

1ST EDITION

Understanding Anesthesia

A Learner's Handbook

AUTHOR

Karen Raymer, MD, MSc, FRCP(C)
McMaster University

CONTRIBUTING EDITORS

Karen Raymer, MD, MSc, FRCP(C)
Richard Kolesar, MD, FRCP(C)

TECHNICAL PRODUCTION

Eric E. Brown, HBS
Karen Raymer, MD, FRCP(C)

www.understandinganesthesia.ca

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AUTHOR

Dr. Karen Raymer, MD, MSc, FRCP(C)

Clinical Professor, Department of Anesthesia, Faculty of Health Sciences, McMaster University

CONTRIBUTING EDITORS

Dr. Karen Raymer, MD, MSc, FRCP(C)

Dr. Richard Kolesar, MD, FRCP(C)

Associate Clinical Professor, Department of Anesthesia, Faculty of Health Sciences, McMaster University

TECHNICAL PRODUCTION

Eric E. Brown, HBSc

MD Candidate (2013), McMaster University

Karen Raymer, MD, FRCP(C)

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Preface



PREFACE

Getting the most from your book

This handbook arose after the creation of an ibook, entitled, “Understanding Anesthesia: A Learner’s Guide”. The ibook is freely available for download and is viewable on the ipad. The ibook version has many interactive elements that are not available in a paper book. Some of these elements appear as placeholders in this (paper) handbook.

If you do not have the ibook version of “Understanding Anesthesia”, please note that many of the interactive elements, including videos, slideshows and review questions, are freely available for viewing at

www.understandinganesthesia.ca

The interactive glossary is available only within the ibooks version.

Introduction

Many medical students’ first exposure to anesthesia happens in the hectic, often intimidating environment of the operating room. It is a challenging place to teach and learn.

“Understanding Anesthesia: A Learner’s Handbook” was created in an effort to enhance the learning experience in the clinical setting. The book introduces the reader to the fundamental concepts of anesthesia, including principles of practice both inside and outside of the operating room, at a level appropriate for the medical student or first-year (Anesthesia) resident. Residents in other pro-

grams such as Emergency Medicine or Internal Medicine, who require anesthesia experience as part of their training, will also find the guide helpful.

The book is written at an introductory level with the aim of helping learners become oriented and functional in what might be a brief but intensive clinical experience. Those students requiring more comprehensive or detailed information should consult the standard anesthesia texts.

The author hopes that “Understanding Anesthesia: A Learner’s Handbook” succeeds not only in conveying facts but also in making our specialty approachable and appealing. I sincerely invite feedback on our efforts:

feedback@understandinganesthesia.ca

Notice

While the contributors to this guide have made every effort to provide accurate and current drug information, readers are advised to verify the recommended dose, route and frequency of administration, and duration of action of drugs prior to administration. The details provided are of a pharmacologic nature only. They are not intended to guide the clinical aspects of how or when those drugs should be used. The treating physician, relying on knowledge and experience, determines the appropriate use and dose of a drug after careful consideration of their patient and patient’s circumstances. The creators and publisher of the guide assume no responsibility for personal injury.

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Acknowledgements

Many individuals supported the production of this book, including the elements that you can only access at the book’s website (www.understandinganesthesia.ca)

Numerous publishers allowed the use of figures, as attributed in the text. The Wood Library-Museum of Anesthesiology provided the historic prints in Chapter 6.

Representatives from General Electric and the LMA Group of Companies were helpful in supplying the images used in the derivative figures seen in Interactive 2.1 and Figure 5 respectively.

Linda Onorato created and allowed the use of the outstanding original art seen in Figures 3 and 6, with digital mastery by Robert Barborini.

Richard Kolesar provided the raw footage for the laryngoscopy video. Appreciation is extended to Emma Kolesar who modified Figure 9 for clarity.

Rob Whyte allowed the use of his animated slides illustrating the concepts of fluid compartments. The image of the “tank” of water was first developed by Dr. Kinsey Smith, who kindly allowed the use of that property for this book.

Joan and Nicholas Scott (wife and son of D. Bruce Scott) generously allowed the use of material from “Introduction to Regional Anaesthesia” by D. Bruce Scott (1989).

Brian Colborne provided technical support with production of the intubation video and editing of figures 5, 10, 11, 15 and 16.

Appreciation is extended to Sarah O'Byrne (McMaster University) who provided assistance with aspects of intellectual property and copyright.

Many others in the Department of Anesthesia at McMaster University supported the project in small but key ways; gratitude is extended to Joanna Rieber, Alena Skrinskas, James Paul, Nayer Youssef and Eugenia Poon.

Richard Kolesar first suggested using the iBookAuthor app to update our existing textbook for medical students and along with his daughter, Emma, made an early attempt at importing the digital text material into the template that spurred the whole project along.

This project would not have been possible without the efforts of Eric E. Brown, who was instrumental throughout the duration of the project, contributing to both the arduous work of formatting as well as creative visioning and problem-solving.

Karen Raymer

The Role of the Anesthesiologist

Dr. Crawford Long administered the first anesthetic using an ether-saturated towel applied to his patient's face on March 30, 1842, in the American state of Georgia. The surgical patient went on to have two small tumours successfully removed from his neck. Dr. Long received the world's first anesthetic fee: \$0.25.

Since then, the specialty of anesthesiology and the role of the anesthesiologist has grown at a rapid pace, particularly in the last several decades. In the operating room the anesthesiologist is responsible for the well-being of the patient undergoing any one of the hundreds of complex, invasive, surgical procedures being performed today. At the same time, the anesthesiologist must ensure optimal operating conditions for the surgeon. The development of new anesthetic agents (both inhaled and intravenous), regional techniques, sophisticated anesthetic machines, monitoring equipment and airway devices has made it possible to tailor the anesthetic technique to the individual patient.

Outside of the operating room, the anesthesiologist has a leading role in the management of acute pain in both surgical and obstetrical patients. As well, the anesthesiologist plays an important role in such diverse, multidisciplinary fields as chronic pain management, critical care and trauma resuscitation.

CHAPTER 1

The ABC's



In this chapter, you will learn about airway (anatomy, assessment and management) in order to understand the importance of the airway in the practice of anesthesiology. As well, you will develop an understanding of the fluid compartments of the body from which an approach to fluid management is developed. Look for review quiz questions at www.understandinganesthesia.ca

Airway Management

In This Section

- [Airway Anatomy](#)
- [Airway Assessment](#)
- [Airway Management](#)
- [Airway Devices and Adjuncts](#)
- [The Difficult Airway](#)

In order to ensure adequate oxygenation and ventilation throughout the insults of anesthesia and surgery, the anesthesiologist must take active measures to maintain the patency of the airway as well as ensuring its protection from aspiration. A brief discussion of airway anatomy, assessment and management is given below.

Airway Anatomy

The upper airway refers to the nasal passages, oral cavity (teeth, tongue), pharynx (tonsils, uvula, epiglottis) and larynx. Although the larynx is the narrowest structure in the adult airway and a common site of obstruction, the upper airway can also become obstructed by the tongue, tonsils and epiglottis.

The lower airway begins below the level of the larynx. The lower airway is supported by numerous cartilaginous structures. The most prominent of these is the thyroid cartilage (Adam's apple) which acts as a shield for the delicate laryngeal structures behind it. Below the larynx, at the level of the sixth cervical vertebra (C6), the cricoid cartilage forms the only complete circumferential ring in the airway. Below the cricoid, many

horseshoe-shaped cartilaginous rings help maintain the rigid, pipe-like structure of the trachea. The trachea bifurcates at the level of the fourth thoracic vertebra (T4) where the right mainstem bronchus takes off at a much less acute angle than the left.

The airway is innervated by both sensory and motor fibres ([Table 1](#), [Figure 1](#), [Figure 2](#)). The purpose of the sensory fibres is to allow detection of foreign matter in the airway and to trigger the numerous protective responses designed to prevent aspiration. The swallowing mechanism is an example of such a response whereby the larynx moves up and under the epiglottis to ensure that the bolus of food does not enter the laryngeal inlet. The cough reflex is an attempt to clear the upper or lower airway of foreign matter and is also triggered by sensory input.

There are many different laryngeal muscles. Some adduct, while others abduct the cords. Some tense, while others relax the cords. With the exception of one, they are all supplied by the recurrent laryngeal nerve. The cricothyroid muscle, an adductor muscle, is supplied by the external branch of the superior laryngeal nerve.

Table 1 Sensory innervation of the airway

NERVE	AREA SUPPLIED
lingual nerve	anterior 2/3 of tongue
glossopharyngeal nerve	posterior 1/3 of tongue
superior laryngeal nerve (internal branch)	epiglottis and larynx
recurrent laryngeal nerve	trachea, lower airways

Figure 1 Nerve supply to the airway

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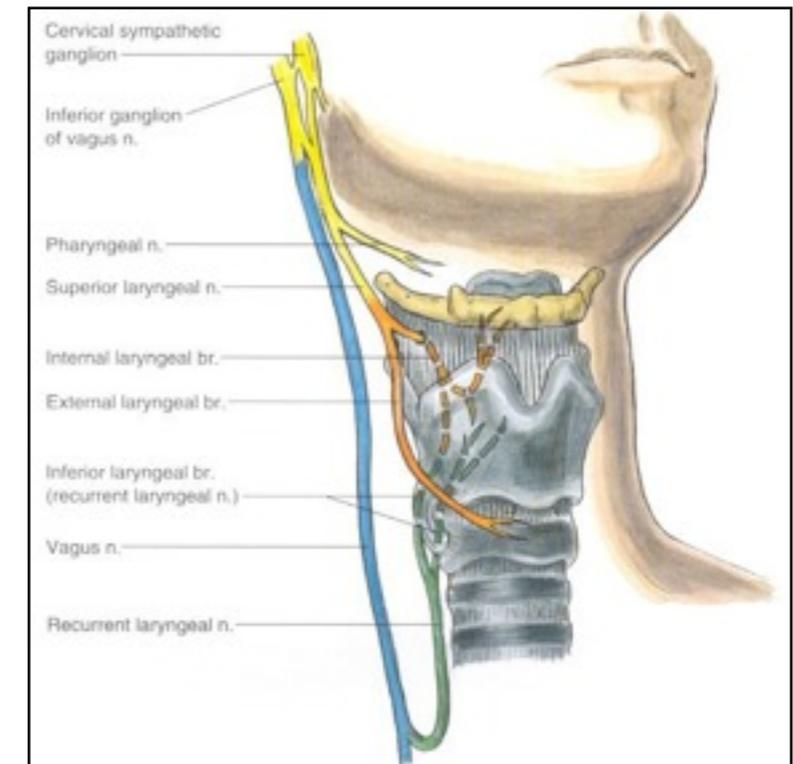
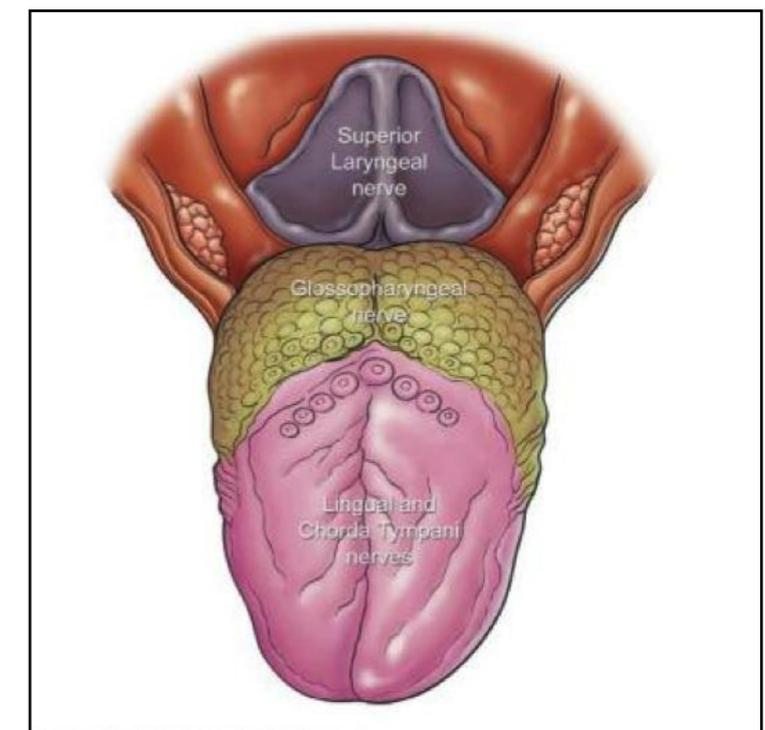


Figure 2 Sensory innervation of the tongue

From the 4th edition (2010) of "Principles of Airway Management". The authors are B.T. Finucane, B.C.H. Tsui and A. Santora. Used by permission of Springer, Inc.



Airway Assessment

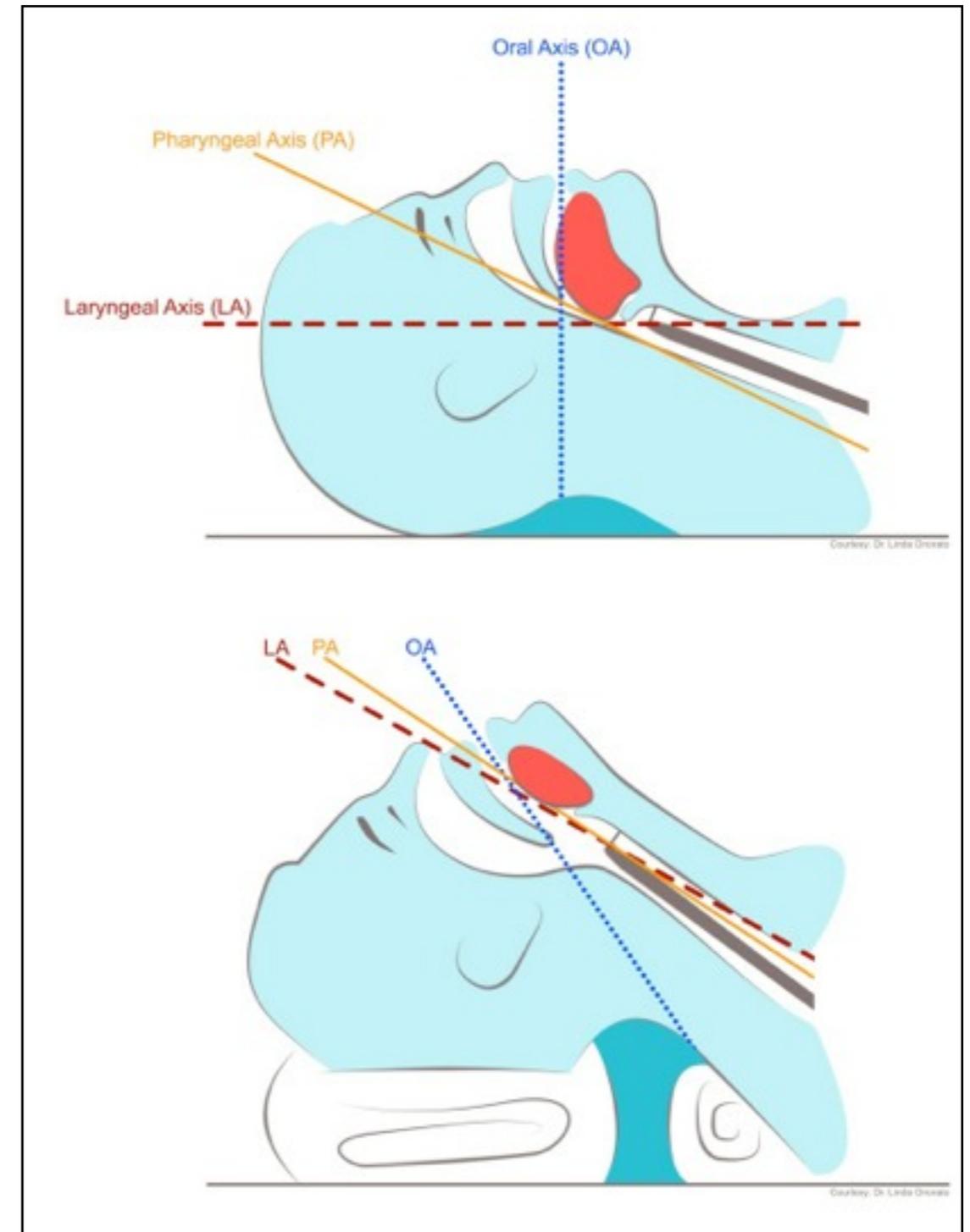
The anesthesiologist must always perform a thorough pre-operative airway assessment, regardless of the planned anesthetic technique. The purpose of the assessment is to identify potential difficulties with airway management and to determine the most appropriate approach. The airway is assessed by history, physical examination and occasionally, laboratory exams.

On history, one attempts to determine the presence of pathology that may affect the airway. Examples include arthritis, infection, tumors, trauma, morbid obesity, burns, congenital anomalies and previous head and neck surgery. As well, the anesthesiologist asks about symptoms suggestive of an airway disorder: dyspnea, hoarseness, stridor, sleep apnea. Finally, it is important to elicit a history of previous difficult intubation by reviewing previous anesthetic history and records.

The physical exam is focused towards the identification of anatomical features which may predict airway management difficulties. It is crucial to assess the ease of intubation. Traditional teaching maintains that exposure of the vocal cords and glottic opening by direct laryngoscopy requires the alignment of the oral, pharyngeal and laryngeal axes ([Figure 3](#)). The “sniffing position” optimizes the alignment of these axes and optimizes the anesthesiologist’s chance of achieving a laryngeal view.

An easy intubation can be anticipated if the patient is able to open his mouth widely, flex the lower cervical spine, extend the head at the atlanto-occipital joint and if the patient has enough anatomical space to allow a clear view. Each of these components should be assessed in every patient undergoing anesthesia:

Figure 3 Axis alignment using the “sniffing position”



Original artwork by Linda Onorato. Digital mastery by Robert Barborini. Used with permission of Linda Onorato.

- Mouth opening: Three fingerbreadths is considered adequate mouth opening. At this point in the exam, the anesthesiologist also observes the teeth for overbite, poor condition and the presence of dental prosthetics.
- Neck motion: The patient touches his chin to his chest and then looks up as far as possible. Normal range of motion is between 90 and 165 degrees.
- **Adequate space:** Ability to visualize the glottis is related to the size of the tongue relative to the size of the oral cavity as a large tongue can overshadow the larynx. The Mallampati classification ([Table 2](#), [Figure 4](#)) assigns a score based on the structures visualized when the patient is sitting upright, with the head in a neutral position and the tongue protruding maxi-

Table 2 Mallampati Classification

Class 1	Soft palate, uvula, tonsillar pillars can be seen.
Class 2	As above except tonsillar pillars not seen.
Class 3	Only base of uvula is seen.
Class 4	Only tongue and hard palate can be seen.

mally. Class 1 corresponds well with an easy intubation. Class 4 corresponds well with a difficult intubation. Classes 2 and 3 less reliably predict ease of intubation. The thyromental distance is also an important indicator. The distance from the lower border of the mandible to the thyroid notch with the neck fully extended should be at least three to four fingerbreadths. A shorter distance may indicate that the oral-pharyngeal-laryngeal axis will be too acute to

Figure 4 Mallampati classification

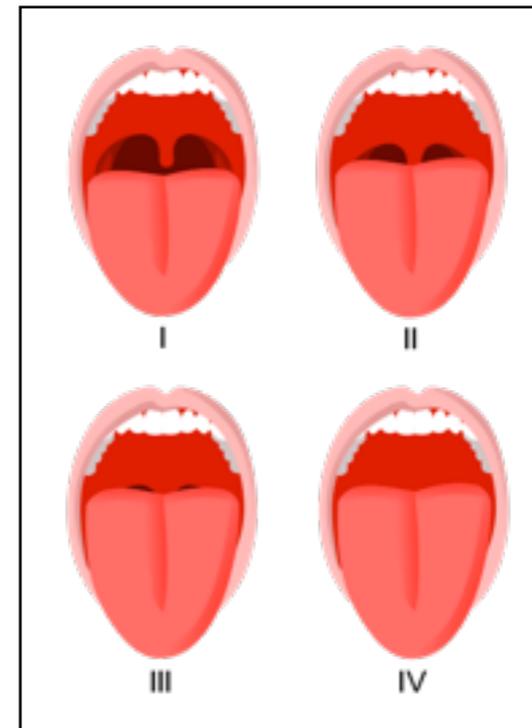


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achieve good visualization of the larynx. As well, a short thyromental distance may indicate inadequate “space” into which to displace the tongue during laryngoscopy.

Combining Mallampati classification with thyromental distance and other risk factors (morbid obesity, short, thick neck, protuberant teeth, retrognathic chin), will increase the likelihood of identifying a difficult airway. No assessment can completely rule out the possibility and so the clinician must always be prepared to manage a difficult airway.

Laboratory investigations of the airway are rarely indicated. In some specific settings, cervical spine x-rays, chest ray, flow-volume loops, computed tomography or magnetic resonance imaging may be required.

Airway Management

Airway patency and protection must be maintained at all times during anesthesia. This may be accomplished without any special maneuvers such as during regional anesthesia or conscious sedation. If the patient is deeply sedated, simple maneuvers may be required: jaw thrust, chin lift, oral airway (poorly tolerated if gag reflex is intact) or nasal airway (well tolerated but can cause epistaxis).

During general anesthesia (GA), more formal airway management is required. The three common airway techniques are:

- mask airway (airway supported manually or with oral airway)
- laryngeal mask airway (LMA)
- endotracheal intubation (nasal or oral)

The choice of airway technique depends on many factors:

- airway assessment
- risk of regurgitation and aspiration
- need for positive pressure ventilation
- surgical factors (location, duration, patient position, degree of muscle relaxation required)

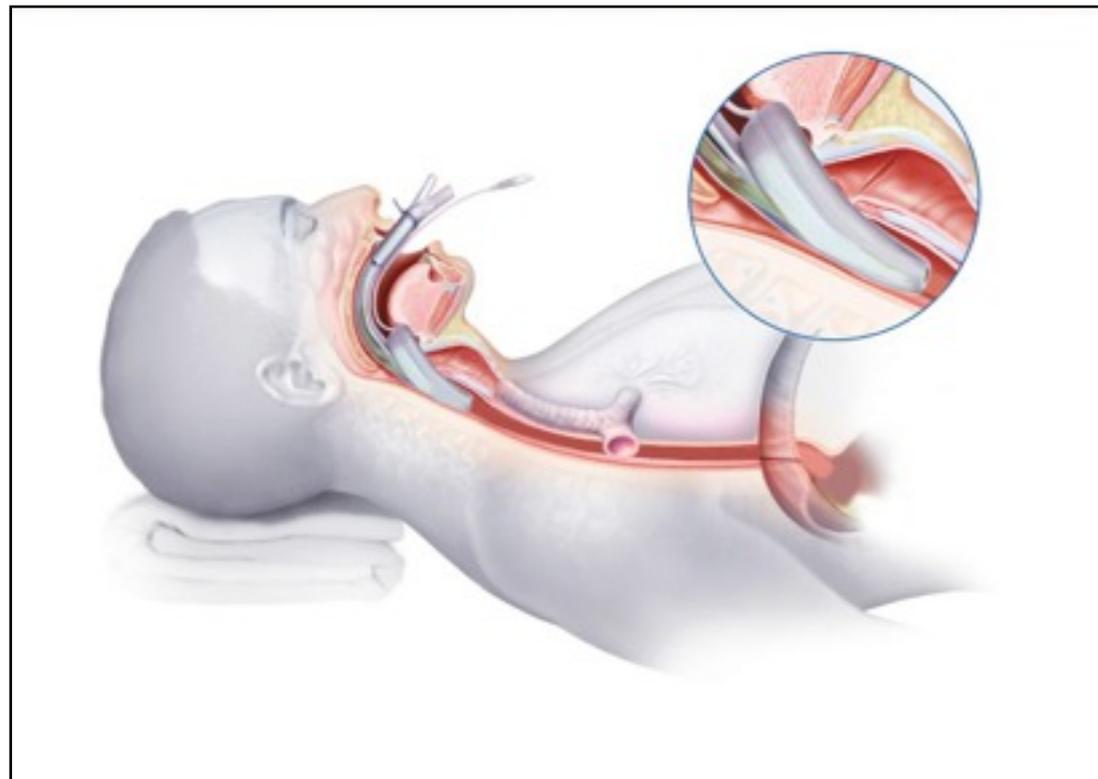
A patient who is deemed to be at risk of aspiration requires that the airway be “protected” with a cuffed endotracheal tube regardless of the nature of the surgery. If the surgery requires a paralyzed patient, then in most cases the patient is intubated to allow mechanical ventilation.

Mask Airway: Bag mask ventilation may be used to assist or control ventilation during the initial stages of a resuscitation or to pre-oxygenate a patient as a prelude to anesthetic induction and intubation. A mask airway may be used as the sole airway technique during inhalational anesthesia (with the patient breathing spontane-

ously) but it is only advisable for relatively short procedures as it “ties up” the anesthesiologist’s hands. It does not protect against aspiration or laryngospasm (closure of the cords in response to noxious stimuli at light planes of anesthesia). Upper airway obstruction may occur, particularly in obese patients or patients with very large tongues. In current practice, the use of a mask as a sole airway technique for anesthesia is rarely-seen although it may be used for very brief procedures in the pediatric patient.

Laryngeal Mask Airway (LMA): The LMA is an airway device that is a hybrid of the mask and the endotracheal tube. It is inserted blindly into the hypopharynx. When properly positioned with its cuff inflated, it sits above the larynx and seals the glottic opening ([Figure 5](#)). It is usually used for spontaneously breathing patients but positive pressure ventilation can be delivered through an LMA. The LMA does not protect against aspiration. Like an endotracheal tube, it frees up the anesthesiologist’s hands and allows surgical access to the head and neck area without interference. While airway obstruction due to laryngospasm is still a risk, the LMA prevents upper airway obstruction from the tongue or other soft tissues. The LMA also has a role to play in the failed intubation setting particularly when mask ventilation is difficult. The #3, #4 and #5 LMA are used in adults. Many modifications have followed the origi-

Figure 5 Laryngeal mask in situ



Images courtesy of the LMA Group of Companies, 2012. Used with permission. Images modified by Karen Raymer and Brian Colborne.

nal “classic” LMA including a design that facilitates blind endotracheal intubation through the LMA (Fastrach LMA™) and one that is specially designed for use with positive pressure ventilation with or without muscle relaxation (Proseal LMA™).

Endotracheal Intubation: There are 3 basic indications for intubation:

1. To provide a patent airway. An endotracheal tube (ETT) may be necessary to provide a patent airway as a result of either patient or surgical factors (or both). For example, an ETT is required to provide a patent airway when surgery involves the oral cavity (e.g. tonsillectomy, dental surgery). An ETT provides a patent airway when the patient must be in the prone position for spinal surgery. Airway pathology such as tumour or trauma may compromise patency, necessitating an ETT.
2. To protect the airway. Many factors predispose a patient to aspiration. A cuffed endotracheal tube, although not 100% reliable, is the best way to protect the airway of an anesthetized patient.
3. To facilitate positive pressure ventilation. Some surgical procedures, by their very nature, require that the patient be mechanically ventilated which is most effectively and safely achieved via an ETT. Mechanical ventilation is required when:

- the surgery requires muscle relaxation (abdominal surgery, neurosurgery).
- the surgery is of long duration such that respiratory muscles would become fatigued under anesthesia.
- the surgery involves the thoracic cavity.

In rare cases, an ETT may be required to improve oxygenation in patients with critical pulmonary disease such as Acute Respiratory Distress Syndrome (ARDS), where 100% oxygen and positive end expiratory pressure (PEEP) may be needed.

While intubation is most commonly performed orally, in some settings nasotracheal intubation is preferable such as during intra-oral surgery or when long-term intubation is required. Nasotracheal intubation may be accomplished in a blind fashion (i.e. without performing laryngoscopy) in the emergency setting if the patient is breathing spontaneously.

Nasotracheal intubation is contraindicated in patients with coagulopathy, intranasal abnormalities, sinusitis, extensive facial fractures or basal skull fractures.

While there are myriad devices and techniques used to achieve intubation (oral or nasal), most often it is performed under direct vision using a laryngoscope to expose the glottis. This technique is called direct laryngoscopy. The patient should first be placed in the “sniffing position” ([Figure 3](#)) in order to align the oral, pha-

Movie 1.1 Intubation technique



Video filmed and produced by Karen Raymer and Brian Colborne; Find this video at www.understandinganesthesia.ca

ryngeal and laryngeal axes. The curved Macintosh blade is most commonly used in adults. It is introduced into the right side of the mouth and used to sweep the tongue to the left ([Figure 6](#)).

The blade is advanced into the vallecula which is the space between the base of the tongue and the epiglottis. Keeping the wrist stiff to avoid levering the blade, the

laryngoscope is lifted to expose the vocal cords and glottic opening. The ETT is inserted under direct vision through the cords. A size 7.0 or 7.5 ETT is appropriate for oral intubation in the adult female and a size 8.0 or 8.5 is appropriate in the male. A full size smaller tube is used for nasal intubation.

[Movie 1.1](#) demonstrates the important technique to use when performing endotracheal intubation.

The view of the larynx on laryngoscopy varies greatly. A scale represented by the “Cormack Lehane views” allows anesthesiologists to grade and document the view that was obtained on direct laryngoscopy. Grade 1 indicates that the entire vocal aperture was visualized; grade 4 indicates that not even the epiglottis was viewed. [Figure 7](#) provides a realistic depiction of the range of what one might see when performing laryngoscopy.

[Movie 1.2](#) shows you the important anatomy to recognize on a routine intubation.

After intubation, correct placement of the ETT must be confirmed and esophageal intubation ruled out. The “gold standard” is direct visualization of the ETT situated between the vocal cords. The presence of a normal, stable end-tidal carbon dioxide (CO₂) waveform on the capnograph confirms proper placement except in the cardiac arrest setting. Both sides of the chest and the epigastrium are auscultated for air entry. Vapour observed moving in and out of the ETT is supportive but not confirmative of correct tracheal placement.

If the ETT is advanced too far into the trachea, a right mainstem intubation will occur. This is detected by noting the absence of air entry on the left as well as by observing that the ETT has been advanced too far. The appropriate distance of ETT insertion, measured at the lips, is approximately 20 cm for an adult female and 22 cm for the adult male.

Complications may occur during laryngoscopy and intubation. Any of the upper airway structures may be traumatized from the laryngoscope blade or from the endotracheal tube itself. The most common complication is damage to teeth or dental prosthetics. It is imperative to perform laryngoscopy gently and not to persist with multiple attempts when difficulty is encountered. Hypertension, tachycardia, laryngospasm, raised intracranial pressure and bronchospasm may occur if airway manipulation is performed at an inadequate

depth of anesthesia. Sore throat is the most common complication that presents post-extubation and is self-limited. Airway edema, sub-glottic stenosis, vocal cord paralysis, vocal cord granulomata and tracheomalacia are some of the more serious consequences that can occur and are more common after a prolonged period of intubation.

Airway Devices and Adjuncts

After performing a history and physical examination and understanding the nature of the planned procedure, the anesthesiologist decides on the anesthetic technique. If a general anesthetic is chosen, the anesthesiologist also decides whether endotracheal intubation is indicated or whether another airway device such as a LMA could be used instead.

When endotracheal intubation is planned, the technique used to achieve it depends in large part on the assessment of the patient's airway. When intubation is expected to be routine, direct laryngoscopy is the most frequent approach. In settings where the airway management is not routine, then other techniques and adjuncts are used. Airway devices that can be used to achieve an airway (either as a primary approach or as a "rescue" method to use when direct laryngoscopy has failed) are categorized below.

