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Preface

As an addition to the European postgraduate training system for young neurosurgeons we began to publish in 1974 this series devoted to Advances and Technical Standards in Neurosurgery which was later sponsored by the European Association of Neurosurgical Societies.

The fact that the English language is well on the way to becoming the international medium at European scientific conferences is a great asset in terms of mutual understanding. Therefore we have decided to publish all contributions in English, regardless of the native language of the authors.

All contributions are submitted to the entire editorial board before publication of any volume.

Our series is not intended to compete with the publications of original scientific papers in other neurosurgical journals. Our intention is, rather, to present fields of neurosurgery and related areas in which important recent advances have been made. The contributions are written by specialists in the given fields and constitute the first part of each volume.

In the second part of each volume, we publish detailed descriptions of standard operative procedures, furnished by experienced clinicians; in these articles the authors describe the techniques they employ and explain the advantages, difficulties and risks involved in the various procedures. This part is intended primarily to assist young neurosurgeons in their post-graduate training. However, we are convinced that it will also be useful to experienced, fully trained neurosurgeons.

The descriptions of standard operative procedures are a novel feature of our series. We intend that this section should make available the findings of European neurosurgeons, published perhaps in less familiar languages, to neurosurgeons beyond the boundaries of the authors countries and of Europe. We will however from time to time bring to the notice of our European colleagues, operative procedures from colleagues in the United States and Japan, who have developed techniques which may now be regarded as standard. Our aim throughout is to promote contacts among neurosurgeons in Europe and throughout the world neurosurgical community in general.

We hope therefore that surgeons not only in Europe, but throughout the world will profit by this series of Advances and Technical Standards in Neurosurgery.

This volume represents the first in which our colleague, Professor Krayenbühl, has no longer acted as Managing Editor. An appreciation to this distinguished figure from his colleague and friend, Professor Yaşargil, is included and all the Editors wish him well in his retirement.

The Editors

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Hugo Krayenbühl — An Appreciation

Professor Hugo Krayenbühl, the founder of Swiss Neurosurgery, has not only been a brilliant surgeon and physician of highest moral standards but also an exceptional teacher. He trained not only young Swiss neurosurgeons but also a large number of foreign pupils who later returned to their countries of origin and are now leaders in their field. Not all became neurosurgeons. Some worked and contributed later to neighbour disciplines as neurology, electroencephalography, neuroanatomy, neurophysiology, and orthopedic surgery. It was his strong belief that progress in every medical field is only possible with international cooperation. Therefore, as a president of the Société de Neurochirurgie de Langue Française he organized the first European Congress of Neurosurgery that took place from July 16 to 19, 1959 in Zürich. This led to the foundation of the European Association of Neurological Surgeons in 1971 in Prague whose primary object is "to promote the free interchange of neurosurgical knowledge and experience among the member Societies".

It was Professor Krayenbühls special and continuing interest to promote the active participation of the young neurosurgeons in the European Congresses. He presided over the special forum "Recent Research by Young Neurosurgeons" during the European Congress in Oxford in 1975 to improve the participation of young neurosurgeons and discussed their presentations actively. The edition of the two book series "Progress in Neurological Surgery" beginning 1966 and "Advances and Technical Standards in Neurosurgery" beginning 1974 helped him in his continuing effort to propagate the results of important research and new techniques among the growing community of neurosurgeons in Europe and in the whole world. The publication of review papers written by authors with great personal expertise allowed a critical appraisal of the value of diagnostic tests, surgical techniques, and basic understanding in our common field of interest. The series "Advances and Technical Standards in Neurosurgery" focused on papers written by European authors whereas the series "Progress in Neurological Surgery" included also contributions from the whole world. The publication of these two series of books allowed him to reach neurosurgeons working in areas remote from the big university centers and allowed them to gain a critical insight into continuing problems and developments in neurosurgery and in neighbouring fields. The section

about "technical standards" reemphasized the value of standard techniques that continue to form the basis of our neurosurgical work.

Professor Krayenbühls approach led to the education of neurosurgeons in the principles established by Harvey Cushing who said in his presidential address at the Annual Meeting of the American Neurological Society

(Boston May 31, 1923): "Whatever his speciality may happen to be, it is only when a surgeon is shouldered with the responsability of acting largely on his own diagnoses that he will be impelled seriously to study his own cases before they come to the operating table and will be inclined to follow the results of his procedures to the end to see wherein his mistakes can be rectified on subsequent occasions. On no other basis he will be likely to see all round his subject; on no other basis he will be likely to contribute anything to it by carrying his problems to the laboratory; on no other basis will he set a safe example for his pupils to follow."

The remaining board of editors has now taken over the burden and the challenge of continuing this series of books that bridges the substantial gap between neurosurgical journals on one side and the (often multivolume) texts and specialized monographs on the other. The board of editors will benefit from the experiences and from the example set by Professor Krayenbühl, one of the great teachers in the field of clinical neurosurgery. We will use our best endeavours to maintain the standards which he has set.

M. Gazi Yaşargil

A. Advances

Nuclear Magnetic Resonance Imaging of the Central Nervous System

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With 16 Figures

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Introduction

Nuclear magnetic resonance (NMR) has been used for many years for spectroscopic chemical analysis, initially in vitro and more recently in vivo.

The use of NMR in imaging began much later and has already generated interest in the medical community as a result of its ability to discriminate between different soft tissues.

Over thirty centres around the world are now using clinical proton NMR imaging systems, and clinical experience with NMR imaging is expanding rapidly although the majority of studies reported so far have been performed with prototype machines.

It appears likely that NMR will have a useful role in neuroradiology in the future and a knowledge of the general principles and results achieved to date is likely to be of interest to these involved in clinical practice.

Physical Principles and Instrumentation

The physical principles underlying NMR have been described previously[1–3] and only a brief review is included. NMR describes the phenomenon whereby the nuclei of some atoms, when placed in certain magnetic fields absorb or emit radiofrequency (RF) energy of a specific frequency. The spectrum of absorbed or emitted RF energy depends upon the nucleus under observation and its chemical environment.

