anesthsia ⇒ $S_{4,5}$ lesion.
5. **Recurrent meningitis** ⇒ a sinus directly/indirectly through dermoid cyst, communicating with subarachnoid space. Causative organisms are atypical, unusual.
6. '**Split spinal cord syndrome**'.

Free ebooks ==> www.Ebook777.com

Possible complications of spina bifida

- Meningitis. *Causes*: Complication of ulcerated sac, infection of a sinus, as a postoperative complication.
- Mental retardation—May be related to associated hydrocephalus/may occur as a separate cerebral deficiency.
- Complications of hydrocephalus: Paralytic squint, optic atrophy with blindness.
- Deafness: A separate unrelated problem.
- Pressure sores.
- UTI. Cystitis-pyelonephritis-progressive renal scarring.
- *Future problems as these children grow*: 1. Small capacity, low-compliance, thick-walled bladder. 2. Deficient function of the external urethral sphincter → incontinence. 3. Fecal incontinence / Constipation.

Adverse factors carrying high mortality

– High-level lesion with complete paraplegia (especially with kyphoscoliosis/hemivertebrae).
– Hydrocephalus clinically present at birth.
– Rapidly progressing hydrocephalus.
– Meningitis, ventriculitis.
– Other severe abnormalities.

CLINICAL PEARLS

- Cervical/high thoracic meningoceles generally do not cause severe neurological deficits.
- Paralysis and sensory loss nearly always occur with meningomyelocele, except when it is low sacral.
- In case of recurrent meningitis, a sinus connecting with subarachnoid space should be sought.
- Children with meningocele and occult spina bifida should be followed up until after puberty since growth of the spine with cord-tethering may → progressive neurological signs.
- Skeletal deformity progresses with age because of imbalance in muscle strength.



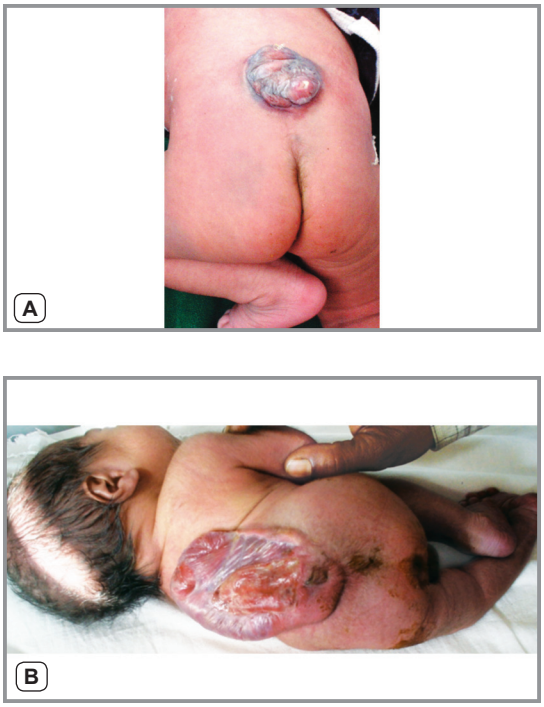


Figs 94-2A and B Thoracic meningocele (Different cases)



Fig 94-3 Lumbosacral meningocele

www.Ebook777.com



Figs 94-4A and B Lumbosacral meningomyeocele (Different cases)

Free ebooks ==> www.Ebook777.com

95 FOREIGN BODIES

FOREIGN BODY IN THE PHARYNX

The most common is a fish bone lodged in the tonsil, the pyriform fossa, or the back of the tongue.
Chief complaint: Pain—constant or felt only during swallowing.
After spraying the fauces and the pharynx with a local anesthetic, the usual sites of impaction can be examined with appropriate instruments and proper illumination, and the object removed with forceps.