- Methods for securing the upper airway only. These methods achieve what is sometimes termed a "non-invasive airway" and include the oral airway with mask; the LMA; and the King Laryngeal Tube™.
- Adjuncts for increasing the likelihood of achieving endotracheal intubation through direct laryngoscopy: alternate laryngoscope blades, endotracheal

introducers (commonly referred to as gum elastic bougies), stylet.

- Methods of achieving endotracheal intubation using "indirect" visualization of the larynx: videolaryngoscope, (the Glidescope™, McGrath™); Bullard™ laryngoscope, fiberoptic bronchoscope.
- Methods of achieving endotracheal intubation in a "blind" fashion (without visualization of the larynx): blind nasal intubation, lighted stylet, retrograde intubation, Fastrach LMA™.

The Difficult Airway

Airway mismanagement is a leading cause of anesthetic morbidity and mortality and accounts for close to half of all serious complications. The best way to prevent complications of airway management is to be prepared. Anticipation of the difficult airway (or difficult intubation) and formulation of a plan to manage it when it occurs, saves lives.

Anticipated difficult intubation: The use of an alternate anesthetic technique (regional or local) may be the most practical approach. If a general anesthetic is chosen, then airway topicalization and awake intubation (with fiberoptic bronchoscope) is the preferred technique. In pediatric patients, neither a regional technique nor an awake intubation is feasible. In this case, induction of anesthesia with an inhaled agent such that the patient retains spontaneous respiration is the safest approach. Efforts are undertaken to secure the airway once the child is anesthetized.

Unanticipated difficult intubation, able to ventilate by mask: In this situation, one calls for help, repositions the patient and reattempts laryngoscopy. The guiding principle is to avoid multiple repeated attempts which can lead to airway trauma and edema resulting in the loss of the ability to ventilate the patient. During the subsequent attempts at intubation, the anesthesiologist considers using alternate airway tech-

niques ([see section on adjuncts](#)) or awakening the patient to proceed with an awake intubation.

Unanticipated difficult intubation, unable to ventilate by mask: This is an emergency situation. One calls for help and attempts to insert an LMA which is likely to facilitate ventilation even when mask ventilation has failed. If an airway is not achievable by non-surgical means, then a surgical airway (either needle cricothyrotomy or tracheostomy) must not be delayed.

When a difficult airway is encountered, the anesthesiologist must respond quickly and decisively. As in many clinical situations which occur infrequently but are associated with high rates of morbidity and mortality, the management of the difficult airway is improved by following well-developed algorithms. The American Society of Anesthesiologists has published a "Difficult Airway Algorithm" which is widely accepted as standard of care. The algorithm is described in a lengthy document such that a full explanation is beyond the scope of this manual. The algorithm, as well as other experts' interpretations, are readily available on the internet.

Fluid Management

In This Section

1. Fluid Requirements
2. Assessment of Fluid Status
3. Vascular Access
4. Types of Fluid

The goal of fluid management is the maintenance or restoration of adequate organ perfusion and tissue oxygenation. The ultimate consequence of inadequate fluid management is hypovolemic shock.

Fluid Requirements

Peri-operative fluid management must take into account the pre-operative deficit, ongoing maintenance requirements and intra-operative losses (blood loss, third space loss).

Pre-operative Deficit: The pre-operative fluid deficit equals basal fluid requirement (hourly maintenance x hours fasting) plus other losses that may have occurred during the pre-operative period.

Maintenance fluid requirements correlate best with lean body mass and body surface area. To calculate maintenance, use the “4/2/1 rule”:

First 10 kilograms (i.e. 0-10 kg):	4 cc/kg/hr
Next 10 kilograms (i.e. 11-20 kg):	2 cc/kg/hr
All remaining kilograms over 20 kg:	1 cc/kg/hr

For example, a 60 kg woman fasting for 8 hours:

10 kg x 4 cc/kg/hr	= 40 cc/hr
10 kg x 2 cc/kg/hr	= 20 cc/hr
+ 40 kg x 1 cc/kg/hr	= 40 cc/hr
	= 100 cc/hr x 8 hr
	= 800 cc

Therefore, the pre-operative deficit (excluding other losses) is 800 cc.

“Other losses” (including fluid lost through sweating, vomiting, diarrhea and nasogastric drainage) are more difficult to estimate. In the febrile patient, maintenance requirements are increased by 10% per degree Celsius elevation in temperature.

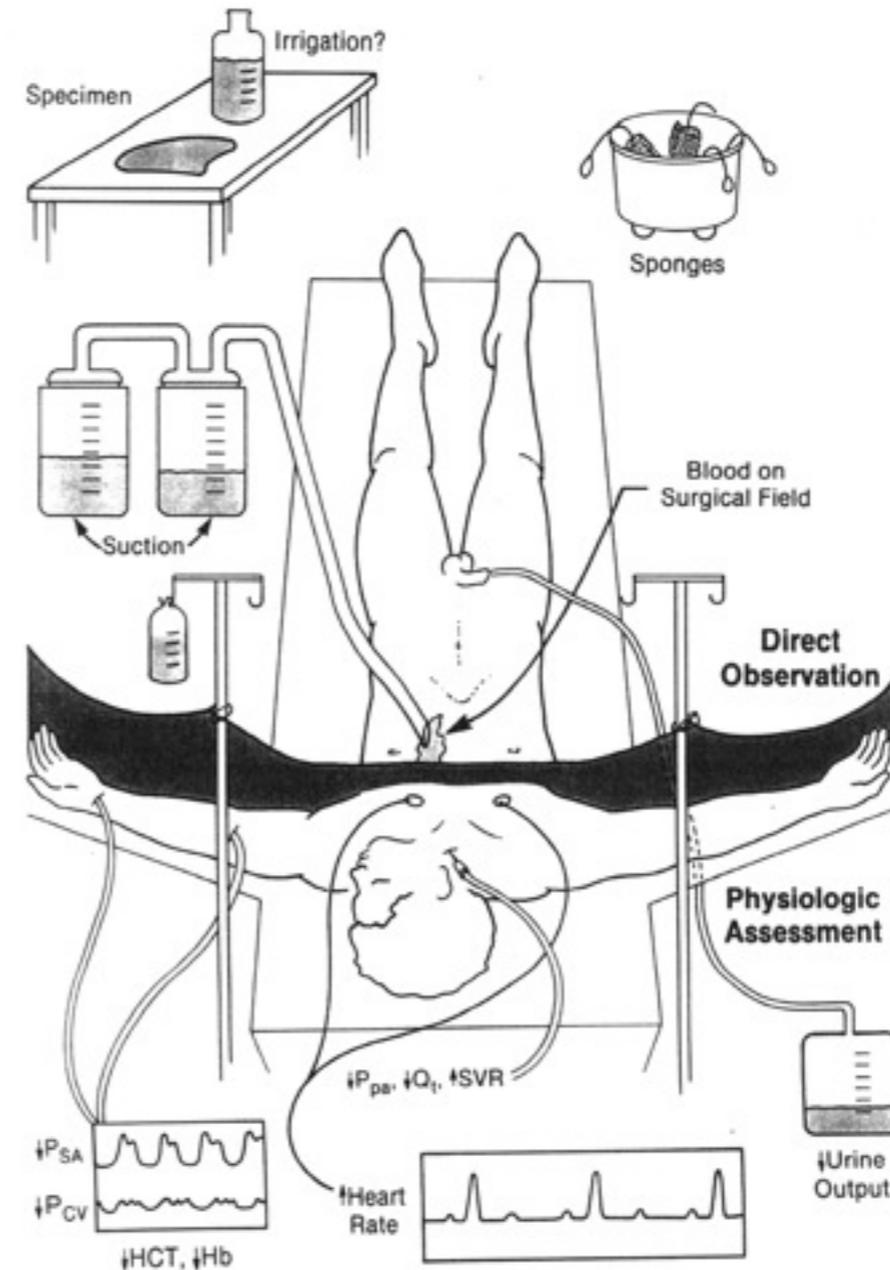
As a rule, half of the deficit should be corrected prior to induction and the remainder replaced intra-operatively. However, if the pre-operative deficit is greater than 50% of the estimated blood

volume, then the surgery should be delayed, if possible, to allow for more complete resuscitation.

Intra-operative losses: Blood loss is usually underestimated. It is assessed by visually inspecting blood in suction bottles, on the drapes and on the floor. Sponges can be weighed (1 gram = 1 cc blood), subtracting the known dry weight of the sponge. Third space loss refers to the loss of plasma fluid into the interstitial space as a result of tissue trauma and can be estimated based on the nature of the surgery:

- 2-5 cc/kg/hr for minimal surgical trauma (orthopedic surgery)
 - 5-10 cc/kg/hr for moderate surgical trauma (bowel resection)
 - 10-15 cc/kg/hr for major surgical trauma (abdominal aortic aneurysm repair)
- These are all crude estimates of fluid requirements. Adequacy of replacement is best judged by the patient's response to therapy. Urine output greater than 1.0 cc/kg/hr is a reassuring indicator of adequate organ perfusion. Hemodynamic stability, oxygenation, pH and central venous pressures are other indicators of volume status, but may be affected by many other factors. [Figure 8](#) depicts the holistic approach to assessing intra-operative blood loss.

Figure 8 Assessment of intra-operative fluid status



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Assessment of Fluid Status

Fluid status is assessed by history, physical exam and laboratory exam. Thorough history will reveal losses of blood, urine, vomit, diarrhea and sweat. As well, the patient is questioned regarding symptoms of hypovolemia, such as thirst and dizziness.

On physical exam, vital signs, including any orthostatic changes in vital signs, are measured. A decrease in pulse pressure and decreased urine output are two of the most reliable early signs of hypovolemia. Poor capillary refill and cutaneous vasoconstriction indicate compromised tissue perfusion. Severely depleted patients may present in shock ([Table 3](#)).

Hemoglobin, sodium, urea and creatinine levels may show the concentration effect which occurs in uncorrected dehydration. When blood loss occurs, hemoglobin and hematocrit levels remain unchanged until intravascular volume has been restored with non-blood containing solutions. Therefore, only after euvolemia has been restored is the hemoglobin level a useful guide for transfusion. Lactic acidosis is a late sign of impaired tissue perfusion.

Table 3 Classification of hemorrhagic shock in a 70 kg person

	CLASS 1	CLASS 2	CLASS 3	CLASS 4
BLOOD LOSS (cc)	<750	750-1500	1500-2000	>2000
BLOOD LOSS (%)	<15	15-30	30-40	>40
PULSE RATE	<100	100-120	120-140	>140
BLOOD PRESSURE	normal	orthostatic drop	decreased	decreased
PULSE PRESSURE	normal	decreased	decreased	decreased
RESPIRATORY RATE	14-20	20-30	30-40	>40
URINE OUTPUT	>30 cc/hr	20-30 cc/hr	<20 cc/hr	negligible
CNS	normal	anxious	confused	lethargic
FLUID REQUIRED	crystalloid	plus colloid	plus blood	plus blood

Vascular Access

Peripheral venous access

Peripheral venous access is the quickest, simplest and safest method of obtaining vascular access. The upper limb is used most commonly, either at the hand or ante-cubital fossa (cephalic and basilic veins). The lower limb can be used if necessary, the most successful site here being the saphenous vein, located 1 cm anterior and superior to the medial malleolus.

Flow through a tube is directly proportional to the pressure drop across the tube and inversely proportional to resistance.

$$\text{Flow} \propto \text{pressure drop} / \text{resistance}$$

Resistance is directly proportional to length and inversely proportional to radius to the fourth power.

$$\text{Resistance} \propto \text{length} / \text{radius}^4$$

From these equations, we can understand how the anesthesiologist achieves rapid administration of fluids. Pressure drop is achieved by using rapid infusers that apply a squeeze to the fluid, usually with an air-filled bladder. A cannula that is of a greater radius makes a significant impact on flow; to a lesser extent, a shorter

cannula allows greater flow than a longer cannula of equivalent bore.

For example, a 16 gauge cannula will allow greater flow (i.e. faster resuscitation) than a (smaller) 18 gauge cannula. Likewise, a 14 gauge peripheral IV cannula will allow greater flow than an equivalent caliber central line, which is, by necessity, significantly longer. From a practical perspective, a 16 gauge cannula is the smallest size which allows rapid administration of blood products.

Starting a peripheral intravenous line

There are several technical points that, when followed, will increase your likelihood of success with “IV starts”. These are itemized below and demonstrated in the video, available for viewing on the website.

- 1) Apply a tourniquet proximal to the site. Apply it tightly enough to occlude venous flow, but not so tightly as to impede arterial flow to the limb.
- 2) Choose an appropriate vein: one that is big enough for the cannula you have chosen and for your fluid administration needs. However, just because a vein is big, doesn't mean it is the best for the IV start. Avoid veins that are tortuous as well as ones with obvious valves. In these cases, threading the cannula will be difficult.
- 3) Prep the area with alcohol.

- 4) Immobilize the vein by applying gentle traction to the surrounding skin with your left hand. Avoid pulling too tightly on the skin, lest you flatten the vein entirely.
- 5) Hold the cannula between the thumb and third fingers of your right hand.
- 6) Approach the vein with the IV cannula in your right hand at an angle that is nearly parallel to the skin. You want to travel within the lumen of the vein, not go in one side and out the other. Another important requirement is to ensure that the planned approach allows the trajectory of the cannula to be identical to the trajectory of the vein. (Once you get more confident with IV starts, you may choose to plan your "puncture site" to be not immediately overlying the vein itself, so that when the IV cannula is ultimately removed, the overlying skin provides natural coverage to the hole in the vein, minimizing bleeding.)
- 7) Watch for the flashback. When you get it, do *not* move your left hand. Just take a breath. Then slowly advance both needle and catheter together within the lumen of the vein, anywhere from 2-4 mm (more with a larger IV cannula). This step ensures that the tip of the catheter (not just the needle) is in the lumen of the vein. Be careful to observe the anatomy of the vein to guide your direction of advancement.
- 8) Thread the catheter using your index finger of your right hand. Your thumb and third finger continue to stabilize the needle in place (stationary). Your left hand continues to stabilize the vein's position. (This part takes lots of practice!)
- 9) Once the catheter is fully threaded, then you can release your left hand which now can be used to release the tourniquet and apply proximal pressure at the IV site.
- 10) Pull out your needle and attach the prepared IV line.
- 11) Secure your IV with appropriate dressing and carefully dispose of your sharp needle.
- 12) Congratulations!

Movie 1.3 Technique for peripheral IV start



This video was filmed by Victor Chu and edited by Karen Raymer. Find this video at www.understandinganesthesia.ca

Central venous access

Central venous access is indicated when peripheral venous access is inadequate for fluid resuscitation, or when central pressure monitoring is required. The internal jugular vein is the most common site used intraoperatively. The external jugular is also useful, but can be technically difficult in some patients due to the presence of valves. The subclavian site is associated with an increased risk of pneumothorax, while the femoral site is associated with an increased risk of infection, embolism and thrombosis. Multiorifaced, 6 c.m., 14 gauge catheters are the most commonly used central lines. Wide bore “introducers” (for example, the 8.5 French Arrow CV Introducer®) are also commonly used for central venous access.

There are many potential complications of central venous cannulation. They include arterial puncture, hemorrhage, pneumothorax, thoracic duct injury, neural injury, air embolism, infection, thrombosis, hydrothorax, catheter misplacement and catheter or wire embolism. The use of ultrasound guidance for central line insertion allows more accurate needle placement and avoidance of complications.

Types of Fluids

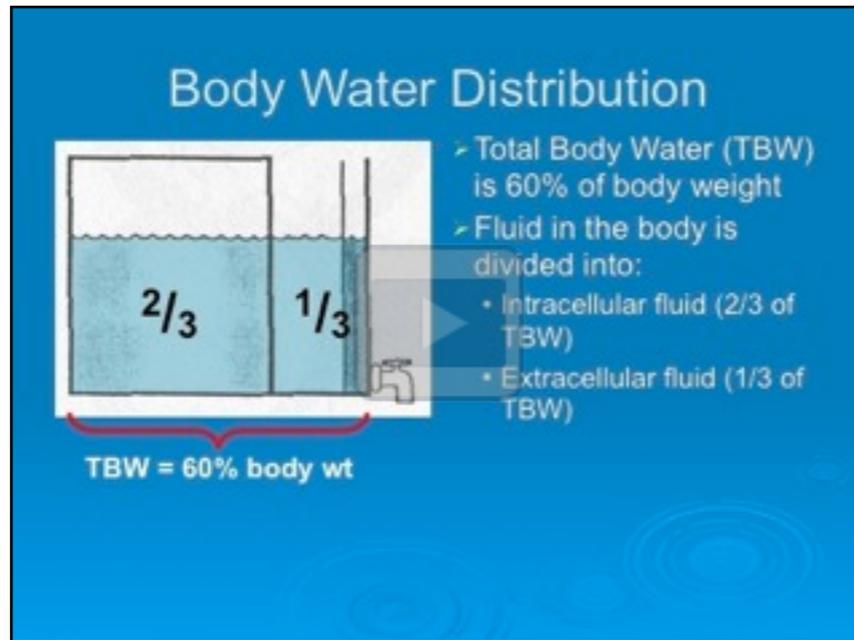
Fluids can be divided into two broad categories: crystalloids and colloids. Crystalloids are solutions of simple inorganic or organic salts and distribute to varying extents throughout the body water. Examples include Ringer’s Lactate (R/L), 0.9% saline (N/S) and 5% dextrose in water (D5W). Sodium chloride, a common constituent of crystalloid solutions, distributes throughout the entire extracellular space. Glucose distributes throughout the entire body water (extracellular and intracellular spaces). Whatever the active solute, water, the ubiquitous solvent, will move across membranes to maintain osmotic equilibrium.

Colloids are suspensions of protein or other complex organic particles. These particles cannot diffuse across capillary membranes and so remain trapped within the intravascular space. Examples of colloids are albumin (5%, 25%), hydroxyethyl starches (Pentaspán®, Voluven®), red cell concentrates, platelets, and plasma.

The partitioning throughout the body’s compartments of some of the various types of fluids for administration is summarized in [Table 4](#) and illustrated in the animated slides, [Interactive 1.1](#).

Normal saline or Ringer’s lactate are the preferred crystalloids for intra-operative fluid administration and resuscitation, as they provide more intravascular volume

Interactive 1.1 Body water distribution and fluid management



“Tank of water” concept and drawing developed by Dr. Kinsey Smith. Animated slides created by Dr. Rob Whyte. Slides used with permission from both. Find this slideshow at www.understandinganesthesia.ca

expansion than D5W or 2/3:1/3. Because of the partitioning in the extracellular compartment, they must be given in a 3-4:1 ratio to the estimated blood loss. Administration of large amounts of N/S results in metabolic acidosis and should be avoided. R/L contains 4 meq/L potassium, and should be avoided in patients with renal failure. Glucose-containing solutions should only be used for specific indications (such as to maintain stable glucose levels in patient with diabetes melli-

tus or hepatic disease), and should be based on known glucose requirements. Finally, the half-life of crystalloid redistribution is only 15-30 minutes, so it must be given at a rate that accounts for its extravasation from the intravascular space.

Colloids replace blood loss in a 1:1 ratio, assuming normal membrane permeability. The use of colloids is gen-

Table 4 Partitioning of various intravenous fluid solutions

TYPE OF FLUID	ECF=1/3 TBW		ICF = 2/3 TBW
	IVF=1/4 ECF	IF=3/4 ECF	ICF
1000 cc D5W	83 cc	250 cc	667 cc
1000 cc 2/3 :1/3	139 cc	417 cc	444 cc
1000 cc N/S OR R/L	250 cc	750 cc	0
500 cc 5% ALBUMIN	500 cc	0	0
100 cc 25% ALBUMIN	500 cc	-400 cc	0
500 cc PENTASPAN	750 cc	-250 cc	0
1 UNIT RCC	400 cc	0	0

TBW = total body water, ECF = extracellular fluid, ICF = intracellular fluid, IVF = intravascular fluid, IF = interstitial fluid, RCC= red cell concentrates.

erally reserved for cases where greater than 20% of the blood volume needs to be replaced or when the consequences of the interstitial edema (which might occur with crystalloid administration) are serious (e.g. cerebral edema).

Blood products are administered for specific indications. Red cell concentrates (RCC) are given to maintain or restore oxygen carrying capacity. As hemoglobin (Hb) concentration falls, oxygen delivery is preserved by compensatory mechanisms: shifting the oxy-hemoglobin dissociation curve to the right and increasing cardiac output (via an increase in heart rate and contractility). When these compensations are inadequate or detrimental, RCC should be transfused. General indications for the transfusion of blood products are outlined in [Table 5](#).

A patient with Class 3 or 4 hemorrhagic shock ([Table 3](#)) should be transfused immediately. For the slow but steady blood loss which occurs during many types of surgery, the lowest allowable hemoglobin, the “transfusion trigger”, is determined on an individual basis. Healthy patients can tolerate Hb levels that are approximately ½ of normal (60-70 g/L). Compensations may be inadequate in patients with pulmonary, cardiac or cerebrovascular disease. Compensation may be harmful in patients with certain types of heart disease such as severe coronary artery disease or aortic steno-

sis. These patients should be transfused to relatively higher Hb levels (80-100 g/L).

Once the lowest allowable hemoglobin has been determined, then the allowable blood loss (ABL) can be calculated as follows:

$$\frac{(\text{Hb initial} - \text{Hb allowable}) \times \text{EBV}}{\text{Hb initial}}$$

Estimated blood volume (EBV) is approximately 60-70 mL/kg in the adult. When blood loss approaches estimated ABL, the anesthesiologist confirms the current Hb and considers transfusing.

Transfusion of plasma, platelets or cryoprecipitate is indicated only for the correction of defective clotting and is not indicated for volume resuscitation. Impaired clotting may be observed or anticipated in a given clinical scenario. For example, after one blood volume of RCC has been transfused (6-12 units in an adult), coagulopathy is likely on a dilutional basis and transfusion of platelets and plasma will be required. Prolonged clotting times or thrombocytopenia alone, without clinical evidence of bleeding, are insufficient indications for transfusion.

Risks and benefits of transfusion should be explained to patients undergoing procedures likely to result in sig-

nificant blood loss. Consent for transfusion should be obtained whenever possible.

Complications of transfusion are numerous and are generally categorized by acuity: early and late. Early complications that can occur when significant volumes of blood are transfused include hypothermia, hyperkalemia and hypocalcemia. With massive transfusion, lung injury may occur.

Transfusion reactions can occur with just a single unit of transfused blood (due to ABO incompatibility). The most common cause of transfusion reaction is clerical error, underscoring the need for careful adherence to safety procedures by all members of the healthcare team. A more complete discussion of the indications and complications of the various blood products is beyond the scope of this manual. Many excellent reviews on the subject can be found in the current anesthesia literature.

Table 5 Indications for blood product administration

BLOOD PRODUCT	DEFICIT
red cell concentrates	oxygen-carrying capacity
platelets	platelet function (quality or quantity)
fresh frozen plasma	clotting factor deficits
cryoprecipitate	fibrinogen
albumin	low protein or colloid volume replacement
factor concentrates	single clotting factor deficit (often hereditary)

CHAPTER 2

The Pre-operative Phase



In this chapter, you will learn how the anesthesiologist assesses a patient who is scheduled to undergo anesthesia and surgery, and how the goal of risk modification is achieved through that process. As well, you will be introduced to the equipment required for the safe delivery of anesthesia: the anesthetic machine and monitors. Find review questions at www.understandinganesthesia.ca

Pre-operative Evaluation

In This Section

1. Pre-operative Visit
2. NPO Status
3. Premedication

Pre-operative Visit

The purpose of the pre-operative assessment is three-fold:

- *To review the medical and psychological status of the patient.*
- *To identify factors which may impact on the peri-operative course, to take measures to optimize those factors where possible, and to delay surgery if necessary.* If the patient's medical condition cannot be altered, then one can take other measures to attempt to reduce risk: substitute a lower-risk surgical procedure, modify the anesthetic technique, intensify the peri-operative monitoring or cancel the surgery altogether.
- *To inform patient, alleviate anxiety and establish rapport.*

This evaluation takes the form of a directed history, physical examination and laboratory exam. On history, the anesthesiologist attempts to elicit symptoms of cardiac or respiratory disease as well as a history of any other major medical illnesses, past or present. Hepatic or renal disease

may impact on metabolism and excretion of anesthetic agents, fluid balance and coagulation status. The patient's medications are reviewed including any history of adverse drug reactions. The patient's and their relative's previous anesthetic experience is reviewed.

The physical examination focuses on the cardiac and respiratory (including airway) systems. Recording baseline vital signs is important, as is detecting any unstable, potentially reversible conditions such as congestive heart failure or bronchospasm. The airway is assessed for ease of intubation.

Routine pre-op laboratory investigations have not been shown to improve patient outcome. Therefore, laboratory studies are ordered only as indicated, according to the medical status of the patient and the nature of the planned surgery. Studies are rarely ordered to establish a "baseline" but rather to detect abnormalities that require correction prior to surgery. The traditional "CBC and urinalysis" is no longer required in healthy patients having minor surgery. An electrocardiogram (ECG) is ordered on patients who

are known to have cardiac disease or in whom risk factors (including age) are present. Routine pre-operative chest x-rays are not required prior to most procedures.

The anesthesiologist will commonly assign an “ASA class” (Table 6) to the patient. The ASA (American Society of Anesthesiologists) classification was defined in the 1940’s as an attempt to identify operative risk. As the patient’s underlying health is the most important determinant of peri-operative risk, the ASA class does correlate (somewhat) to overall peri-operative risk. Though it does not lend itself to inter-rater reliability, it is an accepted method of communicating the overall physical condition of the patient and the learner should become accustomed to applying this scale to the patients he or she encounters.

Table 6 ASA classification

ASA CLASS	DESCRIPTION
1	A normal healthy patient in need of surgery for a localized condition.
2	A patient with mild to moderate systemic disease; examples include controlled hypertension, mild asthma.
3	A patient with severe systemic disease; examples include complicated diabetes, uncontrolled hypertension, stable angina.
4	A patient with life-threatening systemic disease; examples include renal failure or unstable angina.
5	A moribund patient who is not expected to survive 24 hours with or without the operation; examples include a patient with a ruptured abdominal aortic aneurysm in profound hypovolemic shock.

NPO Status

The induction of anesthesia abolishes the normal laryngeal reflexes that prevent inhalation (“aspiration”) of stomach contents. Due to gastric, biliary and pancreatic secretions (which are present even during fasting), a stomach is never “empty”. NPO (*nil per os*) indicates the restriction of oral intake for a period of time prior to surgery, minimizing the volume, acidity and solidity of stomach contents. Such measures reduce both the risk of aspiration occurring as well as the severity of pneumonitis, should an aspiration event occur.

For elective surgery, patients should not have solid food for 8 hours prior to anesthesia. Traditionally, patients were ordered to refrain from all fluids for the 8 hour pre-operative period as well. However, more recent studies have shown that the time of the last (clear) fluid intake bears little relation to the volume of gastric contents present at the induction of anesthesia. Thus, most institutions are allowing unrestricted intake of clear fluids until 2-4 hours prior to scheduled surgery. Guidelines for pediatric patients vary from institution to institution but generally are more liberal than in the adult population. For example, infants may be allowed breast milk up to 4 hours pre-operatively and formula up to 6 hours pre-operatively.

It is important to recognize that some patients remain at risk for aspiration despite strict application of NPO

guidelines. Known risk factors are outlined in [Table 7](#). When it is possible to identify these patients pre-operatively, measures can be taken to reduce their risk of aspiration syndrome. Firstly, pre-medication can be given to increase gastric motility (metoclopramide) or to decrease gastric acidity (ranitidine or sodium citrate). Risk can also be reduced through careful airway management that may include the use of the Sellick Maneuver on its own or as part of a rapid sequence induction.

Table 7 Risk factors for aspiration

RISK FACTORS FOR ASPIRATION
<ul style="list-style-type: none">• Gastroesophageal reflux• Pregnancy• Trauma• Diabetes Mellitus• Recent oral intake• Bowel obstruction• Intra-abdominal pathology• Obesity

Premedication

Pre-medication can include medication that the patient takes routinely as well as medication that may be prescribed specifically for the pre-operative period. Generally speaking, patients should be given their usual medication on the morning of surgery with a sip of water. It is particularly important that patients receive their usual cardiac and antihypertensive medications pre-operatively. Discontinuation of beta-blockers, calcium channel blockers, nitrates or alpha-2 agonists (clonidine) can lead to rebound hypertension or angina. Similarly, most medications taken for chronic disease should be continued on the morning of surgery as well as throughout the peri-operative period. This is particularly important for most antidepressants, thyroid replacement and anticonvulsants.

There are certain medications that may need to be discontinued in the pre-operative period. Examples include monoamine oxidase inhibitors and anticoagulants. Patients on platelet inhibitors such as aspirin represent a special group of patients who must be considered on an individual basis such that the risk of stopping the aspirin is weighed against the risk of surgical-site bleeding. For example, a patient who is on aspirin because of the recent insertion of a coronary stent must receive their aspirin throughout the peri-operative period. On the other hand, if the patient is on aspirin for

primary prevention then it is usually discontinued a full week before surgery to allow return of normal platelet function.

Some medications are ordered specifically for the pre-operative period. Examples include anxiolytics, antibiotics, bronchodilators, anti-anginal medication and anti-emetics. Beta blockers have been used to reduce the incidence of cardiac morbidity and mortality in high-risk patients undergoing high-risk procedures, although the impact of this intervention is not yet fully understood.

Currently, pre-operative sedation is used less frequently than it has been in the past as it can delay awakening at the end of anesthesia. A delayed recovery is particularly undesirable in the outpatient surgical population where a return of cognitive function is required prior to discharge home. Furthermore, a pre-operative visit has been shown to be at least as effective as pharmacologic means in allaying anxiety in surgical patients. Nonetheless, there is a role for pre-operative sedation in very anxious patients or in those for whom anxiety would be deleterious, such as the cardiac patient.

For most types of surgery, antibiotics are ordered pre-operatively to reduce the incidence of wound infection. Antibiotics may also be ordered to reduce the risk of bacterial endocarditis in at-risk patients though the cur-

rent recommendations from the American Heart Association are much more restrictive than they have been in the past.

As discussed, aspiration prophylaxis may be ordered in high risk patients. This includes agents which decrease the volume and/or acidity of gastric secretions (ranitidine, sodium citrate) as well as agents which increase gastric emptying (metoclopramide).

A history of systemic steroid use may require the delivery of a peri-operative course of steroids in order to avoid the consequences of adrenal suppression which may present as an Addisonian crisis. Adrenal suppression occurs when a patient receives longterm exogenous steroids in daily dose equal to or greater than 10 mg. Once adrenal suppression has occurred, the adrenal gland takes approximately 3 months to recover function (after steroid discontinuation). Therefore, steroid supplementation is required for patients who are currently on exogenous steroids or have discontinued a longterm course in the past three months. The amount and duration of supplemental steroid coverage required depends on the invasiveness of the surgery. For minor surgery, a single dose of hydrocortisone (25 mg) suffices, while for major surgery, the patient requires 100 mg of hydrocortisone daily for 2-3 days.

Anesthetic Equipment and Monitoring

In This Section

1. The Anesthetic Machine
2. Monitoring

The Anesthetic Machine

The purpose of the anesthetic machine is to deliver gases to the patient in precise, known concentrations. Although the anesthetic machine has evolved substantially over the years, the essential features have remained remarkably constant. Some of the important components of a modern anesthetic machine are depicted in [Interactive 2.1](#). (Tap the labels for a close-up view as well as a brief description of each component.)