Nuclei suitable for NMR are those which have an odd number of protons or neutrons and therefore possess a net charge. Nuclei which possess both charge and angular momentum behave like magnetic dipoles. Several naturally-occurring NMR responsive nuclei are of biologic interest including hydrogen (proton), phosphorus (^{31}P), sodium (^{23}Na), carbon (^{13}C), fluorine (^{19}F) and potassium (^{39}K). Of these, protons are the most abundant nuclei in the body and have high NMR sensitivity and are therefore of principle interest in imaging.

In the earth's weak magnetic field there is a slight preferential alignment of nuclear magnetic dipoles in the direction of the field producing a net nuclear magnetization. In a strong magnetic field more of these nuclear magnetic dipoles align in the direction of the field producing a larger magnetization in the direction of the field.

The direction of the magnetic field is along the z axis, which is usually along the longitudinal axis of the patient in an NMR imaging machine (Fig. 1). The x and y axes are perpendicular to the z axis and to each other and represent the transverse plane. The strong magnetic field, (Bo) necessary for imaging is usually provided by either a resistive or superconducting magnet. Magnetic field strength used for imaging currently range for 0.08 Tesla (T) to 1.8 T.

At equilibrium, the magnetic dipoles are aligned with the applied magnetic field, but when perturbed away from the z axis, they rotate about this axis at a specific frequency. This rotatory motion is termed precession and the frequency of rotation is directly proportional to the magnetic field

strength. (For protons in a magnetic field of 0.15 T, the precessional frequency is 6.5 MHz, which is in the RF range.)

In order to perturb the protons, a magnetic field rotating at the precessional frequency of the nuclei is used. If the frequency of the RF magnetic field precisely matches the precessional frequency of the nuclei being studied, the net magnetization along the z axis is deviated through an angle which depends upon the strength and duration of the RF magnetic field.

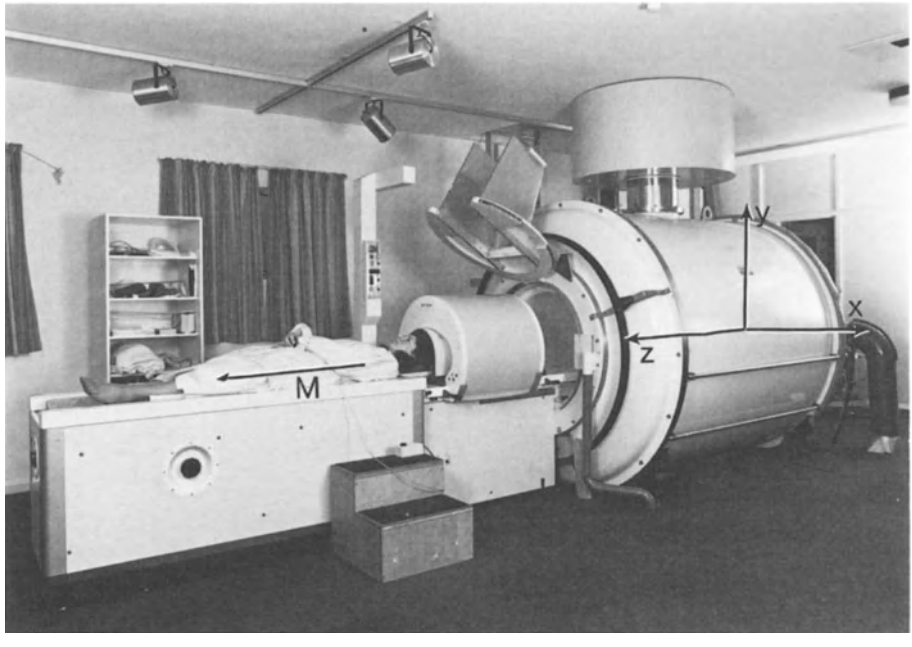

Fig. 1. NMR scanner based on a cryogenic magnet. The x, y, and z are labelled. The net magnetization M is shown in the long axis of the patient

In practice, short RF pulses are used to rotate the magnetization from the z axis. The most commonly used pulse is the 90° or $\pi/2$ pulse which rotates the magnetization through 90°. Following such a pulse, the component of net magnetization in the transverse (x, y) plane is used to generate an NMR signal termed the free induction decay (FID), in a RF coil which surrounds the patient. The size of the received signal depends upon the number of mobile nuclei of interest in the sample. For proton imaging, this is the proton density (ρ).

After a 90° pulse, the magnetization returns to equilibrium in an exponential manner. Recovery in the longitudinal axis of the patient is described by T_1, the spin-lattice (longitudinal) relaxation time constant.

Decay of magnetization in the transverse plane is described by T_2, the spin-spin (transverse) relaxation time constant. Both of these time constants are sensitive to the local chemical environment.

In order to produce an image, spatial localization of the received signal is required. A graduated magnetic field is applied so that the nuclear resonant frequency varies linearly with distance. As a consequence, determinations of the resonant frequency can be used to ascertain the position of the nuclei using a mathematical process termed Fourier transformation.

The NMR signal is received from the entire volume within the receiver coil, so various methods are utilized to restrict data collection to either a point, line, plane, or three-dimensional volume. Most NMR imaging machines are constructed to receive data from a selected plane using specially tailored RF pulses with a varying gradient magnetic field. By manipulation of the gradient magnetic fields, direct sagittal and coronal images can also be obtained.

Two basic methods of image reconstruction are used—the projection reconstruction technique[4] and two-dimensional Fourier transformation[5]. Projection-reconstruction is the technique used in CT, whereas two-dimensional Fourier transformation is a complex process unique to NMR.

Current single slice data acquisition times range from 30 seconds to 12 minutes depending upon the pulse sequence and resolution chosen. Multislice and volume imaging can reduce total examination times (despite the slow data acquisition times per single slice), while maintaining image quality and resolution. Physiological motion results in some loss of resolution.

NMR Pulse Sequences

Unlike CT images where contrast is determined by differences in one parameter, X-ray beam attenuation (μ), multiple parameters influence the NMR signal and, therefore resultant image contrast, *i.e.*, ρ, T_1 and T_2. In addition, flowing material within the image plane alters contrast. T_1 and T_2 variations between tissues are usually greater than proton density variations and thus images with greater dependence on relaxation times have greater contrast. By using different NMR pulse sequences images with varying dependence on ρ, T_1 and T_2 are obtained (Table 1).