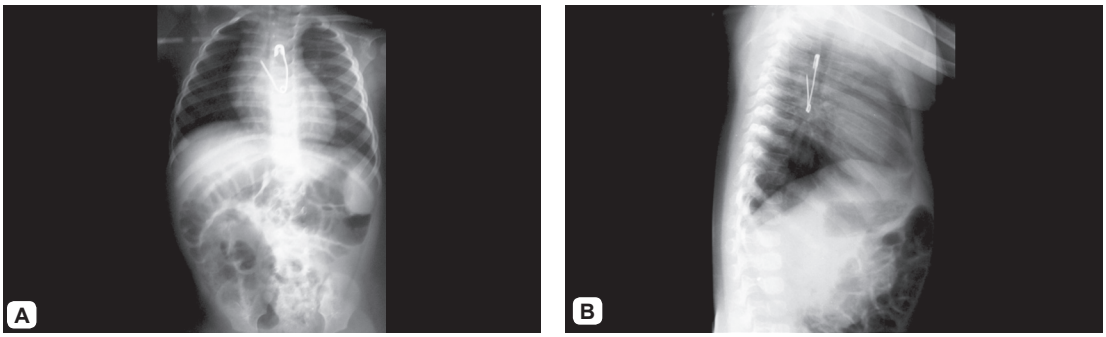
INGESTED FOREIGN BODY

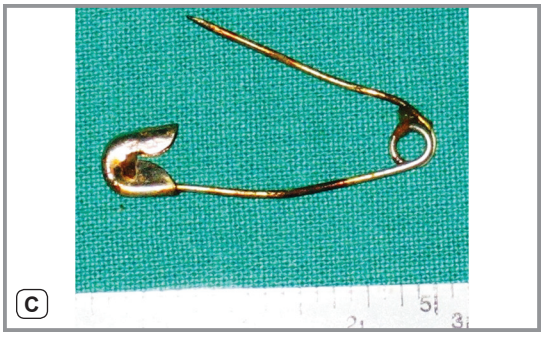
Ingested foreign body can **present** in several ways

- Acute complete upper airway obstruction → Immediately carry out Heimlich maneuver
- Dysphagia especially following poorly chewed food (e.g. meat with bone) in a k/c/o chronic obstructive disease (Pathological narrowing of esophagus)
- Actually witnessed, or suspected ingestion of a foreign body
  - Asymptomatic
    - May pass off without any problem
    - Smaller smooth objects such as coins may remain *in situ* for many days or weeks with minimal consequences
  - Symptoms produced due to impaction and/or complication
- No obvious h/o foreign body ingestion → Presentation straightaway with complication
  - Acute/subacute complication
    - Mallory-Weiss tear
    - Mediastinitis
    - Aspiration pneumonia
    - Peritonitis
  - Chronic complication
    - Progressive dysphagia, anorexia,and weight loss → failure to thrive
    - If the foreign body is large, → progressive dyspnea due to anterior tracheal compression
    - Recurrent aspiration pneumonia
    - Long-standing foreign bodies become embedded in granulation tissue which can be friable, and bleed → hematemesis or may produce increasing obstruction and appear as a pseudotumor on Barium-swallow or endoscopy
    - Stricture
    - Fistula into the trachea leading to recurrent pneumonia
    - Fistula into a blood vessel leading to massive hematemesis
- History concealed Detected later apparently as an incidental finding, especially of multiple sharp objects ⇓ Strong suspicion of child abuse

General remarks
History
Examination
Pitfalls in arriving at the diagnosis
Investigation
Practical situations and principles of management
} Refer to chapter 62.

www.Ebook777.com





Figs 95-1A to C An 8 months old active male child pulled a safety pin of his nappy and swallowed it

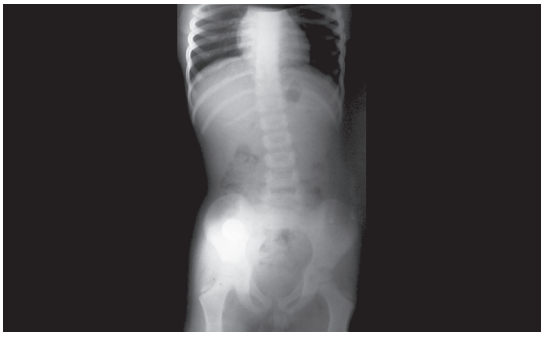


Fig 95-2 This swallowed coin is expected to be passed without consequence

FOREIGN BODY IN THE EAR

Unless there is a definite history, children present later with a blood-stained discharge, irritation, or deafness.

FOREIGN BODY IN THE NOSE

Modes of presentation: Nasal irritation and obstruction, or a purulent sanguinous discharge.
The object can often be seen after the local application of an anesthetic with adrenaline.
X-rays are sometimes necessary.

FOREIGN BODY IN THE TRACHEA/BRONCHI

Clues: Coughing, spluttering, gagging of sudden onset with a residual wheeze.
Clinical picture varies with the size of the object, the site of lodgement and the time elapsed since the object was inhaled.

- Inspiratory stridor and retraction of the supraclavicular, substernal or intercostal areas ⇒ object in the larynx or subglottic area.
- Wheeze ⇒ object in the trachea or bronchus.

Free ebooks ==> www.Ebook777.com

X-ray findings:
– An opaque object will be clearly visualized.
– Indirect evidences: Segmental pulmonary collapse, lobar emphysema.

A pitfall in case of infants: Respiratory signs can occur when the object is impacted in the esophagus, e.g. acute SOB, stridor, cough, wheeze. An orientation of the object in X-ray will give a clue.

FOREIGN BODY IN THE URINARY TRACT

Modes of entry:
1. Most often in the urinary bladder introduced per urethra—in girls.
2. Boys may introduce a foreign object in the lumen of the urethra to attempt to seek erotic stimulation.
3. Rarely, a swallowed foreign body ulcerates through the wall of the duodenum in the right renal pelvis.
4. A fragment of a ureteric catheter may break off and remain lodged.

Complications/Modes of presentation: Pain, hematuria, infection, stone formation.

FOREIGN BODY IN THE PALM

If unrecognized, a painless cystic swelling with a fibrous capsule may develop around the object.