Gases (oxygen, air and nitrous oxide) come from pipelines entering the operating room through the wall ([Figure 9](#)). Tanks on the back of the anesthetic machine provide an alternate source of those gases should the wall supply fail. Although 100% oxygen can be delivered to the patient, usually a mixture of oxygen (with air or nitrous oxide) is selected. The relative concentrations of the gases to be delivered are controlled by flowme-

ters (one flowmeter for each gas) found on the left hand side of the anesthetic machine.

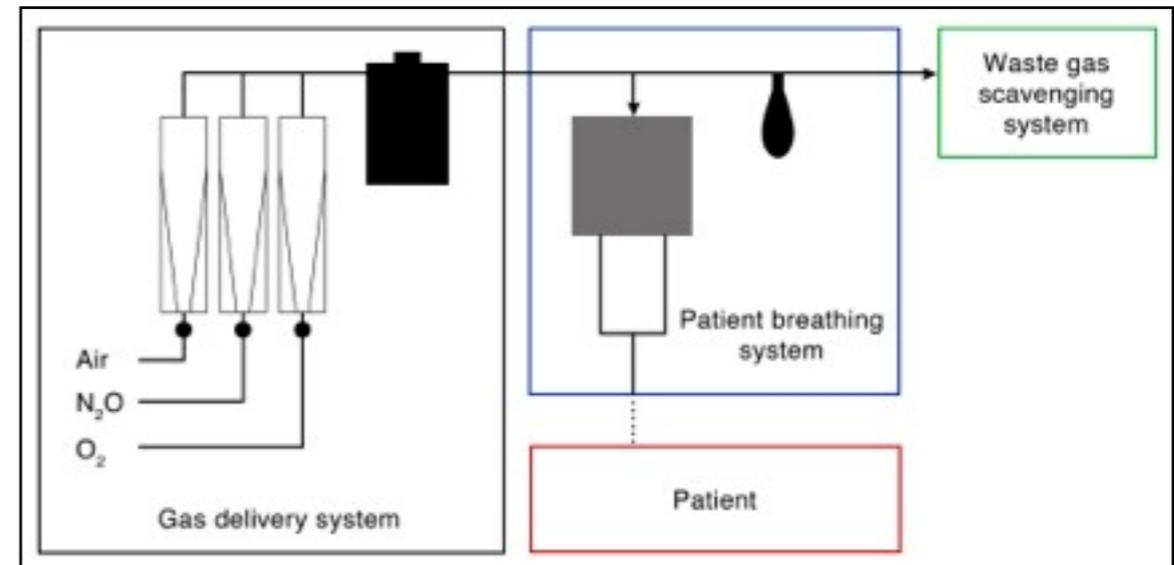
The anesthetic machine also allows the delivery of a precise concentration of volatile agent. The volatile anesthetic gases, such as sevoflurane and desflurane, are contained in liquid form in the vaporizers mounted on the machine. The gas mixture from the flowmeters flows through the vaporizer and the volatile anesthetic agent is added to the mixture in gaseous form. The concentration of the volatile gas in the final mixture is determined by a dial on or near the vaporizer. For safety reasons, only one volatile agent can be delivered at a time.

The ventilator allows positive pressure ventilation of the anesthetized patient. The ventilator can be set to deliver a specific tidal volume (in which case pressure varies according to lung compliance) or to achieve a certain peak inspiratory pressure (in which case volume varies ac-

ording to lung compliance). The ventilator moves the gas mixture through the common gas outlet and into the anesthetic circuit, the tubing that connects to the patient's airway. The vast majority of general anesthetics today are delivered through a circle system. The circle circuit has a CO₂ absorber, a canister containing a hydroxide mixture (soda lime) that absorbs CO₂. The absorption of CO₂ allows the expired gas to be recycled, thus minimizing the excessive cost and pollution that would otherwise result. There are several other types of circuits which are useful in specific clinical situations or are of historical interest. The origin and pathways of gas flow that applies to most anesthetic machines is depicted in schematic form in [Figure 9](#).

It is imperative that all anesthesia equipment undergo regular checks and maintenance. It is the responsibility of the anesthesiologist to ensure that the equipment is in functioning condition prior to the administration of every anesthetic. The pre-operative checklist can be found on every anesthetic machine.

Figure 9 Pathway of gas flow in anesthetic machine



The shaded shapes represent (from left to right): volatile anesthetic vaporizer, ventilator and bag used for bag-mask ventilation. Image by Wikimedia user TwoOneTwo, available under the Creative Commons Attribution-Share Alike 3.0 Licence. Image modified by Emma Kolesar.

Monitoring

The purpose of monitoring during anesthesia is to ensure the maintenance of homeostasis. The best single monitor is a vigilant anesthesiologist. The practice of anesthesia involves the use of some key monitors that are not commonly seen in other health care settings. Examples include the pulse oximeter, the capnograph and the peripheral nerve stimulator. The Canadian Anesthesia Society guidelines for intra-operative monitoring are listed in [Table 8](#).

In some settings, depending on the patient status or the nature of the procedure, monitoring beyond the routine measures listed above may be deemed necessary. There are methods of invasively monitoring the cardiovascular, renal and central nervous systems in the peri-operative period. Examples include arterial catheter, pulmonary artery catheter, transesophageal echocardiography, Foley catheter and 16-channel EEG monitoring.

Table 8 The Canadian Anesthesia Society guidelines for intra-operative monitoring

Monitors which must be continuously used:

- pulse oximeter
- apparatus to measure blood pressure
- electrocardiography
- capnograph when an endotracheal tube or laryngeal mask is in use
- agent-specific anesthetic gas monitor when inhaled anesthetic agents are used

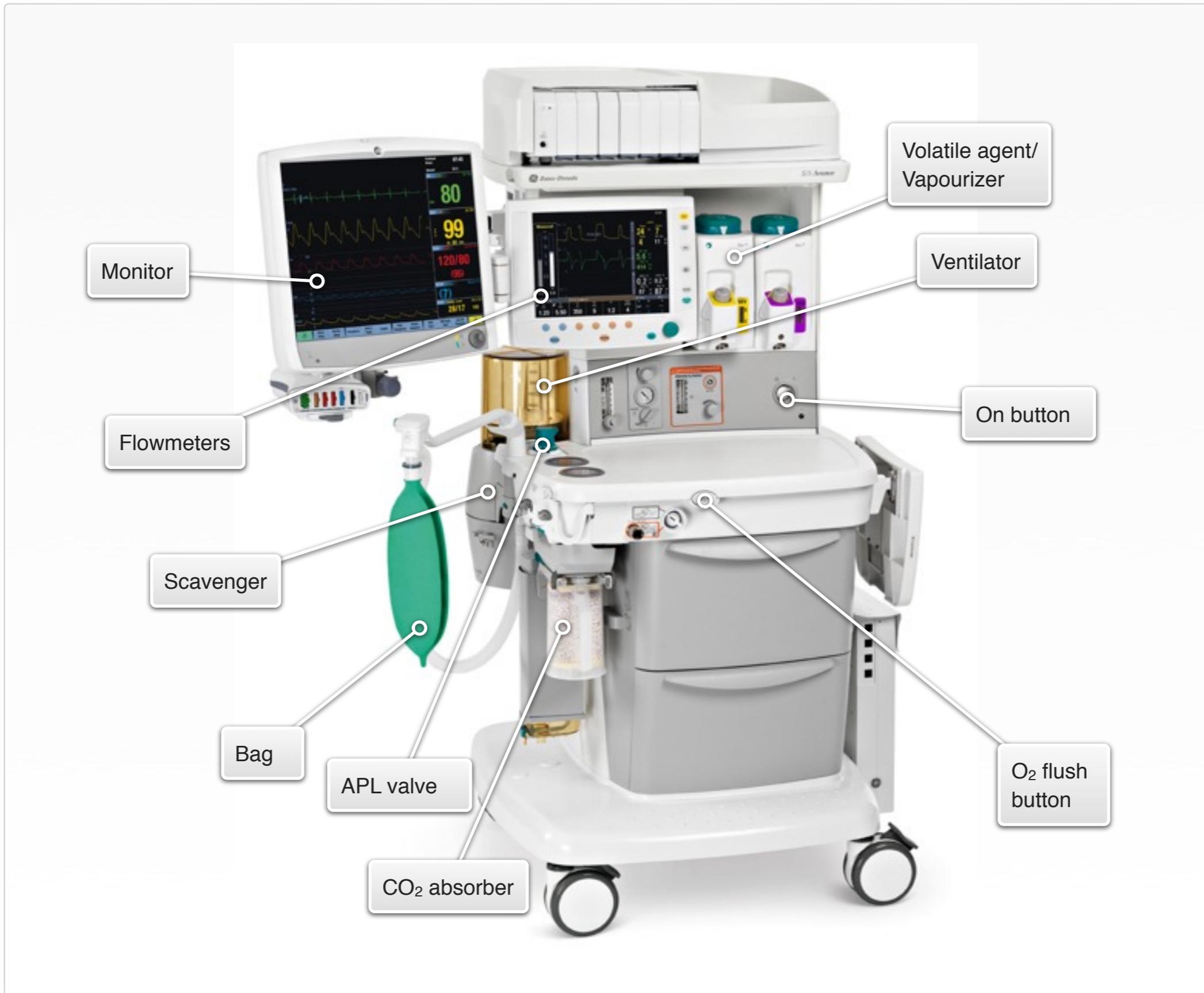
Monitors which must be exclusively available:

- apparatus to measure temperature
- peripheral nerve stimulator (when neuromuscular blockers are used)
- stethoscope (precordial or esophageal)
- visualization of exposed portion of patient with adequate lighting

Monitors which must be immediately available:

- spirometer for measurement of tidal volume

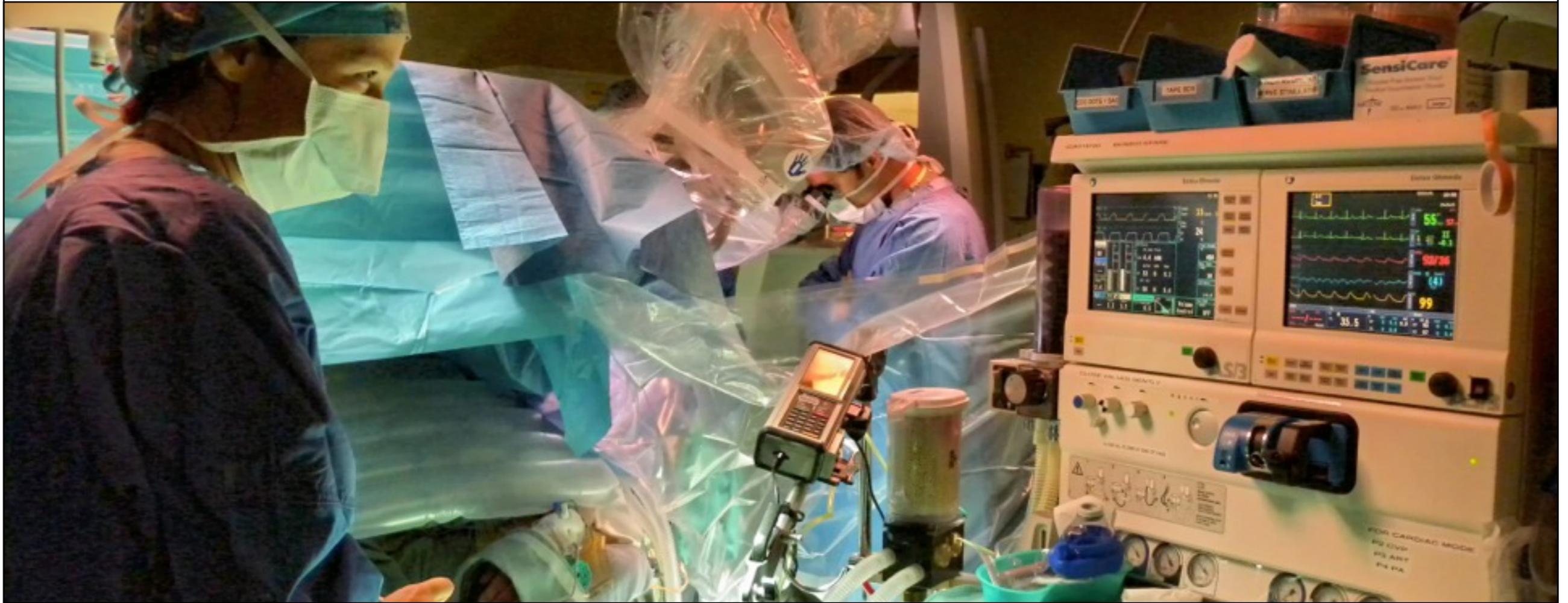
Interactive 2.1 Anesthesia machine



Photograph of anesthetic machine used with permission of GE Healthcare. Interactive (available on iBook only) created by Karen Raymer

CHAPTER 3

The Intra-operative Phase



In this chapter, you will be presented an overview of the range of techniques that can be used to provide anesthesia. Regional and general anesthesia are discussed in greater detail. The pharmacology of each of the important drugs used in the delivery of anesthesia can be found in the “Drug Finder” (Chapter 6). Review questions available at www.understandinganesthesia.ca

Anesthetic Techniques

In This Section

1. Local Anesthesia
2. Sedation
3. Regional Anesthesia
4. General Anesthesia

Except in the most desperate of circumstances, surgical procedures are performed with the benefit of anesthesia. There are four types of anesthesia that may be employed alone or in combination:

- local
- sedation (minimal, moderate or deep)
- regional
- general

The findings on pre-operative assessment, the nature of the surgery and the patient's preference all factor into the choice of anesthetic technique. Contrary to popular belief, studies have failed to identify one technique as superior (lower morbidity and mortality) to the others *in a general patient population*. Regardless of the technique employed, the anesthesiologist must ensure patient comfort, maintenance of physiologic homeostasis and provision of adequate operating conditions.

Local Anesthesia

Local anesthesia refers to the infiltration of a local anesthetic agent at the surgical site and is usually performed by the surgeon. This technique is appropriate for superficial procedures such as dental surgery, breast biopsy or carpal tunnel release. Local anesthesia may be used in an unmonitored setting. However, often it is used in combination with sedation in which case monitoring is required. While local anesthesia is inadequate for more invasive procedures such as those involving the body cavities, local infiltration is often used as an adjunct in post-operative pain management. Care must be taken to avoid intravascular injection and to avoid exceeding the toxic dose of the local anesthetic in use.

Sedation

Sedation involves the delivery of agents (usually intravenous) for the purpose of achieving a calm, relaxed patient, able to protect his own airway and support his own ventilation. The range of physiologic effects of sedation is varied and is dependent on the depth of sedation provided: minimal, moderate or deep. A patient under minimal sedation will be fully responsive to verbal commands although his or her cognitive functions and coordination would be impaired. He or she would appear calm and relaxed and would have normal cardiorespiratory function. At the other extreme, a patient receiving deep sedation would be rousable only to repeated or painful stimuli. In some instances, the patient may require assistance in maintaining a patent airway. In this case, the line between deep sedation and general anesthesia is not easily identifiable. [Table 9](#) outlines the physiologic effects of each of the three levels of sedation as defined by the American Society of Anesthesiologists (ASA). Sedation may be used alone for minimally painful procedures such as endoscopy. Often it is used in combination with local or regional anesthesia to provide a more palatable experience for the patient. In any case, the sedated patient must be monitored due to the depressant effects of the agents used. Care must be taken to reduce the dose administered to the frail, elderly or debilitated patient, in whom depressant effects may be exaggerated. In any patient, sedation may

cause disinhibition, resulting in an uncooperative, agitated patient.

Many different agents have been used for sedation. The term “neurolept anesthesia” refers to the (now historical) use of high doses of droperidol (a butyrophenone, in the same class as haloperidol) in combination with fentanyl (an opioid). Side effects were prominent. Currently, agents are chosen with specific effects in mind. Opioids, such as fentanyl or remifentanyl, may be given alone if analgesia is the primary goal. The short-acting benzodiazepine, midazolam, is a popular choice because it provides amnesia as well as anxiolysis. Propofol, an anesthetic induction agent, can be infused in sub-anesthetic doses to produce a calm, euphoric patient.

Table 9 Sedation

	VERBAL RESPONSE	COGNITIVE FUNCTION	AIRWAY PATENCY	RESPIRATORY FUNCTION	CARDIOVASCULAR FUNCTION
Light Sedation	Normal	Conscious, but cognitive function and coordination would be impaired	Normal	Normal	Normal
Moderate Sedation	Would respond purposefully to verbal commands	Depressed consciousness	Normal	Normal	Normal
Deep Sedation	Not easily aroused; verbal response only to painful or repeated physical stimuli	Depressed consciousness	Airway might require support to remain patent	Spontaneous ventilation may be inadequate	Usually maintained

Regional Anesthesia

Regional anesthesia involves the blockade (with local anesthetics) of the nerve supply to the surgical site. This may be achieved by blocking peripheral nerves (e.g. ankle block) or by blocking the spinal cord and/or nerve roots (spinal, epidural block). A single nerve block may be sufficient (e.g. ulnar nerve block for repair of 5th digit) or a group of nerves may need to be blocked (e.g. brachial plexus block for forearm fasciotomy). While regional techniques are perceived to be “safer” than general anesthesia, they do carry risks of their own. The most serious “early” complication of a peripheral nerve block is local anesthetic toxicity. The most worrisome “late” complication is neuropraxis or nerve injury. The central neuraxial blocks have many potential complications, both early and late, which will be discussed in the next section.

There are some patients in whom a regional technique offers at least short term benefits over general anesthesia. For example, in those undergoing total hip arthroplasty, the use of spinal or epidural anesthesia is associated with less intra-operative blood loss, less post-operative hypoxemia and a lower risk of post-operative deep venous thrombosis formation. While it seems intuitive that physiologic homeostasis is more readily achieved when regional anesthesia is employed, the anesthesiologist must always remain vigilant: numerous

case reports of sudden cardio-respiratory arrest in patients under central nerve blocks emphasize this point all too clearly. Principles of pre-operative assessment and preparation must be applied just as vigilantly to the patient undergoing regional anesthesia.

General Anesthesia

General anesthesia is a pharmacologically-induced, reversible state of unconsciousness. It may involve the use of intravenous agents, inhaled agents or both. The goals and techniques of general anesthesia are discussed in an upcoming chapter. General anesthesia may be used alone or in combination with local anesthesia or a regional technique. An example of such a “combined technique” would be the use of epidural and general anesthesia in a patient undergoing an abdominal aortic aneurysm repair. Such a technique allows the continued use of the epidural for post-operative pain management and may confer a lower morbidity and mortality in high risk patients.

Regional Anesthesia

In This Section

1. Introduction
2. Epidural Anesthesia
3. Spinal Anesthesia
4. Intravenous Regional Block (Bier Block)
5. Brachial Plexus Block

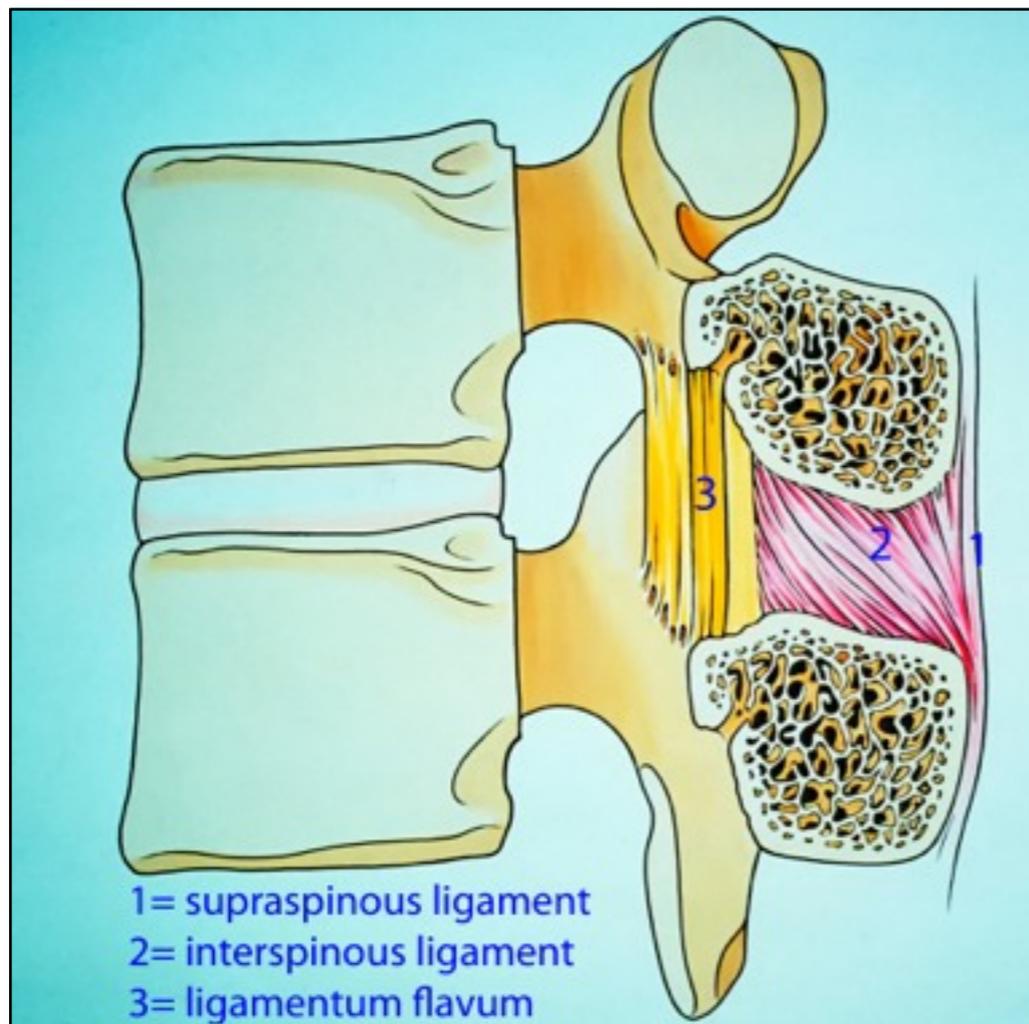
Many different types of surgical procedures can be performed while the nerves that supply the surgical site are rendered insensate. This method is called “neural blockade” or “nerve block”. Various techniques of neural blockade comprise what is known as regional anesthesia. During regional anesthesia, the anesthesiologist must not only monitor and manage the patient’s physiologic status but he or she must ensure that the patient remains calm and cooperative. The anesthesiologist must be alert to the development of complications and must also be prepared to convert to a general anesthetic at any point in the procedure.

A complete description of the scope of regional anesthesia is beyond the scope of this textbook. A brief discussion of four commonly-used techniques is presented below.

Epidural Anesthesia

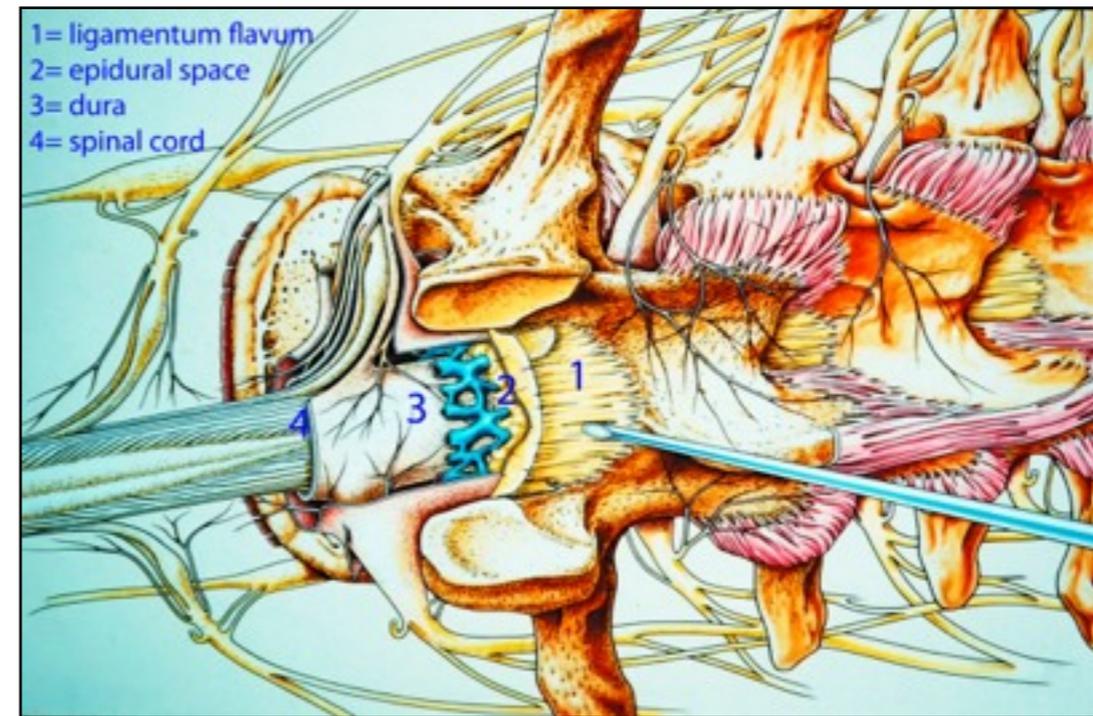
Epidural and spinal anesthesia can be used for procedures involving the abdomen, perineum or lower extremities. The two blocks differ by virtue of the anatomic space into which local anesthetic (LA) is delivered. Understanding the anatomy of the region ([Figure 10](#), [Figure 11](#)) is crucial to understanding the blocks.

Figure 10 Ligamentous anatomy of the spine



From "Introduction to Regional Anaesthesia" by D. Bruce Scott (1989). Used with permission from his wife, Joan and son, Nicholas B. Scott.

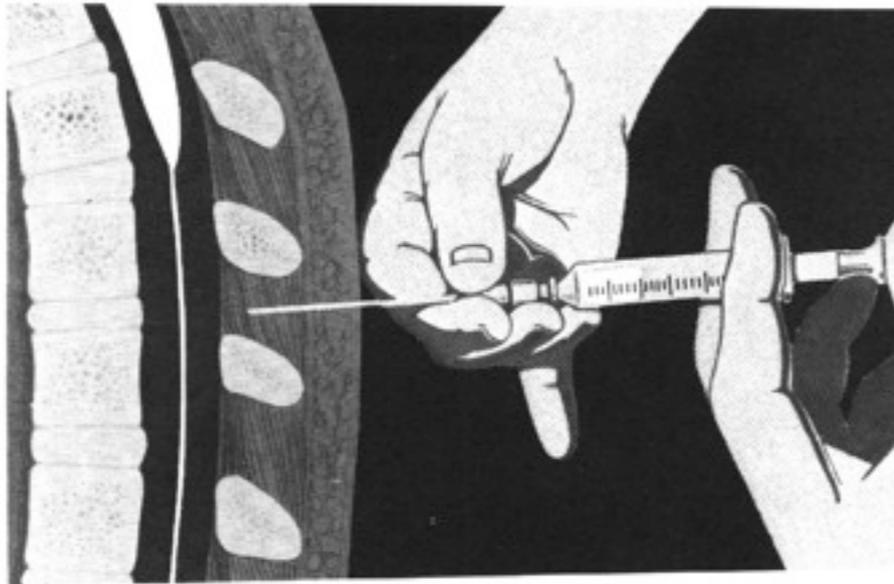
Figure 11 Anatomy relevant to epidural anesthesia



From "Introduction to Regional Anaesthesia" by D. Bruce Scott (1989). Used with permission from his wife, Joan and son, Nicholas B. Scott.

In epidural anesthesia, a tiny plastic catheter is placed into the epidural space, which is the anatomic space located just superficial to the dura. An epidural catheter can be placed at any point along the spinal column. Epidural catheters placed for surgical anesthesia or analgesia are most commonly used at the thoracic or lumbar regions depending on the site of the surgery. A LA solution is delivered through the catheter. From the epidural space, it is slowly absorbed into the subarachnoid space where it blocks the nerves of the spinal cord

Figure 12 Insertion of Tuohy needle into epidural space



Reproduced with permission from Astra Pharma Inc.

Figure 13 Insertion of epidural catheter



Reproduced with permission from Astra Pharma Inc.

and cauda equina. The volume of anesthetic delivered and the site of the catheter determine the level or “height” of the block. The presence of an indwelling catheter allows the block to be extended in height or duration as required.

Insertion of an epidural catheter is done in a strictly sterile fashion. After local infiltration, a specially designed 17 or 18 gauge epidural needle (common trade names Tuohy® or Hustead®) is inserted into the spinous interspace. A special syringe, filled with air or saline is attached to the hub of the needle. While advancing the needle, the anesthesiologist maintains pressure on the syringe in order to sense the resistance of the tissue being traversed ([Figure 12](#)). The epidural space is a “potential space” such that when it is entered with the needle, a sudden loss of resistance is detected. The syringe is then removed so that a catheter can be threaded through the needle into the epidural space ([Figure 13](#)), after which the needle is removed. A labour epidural insertion can be viewed in [Movie 3.1](#).

This movie demonstrates two frequent challenges faced by the anesthesiologist. The first is the difficulty of inserting an epidural in the presence of a dermal tattoo. Inserting an epidural through tattooed skin is undesirable as it may bring a plug of ink into the epidural space, the consequences of which are not known. In this case, the anesthesiologist is able to locate a small

Movie 3.1 Labour epidural insertion



Procedure videotaped and presented with patient permission. Produced by Karen Raymer. This video is available only in the ibook.

area of non-inked skin just slightly off midline, which provides satisfactory access for catheter insertion. The second challenge is performing a technical procedure in a patient who is in active labour. In this case, the anesthesiologist pauses while the patient is having contractions. The patient is able to do an excellent job of remaining still, which is quite important during this delicate procedure. As you are watching the video, watch carefully for the moment of the “loss of resistance”, when the gentle pressure on the hub of the syringe finally gives way, as the needle has entered the “potential” space that is the epidural space.

Once the catheter is in place, the anesthesiologist “tests” the catheter to ensure that neither blood nor cerebral spinal fluid (CSF) can be aspirated. While delivering LA in small (3 ml) aliquots, the anesthesiologist observes for symptoms or signs of incorrect catheter placement. If the catheter is situated in one of the veins of the epidural plexus then the patient may experience symptoms of local anesthetic toxicity: tinnitus, perioral numbness, metallic taste in the mouth and dizziness. If the catheter is situated in the intrathecal space, then the patient will develop a sensory/motor block rapidly after the administration of only a small amount of LA.