Saturation-recovery (SR) pulse sequence utilize a series of equally-spaced 90° RF pulses. Image contrast is primarily dependent upon proton density with some dependence on T_1. As well, flow is highlighted as the repetition time (TR) decreases.

Inversion-recovery (IR) pulse sequences, utilize a 180° pulse followed at time TI later by a 90° pulse to produce images in which contrast is primarily dependent upon the differences in T_1, although contrast is influenced by ρ and T_2 as well.

Table 1. *Image Pixel Value Dependence on* ρ, T_1 *and* T_2

Type of image	Image contrast determinant		
	ρ	T_1	T_2
Saturation-recovery (SR)	proportional to ρ*	decrease if T_1 is very long	
Inversion-recovery (IR)	proportional to ρ	decrease as T_1 increases*	
Spin-echo (SE)	proportional to ρ	decrease if T_1 is very long	increases as T_2 increases*

* Principal image contrast determinant for each type of image.

Table 2. *NMR Pulse Sequences Utilized in This Study*

NMR pulse sequence	TR	TI	TE
Saturation recovery (SR)			
SR_{200}	200		
SR_{1000}	1 000		
Inversion recovery (IR)			
$IR_{1400/400}$	1 400	400	
$IR_{1800/600}$	1 800	600	
$IR_{2400/800}$	2 400	800	
$IR_{1400/400/44}$	1 400	400	44
$IR_{1500/500/44}$	1 500	500	44
Spin echo (SE)			
$SE_{544/44}$	544		44
$SE_{1040/40}$	1 040		40
$SE_{1080/80}$	1 080		80
$SE_{1160/160}$	1 160		160
$SE_{1580/80}$	1 580		80

Spin-echo (SE) pulse sequences utilize a 90° pulse followed at time τ by a 180° pulse with date collection at time τ after the 180° pulse when an echo of the original signal is obtained. [Note: Echo time (TE) is the time between the 90° pulse and echo production and equals 2τ.] This sequence produces

images whose contrast is primarily dependent upon differences in T_2, as well as on ρ and T_1.

Variations in the timing of the RF pulses in these pulse sequences may produce marked differences in image contrast[7,8]. Details of the timing of sequences are given in Table 2 and are described according to the American College of Radiology nomenclature[9].

Safety

The safety of NMR imaging is an important consideration, and as yet no basic hazard has been identified provided the machine is used sensibly[10,11]. The National Radiological Protection Board in Great Britain has published guidelines for the medical use of NMR[12] and these have recently been revised[13]. Three possible adverse effects have been considered. These are the static magnetic field, induced currents due to changing magnetic fields and heating effects due to the RF pulses. Exposure limits have been suggested for each of these.

As the possibility exists that ferromagnetic aneurysm clips may become dislodged by magnetic fields, patients with these in place should not be examined[14]. Patients with cardiac pacemakers should also be excluded. Patients with epilepsy are no longer excluded[13].

Other hazards to be considered include the effect of the magnetic field on external ferromagnetic objects such as scissors and scalpels, when accidentally released near the magnet. As well, the magnetic field may erase magnetic tapes, hard and floppy discs and magnetic stripes on credit cards. Cathode ray tube displays can be distorted by the fringe magnetic fields and their calibration can be affected[15].

Image Interpretation

NMR image contrast is determined by ρ, T_1, T_2, and flow effects, and different pulse sequences can be used to emphasize one or more of these variables (Table 1). A review of normal appearances using the different pulse sequences is useful (Fig. 2). By altering TR, TI, and TE, dependence upon T_1 and T_2 can be varied thus altering image contrast.

Images produced using SR pulse sequences demonstrate areas of high ρ with high signal towards the light end of the gray scale and areas of low ρ and low signal towards the dark end. As well, areas of long T_1 which have not relaxed completely before the next 90° pulse produce a low signal and a dark appearance. Blood flowing into the slice results in a high signal and appears white. By varying the TR, differences in T_1 can be highlighted.

The high level of contrast available with IR sequences is primarily a result of differences in T_1. Areas with short T_1, such as white matter and fat give a high signal and appear white, whereas areas of long T_1, such as CSF,

give a low signal and appear dark, Flowing blood usually produces little or no signal and blood vessels appear dark.

Calculated T_1 images whose pixel values are T_1 measurements can be obtained from an SR and an IR image through the same plane. Using a "region of interest" facility on the visual display unit, direct readings of T_1 can be obtained. Calculated T_1 images are noisy and display areas of short T_1 towards the dark end of the gray scale and areas of long T_1 towards the light end, that is, the reverse of the IR gray scale.

Images produced using SE pulse sequences have pixel values proportional to ρ, and are generally designed to emphasize T_2 dependence. Areas with long T_2 appear white, and short T_2 appear dark. Areas with low ρ and long T_1 usually appear dark.

Many acute and subacute pathological processes (such as inflammation and oedema) result in an increase in T_1 and T_2. A decrease in T_1 is seen in acute haemorrhage, some lipid-containing lesions, fibrosis and pleural thickening. Changes in T_1 and T_2 are generally non-specific, and clinical interpretation requires evaluation of the location of the lesion and associated clinical features. Measurement of T_1 values has been less helpful than initially hoped, as significant overlap between different pathological tissues occurs.

Central and peripheral artefacts are frequently seen on images produced using projection-reconstruction techniques. Small quantities of magnetic materials outside the plane of interest can produce black, inverted U-shaped artefacts. Similar artefacts may be seen with stainless steel clips and ventricular shunt valves within the imaging plane.

The normal appearances on SR, IR, and SE scans have been presented (Fig. 2). The IR images demonstrate a high level of gray-white matter contrast and provide excellent anatomic detail. Pathological change associated with long T_2 is highlighted against the relatively featureless background of SE scans. Sagittal and coronal images are readily obtained and can be useful to demonstrate and localize certain lesions, particularly those which are midline or deep-seated. The absent signal from cortical bone is a particular advantage in examining the posterior fossa, where bone artefact can significantly degrade CT images. The internal anatomy of the brainstem and cerebellum are better demonstrated on NMR than CT. The ventricular system is readily identified on all 3 pulse sequences.

A wide variety of neurological disease has been studied using NMR imaging which has been found to be sensitive in demonstrating pathological change[16—18].

Cerebrovascular Disease

Cerebral infarction presents a well-defined region of loss of gray-white matter contrast with a T_1 value longer than that of grey matter on IR scans.