FOREIGN BODY IN THE NECK

A sharp foreign body may enter through a remarkably small laceration. Such a foreign body may present with a puzzling swelling or tenderness, sometimes remote from the site of laceration, several days or weeks later.

www.Ebook777.com

BIBLIOGRAPHY

1. Bickley L. Bates' guide to physical examination and history-taking. Lippincott Williams & Wilkins, 8th edition, 2003.
2. Cuschieri A, Steele R, Moosa Arnold A (Ed). Essential surgical practice: Higher surgical training in general surgery. A member of Hodder Headline Group, 4th edition, 2002.
3. Cuschieri A , Steele R, Moosa Arnold A (Ed). Essential surgical practice: Basic surgical training. A member of Hodder Headline Group, 4th edition, 2001.
4. Das K. Clinical methods in surgery. Published by Dr S Das, 12th edition, 1982.
5. Garden OJ, Bradbury AW, Forsythe JLR, Parks RW (Ed). Principles and practice of surgery. Edinburgh: Churchill Living Stone/Elsevier, 5th edition, 2007.
6. Hobsley M. Pathways in surgical management. London: Arnold-Heinemann, 1984.
7. Hurt K Joseph, et al (Ed). The Johns Hopkins Manual of gynecology and obstetrics. Wolters Kluwer / Lippincott Williams & Wilkins, 4th edition; 2011.
8. Hutson JM and Beasley SW. The surgical examination of children. Heinemann Medical books, 1988.
9. Jones Peter, Woodward Alan (Ed). Clinical paediatric surgery: Diagnosis and management. Blackwell Scientific Publications, 3rd edition, 1986.
10. Joshi NC. Clinical paediatrics. Elsevier, 2007.
11. Joshi MJ. Surgical diseases in the tropics. MacMillan India Ltd, 1992.
12. King M, Bewes PC, Cairns J, Thornton J, et al (Ed). Primary surgery; Volume one. Non-trauma. Oxford University Press, 1987.
13. King M, Bewes PC, Cairns J, Thornton J, et al (Ed). Primary surgery; Volume two. Trauma. Oxford University Press, 1987.
14. Robbins SL, Kumar Vinay. Basic pathology. WB Saunders Company, 4th edition, 1987.
15. Lindsay KW, Bone I, Callander R. Neurology and neurosurgery illustrated. Churchill Livingstone, 3rd Edition, 1998/1999.
16. Moore K. Clinically oriented anatomy. Williams & Wilkins, 3rd edition, 1992.
17. Nancy Sr. Stephanie's principles and practice of nursing : Volume I. Nursing arts procedures. NR Publishing House (Medical Division), 5th edition; 1999.
18. Parikh CK. Parikh's textbook of medical jurisprudence and toxicology. CBS Publishers and Distributors, 5th edition, 1992.
19. Parulekar SV, Supe AN. Practical surgery. Vora Medical Publications, 1992.
20. Pearson FG, Deslauriers J, Ginsberg RJ, Hiebert CA , Mckneally MF, Urschel HC Jr (Ed). Esophageal surgery. Churchill Livingstone Inc, 1995.
21. Rains AJH, Mann CV. Bailey and Love's short practice of surgery. HK Lewis & Co. Ltd; 20th edition, 1990.
22. Reid R, Roberts F. Pathology illustrated. Elsevier, 6th edition, 2005.
23. Rosenfeld JV, Watters DAK. Neurosurgery in the tropics: A practical approach to common problems. MacMillan Education Ltd, London & Oxford; 2000.
24. Saha ML. Bedside clinics in surgery. Academic Publishers, 2007.
25. Shah PL. A new approach to clinical diagnosis in surgery. Published by Dr SP Shah Trust, 1976.
26. Shrivastava RK. A companion in clinical examination in surgery. Aishwarya and Anuj publications, 1996.
27. Sinnatamby CS. Last's anatomy: Regional and applied. Churchill Livingstone, 11th edition, 2007.
28. Sleisenger & Fordtran's gastrointestinal and liver disease: Pathophysiology / diagnosis / management. WB Saunders, 6th edition.