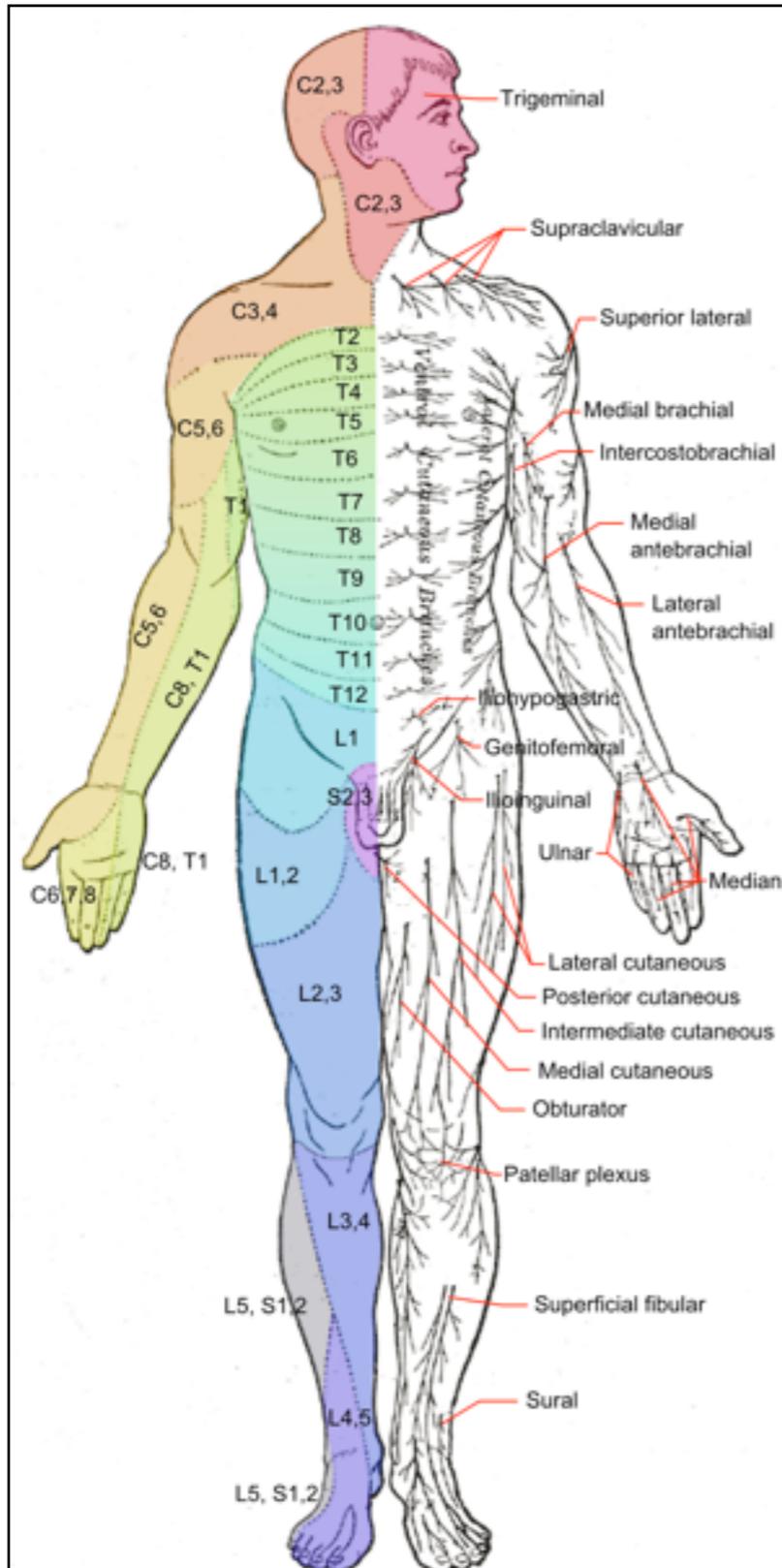
In the absence of any signs or symptoms of incorrect catheter placement, the full dose of LA is delivered over 10-20 minutes. Some LA’s, such as lidocaine, have

a relatively rapid onset of effect but also have a relatively shorter duration of action. Bupivacaine, while possessing a slower onset of effect, has a longer duration of action. The dermatomal level of block is tested by pinprick or ice cube ([Figure 14](#)). It generally takes 20-30 minutes for an adequate epidural block to take effect. The required dose is determined by the range of segments which must be blocked. The higher the surgical site is, the higher the block must be. [Table 10](#) describes the dermatomal level of block required for some of the more common surgical procedures which apply to both spinal and epidural anesthesia. Many other factors influence the amount of local anesthetic required. For example, in pregnancy, there is a significant reduction in the required volume of LA delivered to achieve

Table 10 Required dermatomal levels for various surgical procedures

SURGICAL PROCEDURE	REQUIRED LEVEL OF BLOCK
Caesarian section	T4
inguinal hernia repair	T10
repair fractured hip	L1
total knee arthroplasty (with tourniquet)	L2
hemorrhoidectomy	S4

Figure 14 Sensory dermatomes



Public domain image derived from Gray's Anatomy. Retrieved from Wikimedia Commons.

the desired block. In general, volumes in the range of 10-20 ml are required for most procedures.

Complications of epidural anesthesia can present in the early or late post-operative periods. The early complications are related either to incorrect catheter placement (LA toxicity, or "total spinal" block), to excessive volume of LA delivered (high block, with hypotension, bradycardia, respiratory compromise), or to the unavoidable blockade of sympathetic fibres (hypotension, bradycardia). Late complications are related to needle and catheter insertion, and include nerve injury, epidural abscess or hematoma, and post-dural puncture headache (if the dura is accidentally punctured).

The contraindications to epidural and spinal anesthesia are identical, and are listed in [Table 11](#).

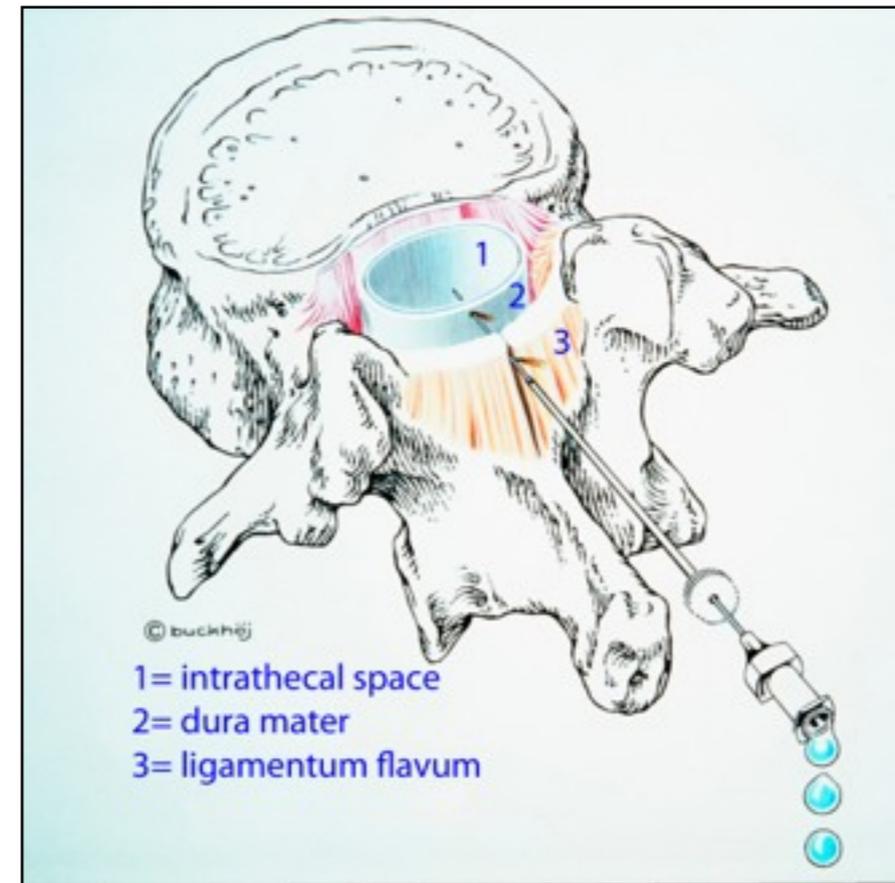
Table 11 Contraindications to central neural blockade

GENERAL	SPECIFIC (ABSOLUTE)	SPECIFIC (RELATIVE)
lack of consent	coagulopathy	evolving neurological deficit
lack of resuscitative equipment	sepsis (systemic or at site of injection)	obstructive cardiac lesion (e.g. aortic stenosis)
known or suspected allergy	increased intracranial pressure (ICP)	spinal hardware
lack of familiarity with technique	shock	
known or suspected allergy		

Spinal Anesthesia

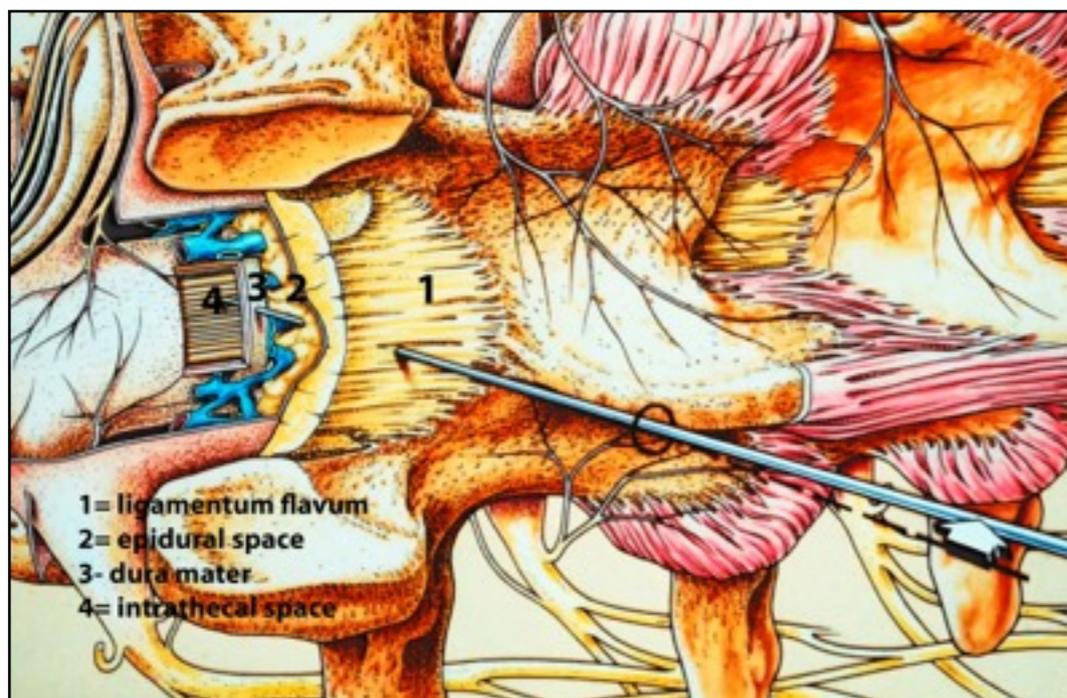
Spinal anesthesia involves the blockade of the nerves of the spinal cord and cauda equina by injection of LA into the intrathecal space. The important anatomy is depicted in [Figure 15](#) and [Figure 16](#). Because the LA is injected in close proximity to its site of action, much smaller volumes are required (1-3 ml) and the onset of effect (within 5 minutes) is rapid relative to epidural an-

Figure 15 Needle placement in spinal anesthesia



From "Introduction to Regional Anaesthesia" by D. Bruce Scott (1989). Used with permission from his wife, Joan and son, Nicholas B. Scott.

Figure 16 Anatomy relevant to spinal anesthesia



From *“Introduction to Regional Anaesthesia”* by D. Bruce Scott (1989). Used with permission from his wife, Joan and son, Nicholas B. Scott.

esthesia. The choice of LA used is based primarily on the anticipated length of procedure. Spinal lidocaine provides surgical anesthesia for procedures lasting up to 75 minutes however its use has been limited by the associated increased incidence of postoperative radiculopathies. Spinal bupivacaine will provide up to 3.5 hours of anesthesia and is considered to be safe. The addition of opioids (e.g. fentanyl) to the local anesthetic solution can extend the duration of block. However, if the block dissipates prior to the end of the procedure there is no way to extend the block at that point. Spinal

anesthesia is performed under strict asepsis. The patient may be sitting or curled in the lateral position. A special small-bore “spinal needle” is used (22-27 gauge). The needle is inserted at a lower lumbar interspace and is advanced through the dura. Because the dura is a tough membrane, a definite “pop” is often felt as the needle passes through into the intrathecal space. The stylet of the needle is removed and cerebrospinal fluid (CSF) is observed in the lumen of the needle. The local anesthetic is then injected and the needle removed. The height of the required block depends on the surgical procedure ([Table 10](#)). The height of the block achieved is determined by many factors including the mass and volume of LA administered, the position of the patient and the baricity or “heaviness” of the LA relative to CSF.

The complications of spinal anesthesia are similar to those of epidural anesthesia with a few exceptions. Because of the small dose of LA required, LA toxicity is not an issue even if intravascular injection occurs. Dural puncture is required for spinal anesthesia therefore “spinal headache” is always a risk, especially in young adult patients. The use of needles which are smaller-bore and have a “pencil-point” tip helps to decrease the incidence of post-dural puncture headache. The contraindications to spinal anesthesia are listed in [Table 11](#).

Intravenous Regional Block (Bier Block)

An intravenous regional anesthetic (IVRA), also known as a Bier Block, is used primarily for relatively short procedures on the distal upper extremity (below the elbow). The monitors are applied and intravenous access is ensured. After cannulating a vein distal to the surgical site, the operative arm is elevated and an elastic bandage is applied to promote venous drainage. After exsanguination, an upper arm tourniquet is inflated. 40-50 ml of dilute lidocaine (without epinephrine) is then injected slowly into the cannula in the operative arm. Diffusion of the local anesthetic from the venous system to the interstitium provides surgical anesthesia within 5 minutes.

The risk of LA toxicity is high especially if the tourniquet leaks or is deflated prior to 20 minutes after injection of LA. If the surgical procedure lasts less than 20 minutes then one must wait until 20 minutes has elapsed prior to deflating the tourniquet. If less than 40 minutes (but more than 20 minutes) has elapsed, then the tourniquet should be deflated and re-inflated intermittently to avoid a sudden bolus of LA entering the systemic circulation. "Tourniquet pain" becomes prominent after 45 minutes and limits the length of time that this technique can be employed.

Brachial Plexus Block

The brachial plexus is formed from the anterior primary rami of the C5-T1 nerve roots ([Figure 17](#)) and supplies all of the motor function and most of the sensory function of the upper extremity ([Figure 18](#)). Throughout their journey to the axilla, the nerve roots merge and divide numerous times ([Figure 17](#)). The nerve roots travel through the intervertebral foramina and emerge between the anterior and middle scalene muscles ([Figure 19](#)). They then travel under the clavicle and enter the axilla as three distinct cords. As they exit the axilla, the plexus divides one final time to form the axillary, radial, median, ulnar and musculocutaneous nerves. The brachial plexus block provides anesthesia for virtually any type of upper extremity surgery.

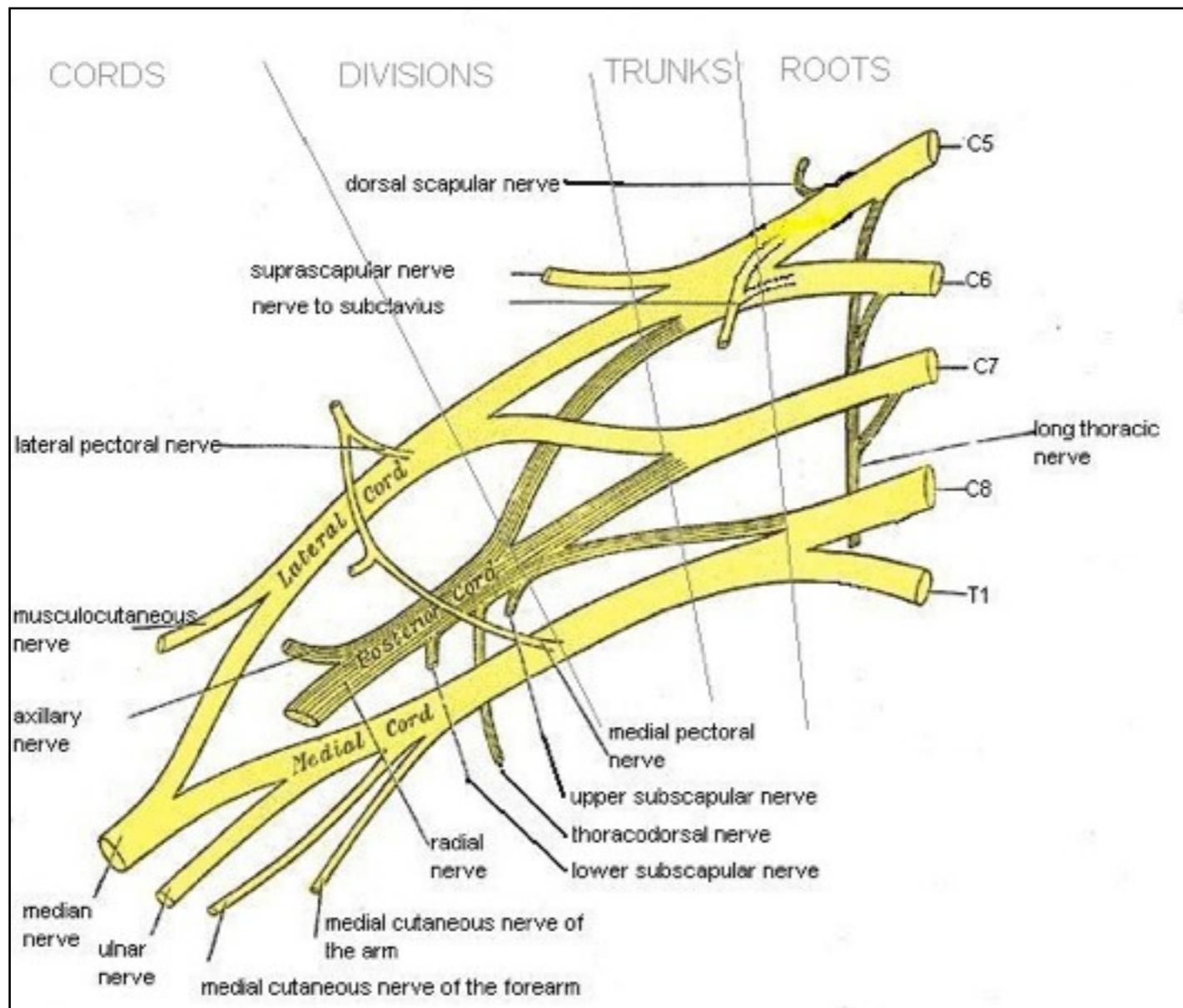
The brachial plexus can be blocked at one of its three most superficial locations: interscalene, supraclavicular or axillary. Sterile technique is used at all times and correct needle placement is ensured through the use of either ultrasound or nerve stimulator. A relatively large volume of LA is injected (30-40 ml). Up to 45 minutes may be required until adequate anesthesia is achieved, depending on how close to the nerve bundle the local anesthetic is deposited. If several hours of anesthesia are required, then bupivacaine or ropivacaine is used. A catheter can be inserted to provide surgical anesthe-

sia or post-operative analgesia for an indefinite period of time.

There are many potential complications of a brachial plexus block. LA toxicity is a risk because of the large volumes of LA used and the proximity of the injection

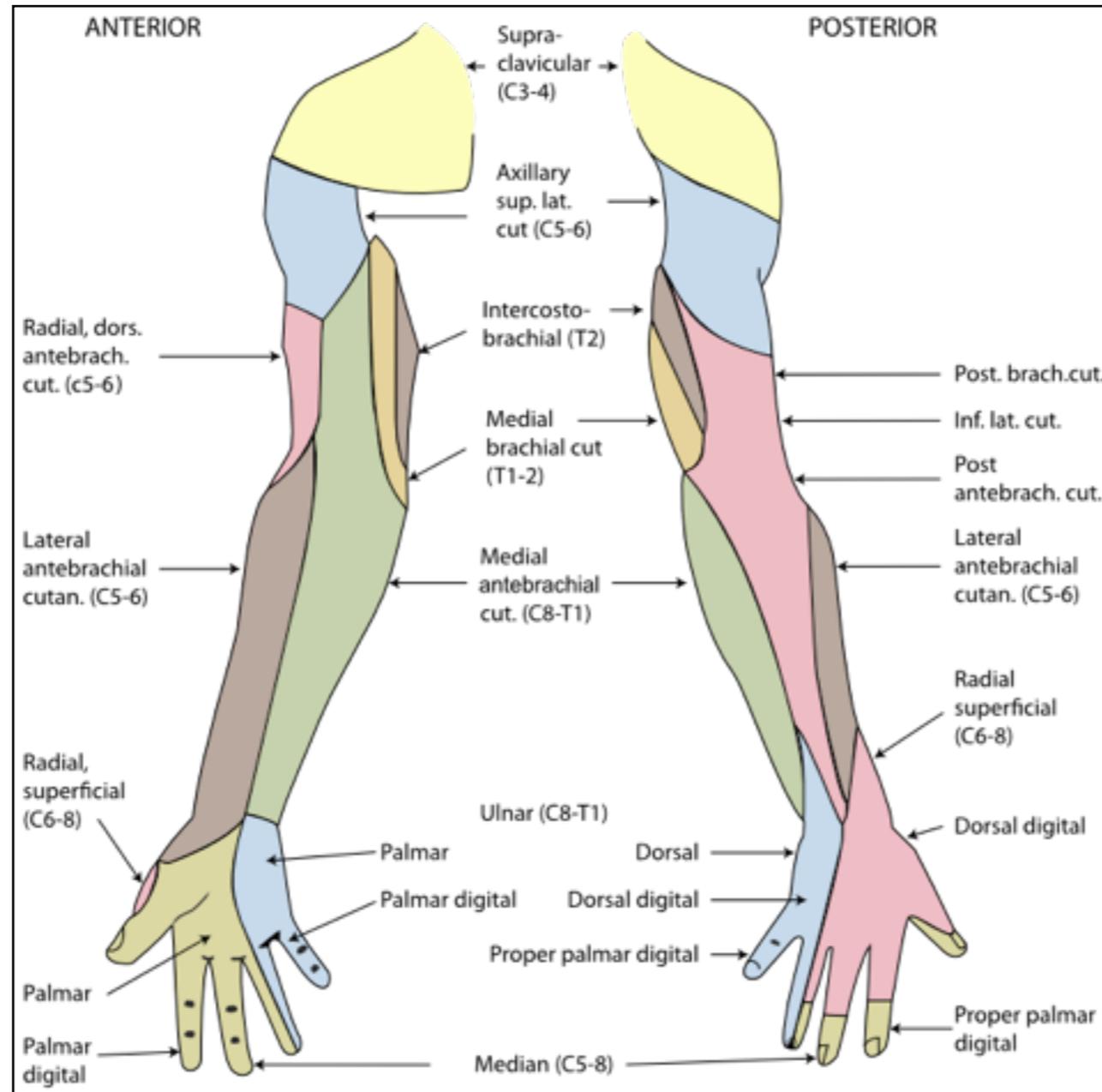
site to major vessels. Any block can lead to hematoma formation, nerve injury or infection. The supraclavicular and interscalene blocks pose the additional risks of pneumothorax, phrenic nerve block and recurrent laryngeal nerve block. Consequently, bilateral blocks are contraindicated. Intrathecal injection is a rare complication of interscalene block.

Figure 17 Brachial plexus: roots, trunks, divisions, cords



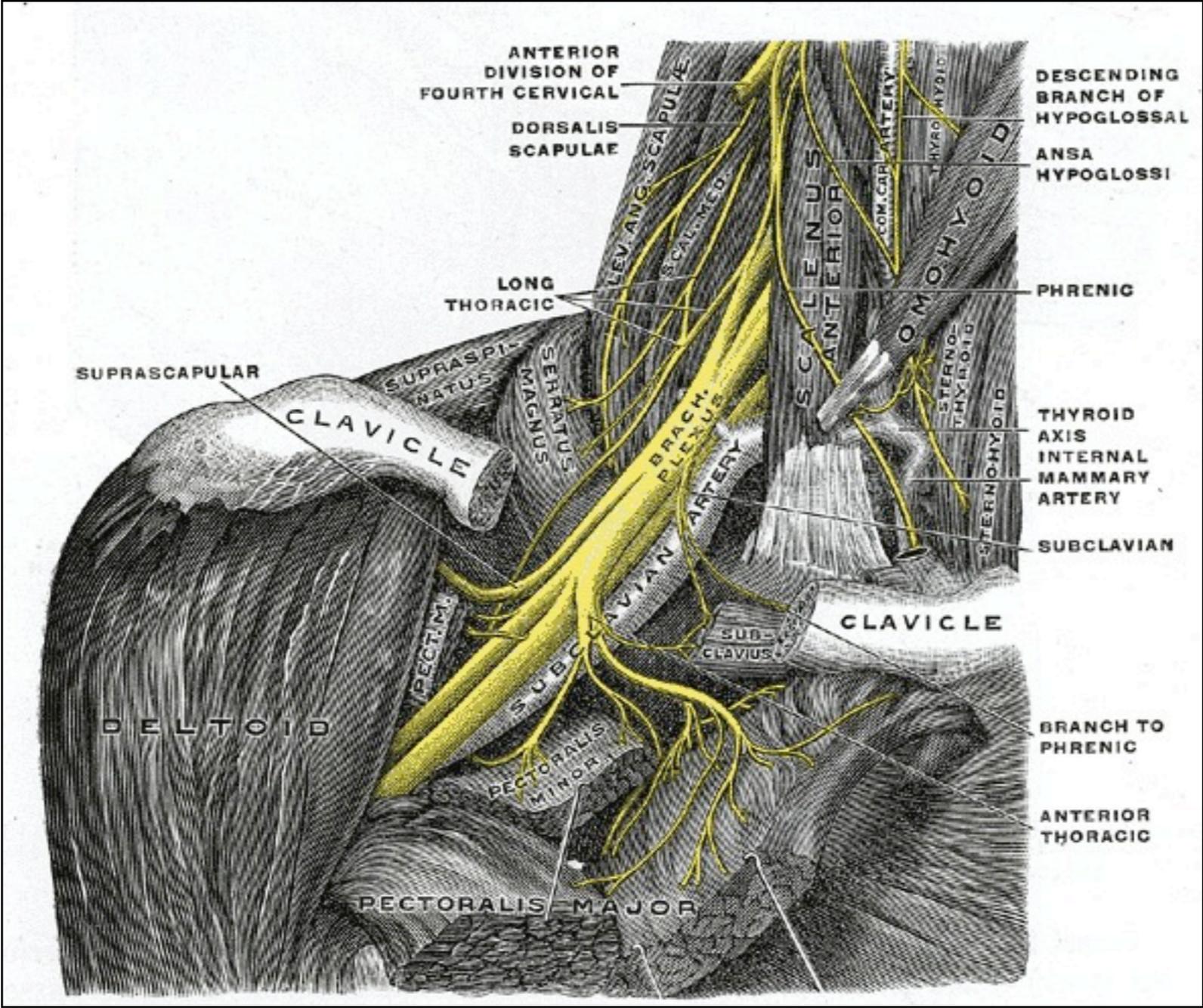
Public domain image derived from Gray's Anatomy image, retrieved from Wikimedia Commons.

Figure 18 Sensory innervation of upper limb



Public domain image derived from Gray's Anatomy, retrieved from Wikimedia Commons.

Figure 19 Brachial plexus: anatomical depiction



Gray's Anatomy image in public domain, retrieved from Wikimedia Commons.

General Anesthesia

In This Section

1. Induction
2. Maintenance
3. Drugs Used in the Maintenance of Anesthesia
4. Emergence

General Anesthesia is a pharmacologically induced reversible state of unconsciousness which is maintained despite the presence of noxious stimuli. With the continual development of new drugs, there is an ever-increasing variety of techniques used to provide general anesthesia. All techniques strive to achieve the following goals, known as the “Four A’s of Anesthesia”:

- Lack of **Awareness**: unconsciousness.
- **Amnesia**: lack of memory of the event.
- Analgesia: the abolition of the subconscious reactions to pain, including somatic reflexes (movement or withdrawal) and autonomic reflexes (hypertension, tachycardia, sweating and tearing).
- **Akinesia**: lack of overt movement. In some cases, the provision of muscle relaxation may be required.

In the past, general anesthesia was achieved using a single agent such as ether or chloroform. Because the above-described goals were achieved by a progressive depression of the central nervous system rather than by any direct or specific

effect, relatively high concentrations of the gases were required. Consequently the associated side-effects were frequent and severe.

In current practice, we have many different agents (both intravenous and inhaled) at our disposal. The intravenous agents in particular have specific effects such as analgesia or muscle relaxation and therefore can be used to achieve the desired effect in a dose-related fashion.

The practice of using combinations of agents, each for a specific purpose, is what is termed “balanced anesthesia”. An example of a balanced technique would be the use of propofol for induction of anesthesia; the administration of desflurane and nitrous oxide for maintenance of unconsciousness; sufentanil for analgesia; and rocuronium for muscle relaxation.

The anticipated benefits of a balanced technique as compared to “ether anesthesia” of the past include:

- improved hemodynamic stability
- more effective muscle relaxation

- more rapid return of respiratory function, consciousness and airway control following the completion of the procedure
- provision of post-operative analgesia with appropriate timing and dosing of opioids administered intra-operatively

A balanced technique is still the most common technique used for the provision of general anesthesia. However, with the development of short-acting intravenous agents such as propofol and remifentanyl, the above-described goals of general anesthesia can be attained with the use of intravenous agents alone, usually by continuous infusion. This is called “Total Intravenous Anesthesia” or “TIVA”.

After the patient has been assessed, the equipment and drugs prepared and the anesthetic technique determined, one can proceed with administering the anesthetic. A general anesthetic consists of four phases: induction, maintenance, emergence and recovery.

Induction

The goal of the induction phase of anesthesia is to induce unconsciousness in a fashion which is pleasant, rapid and maintains hemodynamic stability. If the anesthetic plan includes control of the airway and ventilation then the induction phase also aims to achieve muscle relaxation to facilitate endotracheal intubation.

Anesthesia can be induced by having the patient breathe increasing concentrations of inhaled gases by mask. While there are settings where this is the desired technique, it tends to be slow and can be unpleasant. More commonly, anesthesia is induced with short-acting intravenous agents such as propofol, ketamine, thiopental or etomidate, followed by a muscle relaxant if indicated. In most cases, a non-depolarizing muscle relaxant (NDMR) is used. NDMR are discussed [later in this chapter](#). Understanding the dynamics of induction requires a grasp of the essential pharmacology of these agents; the reader can do so by touching the hyperlink on each drug or by visiting Chapter 6.

Rapid Sequence Induction

Although regurgitation and aspiration are potential complications of any anesthetic, there are factors which place some patients at higher risk ([Table 7](#)). The obvious risk factor is recent intake of solid food. However, even a prolonged period of fasting does not guarantee an “empty stomach” if gastric emptying is delayed. Ex-

amples of conditions which impair gastric emptying include diabetes, trauma, recent opioid administration and bowel obstruction. Finally, decreased integrity of the lower esophageal sphincter, as occurs in pregnancy and obesity, increases the risk of regurgitation of stomach contents. In patients deemed to be at increased risk for aspiration, the time between inducing anesthesia and securing the airway with a cuffed endotracheal tube must be minimized. Such a technique is termed a "rapid sequence induction".

A rapid sequence induction is performed as follows:

1. Suction apparatus is checked and kept readily available.
2. Pre-oxygenation of patient with 100% oxygen for 3-5 minutes.
3. Application of cricoid pressure (Sellick's maneuver) by assistant.
4. Induction with pre-calculated dose of induction agent followed immediately by intubating dose of depolarizing muscle relaxant (succinylcholine). A rapidly acting non-depolarizing agent (e.g. rocuronium) is commonly used in a so-called "modified" rapid sequence induction.
5. Intubation of trachea, cuff inflation and verification of proper tube position.

The purpose of pre-oxygenation is to lessen the risk of hypoxemia occurring during the apneic period after induction. Although pre-oxygenation does increase the patient's arterial PO₂ prior to induction, its most important effect is the de-nitrogenation of the functional residual capacity of the lungs. Traditionally teaching is that the Sellick maneuver provides occlusion of the esophagus between the cricoid cartilage (a complete circumferential cartilage) and the cervical vertebrae thus minimizing the risk of passive regurgitation.

Succinylcholine

Succinylcholine (Sch), a depolarizing muscle relaxant, is a very useful and very powerful drug; the anesthesiologist must understand the effects and contraindications of Sch in order to avoid causing harm or death.

Succinylcholine causes rapid, profound and brief muscular paralysis. It acts by attaching to nicotinic cholinergic receptors at the neuromuscular junction. There, it mimics the action of acetylcholine, thus depolarizing the post-junctional membrane. Neuromuscular blockade (paralysis) develops because a depolarized post-junctional membrane cannot respond to subsequent release of acetylcholine.