Areas of very long T_1 within the infarct may represent cystic components. Mass effects including compression of sulci and displacement of the ventricular system may be seen with acute infarcts. Due to the high level of grey-white matter contrast, subtle mass effects, such as displacement of the external capsule can be seen on IR scans, but not with CT. SE scans demonstrate infarcts as areas of long T_2. Generally, NMR demonstrates the extent of involvement better than CT[17]. Chronic infarcts over two months old may demonstrate associated atrophic changes with widened sulci and expansion of the ipsilateral ventricle, as seen with CT. As with CT, infarcts are generally peripheral and wedge-shaped. In vivo sodium imaging has detected increased sodium signal from an infarcted hemisphere in a cat nine hours following ligation of the middle cerebral artery[19].

Lacunar infarcts (Fig. 3) are usually multiple, small, and deep-seated and present as focal areas of increased T_1 and T_2.

Infarcts within the brainstem are more readily visualized with NMR than CT. These are frequently multiple and demonstrate increased T_1 values. Circular, linear, and branching patterns have been described, as has sparing of a rim of brainstem, possibly corresponding to the territory of circumferential arteries.

Haemorrhagic infarcts are defined by their long T_1 and loss of grey-white matter contrast on IR scans, but the area of haemorrhage may be seen as a region of short T_1 with a longer T_1 centre. Preliminary results suggest infarction in systemic lupus erythematosus is more accurately delineated by NMR than CT. Most lesions appear as areas of increased T_1 and T_2 on IR and SE scans[20].

Aneurysms can be demonstrated by short T_1 areas due to the presence of thrombus[21]. Blood flowing within the aneurysm demonstrates a high signal on SR images, whereas it demonstrates little or no signal on IR images, thus distinguishing the region of flow from clotted blood.

Arteriovenous malformations (Fig. 4) are visualized without the use of iodinated contrast media and appear as areas of long T_1 on IR scans. Vessels with significant flow into the slice are seen because of their high signal on SR images with short TR (SR_{200} or SR_{100}). Associated thrombus may be seen because of its short T_1 and associated oedema and mass effects are identified because of their long T_1 and loss of grey-white matter contrast on IR images.

Intracranial Haemorrhage

Acute intracerebral haemorrhage demonstrates increased ρ on SR scans and an outer short T_1 rim with longer T_1 centre on IR scans. Haemorrhage is seen as an area of long T_2 on SE scans. Surrounding oedema is demonstrated on IR and SE scans and associated mass effects are best seen on IR scans. The central longer T_1 area is not seen on corresponding CT scans and may represent liquefaction of the haemorrhage.

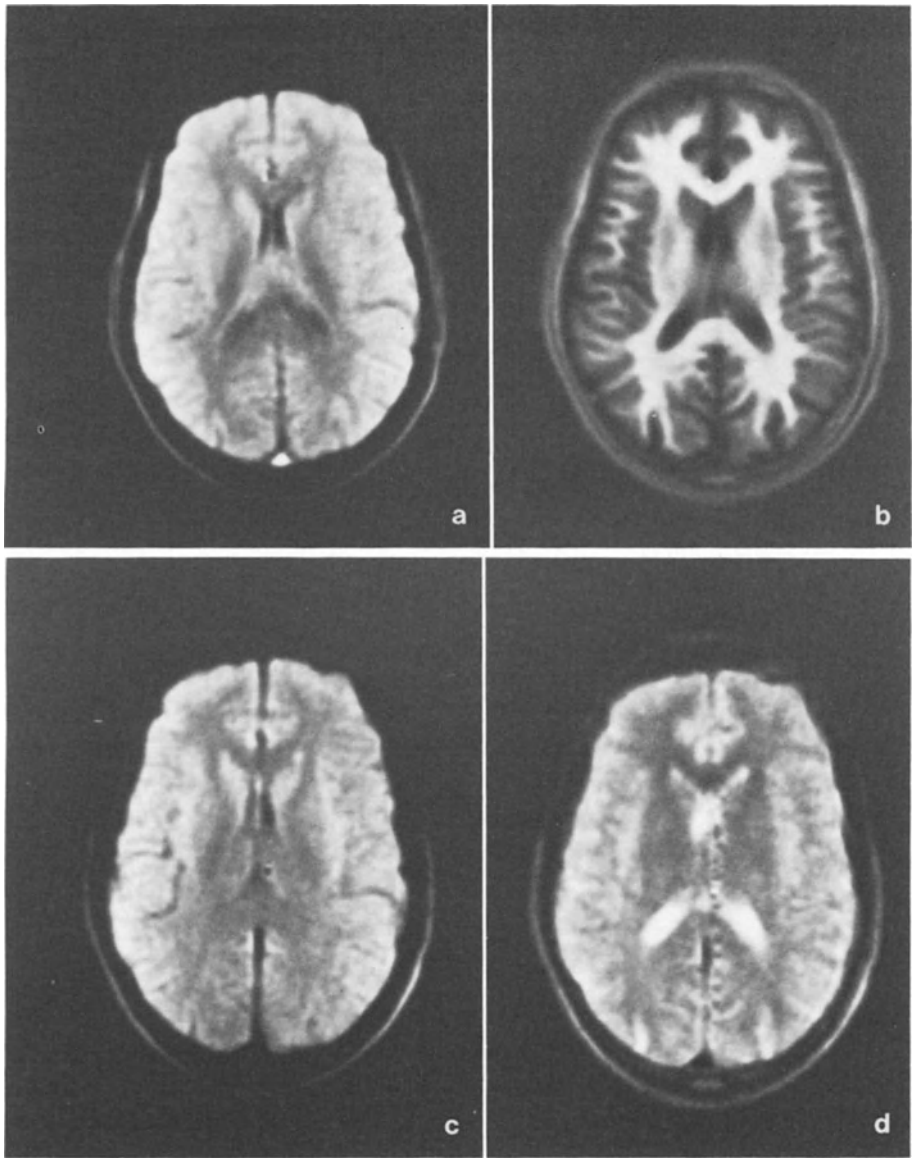

Fig. 2. a Normal SR_{1000} image. Note the high signal from blood in the superior sagittal sinus posteriorly. b Normal $IR_{1400/400}$ image. Note the grey-white matter contrast. c Normal $SE_{1080/80}$ image. Relatively featureless. Note dark appearance of CSF on this relatively short TE scan. d Normal $SE_{1160/160}$ image. Note white appearance of CSF on this long TE scan