Free ebooks ==> www.Ebook777.com

www.Ebook777.com

INDEX

A

Abdomen
  examination of 69, 290, 391, 438, 496, 545, 587, 906
Abdominal
  compartment syndrome 917
  emergencies in mentally retarded children 964
  girth 908
  pain of uncertain origin 964
Abnormalities of
  menstruation 11
  the cervix 62
Absence of orgasm 792
Absolute constipation 606
Acholuric jaundice 550
Actinic keratosis 188
Actinomycosis 108, 154
Acute
  cellulitis or a small abscess at the side or base of a nail 36
  cholestatic jaundice 552
  hepatitis 550
  inflammation 18
  inflammatory swelling due to bacterial infection 146
  lump in abdomen
    causes of 490
  massive bleeding 588
  mesenteric vascular ischemia 484
  necrotizing ulcerative gingivitis 45
  non-infective inflammatory swelling 146
  osteomyelitis v/s other acute conditions 179
  pain in the perineum 790
  paronychia 36
  renovascular occlusive events 766
  retention of urine 988
  scrotal swelling 318
  specific bacterial infections affecting non-visceral soft tissues 98
  swelling 145
Adenoma (fibroadenoma) 192
Adnexal masses 63
Alcohol 13
Allergic swelling 146
Amalgam tattoo 727
Amoebiasis 110
Amputation 691, 926
Aneurysm 225, 229
Anorectal malformations 970
Anterior
  abdominal wall 127
  group of muscles 126
Apparent state of health 31
Appendicitis 963
Articular swelling, basically, a swelling in the region of a joint 149
Ascariasis
  (round-worm infestation) 112
Ascites 536
Aspiration and respiratory symptoms 574
Assessing a ventriculoperitoneal shunt 1005
Assessment of a patient with sudden loss of consciousness 80
Associated disorder 76, 295
Asthma 58
Asymmetry 369
Asymptomatic urinary abnormalities 766
Atelectasis (lobar obstruction) 58
Attitude of the upper extremities 929
Attributes of mobility tested 138
Autoimmune disorder, symptoms of 262
AV malformations 135, 188
Axilla 44

B

Basal-celled carcinoma 183
Belching 570
Benign
  bile duct stricture 555
  oral ulcers 727
  pigmented nevus (mole) 189
  swellings of the nipple 360
Bilateral true parotid swellings 241
Bladder
  injury 923
  outflow obstruction 789
Blast injury 826
Bleeding
  from gums 22
  blood-stained/purulent/mucous discharge apart from defecation 592
Blunt
  abdominal trauma 910
  injuries of the neck 881
  trauma 825
Body
  cavities 74
  mass index 43
  proportion 43

Free ebooks ==> www.Ebook777.com

Boerhaave syndrome 894
Bone and joint injuries 830
Bony swelling 148
Bowen's disease
(carcinoma *in situ*) 189
Breathing
difficulty in 15
normal 52
Breathlessness 15
Burns 926
Bursal swelling 194

C

Cachexia 47, 48
Calcinosis cutis 188
Callosity (callus) 187
Carcinoma
abdominal 393
adenocarcinoma 239
adenoid cystic 237, 239, 254
alveolar 248
basal-celled 183, 627
branchial 258
colloid 599
colorectal 600
duodenal 417, 524
epidermoid 248
fibrosis 352
gallbladder 425
hepatic cholangiocarcinoma 405
hepatocellular 405, 555
inflammatory 358
invasive ductal 358
invasive lobular 353
malignant 239
medullary 377
mesentery and omentum 406
mucinous 635
pancreas 406
primary duodenal 405
scirrhous 347
SCM 950
secondary 514
squamous-cell 239, 601, 627
stomach 416
thyroid 276
ulcerative 586
urethra 341
vaginal wall 342
vulva 342
Cardiac arrhythmias with a regular rhythm 40
Cardiorespiratory arrest 79
Cardiovascular disorders 19
Carotid arterial pulsations should not be mistaken for internal jugular venous pulsations 42
Case-taking
general principles of 3
Cavernous lymphangioma 157
Cervical
esophageal perforation 893
lymph nodes, clinical method of palpation 144
Cervix 71
Chalazion 234
Cheeks 45
Chemical burns 943
Chest
examination of 71
expansion 54
symptoms 52
Cholangiocarcinoma 555
Choledochal cyst 980
Cholelithiasis 980
Chondrodermatitis helicis 236
Chronic
auricular swelling 236
bronchitis 57
case usually with +ve signs 676
cholestatic jaundice 551
circumscribed general swellings arising from muscle 190, 193
skin 180
circumscribed swelling in the region of the head (including scalp) 232
circumscribed swellings related to tendon 195
gingivitis and periodontitis 45
hepatitis 551
inflammation 18
inflammatory swelling 153
mastalgia 362
nonspecific cellulitis / pyogenic abscess 153
or acute-on-chronic 73
osteomyelitis 247
pain in the
breast (chronic mastalgia) 360
perineum 791
paronychia 36
periauricular swelling 234, 235
periorbital swelling 233
peripheral arterial occlusive disease 36
renal failure (CRF) 764
retention of urine 988
scrotal swelling 319
SDH 867
specific swelling
gluteal region 341
perineum 341
swelling 147
breast 355
cheek 731
floor of the mouth 732
male breast 376
nasolabial fold 243
palate 731
parotid region 236
region of the face 233
skin/subcutaneous tissue overlying the breast 359
tongue 733
Chronically inflamed
swelling 153
ulcer 619
Cirrhosis 553
Closely related structures/ organs? 135
Cloudy urine 784
Colon 527
Coma 79
Common anorectal disorders 607
Condyloma acuminatum venereal wart 185
Condylomata 185

www.Ebook777.com

Congenital
anomalies 378
cleft lip 737
cystic lung 955
lobar emphysema 954
sequestration dermoid 190, 152, 233
Constipation 605, 963
Contralateral
breast in usual manner examine 360
limb and other limbs examination of 692
Cord injury 931
Corn 187
Corrosive injury 895
Costochondritis 379
Cough
acute 17
barking 17
brassy 17
causes of 17, 19
chronic 10, 17
episodic 17
Cracked nipple 368
Cysts
benign tumor 151
complications of 153
duplication 970
Cystic
hygroma 156
swelling 241
Cysticercosis 117