Succinylcholine has effects on almost every organ system, most of them being secondary to the depolarization and subsequent contraction of skeletal muscle.

Important effects include increased intracranial pressure, increased intragastric pressure and post-operative myalgia.

The most critical effects of Sch relate to its interaction with muscle cells. Sch elevates serum potassium by 0.5 mEq/L in patients with normal neuromuscular function and is therefore contraindicated in hyperkalemic patients or in patients with renal failure, for whom even a small rise in potassium could have critical implications. Sch can cause an *exaggerated* release of potassium, leading to fatal hyperkalemia, in those with neuromuscular or muscle disease. Examples include recent paralysis (spinal cord injury or stroke), amyotrophic lateral sclerosis, Duchenne's muscular dystrophy and recent burn or crush injury.

Sch is a potent trigger of malignant hyperthermia (MH) and is therefore contraindicated in MH-susceptible patients. Sch is contraindicated in patients with pseudocholinesterase deficiency in whom the paralysis will be prolonged.

A more complete discussion of succinylcholine can be found in Chapter 6.

Maintenance

If no further agents were administered following the induction of anesthesia the patient would awaken within minutes. Therefore, maintenance of anesthesia requires the delivery of pharmacologic agents with the aim of achieving the "four A's of anesthesia" and hemodynamic stability throughout the surgical procedure. A further consideration is the length of the procedure and the need to awaken the patient at the end of the case. The maintenance phase of anesthesia involves the use of inhaled agents, opioids and non-depolarizing muscle relaxants (NDMR). These agents are described later in this chapter.

The anesthesiologist must be ever vigilant. Problems related to the airway, breathing and circulation (ABC's) are most critical and can occur during any phase of anesthesia. Several of the problems that are unique to the maintenance phase of anesthesia are discussed below.

Awareness

"Awareness" refers to a complication of anesthesia whereby a patient who has received a general anesthetic becomes conscious of his or her environment during the surgical procedure. The patient may or may not experience pain and may or may not recall the events post-operatively. The incidence of awareness *with recall* has been estimated at 0.2-1.0%. Some types of surgical procedures such as Caesarian section, cardiac surgery

and trauma surgery pose a higher risk of awareness because of the nature of the anesthetic given for those procedures. It may be prudent to warn such patients of the risk pre-operatively.

Intra-operatively, care should be taken to ensure delivery of adequate amounts of hypnotic drugs such as inhaled agents, propofol, benzodiazepines or ketamine. Opioids alone provide very little hypnosis and muscle relaxants provide none whatsoever! Signs of awareness should be sought. In an un-paralyzed (or partially paralyzed) paralyzed patient, this includes movement. However, a fully paralyzed patient is only able to communicate through the autonomic nervous system with signs of sympathetic hyperactivity, such as hypertension, tachycardia, sweating and tearing. Not surprisingly, the overwhelming majority of cases of awareness have been reported in paralyzed patients.

Positioning

The patient is positioned to facilitate surgical access. Depending on the procedure, the patient may be placed in the supine, prone, lateral, lithotomy, jack-knife, kidney or even the sitting position to name but a few. Most of the consequences of positioning involve the cardiovascular, respiratory and peripheral nervous systems.

Kinking of, or pressure on major vessels leads to decreased venous return, decreased cardiac output and hypotension. This is particularly relevant when the

prone or kidney position is used. In the semi-sitting position, venous pooling in the legs has a similar effect. Very occasionally, surgery is performed in the sitting position which is associated with the risk of venous air embolism.

The airway may become obstructed or dislodged while the patient is in the prone position. The prone, trendelenburg and lithotomy positions may cause an upward displacement of the diaphragm due to an increase in intra-abdominal pressure. This leads to ventilation/perfusion mismatching and decreased lung compliance which may manifest as hypoxemia, hypercarbia or increased airway pressure.

Nerve injury results from compression on pressure points or stretching. Other factors such as prolonged surgery, hypothermia, hypotension, obesity and diabetes may play a role in increasing the risk of a post-operative neuropathy. The ulnar nerve, because of its superficiality, is at risk of compression in almost any position. Padding is commonly used but has not been shown convincingly to be helpful. Careful positioning is probably most important in this regard. The brachial plexus is at risk of stretch injury when arms are abducted in the supine position. The angle of abduction should be kept below 90 degrees and the head should be turned slightly toward the abducted arm. Many nerves including the sciatic, lateral femoral cutaneous

and common peroneal nerves are at risk of either stretch or compression injuries when the lithotomy position is used.

Other organ systems may be vulnerable in the prone position. Pressure on the orbit of the eye can lead to retinal ischemia by either arterial compression or obstruction of venous flow. The eye socket itself provides a natural protection and specially designed head rests are helpful. Constant vigilance must be maintained as patient position may shift during anesthesia. The male patient's genitalia must be free of pressure. Finally, pressure over skin surfaces (e.g. the forehead) must be minimized as skin sloughing can result after prolonged surgery in the prone position.

Hypothermia

Hypothermia has deleterious effects on the cardiovascular, respiratory, central nervous, hematologic and renal systems. As well, it decreases the rate of recovery from the effects of muscle relaxants.

Heat is lost through four mechanisms:

- convection (e.g. exposure to drafts of cool air)
- conduction (e.g. contact with cold operating room table)
- evaporation (e.g. airway mucosa, prep solution, sweat)

- radiation (e.g. temperature gradient between patient and operating room environment)

Furthermore, the normal responses to hypothermia (shivering, vasoconstriction) are abolished under anesthesia. Procedures which are prolonged, involve large abdominal incisions or require administration of large volumes of intravenous fluids can be associated with particularly severe hypothermia.

Heat loss can be minimized by keeping the operating room temperature as high as tolerable (>21 C, preferably). Gas humidifiers or the use of low gas flow minimizes the heat lost through airway evaporation. Fluid warmers should be used whenever blood products or large volumes of intravenous fluids are being given. A forced air warming system should be used routinely except for those cases which are very short in duration.

If significant hypothermia (<35 C) results despite preventative measures then, depending on the underlying patient condition, the anesthesiologist may decide to leave the patient sedated, paralyzed and mechanically ventilated post-operatively until adequate temperature is restored. This is particularly important for patients with cardiac or respiratory disease where shivering (which increases oxygen consumption fourfold) may compromise outcome.

Drugs for Maintenance of Anesthesia

Inhaled Agents

The term “inhaled agent” refers to the volatile agents (desflurane, isoflurane, sevoflurane) and nitrous oxide (N₂O). While the development of intravenous agents has largely eliminated the use of inhaled agents for induction except in the pediatric population, they continue to be the mainstay for maintenance of anesthesia. The volatile agents are so called because with vapour pressures below atmospheric pressure, they exist in equilibrium between liquid and gaseous states at room temperature. N₂O is a gas at room temperature and atmospheric pressure and is therefore not a volatile agent though it is, of course, an inhaled agent.

Mechanism of Action

The mechanism of action of inhaled agents is not well understood.

Dose

While intravenous agents are given in mg (or mcg)/kg doses, the inhaled agents are given in volume percent concentrations. The volatile agents can also be termed “potent” vapours, because concentrations in the range of 0.3-6% are clinically effective while N₂O (not potent) must be given in concentrations of between 30% and 70% to have any effect.

This brings us to the concept of Minimum Alveolar Concentration (MAC). The concentration of a gas in the

alveoli creates an alveolar partial pressure of gas which in turn reflects its partial pressure in the active site (brain). MAC refers to the concentration of the inhaled agent in alveolar gas necessary to prevent movement of 50% of patients when a standard incision is made. This definition hints at the fact that a specific concentration of gas does not correlate to a predictable clinical effect. Many factors influence MAC, and therefore influence the concentrations required to maintain anesthesia. Some of the known factors are listed in [Table 12](#).

Table 12 Factors affecting MAC

FACTORS WHICH DECREASE MAC	FACTORS WHICH INCREASE MAC
advanced age	childhood
pregnancy	hyperthyroidism
hypothermia	hyperthermia
acute alcohol intoxication	chronic alcohol use
drugs: benzodiazepines, opioids, muscle relaxants, central-acting antihypertensives	drugs: amphetamine, cocaine

The MAC values of the commonly used agents are shown in [Table 13](#). Note that N₂O, with a MAC > 100%, can never be used as a sole agent to provide anaesthesia. Even the potent agents are often not administered at 1 MAC concentrations during general anaesthesia. This is because other agents (such as opioids) are also being given. It is generally felt that 0.5 MAC of inhaled agent is the *minimum* level to provide adequate hypnosis and amnesia.

Onset of action, duration of action and elimination

The solubility of a gas in blood determines its rate of onset and offset of effect. An agent such as N₂O, which is relatively insoluble in blood, will build up its alveo-

lar partial pressure (and therefore brain partial pressure) quickly and consequently will have a faster onset and offset of effect. Conversely, a soluble gas such as isoflurane will equilibrate slowly throughout body stores and therefore onset and offset lags. You may notice this theory being put into practice in the operating room: a soluble potent agent is often discontinued 5 or 10 minutes prior to the end of surgery while N₂O is delivered until moments before the desired emergence or wake-up. [Table 13](#) summarizes these pharmacokinetic properties. The termination of effect of inhaled agents depends only on its exhalation from the lungs. The exception, of historical interest, is halothane of which up to 20% can undergo metabolism in the liver.

Table 13 Characteristics of inhaled agents

	N ₂ O	ISOFLURANE	SEVOFLURANE	DESFLURANE
ODOUR	odourless	pungent	sweet	pungent
MAC%	104	1.15	1.8	6.6
BLOOD/GAS PARTITIONING	0.47	1.4	0.65	0.42
VAPOUR PRESSURE 20°C MMHG	38,770	238	157	669

Effects of the inhaled agents

The effects of the volatile agents are quite distinct from those of nitrous oxide. Inhaled agents have effects on almost every organ system and the reader is referred to Chapter 6 for a detailed summary. Several key effects are highlighted below.

All volatile anaesthetics are triggers of malignant hyperthermia while nitrous oxide is not. N₂O expands the volume of gas-containing spaces as N₂O diffuses across membranes more readily than nitrogen can diffuse out. Thus the size of a pneumothorax, an emphysematous bleb or a distended bowel loop will increase when N₂O is used.

Contraindications to the inhaled agents

The use of volatile agents is absolutely contraindicated in patients who are known or suspected to have malignant hyperthermia.

The use of nitrous oxide is contraindicated in patients with pneumothorax or bowel obstruction. As N₂O raises intracranial pressure, its use is avoided in patients with intracranial pathology. Caution should be used in those patients with coronary artery disease or emphysema.

Opioids

Opioids are used intra-operatively to provide analgesia, and to reduce the requirement of other maintenance agents. The commonly used intravenous agents are the synthetic opioids fentanyl, sufentanil, remifentanyl and alfentanil. They are favoured by anesthesiologists over the more familiar agents, such as morphine and meperidine. Their shorter duration of action allows finer titration to provide adequate analgesia during the variable, but intense nature of surgical stimulation, while still allowing for awakening at the end of the procedure. While there are many different opioids available for use, the discussion below is limited to the three synthetic agents which are most commonly used in anesthetic practice.

Usually, an opioid is administered in the form of a loading dose, prior to induction. Not only does this help to

blunt the response to intubation, which is a very stimulating maneuver, but it establishes a plasma level of opioid which can then be supplemented as needed throughout the surgical procedure. In the case of remifentanyl, which is extremely short-acting, a bolus followed by an infusion is most practical.

Mechanism of action

There are five different opioid receptors of which the most clinically relevant are the Mu and Kappa receptors. Binding to different receptors produces distinct responses. Activation of Mu receptors produces analgesia, respiratory depression, bradycardia, euphoria and decreased gastrointestinal motility. Binding to Kappa receptors produces analgesia, sedation and miosis. The major receptors for analgesia are the Mu-1 receptor at the periaqueductal gray area of the midbrain and the Kappa receptor at the substantia gelatinosa of the spinal cord. Each opioid has its own unique profile of agonism and antagonism for each receptor. Unfortunately, an agent which possesses agonism exclusively at the analgesia receptors has not yet been developed.

Dose, onset, and duration

All opioids are relatively lipid soluble. The greater the lipid solubility, the greater the potency. As a general rule:

10 mg Morphine =100 mg Meperidine =100 µg Fentanyl =10 µg Sufentanil

Onset of action is determined by lipid solubility and ionization (pKa). Duration of action is determined by

Table 14 Pharmacology of commonly used opioids

	Fentanyl	Sufentanil	Remifentanil
Induction dose (µg/kg)*	4-20	0.25-2	0.5-1
Intra-op dose for maintenance*	2-5 µg/kg/hr	0.3-2.0 µg/kg/hr	0.1-1 µg/kg/min
Additional boluses*	25-150 µg	2.5-20 µg	0.1-1 µg/kg
Onset of action (min)	5-8	4-6	1-2
Duration after bolus (min)	45	45	1-4

**Loading and maintenance requirements depend greatly on the patient age and status, the doses of other anesthetic agents given, and the nature of the surgical procedure being performed.*

both clearance and volume of distribution. [Table 14](#) summarizes the clinically useful pharmacology of the opioids most commonly used in anesthesia.

Elimination

The relatively short duration of action of these agents is in part attributable to their lipid solubility. This leads to rapid redistribution away from the central nervous system to inactive tissue sites. Fentanyl and sufentanil are metabolized in the liver to (mostly) inactive metabolites which are then excreted in the urine. Remifentanil on the other hand, is susceptible to metabolism by blood and tissue esterases which accounts for its ultrashort duration of action.

Effects

Opioids have effects on almost every system in the body. The reader is referred to Chapter 6 for a detailed discussion.

The most important side effect of the opioids manifests on the respiratory system. Minute ventilation is reduced due to a reduction in respiratory rate. (Tidal volume actually increases.) The responsiveness to raised PCO₂ is diminished such that apnea occurs until the dose-dependent “apneic threshold” of PCO₂ is reached.

Opioids cause nausea and vomiting due to stimulation of the chemoreceptor trigger zone. They also cause constipation due to decreased GI motility.

Contraindications

Opioids must not be given to those with a known allergy. Intravenous opioids must not be given in settings where one is not able to support ventilation.

Caution should be used when administering opioids to patients with hypovolemic or cardiogenic shock, where the blunting of sympathetic tone may exacerbate hypotension.

Non-Depolarizing Muscle Relaxants

The decision to use non-depolarizing muscle relaxants during maintenance of anesthesia depends on both the type of surgical procedure and the type of anesthetic. Some procedures require muscle relaxation to facilitate exposure (e.g. intra-abdominal surgery). In other cases, muscle relaxation is required because patient movement would be detrimental (e.g. neurosurgery, ophthalmic surgery). In a balanced technique, the use of muscle relaxants decreases the requirements of the other agents and facilitates mechanical ventilation.

Historically, the choice of succinylcholine versus a non-depolarizing muscle relaxant (NDMR) for use during induction of anesthesia was a decision which balanced the need for rapid airway control against the side effects of succinylcholine. With the introduction of the rapidly acting non-depolarizing agent, rocuronium, the use of succinylcholine has steadily declined. In the rare circumstance where succinylcholine is used to facilitate

intubation, a NDMR is given after the effects of succinylcholine have worn off. However, in the vast majority of cases NDMR is given at induction to provide relaxation for both intubation and surgery.

Mechanism of action

In order to appreciate how NDMR cause muscular paralysis, one must have a basic understanding of how neuromuscular transmission occurs ([Figure 20](#)). Normally a nerve impulse travels the length of the nerve to arrive at the motor nerve terminal where it causes release of acetylcholine (Ach) into the synaptic cleft. The Ach then binds to post-synaptic nicotinic Ach receptors causing a conformational change in those receptors.

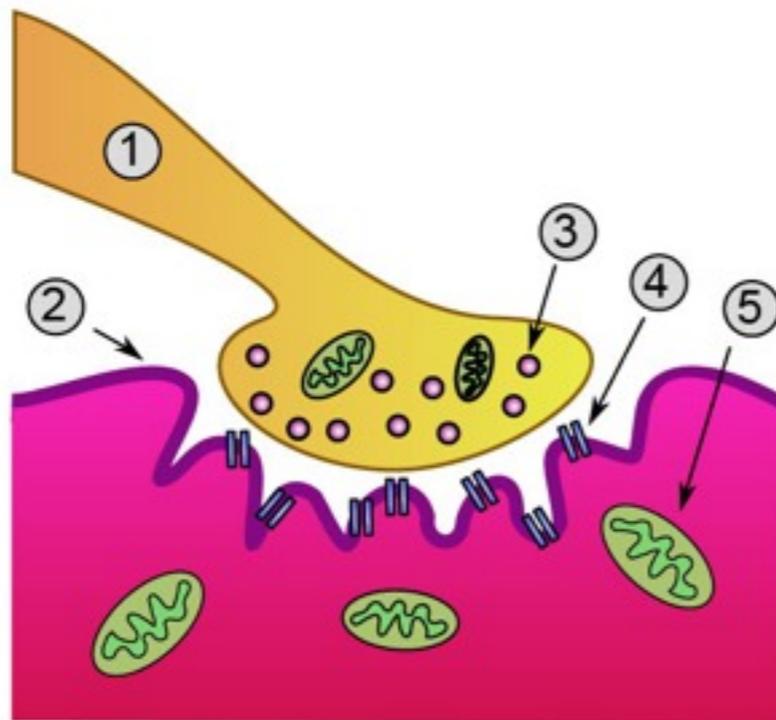
This conformational change leads to a change in membrane permeability of sodium and potassium causing depolarization of the post-junctional membrane. The propagation of this action potential leads directly to muscular contraction. NDMR interfere with this process by binding to the post-synaptic Ach receptors thereby acting as a competitive inhibitor to Ach.

Dose, onset, duration, elimination and effects

The choice of which muscle relaxant to use is influenced by the speed of onset, duration of action, method of elimination and side effect profile of the various agents. Except in the case of very brief procedures, NDMR are used for relaxation during maintenance of anesthesia. Commonly-used NDMR are rocuronium

and cis-atracurium. [Table 15](#) presents the relevant pharmacology and includes an older NDMR, pancuronium which is a long-acting agent that is still in use. Atracurium, another short-acting NDMR, is included in Chapter 6 (Drug Finder) although it is no longer available in many jurisdictions.

Figure 20 Neuromuscular junction



1. nerve terminal 2. sarcolemma 3. acetylcholine vesicles
4. acetylcholine receptors 5. mitochondrion.

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Many factors may exaggerate or prolong the effects of NDMR. These include:

- factors which increase the susceptibility of the neuromuscular junction to NDMR: muscular dystrophies, myasthenia gravis, hypocalcemia, hypermagnesemia, acid-base abnormalities and many drugs (aminoglycosides, lithium, diuretics, volatile anesthetics)
- factors which delay metabolism or excretion: hypothermia, renal insufficiency and liver disease

The reader is referred to Chapter 6 for a more complete discussion of the pharmacology of the non-depolarizing muscle relaxants.

During maintenance of anesthesia, the degree of muscular paralysis is best monitored using a peripheral nerve stimulator. The anesthesiologist observes the magnitude and number of twitches in response to a series of four electrical stimuli (2 per second) applied over the ulnar nerve. The importance of carefully titrating NDMR is two-fold:

- to ensure sufficient muscle relaxation during the procedure
- to ensure the ability to adequately reverse muscle relaxation at the end of the procedure

Table 15 Dose, onset, duration, elimination, and effects of NDMR

	PANCURONIUM	ROCURONIUM	CIS- ATRACURIUM
INTUBATING DOSE (MG/KG)	0.08 - 0.1	0.6 - 1	0.15 - 0.25
REPEAT DOSE (MG/KG)	0.02	0.1	0.02
ONSET (MIN)	4-5	1.5	1.5-3
DURATION AFTER INTUBATING DOSE (MIN)	60 - 90	30 - 60	40 - 75
METABOLISM/ ELIMINATION	80% renal	>70% hepatic	77% Hoffmann elimination
SIDE EFFECTS	muscarinic blocker	weak muscarinic blocker	none

Contraindications

The major contraindication to the use of any muscle relaxant is the inability to provide airway and ventilatory control. A patient who is known or suspected to be a difficult intubation or a patient who has a fixed airway obstruction should not receive muscle relaxants prior to having the airway secured.

Emergence

During the emergence phase of anesthesia, the patient begins to return to his pre-operative state of consciousness. In most cases, the anesthesiologist aims to awaken the patient at the end of the operative procedure prior to transfer to the post-anesthetic care unit (PACU). How “awake” must the patient be? Ideally the patient is conscious enough to obey commands and support his own airway. At the very least, the patient must have adequate spontaneous ventilation but may need minimal assistance to maintain patency of the airway. In between these two states lies a wide spectrum of level of consciousness. Patient factors as well as the anesthetic technique determine the rate at which emergence from general anesthesia occurs.

Spontaneous and Active Reversal

Emergence requires the offset of effect of the anesthetic agents. This is achieved by administering the anesthetic drugs in appropriate doses at the appropriate time according to the anticipated length of the procedure. The anesthesiologist relies on the normal metabolism and excretion of drugs to achieve offset of effect. Active reversal of drug effect through the administration of another drug also plays a role in emergence. The most common example of this is the reversal of muscle relaxation which is discussed in greater detail below. Aside from muscle relaxants, anesthetic agents are

rarely actively reversed in order to achieve emergence. There is no “antidote” to the inhaled agents; offset of effect relies on the timely discontinuation of administration followed by excretion through the lungs. While an opioid antagonist (naloxone) exists, there are several disadvantages to using it to reverse opioid effect at the end of surgery. Firstly, unless very carefully titrated, its use will lead to a startled, hyper-alert patient who complains of pain at the operative site. Hypertension, tachycardia, myocardial ischemia and pulmonary edema may result. Secondly, the duration of effect of the antagonist is shorter than that of many of the opioid agonists therefore “re-narcotization” in the PACU is a risk. Finally, naloxone is an expensive drug whose use adds unnecessarily to the cost of the anesthetic. Flumazenil is a specific benzodiazepine antagonist which may play a role in the occasional surgical patient whose decreased level of consciousness is attributed to benzodiazepines. Like naloxone, flumazenil has a shorter duration of action than most of the benzodiazepine agonists therefore rebound sedation may occur.

Extubation

If an endotracheal tube is used to maintain the airway intra-operatively, it must be removed at some point during the emergence phase of anesthesia. It is important to time the extubation properly, so as to avoid the potential post-extubation complications:

- airway obstruction
- aspiration
- inadequate ventilation
- laryngospasm

If the patient meets three simple criteria, most emergence complications can be avoided. The anesthesiologist must ensure that:

- the patient has regained their drive to breathe. Sufficient offset of effect of opioids is required for the patient to resume and maintain spontaneous respiration.
- the patient has normal muscle strength. A “weak” patient will not have enough strength to keep the tongue from falling to the back of the pharynx and causing airway obstruction. Muscle strength is also required to achieve satisfactory tidal volumes. Adequate muscle strength is required for the cough reflex which protects the airway from aspiration.
- the patient is awake enough to obey commands. An adequate level of consciousness is required in order for the patient to protect his airway from aspiration and to avoid laryngospasm.

Laryngospasm (reflexive closure of the vocal cords) deserves special mention. Laryngospasm is the airway’s response to irritation. It can occur immediately after ex-

tubation, leading to total airway obstruction, particularly in children and young adults. Extubating the patient at a deep plane of inhalational anesthesia (when the reflex is blunted) is one way to avoid laryngospasm but is an approach that is only safely applied to the pediatric patient. In adults (and pediatric patients), performing extubation when the patient is wide awake (where consciousness abolishes the reflex) will decrease the risk of post-extubation laryngospasm. Practicing anesthesiologists understand that extubating the patient at a light plane of anesthesia (not awake, but not “asleep” either) increases the risk of post-extubation laryngospasm.

Reversal of Muscle Relaxation

The action of all non-depolarizing muscle relaxants must be reversed prior to emergence from anesthesia. The anticholinesterase drugs, sometimes termed “reversal agents” are edrophonium, neostigmine and pyridostigmine ([Table 16](#)), with neostigmine being most commonly used.

The anticholinesterases reverse the effects of the NDMR. However, in order for them to be completely effective, some degree of spontaneous recovery from the NDMR block must be present prior to administration of the anticholinesterase. Adequacy of reversal is assessed clinically. The peripheral nerve stimulator is used while the patient is still unconscious. Tradition-

ally, the anesthesiologist “eyeballs” the number of twitches and presence of fade although this technique is known to result in an underestimation of the degree of residual blockade. Newer anesthetic machines are equipped to assess the same indices by measuring mechanical deflection of the thumb. The most important indicators are clinical and are measured in the awake patient. A strong hand grip and the ability to lift the head off the bed for 5 seconds reliably indicate the return of adequate muscular strength.

Mechanism of action

Anticholinesterases act in the synaptic cleft of the neuromuscular junction. Here, they inhibit the action of cholinesterase, thereby decreasing the rate of breakdown of acetylcholine (ACh). The increased concentration of ACh in turn displaces the NDMR from the ACh receptors and thus restores normal neuromuscular transmission.

Dose, onset, duration and elimination

Relevant pharmacokinetic facts are summarized in [Table 16](#).

Effects

Unfortunately, the anticholinesterase drugs potentiate the action of ACh at muscarinic receptors as well as at the nicotinic receptors of the neuromuscular junction. This can lead to all of the symptoms that are associated with excessive parasympathetic tone such as bradycardia, heart block, increased airway secretions, broncho-

spasm, intestinal spasm, increased bladder tone and pupillary constriction. These effects are minimized by administering an anticholinergic (atropine or glycopyrrolate) along with the anticholinesterase.

The reader is referred to Chapter 6 for a more complete discussion of the pharmacology of the anticholinesterase agents as well as the anticholinergics that must accompany their administration.

Contraindications

Anticholinesterases are contraindicated in patients with gastrointestinal obstruction. They should be used with caution in patients with bradycardia, asthma, seizure disorders and Parkinson's disease. An overdose can cause a cholinergic crisis.

Table 16 Dose, onset, duration, elimination of anticholinesterases

	EDROPHONIUM	NEOSTIGMINE	PYRIDOSTIGMINE
DOSE (MG/KG)	0.5 - 1.0	0.025 - 0.075	0.1 - 0.3
ONSET (MIN)	rapid (1)	intermediate (5)	delayed (10)
DURATION (MIN)	40 - 65	55 - 75	80 - 130
RENAL EXCRETION	70%	50%	75%

CHAPTER 4

Post-operative Phase



In this chapter, you will develop an understanding of both the goals and challenges of the recovery phase of anesthesia with a special focus on postoperative pain management. A review quiz is available at www.understandinganesthesia.ca

Recovery

In This Section

1. Goals of Recovery
2. Post-operative Nausea and Vomiting
3. Shivering
4. Pain

Goals of Recovery

At the end of the operative procedure, care and monitoring of the patient is handed over from the anesthesiologist to the nurse in the post-operative care setting as the patient enters the period of recovery. For most patients, this occurs in the Post-Anesthetic Care Unit (PACU). However, some patients, such as those requiring prolonged post-operative ventilation or close hemodynamic monitoring, may instead be admitted directly to the Intensive Care Unit. Prior to transporting the patient from the operating room, the anesthesiologist must ensure the presence of the following:

- patent airway (provided either by an awake patient, oral airway or endotracheal tube)
- adequate ventilation
- stable hemodynamics
- adequate pain control

Any identified problems must be corrected before leaving the operating room to avoid transporting an unstable patient.

On arrival in the PACU, the airway patency, breathing and circulation are immediately assessed. Supplemental oxygen is provided as indicated. Routine monitors are applied. The anesthesiologist then gives the attending nurse pertinent information about the patient's past medical history and intra-operative course. The latter includes details of the nature of the procedure, anesthetic technique, fluid balance and any intra-operative complications. Finally, instructions regarding monitoring, management of fluids, pain and nausea as well as discharge plans are given.

Monitoring in the PACU is an extension of that provided in the operating room. The patient is observed for potential complications, both surgical and anesthetic. When discharge criteria are met, the patient is transferred to their ultimate destination: the ward for inpatients or the same day surgery unit for outpatients.

Criteria for discharge are stratified into "Phase 1" (which determines when the patient is able to be transferred from the PACU to the ward) and "Phase 2" (which addresses home-readiness and only applies to those patients having "same day

surgery”). A scoring system (Aldrete score) has been developed that grades the patient’s colour, respiration, circulation, consciousness and activity on a scale of 0-2.

For Phase 1 recovery, the patient must:

- be showing no signs of respiratory depression for at least 20-30 minutes after last dose of parenteral opioid.
- be easily aroused.
- be fully oriented to person, place and time.
- be able to maintain and protect the airway on his own including evidence of a strong cough.
- have stable vital signs for at least 30 minutes.

It is also important that pain and post-operative nausea and vomiting are controlled prior to PACU discharge and that there are no ongoing surgical concerns such as surgical site bleeding. Most healthy patients undergoing routine surgery will meet the PACU discharge criteria within 60 minutes.

Prior to being discharged home (from the same day surgery unit), the patient must demonstrate the return of cognitive function, ambulation and the ability to take oral liquids.

Many types of complications can occur in the PACU. Some of them, such as airway obstruction, aspiration,

post-operative hemorrhage and myocardial ischemia are life-threatening but occur relatively infrequently. With expertise in airway management and cardiovascular resuscitation, the anesthesiologist is well-positioned to detect and manage these critical events.

Hypertension is commonly seen in the PACU and has many possible underlying causes. The patient may have pre-existing essential hypertension which is poorly controlled and may be exacerbated by the omission of their usual medication on the day of surgery. Other factors can lead to hypertension in the PACU such as full bladder (which the patient may not recognize), pain and importantly, hypoxemia and hypercarbia.

Fortunately, the most common PACU complications are not usually life-threatening but are important to recognize and manage nonetheless. A brief discussion follows below.

Post-operative Nausea and Vomiting

One of the most common problems encountered in the PACU is post-operative nausea and vomiting (PONV). PONV is unpleasant for both patient and staff. Moreover, it places the patient at risk for aspiration of gastric contents, particularly if airway reflexes are blunted due to the residual effects of opioids, inhaled agents and muscle relaxants.

When severe PONV is encountered, it is important to rule out sinister causes such as myocardial ischemia, bowel obstruction or raised intracranial pressure. More commonly, the cause is multifactorial with patient, surgical and anesthetic factors contributing. The many risk factors for PONV are outlined in [Table 17](#), the most important (and statistically-robust) factors highlighted in bold.

The best approach to PONV is prevention. Attention should be paid to the most emetogenic anesthetic drugs which are nitrous oxide and neostigmine. Minimizing the dose of neostigmine to less than 2 mg appears to eliminate its emetogenic effect and should be considered if neuromuscular function allows. The modification of anesthetic factors (such as avoiding the use of nitrous oxide) is at least as effective as the administration of a prophylactic antiemetic agent. Current guidelines recommend that prophylaxis (ondansetron and/or dexamethasone) be administered selectively to

moderate or high risk patients. These agents are administered during anesthesia rather than pre-operatively. Interestingly, ondansetron is much more effective as a treatment for PONV than as a preventive, where its NNT (number needed to treat) is around 5. It is also not free of side effects such as headache, constipation and elevated liver enzymes.

In the PACU, PONV is best treated with hydration and intravenous antiemetics such as ondansetron (if not used as prophylaxis), prochlorperazine or dimenhydrinate. PONV may act as a limiting factor to the delivery of opioid analgesia; patients at high risk of PONV benefit from opioid-sparing analgesic techniques such as peripheral nerve blocks or neuraxial analgesia.