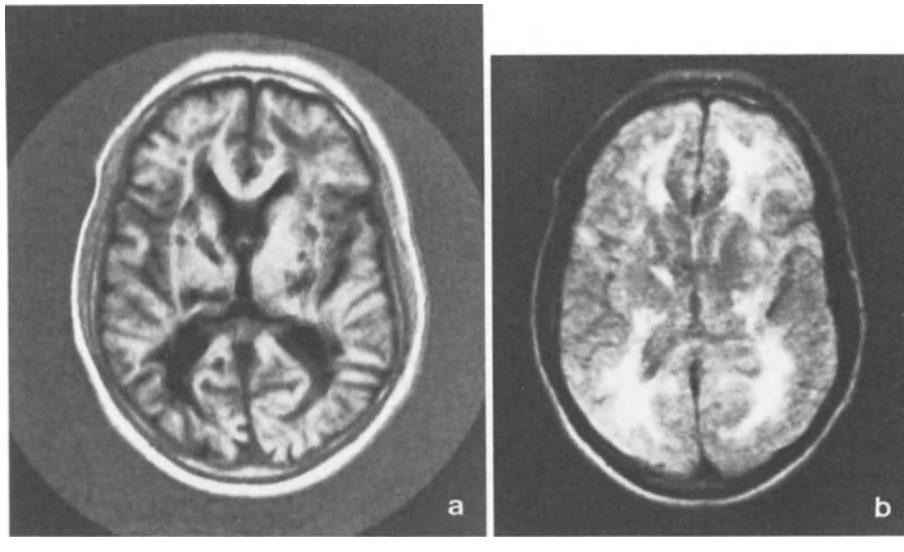

Fig. 3. Lacunar infarcts: a $IR_{1400/400/44}$ scan. Multiple areas of long T_1. b $SE_{1580/80}$ scan corresponding areas of long T_2

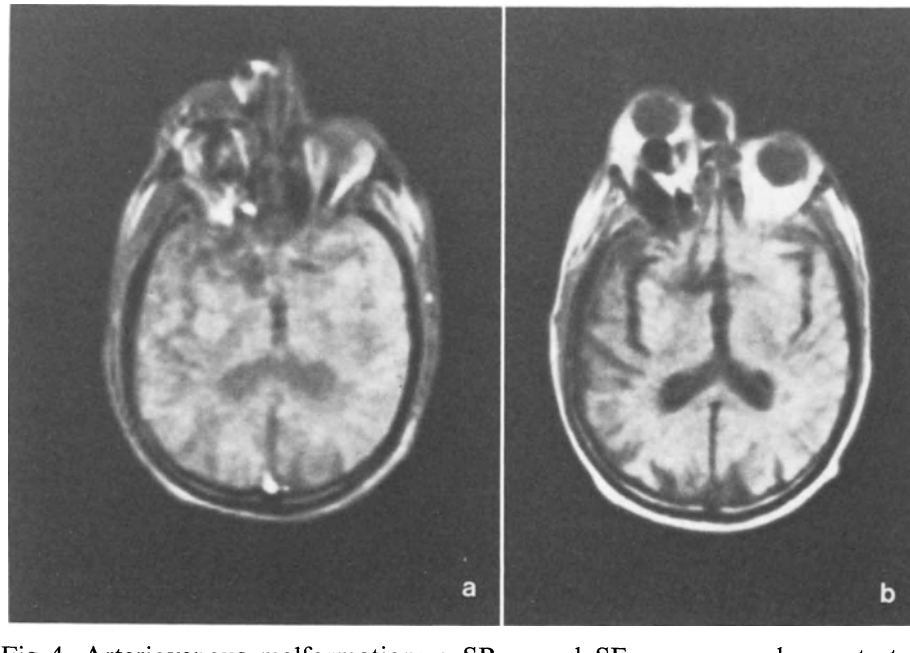

Fig. 4. Arteriovenous malformation: a SR_{200} and $SE_{544/44}$ scans demonstrated abnormally dilated vascular structures in the orbit along the course of the superior ophthalmic vein with associated proptosis

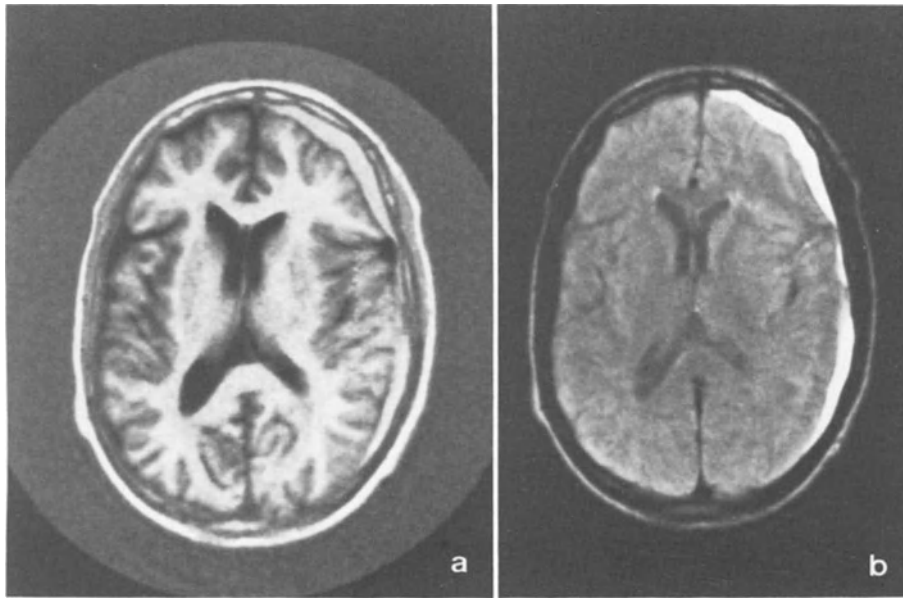

Fig. 5. Subdural haemorrhage. a $IR_{1400/400/44}$ and b $SE_{1580/80}$ scans demonstrate the haemorrhage by its short T_1 (a) and long T_2 (b)

Acute subdural haemorrhage (Fig. 5) is readily demonstrated by NMR and appears as an extra-axial crescentic collection with short T_1 and long T_2. Associated mass effects are identified by displacement of grey-white matter interfaces on IR scans. The medial and lateral margins of the haemorrhage are delineated, unlike CT where only the medial margin is usually identified.