D

Deep muscle of the posterior compartment 127
Deeper hemangioma 156
Deltoid 124
Dermatitis of and around the umbilicus 983
Dermatofibroma (Syn. sclerosing angioma, subepidermal nodular fibrosis) 181
Dermatofibrosarcoma protuberans 182
Desirable specific details in the ems personnels's report 836
Diabetic nephropathy 766
Digital rectal examination 64, 961
Dilated veins 135
Diphtheria 46
Diplopia (double vision) 26
Discharge from nipple/s 364
Discomfort in the breast/s 360
Diseases
acute febrile 579
intra-abdominal 455
Addison's 25, 723
alcoholic liver 548
amebic 466
aortic 664, 648
arthritis 149
autoimmune 241, 275, 276
bilateral 804
bone 389
Bowen's 189
calculous 237, 254
cardiac 40
cervical 12
degenerative 649
disk 653
Chaga's 461
chronic fibrotic 54
liver 484, 511, 518
lung 19, 22, 92
pancreaticobiliary 576
peptic ulcer 577
peripheral arterial occlusive 36, 659
renal 765
systemic 76
collagen 520
vascular 622
colonic diseases 636
colorectal 389
common benign nodular 272
communicable 14
condylomata 603
congenital polycystic 518
congestive 519
Crohn's 415, 524, 527
colitis 605, 606, 607, 619
Cushing's 237
cystic disease of the biliary tract 406
deficiency 520
diverticular 581, 586
esophageal 571, 572, 900
fibroepithelial polyp 600
gallstone 417
gallbladder 13, 384, 402, 507
gallstone disease 466, 542, 545, 547
gastric 571
gastroesophageal reflux 404
Gaucher's 520
Glomerular 765
Grave's 30, 261–267, 271, 705, 706
Hansen's 44
Hashimoto's 268, 272
heart 16
hematological 520, 540, 541
hemorrhagic 487
hepatic 113, 389
hepatobiliary and pancreatic 43
Hirschprung's 392
Hodgkin's 198, 199, 202, 523
hydatid 115, 116
hydrocephalus 44
hypertensive cardiovascular 228
infarction of bone in sickle-cell 180
infectious 15
Infective 520
inflammatory and granulomatous 519
bowel 405, 417
internal ear 25
intra-abdominal 644
Kawasaki 200
laryngeal 563
liver 541, 552, 554, 555, 577
locomotor 700
lung 16
lung disease 56
macrovascular disease 676
Ménière's 25
metastatic 320, 328, 346, 360, 371, 533, 534
Mikulicz 242
multilevel 676, 677, 678, 683
multiple 72

Free ebooks ==> www.Ebook777.com

nature of 74
neoplastic 519
or inflammatory 542
obstructive disease 560, 561, 562
lung 41
occlusive disease 659
overt pulmonary 84
Paget's 44, 345, 348, 349, 360, 367, 368, 370, 371, 617, 655
pancreatic 462, 549
Parkinson's 31, 650
peptic ulcer 25, 384, 415
Peyronie's 778, 809
pruritus ani 603
pulmonary 42
renal 385, 765, 773
restrictive lung 41
rheumatic heart 192
rheumatological 709, 714
severe cardiorespiratory 47
obstructive pulmonary 56, 57
renal 461
respiratory 461
sickle cell 469, 491
Simmond's 25
stage of 74
storage 520, 523
systemic 200
Takayasu 663
thyroid 261
tuberculosis 308
tubulointerstitial 766
urinary stone 382
tract 113
valvular 80
heart 484
varicose vein 649
venereal 11
venereal wart 603
Von Recklinghauson's 192
Wilson's 519, 520, 541, 550
Disorders of the
palate 737
lip 736
tongue 739
Distension cyst 152
Distinguishing vaginal from
cervical discharge 62
Distress
sign of 29
Dorsiflexors of
the foot 127
Dorsum of the
foot 127
hands 35
Dracontiasis
(guinea worm infestation) 116
Ductal stones 554
Duodenal obstruction 970
Dysphagia 560
Dyspnea 15

E

Ear symptoms 26
ECG pattern 40
Eczema of the nipple, usually extending onto the areola 367
Edema 36
Electrical burns 943
Enuresis 788
Epistaxis 22
Esophageal perforation 892
External genitalia 60, 442
Extrahepatic biliary tract excluding the gallbladder 527
Exudative tonsillitis 46
Eye signs 266
Eyes and facial expression 30