Table 17 Risk factors for PONV

PATIENT	SURGICAL	ANESTHETIC
female	laparoscopic	peri-operative opioids
history of PONV	ophthalmic	neostigmine
history of motion sickness	gynecologic	nitrous oxide
non-smoking		sodium thiopental
age <12 years		volatile agent
obesity		

Shivering

Shivering is a response controlled via the hypothalamus aimed at generating endogenous heat. The cost of this heat production is a fourfold increase in oxygen consumption and carbon dioxide production. This can precipitate myocardial ischemia or respiratory failure in patients with limited coronary or ventilatory reserve. Most commonly, shivering is a direct response to hypothermia and is best treated with aggressive rewarming techniques such as forced air warming systems. In the PACU, one often observes normothermic patients who shiver. This is a poorly understood effect of residual volatile gases and often abates with the administration of small doses of intravenous meperidine.

Pain

Pain is a consequence of all but the most minor surgical procedures. Aside from its inherent unpleasantness, it can lead to hypertension, tachycardia, myocardial ischemia and respiratory failure. Post-operative pain is most effectively approached through preventive measures taken in the operating room. Occasionally, this is achieved with a nerve block or the administration of central neuraxial local anesthetics and/or opioids.

More commonly, however, the anesthesiologist titrates the dose and timing of intravenous opioids to anticipate an awake, comfortable patient at the end of the procedure. A brief discussion of the methods of continued post-operative pain control follows in the next chapter.

Post-operative Pain Management

In This Section

1. Intravenous Analgesia
2. Central Neuraxial Analgesia
3. Peripheral Nerve Blocks

Appropriate pain control not only contributes to patient comfort, it decreases the incidence of post-operative complications such as cardiac ischemia, pulmonary atelectasis and delirium. Many factors need to be considered when determining the method of post-operative analgesia. The most important factors are the patient's medical history and the nature of the surgical procedure. Oral analgesics (acetaminophen, codeine phosphate, non-steroidal anti-inflammatories) or intramuscular opioids may be sufficient in many cases. However, often, more sophisticated techniques are required.

Patient-controlled analgesia

Patient-controlled analgesia (PCA) permits the patient to administer the delivery of his own analgesic by activating a button, which then triggers the intravenous delivery of a predetermined dose of an opioid such as morphine. Limits are set on the number of doses per four-hour period and on the minimum time that must elapse between doses (lockout interval). The pharmacokinetic advantage of PCA is that by self-administering frequent, small doses, the patient

is able to come closer to achieving a steady state analgesic level in the blood, avoiding the high peaks and low troughs that can be found with intermittent (intramuscular) opioid administration. Indeed, PCA has been shown to provide equivalent analgesia with less total drug dose, less sedation, fewer nocturnal disturbances and more rapid return to physical activity. In addition, patient acceptance is high since patients have a significant level of control over their pain management.

PCA analgesia is not without side effects, the most common of which is nausea and vomiting. Excessive sedation and pruritis may also be seen. Standardized orders provide "as needed" orders for medications to counteract both nausea and pruritis.

Although it does not obviate the need for close monitoring, PCA frees nursing personnel from administering analgesic medication. Since patients titrate their own therapy with PCA, they must be capable of understanding the principle, willing to participate and physically able to activate the trigger. Consequently, use is prohibited

at the extremes of age as well as in very ill or debilitated patients.

PCA would be appropriately used for patients recovering from breast reconstruction or lumbar spine decompression and fusion. Typically, the PCA modality is used for 24-72 hours. The patient must be capable of oral (fluid) intake prior to converting from PCA to oral analgesics, a factor which is most relevant for those recovering from bowel surgery.

Central Neuraxial Analgesia

Central neuraxial analgesia involves the delivery of local anesthetics and/or opioids to either the intrathecal (spinal) space or the epidural space.

Because intraspinal catheters are rarely used, intrathecal analgesia is usually an extension of a “one-shot” spinal anesthetic used intra-operatively. Opioids added to the (spinal) local anesthetic solution provide long-lasting analgesia after a single injection, lasting well into the post-operative period. The duration of effect is directly proportional to the water-solubility of the compound, with hydrophilic compounds such as morphine providing the longest relief.

Epidural catheters are safe and easy to insert. Contraindications can be reviewed in [Table 11](#). Epidural analgesia can be used to provide pain relief for days through the infusion of a solution containing local anesthetic, opioid or both. The infusion is usually delivered continuously. Intermittent or “bolus” doses lack titratability and are associated with a higher incidence of side effects such as respiratory depression. Continuous epidural infusions provide a steady level of analgesia while reducing the side-effects associated with bolus administration.

Overall, epidural analgesia can provide highly effective management of post-operative pain. It is believed to

lead to a decreased stress response to surgery, improved post-operative pulmonary function and in high risk patients, decreased cardiac morbidity. Successful management relies on proper patient selection, appropriate catheter placement (depending on the level of the surgical site), adequate post-operative monitoring and specific training of personnel to identify and treat complications (including inadequate analgesia).

Epidural analgesia is commonly used after major intra-abdominal or thoracic surgery. A common use would be following (open) abdominal aortic aneurysm repair where the catheter might be left in for 48-72 hours. Occasionally, the need for post-operative thrombosis prophylaxis triggers the removal of the catheter as catheters should not be removed or left indwelling in the anticoagulated patient.

Peripheral Nerve Blocks

Almost any peripheral nerve that can be reached with a needle can be blocked with local anesthetics. The brachial plexus, intercostal and femoral nerves are examples of nerves which are commonly blocked to provide post-operative analgesia. A block may be used as the sole method of post-operative analgesia or it may be useful as an adjunct to decrease the required dose of systemic opioids. Some peripheral nerve sites (e.g. the brachial plexus) lend themselves to the insertion of catheters for the continuous infusion of local anesthetics. In the absence of catheter insertion, the major drawback of this method of post-operative analgesia is that the duration of effect of a single block is limited, usually to less than 18 hours.

A typical example of the use of a peripheral nerve block for post-operative pain would be the use of a femoral/sciatic nerve block for a patient undergoing total knee arthroplasty. The block would be augmented with oral opioids and other adjuncts.

CHAPTER 5

Special Patients



In this chapter, you will be introduced to several subsets of patients who present anesthetic concerns in addition to the ones previously discussed. You will develop an understanding of how anesthetic care is modified to accommodate these “special patients”. A review quiz is available at www.understandinganesthesia.ca

Malignant Hyperthermia

In This Section

1. Malignant Hyperthermia

Malignant Hyperthermia (MH) is a potentially life-threatening pharmacogenetic disorder characterized by the onset of a hypermetabolic crisis in response to certain triggers. Since the usual triggers are succinylcholine and volatile anesthetics, MH is known as “the anesthesiologist’s disease”.

Although MH is now known to be a genetically heterogeneous disorder, up to 70% of cases involve a mutation in a gene on chromosome 19 which encodes the ryanodine receptor protein. The ryanodine receptor is located on the sarcoplasmic reticulum. This particular mutation shows autosomal dominant inheritance pattern with variable penetrance. The other known causative gene for MH is CACNA1S, which is responsible for a voltage-gated calcium channel α -subunit. Several other chromosomal loci have been linked to MH although the specific genes have not yet been identified.

The MH-associated mutations cause an abnormality in skeletal muscle metabolism whereby uncontrolled intracellular release of calcium leads to sustained muscular contraction and cellular hypermetabolism. One very rare neuromuscular dis-

ease, called central core disease, is known to be associated with MH, while other more common neuromuscular disorders, such as Duchenne Muscular Dystrophy, are *possibly* associated with MH. Although rare (the incidence is reported to be 1 in 126,000 general anesthetics), MH does occur in geographical clusters.

The clinical manifestations of an MH crisis reflect the hypermetabolic state and may occur intra-operatively or post-operatively. The earliest sign is tachycardia followed by evidence of increased carbon dioxide (CO₂) production. Increase CO₂ production manifests as tachypnea in a spontaneously-breathing patient or raised end-tidal CO₂ levels in a mechanically-ventilated patient. Skeletal muscle rigidity is prominent. Hyperthermia is often delayed. Hypoxemia, acidosis, hyperkalemia, dysrhythmias and hemodynamic instability may ensue as the reaction progresses. Without treatment, the mortality rate for MH reaction is exceedingly high; even with prompt treatment, mortality may be as high as 10%.

Successful treatment of an MH crisis requires prompt recognition, discontinuation of triggering agents and administration of dantrolene. Dantrolene is a direct skeletal muscle relaxant which acts at the muscle cellular level. It is administered intravenously in 2.5 mg/kg doses until clinical signs show reversal of the hypermetabolic state. In most cases the symptoms will abate with a total dose of less than 20 mg/kg but the anesthesiologist must not hesitate to administer more dantrolene if the clinical indicators warrant. The remainder of treatment is supportive and involves hyperventilation with 100% oxygen, fluid administration and active cooling if temperature is elevated. One should be prepared to treat hyperkalemia and cardiac dysrhythmias. The surgical procedure should be terminated as quickly as is feasible after which the patient is transferred to the intensive care unit. Dantrolene should be continued in 1-2 mg/kg doses, every four hours for at least 24 hours. Patients should be monitored for recrudescence of the reaction as well as for complications such as myoglobinuria, renal failure and disseminated intravascular coagulation (DIC).

A very important component of care for the patient who has had an unexpected MH reaction is counseling and education for both patient and family. While the patient himself is known to be MH susceptible, his family members must be assumed to be MH susceptible un-

til it is proven otherwise. A diagnosis of MH has implications for employment, life insurance premiums and for future anesthetic management. Specialists at "MH clinics" are best able to advise the patient and his family; it is the duty of the attending anesthesiologist to make that referral. At the MH clinic, the appropriateness of muscle biopsy will be discussed. Muscle biopsy can rule out (or in) MH susceptibility in a family member but is painful and requires an anesthetic. It is expected that in the near future, the development of a genetic blood test will obviate the need for the invasive muscle biopsy in the majority of patients.

The anesthetic management of a patient known to be MH-susceptible is straightforward. Dantrolene prophylaxis can be given preoperatively to high risk patients. Intra-operatively, standard monitoring is used with an emphasis on end-tidal CO₂, O₂ saturation and temperature measurement. Triggers are avoided by using a "trigger-free" anesthetic machine which is free of vapourizers, and has been flushed clear of residual volatile gases. An anesthetic technique which does not involve the use of succinylcholine or volatile anesthetic gases is chosen. Post-operatively, the patient is usually observed in the post-anesthetic care unit for an extended period of time (e.g. 4 hours). If no suspicious signs (such as fever or unexplained tachycardia) are detected, routine post-operative care follows. The patient

can be discharged home if instructions regarding worrisome symptoms have been given and understood, and if the patient has reasonable access to the hospital from home, should problems arise.

Obstetrical Anesthesia

In This Section

1. Physiologic Changes of Pregnancy
2. Labour Analgesia
3. Anesthesia for Operative Delivery

Physiologic Changes of Pregnancy

Physiologic and anatomic changes develop across many organ systems during pregnancy and the postpartum period. Metabolic, hormonal and physical changes all impact on anesthetic management. To the anesthesiologist, the most important changes are those that affect the respiratory and circulatory systems.

Respiratory System

There is an increased risk of difficult or failed intubation in the parturient. This is primarily due mucosal vascular engorgement which leads to airway edema and friability. Laryngoscopy can be further impeded by the presence of large breasts.

In addition, the parturient is at risk for aspiration of stomach contents. During pregnancy, the stomach is displaced cephalad and intragastric pressure increases. Gastric motility is decreased and gastric secretions increase. This, combined with a decrease in the integrity of the gastroesophageal junction predisposes to pulmonary aspiration of gastric contents. In fact, airway complications (difficult intubation, aspiration) are the most common

anesthetic cause of maternal mortality. The best means of avoiding this outcome is to avoid general anesthesia (by using a regional technique) and thus maintain intact laryngeal reflexes. If a general anesthetic is required, NPO status for eight hours is preferred although not achievable in an emergency situation. Pretreatment of all parturients with a non-particulate antacid (30 cc sodium citrate p.o.) as well as with a histamine blocker (ranitidine 50 mg IV) is important. Finally, a rapid sequence induction with cricoid pressure is mandatory.

With the apnea that occurs at induction of anesthesia, the parturient becomes hypoxic much more rapidly than the non-pregnant patient. The reason for this is two-fold. Firstly, oxygen requirement has increased by 20% by term. Secondly, the functional residual capacity (FRC), which serves as an "oxygen reserve" during apnea, has decreased by 20% due to upward displacement of the diaphragm.

Adequate ventilation must be maintained during anesthesia. By term, minute ventilation has increased to 150% of baseline. This results in a de-

crease in PaCO₂ (32 mmHg). The concomitant rightward shift in the oxyhemoglobin dissociation curve allows increased fetal transfer of O₂.

Circulatory Changes

Blood volume increases by 40% during pregnancy in preparation for the anticipated 500-1000 cc average blood loss during vaginal or Caesarian delivery, respectively. This is significant for two reasons. Firstly, the normal signs of hypovolemia may not be seen until a relatively greater blood loss has occurred. Secondly, the expanded intravascular volume may not be tolerated by parturients with concomitant cardiovascular disease, such as mitral stenosis.

Due to the increasing uterine size, aortocaval compression (obstruction of the inferior vena cava and aorta) becomes relevant in the third trimester. When the pregnant patient is in the supine position, the heavy gravid uterus compresses the major vessels in the abdomen leading to maternal hypotension and fetal distress. Left lateral tilt, usually achieved with a pillow under the woman's right hip, is an important positioning maneuver.

Labour Analgesia

There are many methods of relieving the pain and stress of labour. The non-invasive methods, such as transcutaneous electrical nerve stimulation (TENS), hypnosis and massage require a well-prepared patient who is able to accept the incomplete relief that such methods inevitably provide. Invasive methods, such as inhaled (nitrous oxide), intravenous (opioids) or regional (epidural) are associated with side effects and risks to both fetus and mother. Epidural labour analgesia will be discussed briefly in this section.

The pain of the first stage of labour is referred to the T10-L1 somatic areas. This extends to include sacral segments (S2-4) during the second stage. Thus, the principle of epidural analgesia is to administer local anesthetics (with or without opioids) into the epidural space to block the aforementioned spinal segments.

The primary advantages of epidural analgesia are its high degree of effectiveness and safety. The patient remains alert and cooperative. In the absence of complications, there are no ill effects on the fetus. Epidural analgesia can be therapeutic for patients with pre-eclampsia or cardiac disease where a high catecholamine state is detrimental. Finally, the level and intensity of an epidural block can be extended to provide anesthesia for operative delivery (Caesarian section).

As well as blocking sensory fibres, local anesthetics in the epidural space interrupt transmission along sympathetic and motor neurons. The hypotension associated with sympathetic blockade can be minimized by a one litre bolus of crystalloid prior to institution of the block, slow titration of the local anesthetic, the use of lower concentrations of local anesthetic and vigilant guarding against aortocaval compression.

There is good evidence that a labour epidural is associated with a prolongation of the second stage of labour, due to the associated motor block. Whether it also leads to an increased incidence of operative delivery remains controversial. The degree of motor block can be minimized by using lower concentrations of local anesthetics along with opioid adjunct. The use of a local anesthetic infusion (as opposed to boluses or “top-ups”) may give a more consistent level of block, lower total dose of local anesthetic, less motor block and less risk of drug toxicity.

Anesthesia for Operative Delivery

The major causes of anesthetic morbidity and mortality in the pregnant patient are those related to the respiratory system. Because of the risks of aspiration and failed intubation, and the depressant effects of anesthetic agents on the fetus, general anesthesia is avoided (where possible) in the parturient undergoing Caesarian section. Regional anesthesia is the preferred technique and can be provided by administering spinal anesthesia or by extending the depth and height of an existing epidural block.

There are two situations where a regional technique would not be chosen for Caesarian section. The first would be in the presence of an absolute contraindication to regional anesthesia ([Table 11](#)). These include coagulopathy, hypovolemia, infection, certain cardiovascular conditions and patient refusal.

The second situation where a regional technique may not be appropriate is in the setting of severe fetal distress. In this setting, general anesthesia almost always allows the most rapid delivery of the compromised fetus. If the fetal heart rate is very low and the maternal airway appears favourable, then general anesthesia will be quickly induced.

General anesthesia in the parturient is unique in several respects which reflects the many physiologic changes in this patient population. The pregnant patient has a lower anesthetic requirement (MAC) and yet, paradoxically, is at higher risk of experiencing awareness under anesthesia. Other important considerations are the risk of aspiration, rapid desaturation and the need to avoid both neonatal depression and uterine atony.

With the patient in left lateral tilt, adequate IV access is ensured and the monitors are applied. The patient is prepped and draped *prior* to induction. After careful pre-oxygenation, a rapid sequence induction with cricoid pressure is performed. Generally speaking, no opioids are administered until delivery of the infant in order to avoid unnecessary neonatal depression. The patient is maintained on a 50% mixture of nitrous oxide and oxygen, and a low dose of volatile agent. The volatile anesthetics, in higher doses, can decrease uterine tone, which can lead to increased blood loss. After delivery of the fetus, a moderate dose of intravenous opioid is administered. As well, oxytocin is administered to augment uterine tone. The parturient must be extubated when fully awake so that intact laryngeal reflexes will protect against aspiration.

Post-operative pain management in the post-Caesarian section patient is usually straightforward as the lower abdominal incision is relatively well-tolerated. In the instance where intrathecal morphine was administered to the patient undergoing spinal anesthesia, up to 24 hours of pain relief can be achieved.

Pediatric Anesthesia

In This Section

1. Physiology of the Pediatric Patient

Not just a small adult...

The principles of pre-operative assessment, anesthetic management and post-operative care described earlier apply equally well to the pediatric patient. Specific variations in management of the pediatric patient result from differences in anatomy and physiology in this patient population, as compared to adult patients. Some of these differences are discussed briefly below.

Respiratory System

The pediatric airway differs from the adult airway in several respects. The occiput is relatively prominent in infants and young children. This means that the “sniffing position” is often best achieved with the head in the neutral position, without the use of a pillow. The relatively large tongue may hinder visualization of the larynx or contribute to upper airway obstruction under anesthesia. The epiglottis is long, angled and mobile. Because of this, a Magill blade is often used (in infants and young children) to lift the epiglottis directly to expose the larynx. The larynx itself is positioned higher (C4 vs. C6 in adult) and more anteriorly. The narrowest part of the pediatric air-

way is the subglottic region, at the level of the cricoid cartilage. Therefore, the use of a cuffed endotracheal tube (ETT) in a child less than 10 years of age is unnecessary and undesirable, as the narrow subglottic region provides its own seal. Because the trachea is narrowed, short and easily traumatized, appropriate selection of an ETT is critical. Recommended sizes of ETT by age are indicated in [Table 18](#). Generally, the formula below predicts the correct tube size for children over one year of age.

$$\text{ETT size} = 4 + (\text{age}/4)$$

The pediatric airway is relatively more prone to obstruction than the adult airway. Infants are obligate nose breathers and the nares are small and easily obstructed by edema or mucous. Due to subglottic narrowing, a small amount of edema resulting from ETT trauma or pre-existing infection (tracheitis or croup) can seriously compromise airway patency. Finally, laryngospasm is common in children. This complex and potentially life-threatening phenomenon can result

from non-specific stimuli as well as from direct irritation of the vocal cords by blood or secretions. In order to avoid laryngospasm, pediatric patients are extubated either at a deep plane of anesthesia or wide awake.

The pediatric patient is more prone to hypoxemia than most adults. Like the obstetric patient, children have a slightly smaller functional residual capacity (FRC) ([Table 19](#)). The FRC acts as a reserve tank of oxygen during apneic periods. In addition, the pediatric patient has a markedly increased oxygen consumption which

Table 18 Pediatric ETT sizes

AGE	ETT SIZE
TERM NEWBORN	3.0-3.5
0-12 MONTHS	3.5
1-2 YEARS	4.0
3 YEARS	4.5
4-5 YEARS	5.0
6-8 YEARS	5.5
8-9 YEARS	6.0
10-12 YEARS	6.5

Table 19 Oxygen reserve, delivery and consumption

	CHILD	ADULT
FRC (mL/kg)	30	35
Va (mL/kg)	150	60
Va/FRC	5	1.5
VO ₂ (mL/kg./min)	7.5	3.5

FRC=functional residual capacity, VA =minute alveolar ventilation, VO₂=minute oxygen consumption

is usually maintained with an increased minute ventilation. The result of both of these factors is that the pediatric patient will desaturate much more rapidly during apnea. Adequate pre-oxygenation is key to the airway management of the pediatric patient.

Cardiovascular

Infants and young children have a heart-rate dependent cardiac output. This means that with bradycardia, their stiff left ventricles are unable to increase stroke volume to maintain cardiac output. This explains why bradycardia is undesirable in pediatric patients. Curiously, the pediatric patient is relatively “vagotonic”. In other words, their vagus nerve is dominant and they are prone to developing bradycardia in response to cer-

tain types of noxious stimuli. Examples include hypoxemia and laryngoscopy. It is common practice, therefore, to pre-treat infants and young children with atropine just prior to the induction of anesthesia. Bradycardia in the pediatric patient must always be assumed to be a result of hypoxemia until proven otherwise.

Fluids and Metabolism

Management of fluid requirements follows the same principles described in the chapter on fluid management. The "4/2/1 rule" to calculate maintenance requirements applies equally well to the pediatric patient. There are some important differences, however. The blood volume of a child is greater, relative to their weight, compared to the adult ([Table 20](#)). This becomes important when calculating estimated blood loss as a percentage of the estimated blood volume as is done to guide to transfusion therapy.

The second important issue involves the type of maintenance fluid used. Because of its glucose and sodium concentrations, 2/3 D5W-1/3 N/S is appropriate for maintenance fluid administration in adults and children. In the operating room, we routinely administer N/S or R/L for maintenance because it is the crystalloid of choice for replacing blood volume and third space losses, which make up the bulk of the fluid needs in the intra-operative period. In infants and young children, however, it is less appropriate to use N/S or R/L

for maintenance especially during prolonged cases. Firstly, the immature kidney is unable to handle an excessive sodium load. Secondly, the child's liver glycogen stores may be insufficient to maintain normal serum glucose during a more prolonged period of fasting.

Table 20 Blood volume

AGE	BLOOD VOLUME (CC/KG)
preterm neonate	90
term neonate	80
infant	75
child	70
adult	60 - 70

Gastrointestinal

Children, generally speaking, present a lower risk of regurgitation and aspiration than adult patients. As well, they will become dehydrated more readily during a period of fasting. Thus, NPO guidelines for pediatric patients are more liberal than in the adult population. For

example, it is common practice to allow clear fluids from 2-4-hours pre-operatively in children under 12 years of age. Infants may be allowed breast milk up to 4 hours pre-operatively and formula up to 6 hours pre-operatively, breast milk being more readily digestible than formula.

Central Nervous System, Behaviour

Anesthetic requirements (MAC) are higher in infants and children, compared to adults, with the peak occurring at 6 months of age.

By 6 months to a year of age, infants become sufficiently aware of their surroundings to feel anxiety in the immediate pre-operative period. They are generally less inhibited about expressing their anxiety than their adult counterparts. There are many different approaches to minimizing this anxiety which must be individualized according to the needs of the patient, her parents and the anesthesiologist. Pediatric patients may be pre-medicated with benzodiazepines, opioids or anticholinergics. Unfortunately the administration of a pre-medication (even orally) can be distressing for these patients. Furthermore, depending on the agent chosen, recovery may be delayed. In many centers a parent is allowed in the operating room for induction to avoid separation anxiety for the child. This requires additional personnel to prepare and stay with the parent throughout. Studies have failed to show a clear benefit

to the child with this technique although the parents, for the most part, seem to prefer it. Inhalation ("mask") inductions are often used in order to avoid having to insert an IV in the awake child. However, for a struggling child, a mask induction may be more traumatic than an IV induction. The use of topical tetracaine (a local anesthetic) has made awake IV starts more feasible in this patient population.

Anesthesia Outside the Operating Room

In This Section

1. Anesthesia Care in Remote Locations

As diverse and ever-evolving as operating room practice is, anesthesiologists also have many opportunities to expand their practice outside of this realm. The ensuing discussion will look more closely some these challenging roles.

Anesthesiologists may provide conventional anesthetic services in locations remote from the operating room, such as radiology, burn center, endoscopy unit, lithotripsy unit, electrophysiology lab, or cardiac investigation unit. The anesthesiologist is often requested to monitor and sedate patients in order to render interventional procedures safer and more palatable to the patient. General anesthesia outside the operating room poses unique problems for the anesthesiologist and certain risks for the patient. Nonetheless, with adequate preparation and appropriate care, general anesthesia can be carried out almost anywhere.

As an expert in airway management, vascular access and fluid resuscitation, the anesthesiologist is a key member of the trauma team. The team resuscitates the patient, establishes the extent of injury and carries out appropriate investigations. These initial steps are followed by definitive therapy,

when necessary. The anesthesiologist's involvement in the care of the trauma victim does not end in the emergency room. Often, these victims arrive in the operating room urgently, where resuscitation is vigorously continued before and during the anesthetic.

Many anesthesiologists spend a proportion of their clinical time working in the intensive care setting. With the use of sophisticated physiologic monitoring, ventilatory care, pharmacologic support and acute pain management, critical care medicine is a natural extension of the anesthesiologist's role in the operating room. Many anesthesiologists pursue specialty training in critical care after their residency training.

Most hospitals have an Acute Pain Service (APS) run by the Anesthesia departments, for post-operative surgical patients. The APS initiates and supervises various pain management therapies such as patient-controlled analgesia (PCA) and epidural analgesia. These interventions are intensive and brief; they require constant and ongoing assessment to ensure safety and effectiveness. Acute pain management requires a special set of

skills that contributes greatly to patient recovery and satisfaction.

Large hospitals usually involve anesthesiologists in managing patients with chronic pain. Typically, the approach to chronic pain is multidisciplinary in that patients are assessed by a social worker, psychologist or psychiatrist, orthopedic surgeon and physiotherapist. Most often, these very complicated patients are referred to the clinic after seeing many other physicians. After taking a detailed history and performing a physical exam, the anesthesiologist may institute a nerve block, using local anesthetic and/or steroids. Examples of the indications for some of the more commonly performed blocks are given below:

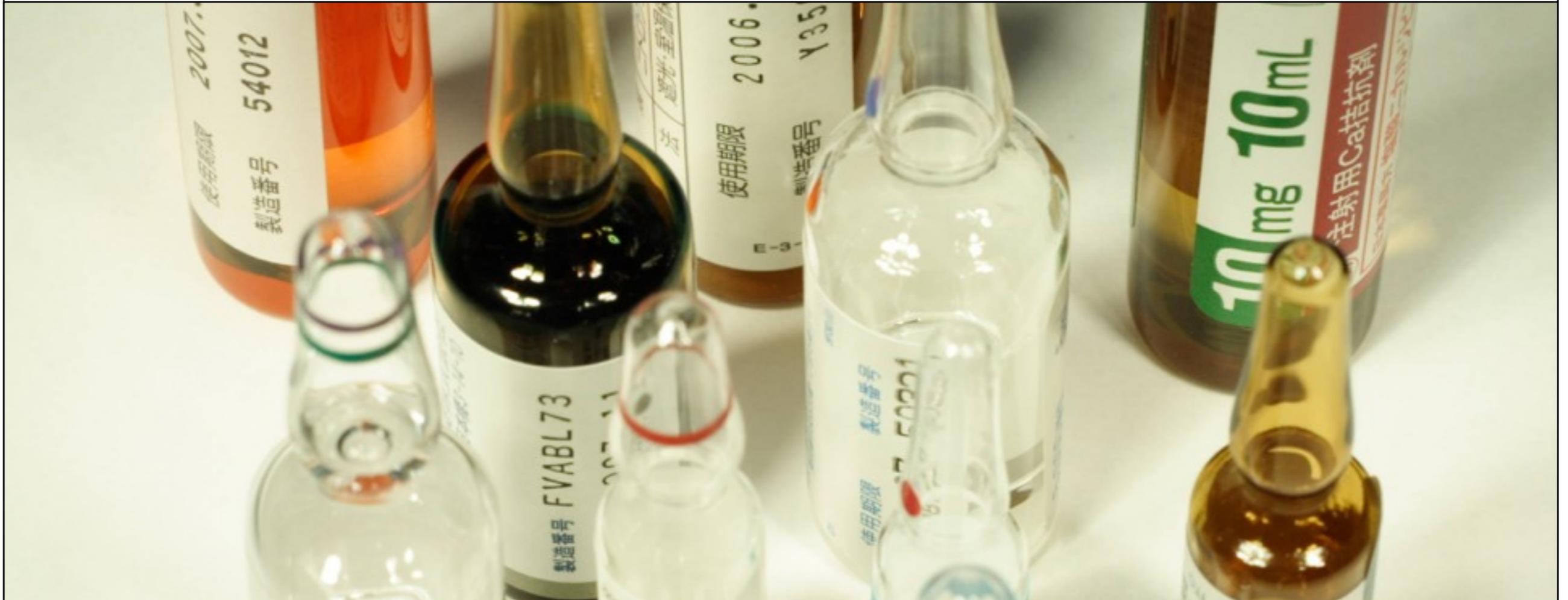
- Complex Regional Pain Syndrome (formerly Reflex Sympathetic Dystrophy): stellate ganglion block, lumbar sympathetic block
- Chronic Back Pain: caudal or lumbar epidural steroid injection
- Occipital Headaches: occipital nerve block
- Myofascial Pain: trigger point injections
- Chronic pancreatitis: celiac plexus block

Often, these patients are followed over a period of time, giving the anesthesiologist an opportunity to establish the long-term rapport not feasible in operating

room practice. Despite the notion that the anesthesiologist doesn't talk to his patients, this patient population must be treated with a good ear and a sympathetic tone if any treatment modality is to be successful.

CHAPTER 6

Drug Finder



In this chapter, the drugs that are commonly used in anesthetic practice are presented in reference format, grouped together by class. A review quiz is available at www.understandinganesthesia.ca

Opioid agonists and antagonists

- Fentanyl
- Sufentanil
- Remifentanyl
- Alfentanil
- Morphine Sulfate
- Meperidine
- Naloxone

Muscle relaxants

- Rocuronium
- Cis-Atracurium
- Pancuronium bromide
- Atracurium
- Succinylcholine chloride

Anticholinesterases and anticholinergics

- Neostigmine
- Glycopyrrolate
- Atropine Sulfate

Induction agents

- Propofol
- Sodium Thiopental
- Ketamine
- Etomidate

Inhaled agents

- Desflurane
- Sevoflurane
- Isoflurane
- Nitrous Oxide

Anxiolytics

- Midazolam

Antiemetics

- Ondansetron
- Dimenhydrinate
- Prochlorperazine

Vasoactive agents

- Phenylephrine
- Ephedrine sulfate
- Epinephrine

Local anesthetics

- Bupivacaine
- Lidocaine

Miscellaneous

- Ketorolac tromethamine
- Diphenhydramine
- Dantrolene

Opioid agonists and antagonists

Drugs

1. Fentanyl
2. Sufentanil
3. Remifentanyl
4. Alfentanil
5. Morphine Sulphate
6. Meperidine
7. Naloxone



Image courtesy of the Wood Library-Museum of Anesthesiology, Park Ridge, Illinois. Used with permission.

FENTANYL

Class

Synthetic opioid analgesic (intermediate-acting); adjunct to anesthesia. Fentanyl can be used as an additive to spinal and epidural anesthesia/analgesia.

Mechanism of Action

Acts at the mu-and kappa opioid receptors.

Dose

General anesthesia: 1-20 ug/kg IV according to physical status, other agents used, duration and nature of surgery.