Subarachnoid haemorrhage is seen as short T_1 due to blood within the cerebral sulci. The absent bone artefact may allow haemorrhage adjacent to bone within the basal cisterns and the posterior fossa to be more readily identified than with CT.

Intracranial Infection

IR Scans in meningitis have demonstrated peripheral areas of infarction[30]. Patients with herpes encephalitis have demonstrated extensive areas of increased T_1 and T_2 on IR and SE scans. The absence of bone artefact around the anterior temporal lobes may be advantageous relative to CT, in herpes encephalitis.

Patients with postinfectious encephalitis have demonstrated encephalomalacia and foci of demyelination which appear as long T_2 areas on SE scans[17].

A resolving brain abscess appeared as a low intensity lesion on IR scans with prolonged T_2 on SE scans suggesting oedema adjacent to the lesion.

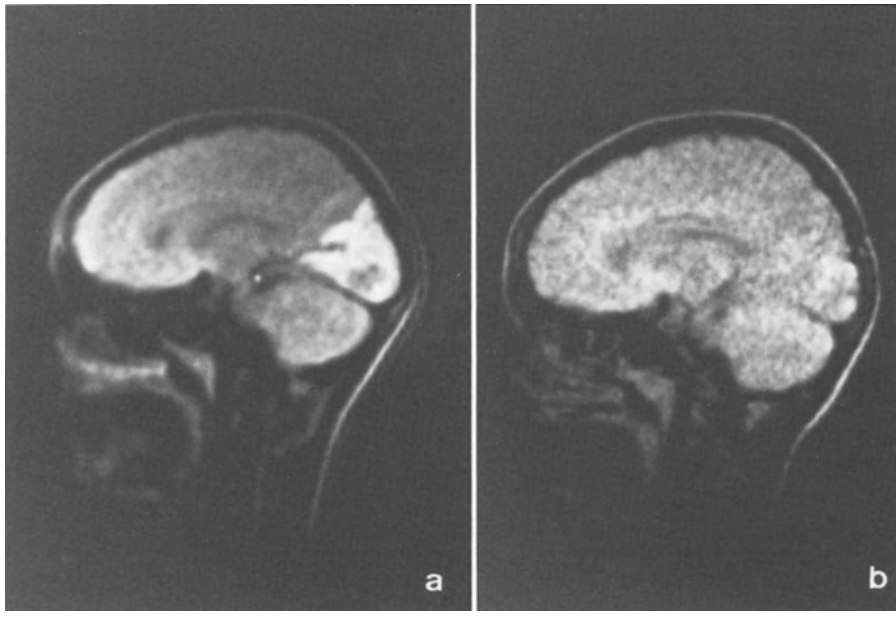

Fig. 6. Probable fungal abscess. a $SE_{1080/80}$ scan pre-drainage. b $SE_{1580/80}$ scan post-drainage. Initial scan (a) demonstrated an area of increased T_2 stopping at the parieto-occipital junction suggesting oedema. Within is an area of shorter T_2 probably representing the abscess itself. There is considerable resolution post-drainage (b)

NMR imaging of an experimental brain abscess in dogs has shown some advantages over CT[22]. Tuberculous abscesses are seen as areas of long T_1 with associated mass effect on IR scans. Calcification is not identified on IR scans as it appears dark due to its low ρ and gives little contrast with the remainder of the tissue in the abscess which also appears dark due to its long T_1. A probable fungal abscess in the occipital lobe demonstrated loss of grey-white matter contrast and increased T_1 in the involved area on IR scans. Transverse and sagittal SE images demonstrated long T_2 in the corresponding location which was seen to stop abruptly at the parieto-occipital junction, suggesting oedema. Within this region of long T_2 was an area of shorter T_2 representing the abscess itself. Resolution followed surgical drainage[23] (Fig. 6).

A presumed ventricular shunt infection was identified by its long T_2 along the tract of the shunt.

White Matter Disease

NMR imaging has been shown to be more sensitive than CT in the detection of plaques of multiple sclerosis (MS)[24—26]. The lesions appear as

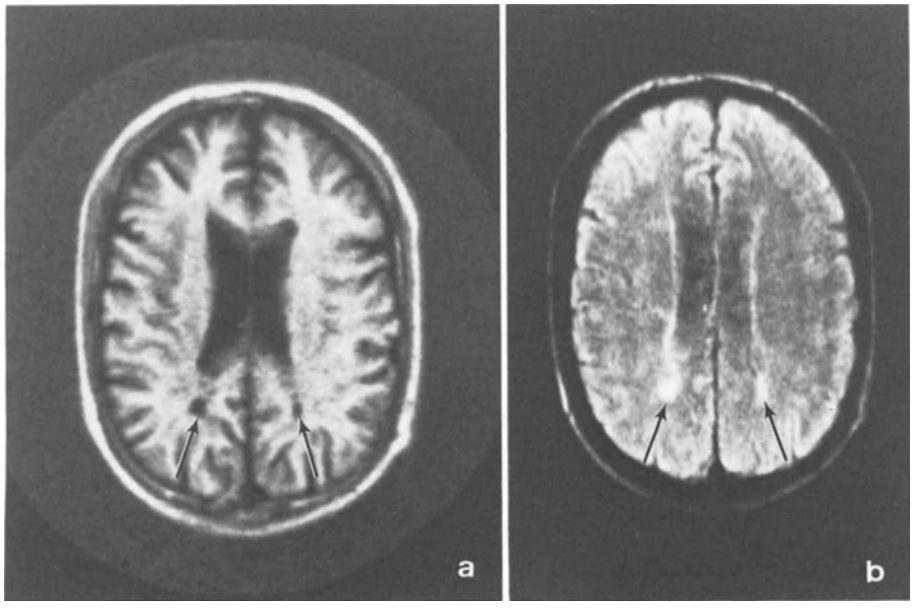

Fig. 7. Multiple sclerosis. a $IR_{1400/400/44}$. b $SE_{1580/80}$ scans. Plaques (arrows) demonstrated as areas of long T_1 (a) and long T_2 (b) are more readily recognized on the SE scan (b)

focal areas of long T_1 and T_2 on IR and SE scans predominantly in periventricular white matter (Fig. 7) as well as in the brainstem and cerebellum, areas which are poorly seen on CT. SE scans are more sensitive than IR scans in the detection of MS lesions in the supratentorial compartment as partial volume effects can occur at grey-white matter interfaces and CSF-white matter interfaces on IR scans, producing apparent long T_1 lesions in white matter. The long T_2 of MS lesions allows them to be readily identified against the relatively featureless background with SE scans. However, caution is required in interpretation of areas of long T_2 at the anterolateral angles of the lateral ventricles where increased T_2 may be noted in normal subjects. In the brainstems, MS lesions may be more readily identified on IR scans.