F

Face 44
Fall from height 825
Fallacies 137, 141, 142
Fatigue 22
Fecal incontinence 606
Female genitalia 992
Fetal movements 70
Fever 15, 791
usually suggests infection 132
Fibroma 155, 181
Fibrosarcoma 155
Filariasis 114
First aid 827
Fistula 230
Fluctuation? 140
Forced expiratory time 57
Forearm 125
Foreign body in the pharynx 1010
Fracture of the vertebral bodies types of 931
Fungal infections of surgical importance 108

G

Gait and general activity 29
Gallbladder 527
Ganglion 152
Gastroesophageal reflux disease 572
GE reflux 976, 963
Gigantism 42
Globus 574
Glomerular diseases 765
Gluteal muscles 126
Gnathostomiasis 117, 191
Groins and external genitalia examination of 71, 402
Gumma (nonulcerated) 184
Gums and teeth 45

H

Hair on the scalp, face, axilla, and pubis 44
Head 119
Headache 22, 23
Health maintenance practices 13
Heartburn 570
Hemangioma 155
Hematospermia 789
Hepatic metastases 556
Hepatosplenomegaly 523
Hernia of the muscle 194
Hirschprung's disease 970
Hoarseness of voice 26
Homeostasis 76, 77
Hydatid cyst 115, 142, 191, 193
Hydatiduria 785
Hydrocephalus 1002
Hypersensitivity 13
Hypertrophic scar 187
Hypothesis of diagnosis 76
Hypovolemia and hypervolemia signs of 47

www.Ebook777.com

I

Ileocecal region 524
Immunization 947
Imperforate hymen 61
Implantation dermoid 152, 191
Impulse-on-coughing? 137
Inability to open the mouth 743
Incomplete contusion 931
Incontinence of urine 787
Infection of
  the stump of the umbilical cord 982
  soft tissues 380
  the bone 379
Infective wart 185
Infertility 792
Ingested foreign body 1010
Inspection of the external genitalia 71
Intermittent pains proximal to foot with all peripheral pulses palpable even after exercise 680
Internal examination when the introitus is small 61
Intestinal atresia 970
Intra-auricular swelling 236
Intradermal type of nevus 189
Intussusception 965
Itching 26

J

Jaundice, types of 540
Jiggers 118
Junctional nevus 189

K

Keloid 187, 236
Keratoacanthoma 188
Kidney 529
Koilonychia 36

L

Lacrimal gland swelling 234
Laparotomy
  general indications for 914
Large normal tonsils 45
Lateral aspect of the distal portion of the arm, and the elbow 124
Left-sided heart failure (early) 57
Lepromatous leprosy 236
Leprosy 107
Lesion in the oral cavity, general guideline for arriving at the diagnosis of 720
Lesser sac 529
Liposarcoma 155
Liver 515
Loa loa (eye worm) 115
Locoregional examination 49, 281, 303, 334, 346, 361, 363, 364, 371, 645, 648, 707, 869, 935
Loose teeth 45
  of libido 792
Lower jaw
  examination of the 246
Lumbar (cold) abscess 514
Lump caused by the gravid uterus 70
Lung hernia 379
Lupus vulgaris 185
Lymphadenopathy 46, 69
Lymphangioma
  (lymphangioma simplex) 157, 193
Lymphangiosarcoma 157
Lymphatic cyst 191

M

Malaria 117
Male
  genitalia 992
  urethral injury 924
Malignant
  melanoma 183, 727
  synovioma 158
  transformation of a pre-existing nevus 184
  tumor 242
Mammary duct fistula 367
Marginal gingivitis 45
Mastectomized patient
  examination of 360
Meconium
  ileus 970
  plug syndrome 970
Meningocele / encephalocele 234
Menopause 11
Method of palpation of axillary lymph nodes 144
Microvascular disease 678
Micturition 988
Mikulicz disease 242
Minimum equipment necessary for a clinical case-taking 5
Mobility and turgor 35
Modes of clinical presentation 922, 986
Molluscum contagiosum 185
Monitoring and revaluation 491
Mucocele of
  the frontal sinus 234
  the lacrimal sac 234
Multiple organ dysfunction syndrome (MODS) 85
Muscle
  power 930
  tone 82
  in the pectoral region 123
Muscles
  of the back 120
  of the lateral compartment 127
Mycetoma 154
Mycobacterial infection 185
Myiasis 118
Myositis ossificans 194
Myxoma 192

N

Natural history of
  (infective) warts 185
  swallowed foreign body 898
  untreated disease 271
Neck 32
Necrotizing enterocolitis 969
Nervous system
  examine 819
Neural tumors 157
Neurofibroma 192
Neurological and focused general examination 81
Nodular leprosy 187
Non-reflux esophagitis 573
Nonvisceral soft tissue injuries 828
Nose and paranasal sinuses 46

O

Obesity 48
Obstetric history 12
Obstructive sialectasis 241
Occult spina bifida 1007