Onset

IV 4-6 minutes

Duration

IV 30-45 minutes

Elimination

Hepatic

Effects

CNS

Potent analgesic effects; some sedative effect. Rarely causes blurred vision, seizures. All of the depressant effects of fentanyl are potentiated by concurrent use of sedatives, volatile anesthetics and nitrous oxide.

CVS

Hypotension, bradycardia. The synthetic opioids are not direct myocardial depressants but they do reduce sympathetic drive which may result in decreased cardiac output in patients who are relying on sympathetic tone to

support their circulation such as those in hypovolemic or cardiogenic shock.

Respiratory

Respiratory depression which at the extreme leads to apnea.

GI

Nausea, vomiting, biliary tract spasm, constipation.

Misc

Muscle rigidity

SUFENTANIL

Class

Synthetic opioid analgesic (intermediate-acting), adjunct to anesthesia.

Mechanism of Action

Acts at the mu-and kappa opioid receptors.

Dose

General anesthesia: 0.3-1 ug/kg IV, depending on patient condition, other agents used, nature and duration of surgery.

Infusion dose: 0.3-1 ug/kg/hour

Onset

1-2 minutes

Duration

20-40 minutes

Elimination

Hepatic

Effects

CNS

Potent analgesic properties and some sedative effect. All of the depressant effects of sufentanil are potentiated by concurrent use of sedatives, volatile anesthetics and nitrous oxide.

CVS

Bradycardia, hypotension. The synthetic opioids are not direct myocardial depressants but they do reduce sympa-

thetic drive which may result in decreased cardiac output in patients who are relying on sympathetic tone to support their circulation, such as those in hypovolemic or cardiogenic shock.

Respiratory

Respiratory depression, which at the extreme leads to apnea.

GI

Nausea, vomiting, biliary tract spasm, constipation.

Misc.

Muscle rigidity

REMIFENTANIL

Class

Synthetic opioid analgesic (ultra short-acting); adjunct to anesthesia.

Mechanism of Action

Acts at the mu-and kappa opioid receptors.

Dose

On induction of general anesthesia: 0.3-1 ug/kg

For maintenance of general anesthesia: 0.1-1 µg/kg/minute (by infusion)

For sedation: infusion 0.05 – 0.1 µg/kg/minute

Onset

After single bolus: 1-1.5 minutes

After initiation of infusion: 3-5 minutes

Duration

5-10 minutes; context sensitive half time 3 minutes

Elimination

Non-specific blood-tissue esterases (end-organ independent)

Effects

CNS

Potent analgesic effects, sedation. "MAC sparing" allows up to 75% reduction in dose of co-anesthetics. All of the depressant effects of remifentanil are potentiated

by concurrent use of sedatives, volatile anesthetics and nitrous oxide.

CVS

Exaggerated bradycardia, hypotension (compared with other opioids). The synthetic opioids are not direct myocardial depressants but they do reduce sympathetic drive, which may result in decreased cardiac output in patients who are relying on sympathetic tone to support their circulation, such as those in hypovolemic or cardiogenic shock.

Respiratory

Profound respiratory depressant which often leads to apnea.

GI

Nausea, vomiting.

Misc.

Can cause profound muscle rigidity. Not suitable for spinal or epidural use due to glycine additive. Rapid elimination requires initiation of post-operative analgesia (usually morphine) prior to emergence.

ALFENTANIL

Class

Synthetic opioid analgesic (short-acting); adjunct to anesthesia.

Mechanism of Action

Acts at the mu-and kappa opioid receptors.

Dose

5-50 ug/kg IV, according to physical status, other agents used, nature and duration of surgery.

Onset

1-2 minutes

Duration

20 minutes

Elimination

Hepatic

Effects

CNS

Analgesia, sedation. All of the depressant effects of alfentanil are potentiated by concurrent use of sedatives, volatile anesthetics and nitrous oxide.

CVS

Bradycardia, hypotension. The synthetic opioids are not direct myocardial depressants but they do reduce sympathetic drive, which may result in decreased cardiac output in patients who are relying on sympathetic tone to support their circulation, such as those in hypovolemic or cardiogenic shock.

Respiratory

Potent respiratory depression which at the extreme, leads to apnea.

GI

Nausea, vomiting, biliary tract spasm.

Misc.

Muscle rigidity, pruritis.

MORPHINE SULFATE

Class

Opioid analgesic (long acting). In anesthetic practice, its main use is for postoperative analgesia. Morphine is commonly used intravenously and for spinal or epidural anesthesia/analgesia.

Mechanism of Action

Active at the mu and kappa opioid receptors.

Dose

Adults: 2.5-15 mg IV/IM/SC

Children: 0.05-0.2 mg/kg IV/IM/SC

Onset

IV 5-10 minutes

IM 15-30 minutes

Duration

2-5 hrs IV/IM/SC

Elimination

Hepatic

Effects

CNS

Reliable analgesic effects; sedation. May cause blurred vision, syncope, euphoria, dysphoria. All of the depressant effects of morphine are potentiated by concurrent use of sedatives, volatile anesthetics, nitrous oxide and alcohol. Morphine's depressant effects are also potenti-

ated by antihistamines, phenothiazines, butyrophenones, MAOIs and TCAs.

CVS

May cause hypotension, hypertension, bradycardia, arrhythmias.

Respiratory

Respiratory depression which at the extreme leads to apnea. May cause bronchospasm or laryngospasm.

GI

Nausea, vomiting, constipation, biliary tract spasm.

Misc.

Releases histamine. May cause pruritis, urticaria, muscle rigidity, urinary retention.

MEPERIDINE

Class

Opioid analgesic (long acting). Traditionally used for postoperative pain but currently its use is restricted (in many hospitals) to the treatment of postoperative shivering.

Mechanism of Action

Acts at the mu and kappa opioid receptors.

Dose

In adults: 25-75 mg IV / IM (0.5-2 mg / kg)

Onset

IV: 3-8 minutes

IM: 10-20 minutes

Duration

2-4 hours IV / IM

Elimination

Hepatic

Effects

CNS

Causes dose-related sedation; variable analgesic effect. Delirium in older patients is often seen. May cause seizures if used in large doses or over an extended time frame due to the accumulation of its excitatory metabolite, normeperidine. May cause euphoria and dysphoria. All of the depressant effects of meperidine are potenti-

ated by concurrent use of sedatives, volatile anesthetics, nitrous oxide and tricyclic antidepressants.

Respiratory

Respiratory depression which at the extreme leads to apnea. May promote bronchospasm in susceptible patients (those with asthma or COPD).

GI

Nausea, vomiting, biliary tract spasm, constipation.

Misc.

Effective in the treatment of postoperative shivering. May cause muscle rigidity, urticaria, pruritis.

Contraindications:

Meperidine must not be used in patients on monoamine oxidase inhibitors in whom it can cause a fatal reaction.

NALOXONE

Class

Opioid antagonist. Used to counteract the effects of opioids.

Mechanism of Action

Agonist at the opioid receptors.

Dose

For postoperative opioid depression: 1-2 $\mu\text{g}/\text{kg}$ IV in 0.5-1 $\mu\text{g}/\text{kg}$ boluses, q 2-3 minutes

For neonatal opioid depression: 10 $\mu\text{g}/\text{kg}$, q 2-3 minutes IV. Infusion: 1-5 $\mu\text{g}/\text{kg}/\text{hr}$

Onset

1-2 minutes

Duration

30-60 minutes

Elimination

Hepatic

Effects

CNS

Rapid reversal of opioid effect can cause delirium and severe pain.

CVS

When opioid effect is abruptly antagonized there can be significant sympathetic activation leading to hypertension, tachycardia and in susceptible individuals, myocardial ischemia and pulmonary edema.

Misc.

Due to the relatively short duration of action of naloxone, "re-narcotization" can be seen when it is used to treat respiratory depression caused by long acting opioids such as morphine. In this case, close monitoring is indicated and supplemental doses may be necessary.

Muscle Relaxants

Drugs

1. Rocuronium
2. Cis-atracurium
3. Pancuronium bromide
4. Atracurium
5. Succinylcholine



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ROCURONIUM

Class

Non-depolarizing muscle relaxant (NDMR); short-acting

Mechanism of Action

Competitive inhibitor at the acetylcholine receptors of the post-synaptic cleft of the neuromuscular junction.

Dose

Intubation: 0.45-0.9 mg/kg

Maintenance bolus 0.1-0.2 mg/kg

Not usually administered by infusion

Onset

Dose-dependent:

1-1.5 minutes (0.6 mg/kg)

0.5-1.0 minutes (0.9 mg/kg)

Higher dose is therefore suitable for rapid sequence induction.

Duration

Dose-dependent:

31 minutes (0.6 mg/kg)

60 minutes (0.9 mg/kg)

Elimination

Hepato-biliary (70%); renal (10%)

Effects

CVS

Very weak vagolytic effect.

MSK

The neuromuscular blockade effects of non-depolarizing muscle relaxants are potentiated by succinylcholine, volatile anesthetics, aminoglycosides, lithium, loop diuretics, lidocaine, magnesium, lithium, ganglionic blockers, hypothermia, hypokalemia and respiratory acidosis.

Enhanced neuromuscular blockade is seen in patients with myasthenia gravis or myopathies.

The effects of NDMR are antagonized by cholinesterase inhibitors. Increased resistance to NDMRs is seen in patients on theophylline, burn patients and those with paresis or paralysis.

Misc.

Muscle relaxants are the most common cause of anaphylactoid reactions under general anesthesia.

CIS-ATRACURIUM

Class

Non-depolarizing skeletal muscle relaxant (NDMR); intermediate-acting

Mechanism of Action

Competitive inhibitor at the acetylcholine receptors of the post-synaptic cleft of the neuromuscular junction.

Dose

Intubation: 0.15-0.2 mg/kg

Maintenance bolus: 0.03 mg/kg

Maintenance infusion: 1-2 µg/kg/minute

Onset

Dose-dependent:

2 minutes (0.15 mg/kg)

1.5 minutes (0.2 mg/kg)

Duration

Dose dependent:

55 minutes (0.15 mg/kg)

65 minutes (0.2 mg/kg)

20 minutes (maintenance bolus 0.03 mg/kg)

Elimination

Hoffman elimination (77%), renal (16%)

Effects

MSK

The neuromuscular blockade effects of non-depolarizing muscle relaxants are potentiated by succinylcholine, volatile anesthetics, aminoglycosides, lithium, loop diuretics, lidocaine, magnesium, lithium, ganglionic blockers, hypothermia, hypokalemia and respiratory acidosis.

Enhanced neuromuscular blockade is seen in patients with myasthenia gravis or myopathies.

The effects of NDMR are antagonized by cholinesterase inhibitors. Increased resistance to NDMR is seen in patients on theophylline, burn patients and those with paresis or paralysis.

Misc.

Histamine release may occur with rapid administration or higher dosages. Produces 5-10x less laudanosine metabolite than atracurium. Muscle relaxants are the most common cause of anaphylactoid reactions under general anesthesia.

PANCURONIUM BROMIDE

Class

Nondepolarizing skeletal muscle relaxant (NDMR); long-acting

Mechanism of Action

Competitive inhibitor at the acetylcholine receptors of the post-synaptic cleft of the neuromuscular junction.

Dose

Intubation: 0.1 mg/kg IV

Maintenance bolus: 0.01-0.03 mg/kg

Onset

4-5 minutes

Duration

45-65 minutes

Elimination

Renal (80%), hepatic (minor)

Effects

CVS

Pancuronium has a vagolytic effect and therefore causes tachycardia and hypertension. Increased risk of arrhythmias in patients receiving tricyclic antidepressants and volatile anesthetics.

Respiratory

May promote bronchospasm, salivation.

MSK

The neuromuscular blockade effects of non-depolarizing muscle relaxants are potentiated by succinylcholine, volatile anesthetics, aminoglycosides, lithium, loop diuretics, lidocaine, magnesium, lithium, ganglionic blockers, hypothermia, hypokalemia and respiratory acidosis. Enhanced neuromuscular blockade is seen in patients with myasthenia gravis or myopathies.

The effects of NDMR are antagonized by cholinesterase inhibitors. Increased resistance to NDMR is seen in patients on theophylline, burn patients and those with paresis or paralysis.

Misc.

Muscle relaxants are the most common cause of anaphylactoid reactions under general anesthesia.

ATRACURIUM

Class

Nondepolarizing skeletal muscle relaxant (NDMR); short-acting

Mechanism of Action

Competitive inhibitor at the acetylcholine receptors of the post-synaptic cleft of the neuromuscular junction.

Dose

Intubation : 0.5-0.6 mg/kg IV

Maintenance bolus: 0.1-0.3 mg/kg IV

Onset

3-4 minutes

Duration

20-35 minutes

Elimination

Hoffman elimination, ester hydrolysis

Effects

MSK

The neuromuscular blockade effects of non-depolarizing muscle relaxants are potentiated by succinylcholine, volatile anesthetics, aminoglycosides, lithium, loop diuretics, lidocaine, magnesium, lithium, ganglionic blockers, hypothermia, hypokalemia and respiratory acidosis.

Enhanced neuromuscular blockade is seen in patients with myasthenia gravis or myopathies.

The effects of NDMR are antagonized by cholinesterase inhibitors. Increased resistance to NDMR is seen in patients on theophylline, burn patients and those with paresis or paralysis.

Misc.

Histamine release may occur with rapid administration or higher dosages. Produces an excitatory metabolite called laudanosine. Muscle relaxants are the most common cause of anaphylactoid reactions under general anesthesia.

SUCCINYLMCHOLINE CHLORIDE

Class

Depolarizing muscle relaxant; ultra short-acting; Used for rapid sequence induction.

Mechanism of Action

Succinylcholine (Sch) attaches to **nicotinic** cholinergic receptors at the neuromuscular junction. There, it mimics the action of acetylcholine thus depolarizing the post-junctional membrane. Neuromuscular blockade (paralysis) develops because a depolarized post-junctional membrane cannot respond to subsequent release of acetylcholine.

Dose

Intubaton: 1-1.5 mg/kg IV or 2.5-4 mg/kg IM

Onset

30-60 seconds after IV administration

2-3 minutes after IM dose

Duration

Duration is 4-6 minutes after IV dose

10-30 minutes after IM dose

Elimination

Hydrolysis by plasma pseudocholinesterase

Effects

CNS

Raised intracranial pressure and raised intraocular pressure.

CVS

Because of cross-reactivity at the muscarinic acetylcholine receptors, Sch causes vagal cardiac dysrhythmias. Bradycardia, junctional rhythm and sinus arrest can occur particularly if a second dose is administered and particularly in children.

Respiratory

Occasionally leads to bronchospasm and excessive salivation due to muscarinic effects. Intra gastric pressure is increased thereby theoretically increasing the risk of regurgitation.

Misc.

Most of the other effects are secondary to the depolarization and subsequent contraction of skeletal muscle. Sch elevates serum potassium 0.3-0.5 mEq/L in normal patients It can cause an exaggerated release of potassium (leading to fatal hyperkalemia) in those with neuromuscular or muscle disease. Post-operative myalgia is common particularly in young adults. Succinylcholine is a potent trigger of malignant hyperthermia.

Contraindications

There is a long list of absolute and relative contraindications which can be found in any Anesthesia text. A brief summary follows:

- Malignant Hyperthermia (MH) or presence of conditions associated with MH.

- Pseudocholinesterase deficiency. Deficiency can result as a genetic defect, as a consequence of various medications or a result of liver disease. The latter two causes are usually relative while the genetic defect can produce a complete lack of pseudocholinesterase activity in homozygous individuals. The use of succinylcholine in a patient with pseudocholinesterase deficiency leads to prolonged paralysis.
- Hyperkalemia.
- Presence of neurologic or muscular condition which would predispose to hyperkalemia after Sch-induced muscle contraction. Examples include recent paralysis (spinal cord injury or stroke), amyotrophic lateral sclerosis (ALS), Duchenne's muscular dystrophy and recent burn or crush injury. Myotonia congenita or myotonia dystrophica can manifest sustained contraction with Sch.

Anticholinesterase and Anticholinergics

Drugs

1. Neostigmine
2. Glycopyrrolate
3. Atropine sulfate



Atropa belladonna. Public domain image by Franz Eugen Köhler, Köhler's Medizinal-Pflanzen. Retrieved from Wikimedia Commons.

NEOSTIGMINE

Class

Anticholinesterase. In anesthesia practice, neostigmine is used for the reversal of neuromuscular blockade. Internal Internal Medicine specialists use neostigmine (or its relative, pyridostigmine) for the treatment of myasthenia gravis.

Mechanism of Action

Anticholinesterases inhibit the breakdown of acetylcholine (Ach) in the synaptic cleft by inhibiting the cholinesterase enzyme. As a result, Ach concentrations in the synaptic cleft are increased. Ach is then better able to compete with muscle relaxants for the Ach receptors and achieve depolarization of the muscle cell.

Dose

For reversal of neuromuscular blockade: 0.05 mg/kg

Dose should not exceed 5 mg

Must be administered with with atropine 0.015 mg/kg or glycopyrrolate 0.01 mg/kg

Onset

5 minutes

Duration

55-75 minutes

Elimination

Hepatic, plasma esterases

Effects

Most of neostigmine's effects are related to its cholinergic action. It must be given with an anticholinergic (atropine or more commonly glycopyrrolate) in order to minimize these effects.

CNS

Seizures

CVS

Bradycardia, AV block, nodal rhythm, hypotension

Respiratory

Increased oral and bronchial secretions, bronchospasm

GI/GU

Increased peristalsis, urinary frequency

Misc.

Overdose may produce cholinergic crisis. Neostigmine does not antagonize succinylcholine and may prolong phase 1 block of succinylcholine.

GLYCOPYRROLATE

Class

Anticholinergic. Clinical uses in anesthesia include the treatment of bradycardia; as a antisialagogue for awake intubation; or (most commonly) for counteracting the muscarinic effects of the anticholinesterases used for the reversal of neuromuscular blockade.

Mechanism of Action

An acetylcholine receptor blocker active at the muscarinic (not nicotinic) acetylcholine receptors. Therefore, glycopyrrolate has an anti-parasympathetic effect.

Dose

Antisialagogue: 0.1-0.2 mg IV/IM/SC in adults or 4-6 ug/kg IV/IM/SC in children

With anticholinesterase: 0.01 mg/kg IV

Onset

IV: <1 minute

IM/SC: 30-45 minutes

Duration

Vagal blockade: 2-3 hrs

Antisialagogue effect: 7 hours

Elimination

Renal, hepatic

Effects

Most effects result from the anticholinergic action of glycopyrrolate.

CNS

Confusion is less common than with atropine, as glycopyrrolate does not cross the blood brain barrier. May cause headache, dizziness, mydriasis, blurred vision, increased intraocular pressure.

CVS

Causes tachycardia at high doses and may cause bradycardia at low doses.

GU

Urinary hesitancy, retention

Misc.

Must be used in caution in patients with glaucoma, gastrointestinal or genitourinary obstruction.

ATROPINE SULFATE

Class

Anticholinergic. Clinical use in anesthesia includes the treatment of bradycardia and asystole; as a antisialagogue for awake intubation; or for counteracting the muscarinic effects of the anticholinesterases used for the reversal of neuromuscular blockade.

Mechanism of Action

An acetylcholine receptor blocker active at the muscarinic (not nicotinic) acetylcholine receptors. Therefore, atropine has an anti-parasympathetic effect.

Dose

Premedication 0.4-0.6 mg IV/IM in adults, 10-20 ug/kg IV/IM in children. Reversal 0.015 mg/kg IV with neostigmine 0.05 mg/kg IV.

Onset

Immediate

Duration

1-2 hours

Elimination

Hepatic, renal

Effects

Most effects result from the anticholinergic action of atropine.

CNS

Confusion, hallucinations, mydriasis, blurred vision, increased intraocular pressure

CVS

Tachycardia (high doses), bradycardia (low doses)

GI

Gastroesophageal reflux

GU

Urinary hesitancy, retention

Misc.

Has additive anticholinergic effects with antihistamines, phenothiazines, tricyclic antidepressants, mono-amine oxidase inhibitors and benzodiazepines. Potentiates sympathomimetics. May produce central anticholinergic syndrome.

Contraindications

Contraindicated in patients with narrow-angle glaucoma, gastrointestinal or genitourinary obstruction.

Induction Agents

Drugs

1. Propofol
2. Sodium thiopental
3. Ketamine
4. Etomidate



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PROPOFOL

Class

Alkylphenol intravenous anesthetic agent. Used for induction of general anesthesia. Can also be used for maintenance of anesthesia or for sedation, in each case by continuous infusion.

Mechanism of action

Not well described.

Dose

Induction: 2-2.5 mg/kg IV for adults

Induction: 3-4 mg/kg IV for children

Maintenance of anesthesia: 100-200 ug/kg/minute

Sedation: 40-100 ug/kg/minute

Onset

Within one arm-brain circulation time (approximately 20 seconds).

Duration

Approximately 5-8 minutes after single induction dose. Offset of effect is more prolonged when administered as a continuous infusion.

Elimination

Rapid redistribution away from central nervous system (CNS) into lean body compartment accounts for prompt awakening. Metabolized by liver and extra-hepatic sites then excreted by kidney.

Effects

CNS

Profound CNS depressant, potentiating the depressant effects of opioids, sedatives and volatile anesthetics. Decreases cerebral metabolic rate and intracranial pressure. Occasionally excitement, tonic-clonic movements or opisthotonus is seen on induction with propofol.

CVS

Causes direct myocardial depression and vasodilation leading to hypotension. Propofol must be used with caution in patients with poor left ventricular function or critical coronary artery insufficiency or in those who are seriously ill or debilitated.

Respiratory

Depression of respiratory centre leads to brief apnea. Propofol effectively blunts the airway's response to manipulation thus hiccoughing and bronchospasm are rarely seen.

Misc.

Pain on injection seen in up to 20%. Mild anti-emetic properties. Patients often experience pleasant dreams under anesthesia followed by a smooth, clear-headed emergence. Strict aseptic technique must be used when handling propofol as the vehicle is capable of supporting rapid growth of micro-organisms.

Contraindications

Egg or soy allergy.

SODIUM THIOPENTAL

Class

Short-acting barbiturate. Was used as an anesthetic induction agent but has largely been replaced by propofol. It is also useful as an anticonvulsant or for the rapid reduction of elevated intracranial pressure.

Mechanism of action

Decreases the rate of dissociation of the inhibitory neurotransmitter GABA from its receptors resulting in depression of the reticular activating system.

Dose

3-5 mg/kg IV for healthy adults

5-6 mg/kg IV for children

7-8 mg/kg IV for infants

Dose must be reduced considerably in unstable or fragile patients.

Onset

Within one arm-brain circulation time (approximately 20 seconds).

Duration

Approximately 5-10 minutes after single induction dose.

Elimination

Rapid redistribution of drug from the central nervous system (CNS) to lean body tissue accounts for the

prompt awakening. The final elimination from the body depends on hepatic metabolism and excretion by the kidneys.

Effects

CNS

Profound CNS depressant. Decreases cerebral metabolic rate and intracranial pressure. May cause hypertonus, twitching and tremors during induction. May contribute to post-operative confusion and delirium. Potentiates the depressant effects of opioids, sedatives, alcohol and volatile anesthetics.

CVS

Depression of myocardial contractility and vasodilation leads to decreased cardiac output and blood pressure with a mild compensatory tachycardia. Must be used with caution in patients with poor left ventricular function or critical coronary artery insufficiency or in those who are seriously ill or debilitated.

Respiratory

Depresses the rate and depth of breathing leading to brief period of apnea. Does not blunt the airway's response to manipulation therefore coughing, hiccoughing, laryngospasm and bronchospasm may be seen at light planes of anesthesia.

GI

Nausea and vomiting

Misc.

Incompatible with drugs with acidic pH. For example, if given in the IV line with vecuronium (a NDMR no longer in use), precipitation would occur. Arterial or extravascular injection produces necrosis.

Contraindications

Porphyria

KETAMINE

Class

Phencyclidine derivative. Can be used as an induction agent (usually in hemodynamically-compromised patients) or for sedation during painful procedures.

Mechanism of action

Acts at numerous central nervous system receptor sites, including the *N*-methyl-D-aspartate (NMDA) receptor.

Dose

Induction of anesthesia: 2 mg/kg IV

Induction of anesthesia: 5 mg/kg IM

Onset

Within one arm-brain circulation time (approximately 20 seconds).

Duration

Approximately 10-15 minutes after single induction dose, with full orientation occurring after 15-30 minutes.

Elimination

Redistribution from central nervous system (CNS) to inactive tissue sites accounts for termination of unconsciousness. Ultimate clearance is via hepatic metabolism and renal excretion.

Effects

CNS:

Produces "dissociative anesthesia" with patient in a cataleptic state. Ketamine provides a state of unconsciousness and intense analgesia however the patient's eyes may remain open and roving, and their limbs may move purposelessly. Cerebral metabolic rate and intracranial pressure are increased.

CVS

Ketamine increases sympathetic outflow from the CNS leading to increased heart rate, blood pressure and cardiac output. Because of this effect, ketamine plays an important role in the management of patients with hypovolemic shock or cardiac tamponade. However, ketamine does possess direct myocardial depressant effects which may lead to worsened hypotension in patients in a prolonged shock state.

Respiratory

Some degree of airway protection is maintained. The patient may cough or swallow. Airway secretions increase. Bronchodilatory effect is secondary to increased sympathetic tone. Apnea is rare as respiratory drive is maintained.

Misc.

Undesirable psychological reactions are common on emergence: vivid, unpleasant dreams, excitement, confusion, fear. They tend to occur in the first hour of emergence and abate within one to several hours. Pretreat-

ment with benzodiazepines may help minimize this effect.

Contraindications

Raised intracranial pressure, coronary ischemia, psychiatric disease, eye surgery.

ETOMIDATE

Class

Short-acting hypnotic; anesthetic induction agent. Useful in hemodynamically-compromised patients.

Mechanism of action

Potentiates the inhibitory GABA neurotransmitter resulting in depression of the reticular activating system.

Dose

Induction: 0.2-0.6 mg/kg IV

Onset

Within one arm-brain circulation time (approximately 20 seconds).

Duration

Approximately 5-10 minutes after single induction dose.

Elimination

Rapid redistribution from central nervous system (CNS) to lean body tissue accounts for brief duration of action. Ultimately metabolized by hepatic and plasma esterases to inactive products.

Effects

CNS

CNS depressant, potentiating the depressant effects of opioids, sedatives and volatile anesthetics. Decreases cerebral metabolic rate and intracranial pressure. The cerebroprotective effects of etomidate make it useful in the management of the head-injured patient. Can cause seizure-like activity.

CVS

Etomidate is notable for the lack of significant cardiovascular depression that it causes. Therefore it is commonly chosen to facilitate intubation in the trauma patient, patients with hypovolemic shock or other unstable patients.

Respiratory

Etomidate causes a brief period of apnea.

GI

Nausea and vomiting

Misc.

Etomidate suppresses corticosteroid synthesis in the adrenal cortex and can lead to primary adrenal suppression. For this reason, its use in patients with sepsis is controversial. Etomidate can result in trismus if administered too quickly.

Inhaled Agents

Drugs

1. Desflurane
2. Sevoflurane
3. Isoflurane
4. Nitrous oxide

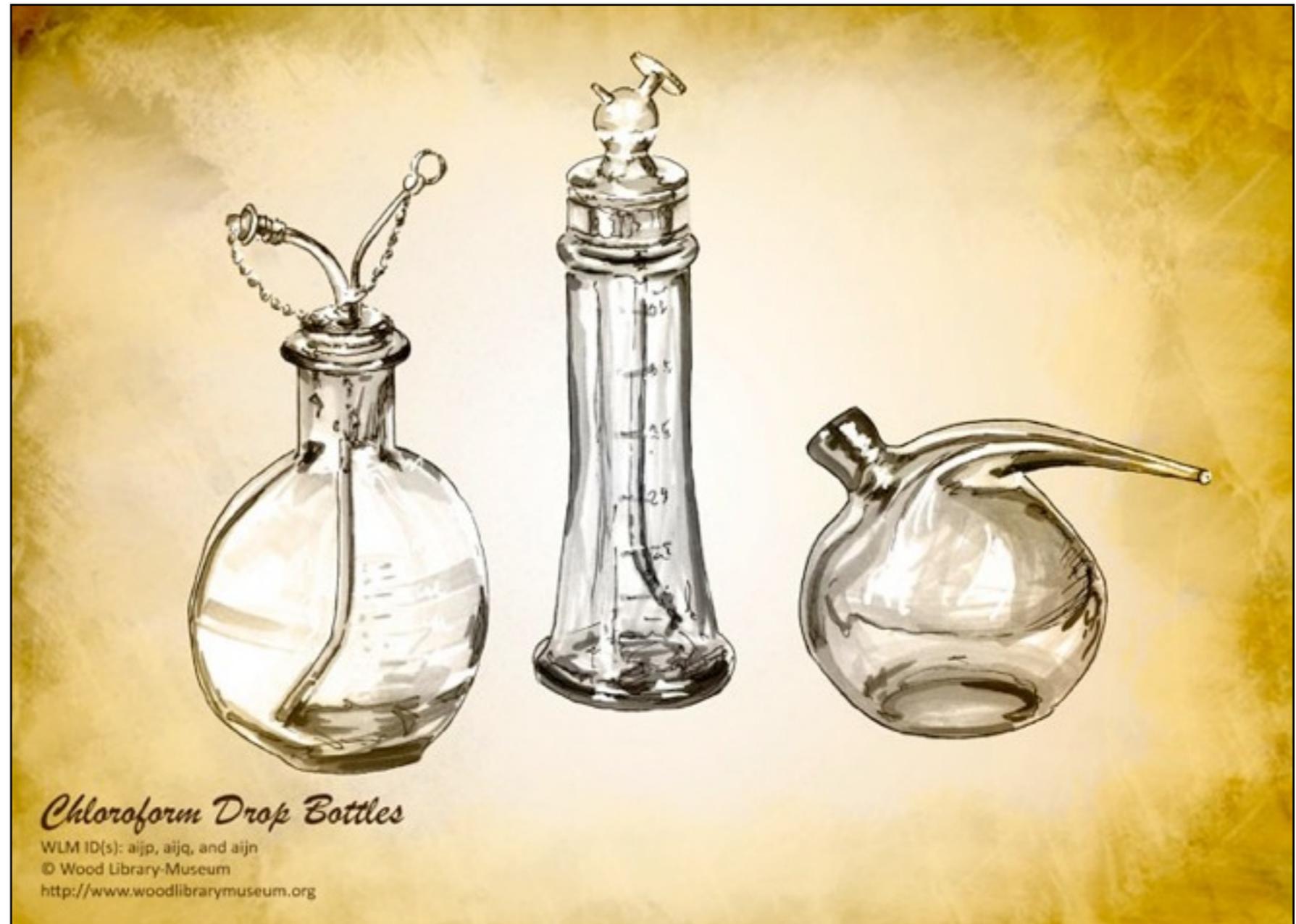


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DESFLURANE

Class

Volatile inhaled anesthetic. Used for maintenance of anesthesia.

Mechanism of Action

Uncertain

Dose

Titrated to effect; MAC (age 40) = 6.0%

Onset

Low solubility allows rapid uptake and equilibration. Onset of effect is hastened by using higher flows of carrier gases and by using higher concentrations of volatile agent.

Duration

Clinical recovery in less than 10 minutes (2-2.5 x faster washout than Isoflurane)

Elimination

Pulmonary (major); negligible hepatic (0.02%)

Effects

CNS

Desflurane produces an additive central nervous system (CNS) depressant effect along with other sedative/hypnotics and analgesics. Sympatho-excitation can occur with rapid increase in concentration of desflurane. Has the potential to increase intracranial pressure which can be mitigated with hyperventilation. May cause headache, agitation, dizziness.