Follow-up scans in patients with MS usually reveals decreased size but not disappearance of lesions following acute episodes. During relapses, new lesions can appear while existing lesions may become larger.

Other rare conditions associated with demyelination and disease of white matter can be recognized on NMR. Patients with Binswanger disease (Fig. 8) demonstrate extensive areas of long T_1 and T_2 throughout white matter on IR and SE scans[40]. A case of adrenoleukodystrophy demonstrated dilated posterior horns of the lateral ventricles as well as increased T_1

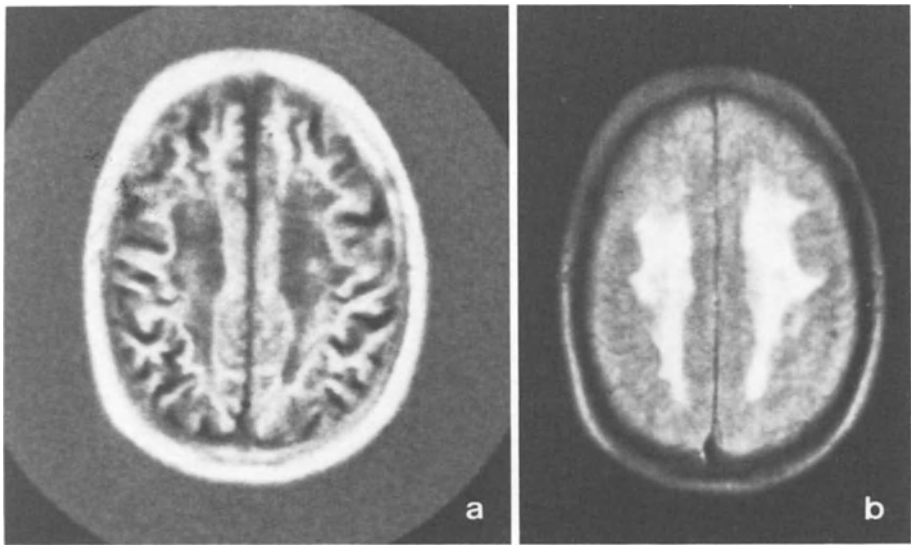

Fig. 8. Binswanger disease. a $IR_{1400/400/44}$. b $SE_{1580/80}$. There is extensive involvement of white matter demonstrating long T_1 (a) and long T_2 (b)

and T_2 and loss of grey-white matter contrast in the occipital lobes. Changes were more extensive than those seen on CT. More generalized increase in T_1 and T_2 of white matter were demonstrated on NMR than CT in a child with leukodystrophy in association with congenital muscular dystrophy.

Trauma

Abnormalities secondary to trauma have been described including loss of grey-white matter contrast in the temporal lobe below the site of impact in a patient in whom CT was normal. This suggests a possible role for NMR in evaluation of white matter shearing injuries. Long T_1 lesions in the pons have also noted.

Postsurgical changes following evacuation of a traumatic left frontal intracerebral haematoma have also been described including both subdural and epidural fluid collections as well as a resolving haematoma in the mastoid air cells.

Bilateral subdural hygromas have been described in two patients, the first postoperatively following ventricular shunt placement, the second in an infant following non-accidental trauma.

Degenerative Diseases and Diseases of the Basal Ganglia

Cerebral atrophy is identified in a similar way as on CT and appears to involve grey and white matter equally on IR scans. Selective grey matter loss

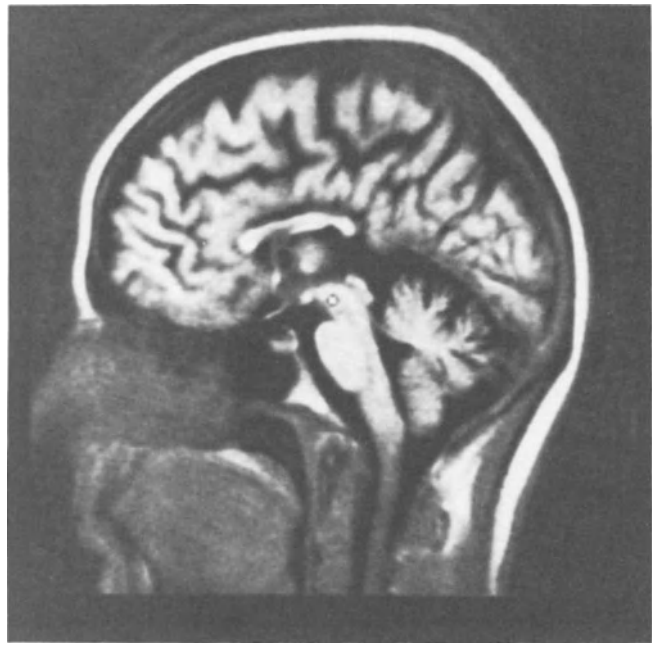

Fig. 9. Partial agenesis of the corpus callosum demonstrated on sagittal $IR_{1\,500/500/44}$ scan

as well as areas of peripheral infarction have been demonstrated on IR scans in a case of neurosyphilis.

Cerebellar hemisphere atrophy is readily identified by increased distance between the cerebellum and adjacent bone. Atrophy of the vermis is recognized by decrease in its size on IR scans and increased size of the adjacent cisterns. The absence of bone artefact in the posterior fossa allows the cerebellum to be more readily identified.