Free ebooks ==> www.Ebook777.com

Odor of the body / breath 46
Odynophagia 570
Oliguria / anuria 813
Onchocerca volvulus 117
Onycholysis 36
Oral
  cancer 734
  manifestations of systemic disease 736
  mucosa, color changes 723
  swellings 730
Organomegaly 240
Orofacial pain 721
Orus palatinus 46
Ovary and broad ligament 528

P

Pain
  and hypersensitivity in the nipple 368
  in the chest, causes and DD of, 20
  in the penis 790
Painful micturition 782
Palm 36
Palpate the opposite side wherever applicable 144
Palpating agent 49
Palpation of
  bladder 988
  inguinal lymph nodes 144
  perianal area 64
  the skin: Temperature, texture, moisture, thickness 32
Pancreas 527
Paralytic ileus 969
Parasitic infestations of surgical importance 110
Parietal swellings 510
Passage
  of fecal matter / worms in urine 791
  of gas in urine 791
Pathological classification of chronic inflammatory swellings 153
Pediatric gynecologic emergencies 964
Pelvic
  examination 59, 71, 402, 443, 501, 587, 778
  and external genitalia examination of 907
Pemberton's sign 266
Penetrating
  abdominal trauma 911
  injuries of the neck 880
  trauma 826, 925
Penile injury 925
Penis
  examination of 777
Periampullary cancer 555
Perineum
  examination of 778
Peritoneal cavity 529
Peritonitis 962, 964
Pharyngitis 46
Pitfalls
  in arriving at the diagnosis 900
  in pediatric trauma 847
Pleural effusion 58
Pneumomediastinum 955
Pneumothorax 58
Polymazia 369
Portal hypertension 554, 980
Postauricular
  pseudodermoid 235
  dermoid cyst results from inclusion of epithelium 235
Posterior group of muscles 126
Pregnant patient
  examination of the 68
Presacral lump 533
Primary syphilitic sore on the penis 621
Proctoscopic examination 66
Prosthesis, examination of 692
Pseudoreducible swelling 151
Psoriasis 36
Pulsatile swelling 137, 224
Pulse rate 40
Pure bleeding per urethra independent of micturition 784
Purple
  violet lesion 727
Pyoderma gangrenosum 619
Pyogenic granuloma 156

R

Rectal examination 63
Rectus sheath swellings 514
Recurrent
  obscure bleeding 591
  subacute or chronic parotitis 241
Red / white lesion 726
Reducibility 142
Reducible swelling 151
Regional lymph nodes examine 317
Renal injury 919
  trauma
    complications of 921
Respiration 40
Respiratory distress
  frothy baby 954
  relieved by crying 953
  system examination 52
Retention
  cyst 61, 152
  of urine 812
Retroperitoneal
  tissue 533
  acute pyogenic abscess v/s tuberculous abscess 179
Rhabdomyosarcoma 194
Rheumatic nodules 193
Rheumatoid nodules on helix 236
Rhythm and pattern of respiration 81

S

Scalp 44
Scar 135
Schistosomiasis (bilharziasis) 113
Scrofuloderma 185
Scrotal
  pain 333
  swelling 310
Sebaceous cyst 180
Seborrheic keratoses 188
Selected common symptoms in clinical practice 15
Seminal vesicles 65
Sepsis 84
Septic shock 84