CVS

Dose-related hypotension (vasodilation). Tachycardia and hypertension may be seen due to sympathetic nervous system activation.

Respiratory

Respiratory depression with a rapid, shallow respiratory pattern. Loss of intercostal muscle function creates a rocking boat appearance. Desflurane is irritating to the airways and can cause breath-holding, cough, laryngospasm or bronchospasm in susceptible individuals, especially if used as sole agent for induction.

GI

Potential immune-mediated hepatotoxicity. Nausea, vomiting.

MSK

Potentiates neuromuscular blockade; malignant hyperthermia trigger.

Misc.

Significant carbon monoxide production occurs on exposure to desiccated CO₂ absorbing agents therefore must not be used with low-flow anesthesia. Rapid elimination requires initiation of post-operative analgesia prior to emergence.

Contraindications

Malignant hyperthermia susceptibility

SEVOFLURANE

Class

Volatile inhaled anesthetic. Used for maintenance of anesthesia. Can be used for induction of anesthesia particularly in children. Rarely may be used as a treatment for status asthmaticus.

Mechanism of Action

Uncertain

Dose

Titrated to effect; MAC (age 40) = 2.1%.

Onset

Low solubility allows rapid uptake and equilibration. Onset of effect is hastened by using higher flows of carrier gases and by using higher concentrations of volatile agent.

Duration

Clinical recovery in less than 10 minutes (usually). If given for prolonged periods, wake-up will be slower as adipose stores have been saturated and are slow to off-load.

Elimination

Pulmonary (major); hepatic (2-5%); renal (metabolites excretion only)

Effects

CNS

Sevoflurane produces an additive central nervous system (CNS)-depressant effect along with other sedative/

hypnotics and analgesics. Has the potential to increase intracranial pressure which can be mitigated with hyperventilation. Delirium.

CVS

Dose-related hypotension (vasodilation).

Respiratory

Respiratory depression with a rapid, shallow respiratory pattern. Loss of intercostal muscle function creates a rocking boat appearance. Causes bronchodilation. Sevoflurane is sweet-smelling and not as irritating to the respiratory tract as desflurane.

GI

Nausea, vomiting.

MSK

Potentiates neuromuscular blockade. Malignant hyperthermia trigger.

Misc.

Potential nephrotoxicity due to Compound A which is produced through contact with soda lime. Compound A can be produced if sevoflurane is used with very low fresh gas flows or for long MAC-hours. Therefore, sevoflurane must be used with a minimum of 2 litres/minute of fresh gas flow.

Contraindications

Malignant hyperthermia susceptibility

ISOFLURANE

Class

Volatile inhaled agent. Used for maintenance of anesthesia.

Mechanism of Action

Uncertain

Dose

Titrated to effect; MAC (age 40)=1.15

Onset

Higher solubility than sevoflurane and desflurane therefore uptake is slower than the modern agents. Onset of effect is hastened by using higher flows of carrier gases and by using higher concentrations of volatile agent.

Duration

Clinical recovery in less than 15 minutes (usually). Theoretically a slower wake-up than the modern agents due to higher solubility.

Elimination

Pulmonary

Effects

CNS

Isoflurane produces an additive central nervous system (CNS)-depressant effect along with other sedative/hypnotics and analgesics. Has the potential to increase intracranial pressure which can be mitigated with hyperventilation. Delirium.

CVS

Dose-related hypotension (vasodilation).

Respiratory

Respiratory depression with rapid, shallow respiratory pattern. Loss of intercostal muscle function creates a rocking boat appearance. Isoflurane is irritating to the airways and can cause breath-holding, cough, laryngospasm or bronchospasm. Its pungent quality makes it unsuitable for use with a mask induction.

GI

Nausea, vomiting.

MSK

Potentiates neuromuscular blockade. Malignant hyperthermia trigger.

Contraindications

Malignant hyperthermia susceptibility

NITROUS OXIDE

Class

Nitrous oxide is an inhaled agent but *not* a volatile agent. It is used as an adjunct to general anesthesia. It has a weak effect and therefore cannot be used as the sole agent for general anesthesia and is most commonly used in combination with a volatile agent. It can be used on its own for sedation or analgesia as can be seen in the obstetric or dental setting.

Mechanism of Action

Uncertain

Dose

Delivered in concentrations of up to 70% in oxygen. Actual MAC is 104%.

Onset

Immediate due to very low solubility.

Duration

Offset of effect is rapid after discontinuation.

Elimination

Pulmonary

Effects

CNS

N₂O is a potent analgesic. It increases cerebral metabolic rate, cerebral blood flow and intracranial pressure and is therefore not a good choice for patients with decreased intracranial compliance.

CVS

N₂O has a mild sympathomimetic effect but causes direct myocardial depression. The net effect is a modest decrease in blood pressure and heart rate. Increased coronary tone may exacerbate ischemia in susceptible patients.

Respiratory

N₂O produces mild respiratory depression which is potentiated by opioids, hypnotics and volatile anesthetics. It has no bronchodilatory effect. It exacerbates pulmonary hypertension.

Misc.

N₂O expands the volume of gas-containing spaces as N₂O diffuses across membranes more readily than nitrogen can diffuse out. Thus the size of a pneumothorax, emphysematous bleb or distended bowel loop will increase when N₂O is used. Bone marrow suppression due to inhibition of methionine synthetase, can occur if N₂O is used for extended periods. N₂O enhances opioid-induced rigidity. Finally, N₂O is an operating room pollutant; N₂O levels (in parts per million) in the operating room environment are measured regularly to comply with workplace safety regulations.

Contraindications

Raised intracranial pressure, pneumothorax or bowel obstruction. Should be used with caution in patients with coronary disease or emphysema.

Anxiolytics

Drugs

1. Midazolam

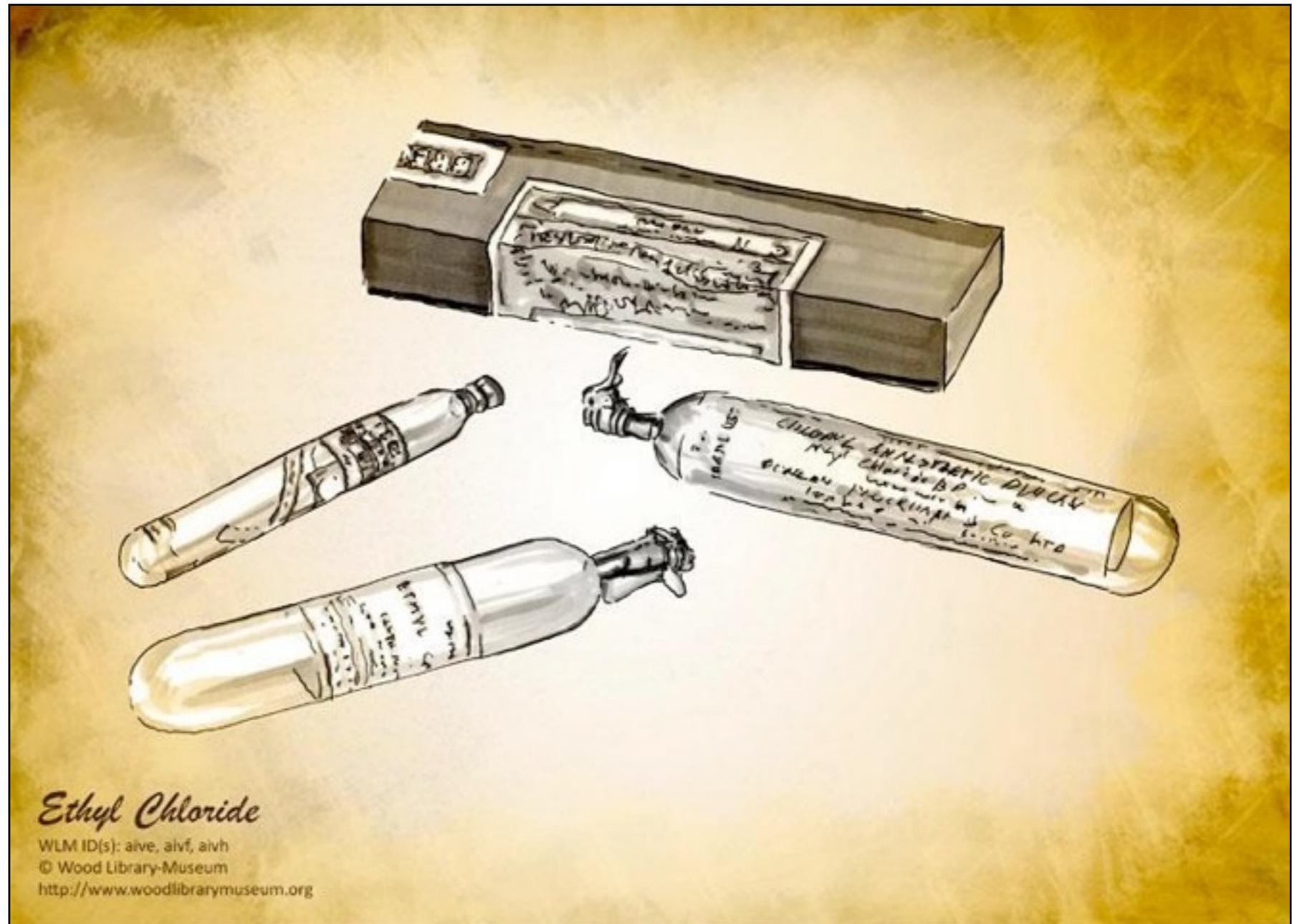


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MIDAZOLAM

Class

Short-acting benzodiazepine. Used for sedation or as an adjunct during general anesthesia. Midazolam has anxiolytic and sedative (but not analgesic) properties.

Mechanism of Action

Agonism at the inhibitory GABA receptor.

Dose

For sedation: 0.03-0.08 mg/kg IV

Can also be given intramuscularly, intranasally and orally.

Onset

Within 3-5 minutes

Duration

Elimination half-time is 1-4 hours, making midazolam a much shorter acting agent than diazepam.

Elimination

Metabolized in the liver by microsomal enzymes and excreted in the urine.

Effects

CNS

Induces anxiolysis, amnesia, hypnosis. Decreases cerebral blood flow. Minimal effects on intracranial pressure. May contribute to post-operative delirium in the elderly.

CVS

In larger doses, in the presence of hypovolemia or when used in combination with opioids, midazolam can lead to decreased blood pressure and increased heart rate. Cardiac output is unchanged.

Respiratory

Dose-related respiratory depression occurs. This response is exaggerated in the elderly, in those with COPD or when used in combination with opioids.

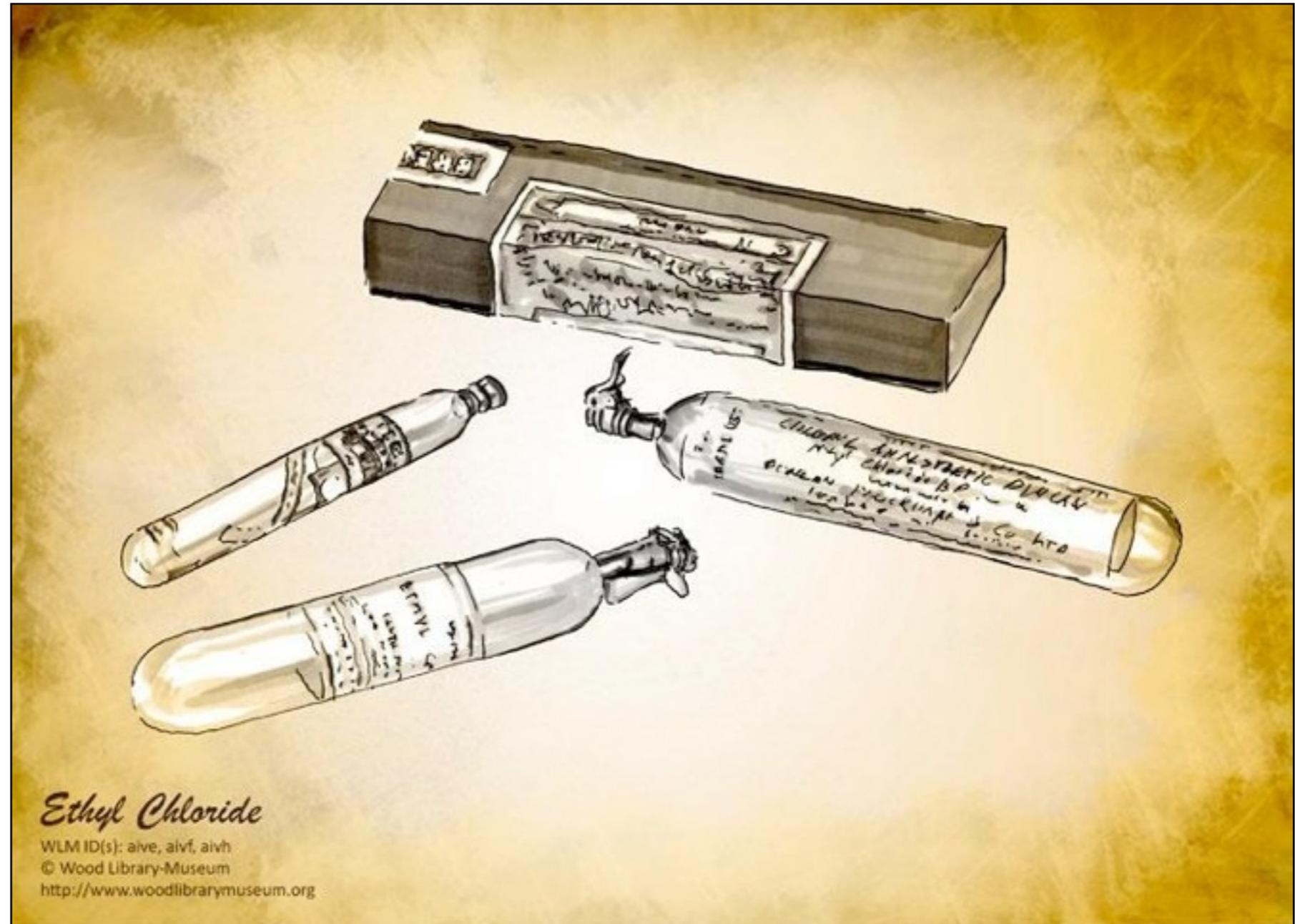
Misc.

Midazolam is water-soluble therefore the pain on injection and phlebitis that are seen with diazepam are uncommon.

Antiemetics

Drugs

1. Ondansetron
2. Dimenhydrinate
3. Prochlorperazine



Ethyl Chloride

WLM ID(s): aive, aivf, aivh
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ONDANSETRON

Class

Serotonin (5-HT₃) antagonist. Clinical use is as an antiemetic for post-operative nausea and vomiting or for patients receiving chemotherapy.

Mechanism of Action

Ondansetron is a highly selective competitive antagonist of the serotonin receptor. It is believed to have its effect centrally, possibly in the area postrema of the brainstem where the chemoreceptive trigger zone is located.

Dose

Prophylaxis (adults): 4 mg IV prior to emergence.

Prophylaxis (children): 50-150 µg/kg IV

Treatment (adults): 1-2 mg IV

Onset

Less than 30 minutes

Duration

9 hours

Elimination

Hepatic (95%)

Effects

CNS

Headache

CVS

May cause cardiac rhythm or ECG changes by prolongation of the QT interval.

GI

Constipation, elevation of liver enzymes.

Misc.

Elimination of ondansetron is prolonged when given with other drugs metabolized by cytochrome P450 system.

DIMENHYDRINATE

Class

Antihistamine, antiemetic. In anesthetic practice, used as a second or third line treatment of post-operative nausea and vomiting (PONV). No role in prevention of PONV.

Mechanism of Action

Dimenhydrinate is a competitive antagonist at the histamine H1 receptor. The antiemetic effects is related to central anticholinergic actions as well as histamine antagonism in the vestibular system in the brain.

Dose

50-100 mg IV q4-6h, max. 400 mg/day (adults)

1.25 mg/kg IV q6h (children)

Onset

5 minutes after IV administration

Duration

4-6 hours

Elimination

Hepatic

Effects

CNS

Sedation (which is additive with alcohol and sedative hypnotics), dizziness, restlessness.

Misc.

May cause dry mouth, blurred vision, difficult urination; more rarely causes acute glaucoma or worsening of asthma. These side effects reflect its anticholinergic activity which is additive with other anticholinergics and monoamine oxidase inhibitors (MAOI).

PROCHLORPERAZINE

Class

Although it has several uses, in anesthetic practice it is used as an antiemetic for post-operative nausea and vomiting (PONV).

Mechanism of Action

Central inhibition of the dopamine D₂ receptors in the medullary chemoreceptor trigger zone. Prochlorperazine also inhibits the vagus nerve in the gastrointestinal tract. The anticholinergic, sedative and antihistaminic effects of prochlorperazine also contribute to its antiemetic action.

Dose

2.5-10 mg IV, max. 40 mg/day (adults)

Onset

10-20 minutes

Duration

3-4 hours

Elimination

Enterohepatic

Effects

Prochlorperazine has anticholinergic properties which are additive to the anticholinergic effects of other drugs. As a phenothiazine, it also has the potential to cause extrapyramidal symptoms.

CNS

Sedative effects which are additive to other-hypnotics> May cause extra-pyramidal syndromes (motor restlessness, oculogyric crisis, opisthotonus, dystonias), especially in young male patients.

CVS

Hypotension caused by α -adrenergic blocking effect. May potentiate hypotensive effect of vasodilators and diuretics. Causes QT interval prolongation.

Misc.

Diminishes effects of anticoagulants. Possible hyperthermia in the presence of hypothalamic dysfunction. Neuroleptic malignant syndrome.

Vasoactive Agents

Drugs

1. Phenylephrine
2. Ephedrine sulfate
3. Epinephrine



Ephedra distachya. Public domain image by Prof. Dr. Otto Wilhelm Thomé Flora von Deutschland, retrieved from Wikimedia Commons.

PHENYLEPHRINE

Class

Sympathomimetic; vasopressor. Used in the treatment of hypotension.

Mechanism of Action

Direct agonist at the α -adrenergic receptor.

Dose

Bolus dose: 50-100 μ g IV (adults)

Infusion: 0.1-1.0 μ g/kg/minute

Onset

<1 minute

Duration

<5 minutes

Elimination

Re-uptake by tissue, liver and gut (monoamine oxidase)

Effects

CVS

Main effect is peripheral vasoconstriction, causing an increase in blood pressure. It is most appropriately used to raise the blood pressure in patients who are peripherally vasodilated (as a result of anesthesia, for example). It has the potential to cause myocardial ischemia, and left and right ventricular failure. It routinely causes a reflex bradycardia. If used in a patient in cardiogenic or hypovolemic shock, it may lead to a further reduction in vital organ blood flow.

Misc.

The clinician may observe diminished response of phenylephrine in patients receiving α -adrenergic blockers or drugs with α -blocking action such as phenothiazines. On the other hand, there may be augmented response when given with other vasopressors such as vasopressin and ergonovine. Phenylephrine has prolonged action in patients using monoamine oxidase inhibitors.

EPHEDRINE SULFATE

Class

Sympathomimetic (indirect-acting); vasopressor. Used in the treatment of hypotension.

Mechanism of Action

Ephedrine causes more norepinephrine to be released from the storage vesicles in the terminal of neurons thus increasing the amount of norepinephrine in the synaptic space. Ephedrine is (mostly) an “indirect-acting” catecholamine because it doesn’t act at the post-synaptic norepinephrine receptors.

Dose

5-20 mg IV (adults)

25-50 mg IM (adults)

Onset

IV: immediate

IM: minutes

Duration

IV: 10-minutes

IM: 60 minutes

Elimination

Hepatic, renal

Effects

CNS

Increases MAC of volatile anesthetics

Respiratory

Bronchodilator

CVS

Increases heart rate, contractility and therefore cardiac output (through its β adrenergic effect). Overall effect is to increase systemic vascular resistance through its α -adrenergic effect. May cause arrhythmias especially when used with volatile anesthetics. As the mechanism of action involves the release of intracellular catecholamines, there is an unpredictable effect in patients with depleted endogenous catecholamines.

Misc.

Excessive catecholamine effects may lead to hypertension, tachycardia, arrhythmias, pulmonary edema, anxiety, tremors, hyperglycemia and transient hyperkalemia followed by hypokalemia. Skin necrosis may occur at site of injection.

Contraindications

Ephedrine should not be used in patients on monoamine oxidase inhibitors (MAOIs) or those using cocaine. In these patients, phenylephrine is a safer choice for raising blood pressure. Ephedrine should be used with caution in patients who take SSRIs (serotonin-norepinephrine re-uptake inhibitors), as it may increase the risk of “serotonin syndrome”.

EPINEPHRINE

Class

Sympathomimetic. Epinephrine has many uses:

- 1) Inotropic support in the patient in cardiogenic shock.
- 2) Bronchodilation in status asthmaticus.
- 3) Treatment of allergic reactions.
- 4) Treatment of croup.
- 5) Resuscitation in cardiovascular collapse of any cause.
- 6) Prolongation of action of anesthetic solutions.

Mechanism of Action

Epinephrine is a direct-acting sympathomimetic. It stimulates α - and β -adrenergic receptors resulting in a wide range of effects attributable to the sympathetic nervous system.

Dose

Cardiac arrest (adults): 0.5-1.0 mg IV q5min prn

Inotropic support (adults): 0.1-1.0 ug/kg/min

Anaphylaxis/severe asthma:

Adults: 0.1-0.5 mg SC/IM or 1-5 ug/kg IV prn

Children: 0.01 mg/kg SC/IM, maximum 0.5 mg

Onset

IV: immediate

SC/IM: 6-15 minutes

Duration

IV: 5-10 minutes

SC: 1-3 hours

Elimination

Enzymatic degradation

Effects

CNS

Anxiety, headache, stroke

CVS

Hypertension, tachycardia, arrhythmias, angina, pulmonary edema. Increased risk of ventricular arrhythmias is seen when used with volatile anesthetics.

Respiratory

Bronchodilation

Misc.

Skin necrosis at site of injection, hyperglycemia, transient hyperkalemia, followed by hypokalemia.

Local Anesthetics

Drugs

1. Bupivacaine
2. Lidocaine



Cocaine

WLM ID(s): aiuz, aiuy, aiva, aivb
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BUPIVACAINE

Class

Local anesthetic. Used in infiltration anesthesia, spinal and epidural anesthesia and other regional anesthesia techniques.

Mechanism of Action

Sodium channel blocker

Dose

Maximum 2mg/kg without epinephrine

Maximum 3 mg/kg with epinephrine

Safe dose depends on where and how it is being administered. For example, absorption from intercostal administration is greater than for administration in adipose tissue.

Onset

Infiltration: 2-10 minutes

Epidural: 10-30 minutes

Spinal: <5 minutes

Duration

Infiltration: 2-5 hours

Epidural and spinal: up to 3.5 hours

Elimination

hepatic, pulmonary

Effects

Local anesthetics should not have systemic effects if used appropriately. If high plasma levels are achieved due to incorrect dosing or inadvertent intravascular injection then the symptoms manifest firstly in the central nervous system and then in the cardiovascular system where hypotension, heart block and other arrhythmias may occur. Premonitory signs and symptoms are perioral numbness, metallic taste, tinnitus, restlessness, dizziness and tremors. Seizures, respiratory and circulatory depression / arrest may occur. The treatment is supportive care and the use of Intralipid. Administration of benzodiazepines will increase the seizure threshold.

High intravascular concentrations of local anesthetics may potentiate the effects of muscle relaxants (both depolarizing and non-depolarizing).

LIDOCAINE

Class

Local anesthetic. Used in infiltration anesthesia and regional anesthesia (e.g. intravenous regional anesthesia). Lidocaine is still used for epidural anesthesia, especially for Caesarian section. Lidocaine is rarely used in spinal anesthesia due to associated nerve irritation. Lidocaine is occasionally used in the treatment of ventricular arrhythmias.

Mechanism of Action

Sodium channel blocker

Dose

Anesthetic:

Maximum 4 mg/kg without epinephrine

Maximum 7 mg/kg with epinephrine

Anti-arrhythmic:

1 mg/kg IV bolus followed by 0.5 mg/kg q 2-5 minutes to maximum 3 mg/kg/hr

By infusion (of 0.1% solution): 1-4 mg/min (20-50 µg/kg/min)

Onset

IV: 45-90 seconds

Infiltration: 0.5-1 minute

Epidural: 5-15 minutes

Spinal: <1 minute

Duration

IV: 10-20 minutes

Infiltration: 0.5-1 hour

Epidural and spinal: 1-3 hours

Elimination

hepatic, pulmonary

Effects

Local anesthetics should not have systemic effects if used appropriately. If high plasma levels are achieved due to incorrect dosing or inadvertent intravascular injection then the symptoms manifest firstly in the central nervous system and then in the cardiovascular system where hypotension, heart block and other arrhythmias may occur. Premonitory signs and symptoms are perioral numbness, metallic taste, tinnitus, restlessness, dizziness and tremors. Seizures, respiratory and circulatory depression / arrest may occur. The treatment is supportive care and the use of Intralipid. Administration of benzodiazepines will increase the seizure threshold.

High intravascular concentrations of local anesthetics may potentiate the effects of muscle relaxants (both depolarizing and non-depolarizing).

Miscellaneous

Drugs

1. Ketorolac tromethamine
2. Diphenhydramine
3. Dantrolene

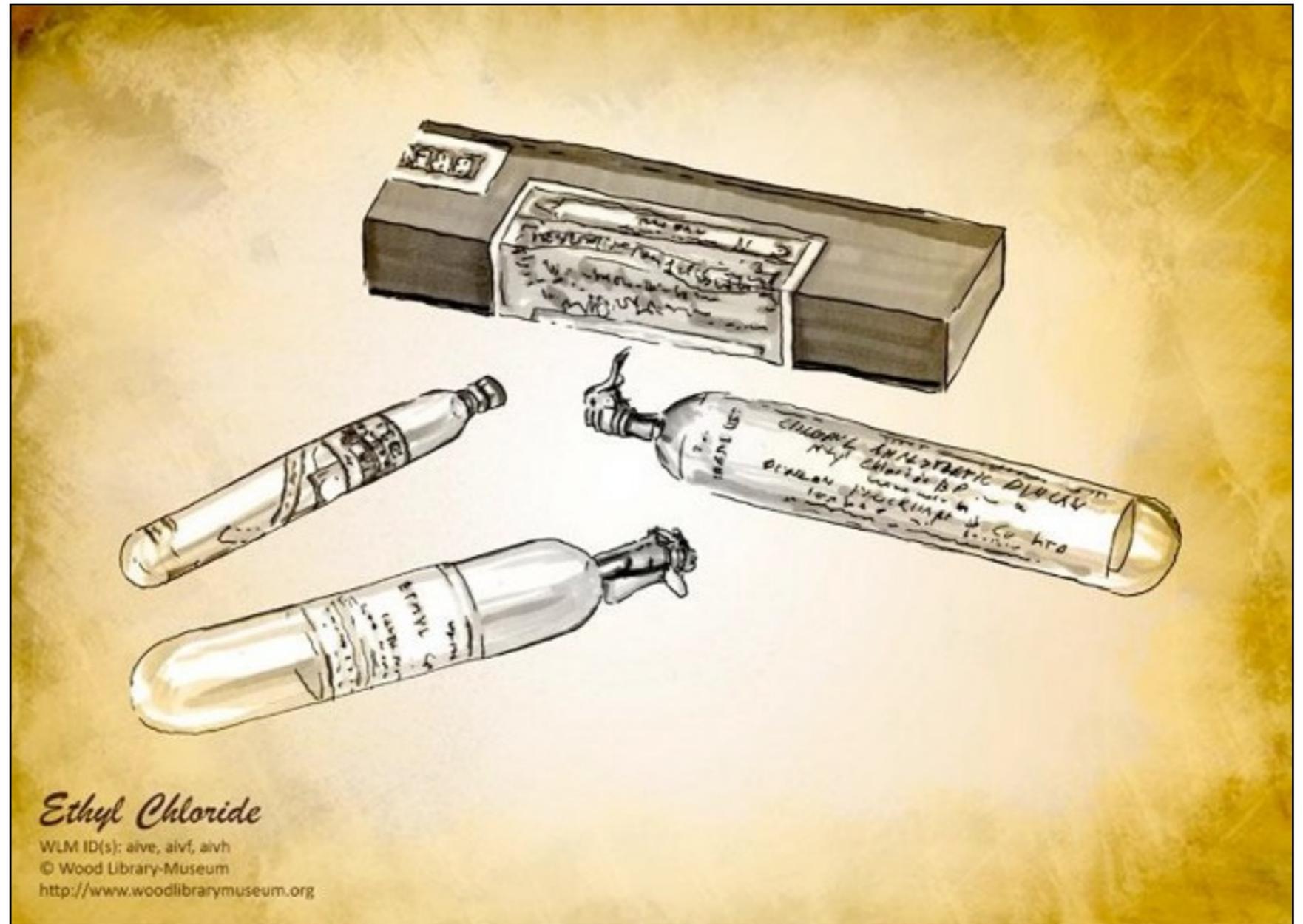


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KETOROLAC TROMETHAMINE

Class

Non-steroidal anti-inflammatory analgesic. Can be used orally or intravenously.

Mechanism of Action

COX-1 inhibitor

Dose

15-30 mg IV (adults)

0.5 mg/kg, max. 15 mg/dose (children > 2 years)

IV therapy should not exceed 2 days.

Onset

<30 minutes; peak effect in 1-2 hours.

Duration

4-6 hours

Elimination

Renal (92%); enterohepatic (6%)

Effects

CNS

Analgesic with an opioid-sparing effect.

GI

Gastrointestinal bleeding, ulcer, perforation, nausea, dyspepsia.

Renal

May precipitate renal failure in patients with renal insufficiency or those using ACE inhibitors.

Hematologic

May potentiate effects of anticoagulants. Rarely may cause hemorrhage due to platelet inhibition.

DIPHENHYDRAMINE

Class

Antihistamine, antiemetic. Used in the treatment of pruritis, allergic reactions and drug-induced extrapyramidal reactions.

Mechanism of Action

Diphenhydramine is a competitive inhibitor at the histamine H1 receptor. The antiemetic effects is related to central anticholinergic effect as well as histamine antagonism in the vestibular system in the brain.

Dose

Adults: 25-50 mg PO q6-8 hours; 10-50 mg IV/IM q 6 hours; maximum daily dose 400 mg.

Onset

IV:5 minutes

PO :<15 minutes

Duration

4-6 hours

Elimination

Hepatic

Effects

CNS

Sedation (which is additive with alcohol and sedative hypnotics), dizziness ,restlessness.

CVS

Rarely causes hypotension, arrhythmias

Misc.

May cause dry mouth, blurred vision, difficult urination; more rarely causes acute glaucoma or worsening of asthma, and GI or GU obstruction. These side effects reflect its anticholinergic activity, which is additive with other anticholinergics. and monoamine oxidase inhibitors (MAOI).

DANTROLENE

Dantrolene is used in the treatment of malignant hyperthermia. It is the only specific and direct treatment of malignant hyperthermia. It is also used in the treatment of neuroleptic malignant syndrome.

It is a direct skeletal muscle relaxant which acts at the muscle cellular level, possibly at the ryanodine receptor. It is administered intravenously in 2.5 mg/kg doses until clinical signs show reversal of the hypermetabolic state (usual total dose <20 mg/kg).

Dantrolene can cause generalized muscle weakness in higher doses. Other common side effects include sedation, dizziness and constipation.

Dantrolene is supplied as a powder that must be mixed with sterile water. Its dissolution in water is very slow, difficult and time-consuming. Special "guns" have been devised to speed the preparation of dantrolene so as to minimize any delay in administration in the urgent situation.