www.Ebook777.com

Septicemia 84
Shapes of the cervical 61
Short notes on selected clinical conditions 273
Shortness of breath (Syns. Breathlessness, dyspnea, difficulty in breathing) 15
Silent chest 56
Sjögren's syndrome 242
Skin lesions 34, 35
Smoking/consumption of tobacco in any other form 13
Soft tissue tumors 154
Sore throat 27
  tongue 27
Sparganosis 118
Specific bacterial infections of surgical importance 98
Spectrum of disease causing 'acute abdomen' of particular importance in tropical countries 490
  of symptomatic gallstone disease 554
Speculum examination 71
Spinal cord injury
  examination for 929
Spine
  examine 748, 908
Spleen 518
Splinter hemorrhages under the nails 36
Squamous-celled carcinoma-protuberant type 182
Stomach 523
Strangury 782, 812
Stump of the umbilical cord or omphalitis, complications of infection 982
Subdiaphragmatic spaces 529
Submandibular sialomegaly 253
Submucous fibrosis 727
Sudden
  loss of consciousness 79
  unresponsiveness 79
Superficial
  and intermediate layer muscles of the anterior compartment 125
  hemangioma (Strawberry nevus) 156
  muscles in the neck 119
  muscles of the medial group (Adductors) 126
  muscles of the posterior compartment 127
Supine in horizontal position 37
Surface of the swelling is of little help in arriving at the diagnosis 238
Swelling in
  the region of the breast 345, 353, 370
Syndromes
  abdominal compartment 917
  acute respiratory distress 90
  anterior tibial 657
  antiphospholipid 679
  Boerhaave 894, 895
  Budd-Chiari 405, 423, 517, 537
  burning mouth 722
  carcinoid 417
  Caroli's 405
  Carpal tunnel 262, 274, 654, 656
  chronic adhesion 406, 417
    gastrointestinal 428
  compartment 657, 712
  complex regional pain 658
  crush 830, 835
  Cushing's 31, 43, 48, 276, 393
  Dandy-Walker 1003
  Dieulafoy 578
  Down's 30
  Dubin-Johnson 540
  focal 818
  Fowler's 789, 796
  Gardner's syndrome 193
  Gilbert 540, 550
  Horner's 30, 166, 263
  hyperventilation 41
  irritable bowel 387, 405, 411, 428, 606
  Katayama 113
  Klinefelter 43, 793
  malabsorption 47, 386, 405, 417
  Mallory-Weiss 578, 894
  Marfan's 43
  McArdle's 680
  meconium plug 956, 970
  megacystitis 406
  multiple organ dysfunction 85
  nephrotic 30, 31, 412, 765, 766, 806
  neurological 867
  orbital apex 162
  peripheral nerve 656
  Peutz-Jeghers 405, 418, 601
  Pierre Robin 952
  Plummer-Vinson 569
  Poland's 1000
  polycystic ovary 48
  popliteal artery entrapment 677
  post-cholecystectomy 406, 425
  postthrombotic 222, 226
  premenstrual 11, 12
  pseudoulcer 405
  restless legs 657
  Rotor's 540, 552
  sham 986
  Sjögren's 237, 241, 242, 243, 254
  small stomach 388, 389
  split spinal cord 1007
  subclavian steal 80
  SVC 261–263
  systemic inflammatory response 84
  thoracic outlet 652, 677
  tunnel 656
  various obstruction 406
Syphilis (gumma) 154
Systemic inflammatory response syndrome (SIRS) 84

T

Tenderness 49, 136
Teres major 124
Test
  multiple tourniquet 700
  Perthe's 700
  Trendelenberg 700
Testicular injury 926
Thermal burns 938
Thoracic and abdominal esophageal perforation 893
Throat 45
Thyroid cancers 275
Tight phimosis 809

Free ebooks ==> www.Ebook777.com

Tinnitus 27
Tips for a successful examination 59
Tongue 31
Torsion 486
Toxemia
degree of 99
Transillumination 141
Traumatic swelling 145
Trivial bleeding 591
Tuberculosis
cutaneous 187
ileocecal 418
intestinal 459
miliary 200, 205
renal 311
small intestinal 417
tenosynovitis: 2 types 195
Tubulodermoid 152
Tubulointerstitial diseases 766
Tumors
acinic cell 238
adnexal 65
benign 10, 131, 132, 133, 134, 136, 138, 151, 152
bilateral 239
bone 158, 218
brain 24
carotid body 255, 257
chromaffin 64
Cock's peculiar 180
cystic 254
desmoid 154, 193
ethmoidal sinus 235
Ewing's 202
glomus vagale 253
high-grade 155
Krukenberg 63
lacrimal gland 234, 235
malignant 62, 63, 132, 133, 134, 136, 138, 151, 152
mandibular 249
metastatic 158
mixed 237, 238
neural 157
ovarian 44
paratesticular 194
parotid 239
rectal 66
reddish brown 135, 195
salivary gland 239
soft tissue 154
solid 141
testicular 305, 308, 315, 323, 327
thyroid 268
urinary tract 763
uterine 62
vascular 155, 215
Wilm's 8
Turbid urine 784

U

Ulceration and crusting 236
Umbilical
fecal fistula in newborn and infants 983
granuloma 983
sepsis, omphalitis 982
stone 983
Underlying bone and joint 142
Undernutrition
signs of 47
Unilateral true parotid swelling 240
Upper jaw
examination of 245
Urachal
fistula 983
injury 921
fistula 791
injury, complications of 925
Urinary
bladder 528
stones 986
tract disorder, symptoms of 766
tract infection 761, 964
tract infection 986
Urine examination
analysis and interpretation of 793
Urolithiasis 761
Urologic trauma in pregnancy 926
Urological causes of painful defecation 782
Uterine
contractility 70
tube 528
Uterus 62, 528

V

Vagina 71
Vaginal
discharge, physiological 62
examination in a female patient 59
Vaginum examination 71
Vanishing heartburn 571
Various injuries 929
Vascular
swelling 150
thrill 136
Veins on the dorsum of the feet in a patient lying supine in horizontal position 37
Venereal disease 11
Vertebral column
examination of the 930
Vertigo 25
Visceral swelling 149
Vital parameters and JVP 37
Volvulus neonatorum 970
Vomiting 25
Vulva
external examination 60
Vulvovaginal symptoms 11

W

Waterbrash 572
Weakness 22
Weight gain 43
Wetting 988
White lesion 723
Withdrawal of the nipple 36

X

Xanthelasma 234
Xanthomas 188
X-ray
abdomen 969
spine—ap and lateral 930

Y

Yaws 108

www.Ebook777.com