Several patients with diseases involving the basal ganglia have been studied. Involuntary movement has been found to produce less image degradation with NMR than CT. Patients with Huntington disease have demonstrated atrophy of the head of the caudate nucleus. Patients with Wilson disease have demonstrated changes in the basal ganglia as well as areas of increased T_1 and T_2 in the thalami on IR and SE scans[27]. Calcification seen in the lenticular nuclei may not be demonstrated on NMR. IR and SE scans in a patient with Hallervorden-Spatz disease demonstrated areas of long T_1 and T_2 in the lenticular nuclei.

Most patients with Parkinson disease have demonstrated no abnormality, while in two patients, the substantia nigra could not be identified. A patient with postencephalitic Parkinson disease demonstrated a long T_1 lesion in the upper mesencephalon, and a patient treated by bilateral thalomotomy demonstrated two long T_1 lesions within the thalamus.

Congenital and Inherited Diseases

Semilobar holoprosencephaly has been recognized on NMR by abnormal ventricular configuration. Sagittal scanning is particularly useful in assessment of midline and craniovertebral malformations (Fig. 9). Arnold-Chiari malformation has been recognized by the low position of the cerebellum[28].

Long T_1 lesions in a periventricular location are recognized on IR scans in tuberous sclerosis. Calcification identified on CT is not seen on NMR. Cases of Freidrich ataxia and olivopontocerebellar degeneration have shown vermis atrophy. IR scans in a case of Sturge-Weber disease demonstrated loss of grey-white matter contrast outside the area in which calcification was demonstrated on CT.

Hydrocephalus

The ventricular system is readily identified using all three pulse sequences and enlargement is recognized as with CT. Using SE scans, periventricular oedema is identified as increased T_2 regions at the margins of the enlarged ventricles (Fig. 10). This has been identified in acute and subacute cases of hydrocephalus but not in atrophy, and it has been seen to regress following successful ventricular shunt placement.

Tumour

Several reports of the appearance of a variety of tumours using NMR imaging have now appeared[16—18, 21, 23, 26, 28, 30—32]. The majority of tumours are recognized by increased T_1 and loss of grey-white matter contrast on IR scans and increased T_2 on SE scans (Fig. 11). Exceptions include tumours containing lipid, haemorrhage or free radicals (*e.g.*, melanoma), which are recognized by short T_1 and long T_2 on IR and SE scans (Fig. 12). Malignant tumours tend to be associated with greater mass effect as well as increased structural change within the tumour than benign tumours. Tentorial herniation can be recognized by displacement of the brainstem at tentorial level with associated oedema of the adjacent temporal lobe. Subfacial herniations can be recognized on coronal scans.

More structure is generally recognized within tumours on NMR than CT. However, calcification is not as readily seen with NMR as with CT. Calcification, with its low ρ is dark and is not seen within the dark (long T_1) tumour in IR scans. However, contrast may be present between the low signal of calcification and the high singal (long T_2) of tumour on SE scans.

Contrast-enhanced CT delineates tumour from surrounding oedema better than unenhanced NMR, although SE scans may show the tumour to have a shorter or longer T_2 than the surrounding oedema allowing the

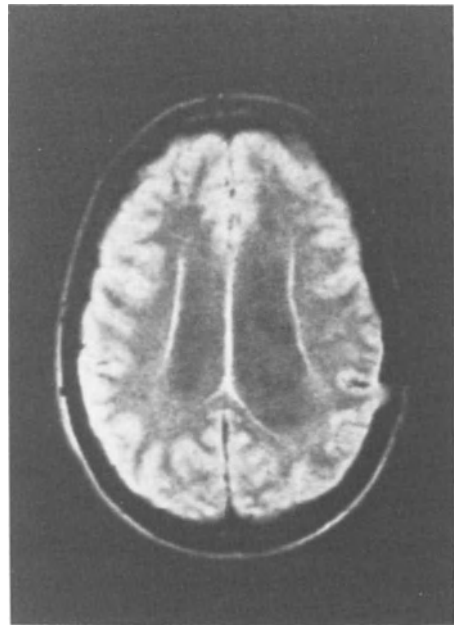

Fig. 10. Hydrocephalus and periventricular oedema. The oedema is identified by its long T_2 along the ventricular margins on the $SE_{1500/80}$ scan

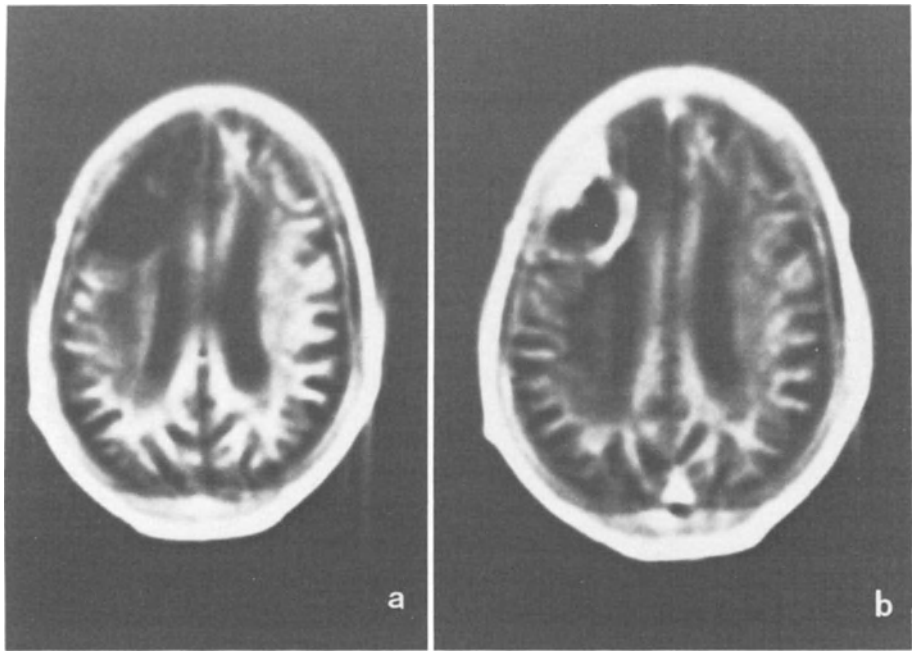

Fig. 11. Astrocytoma Grade IV. $IR_{1400/400}$ scans before (a) and after (b) enhancement with a paramagnetic contrast agent. Ring enhancement is seen in (b)

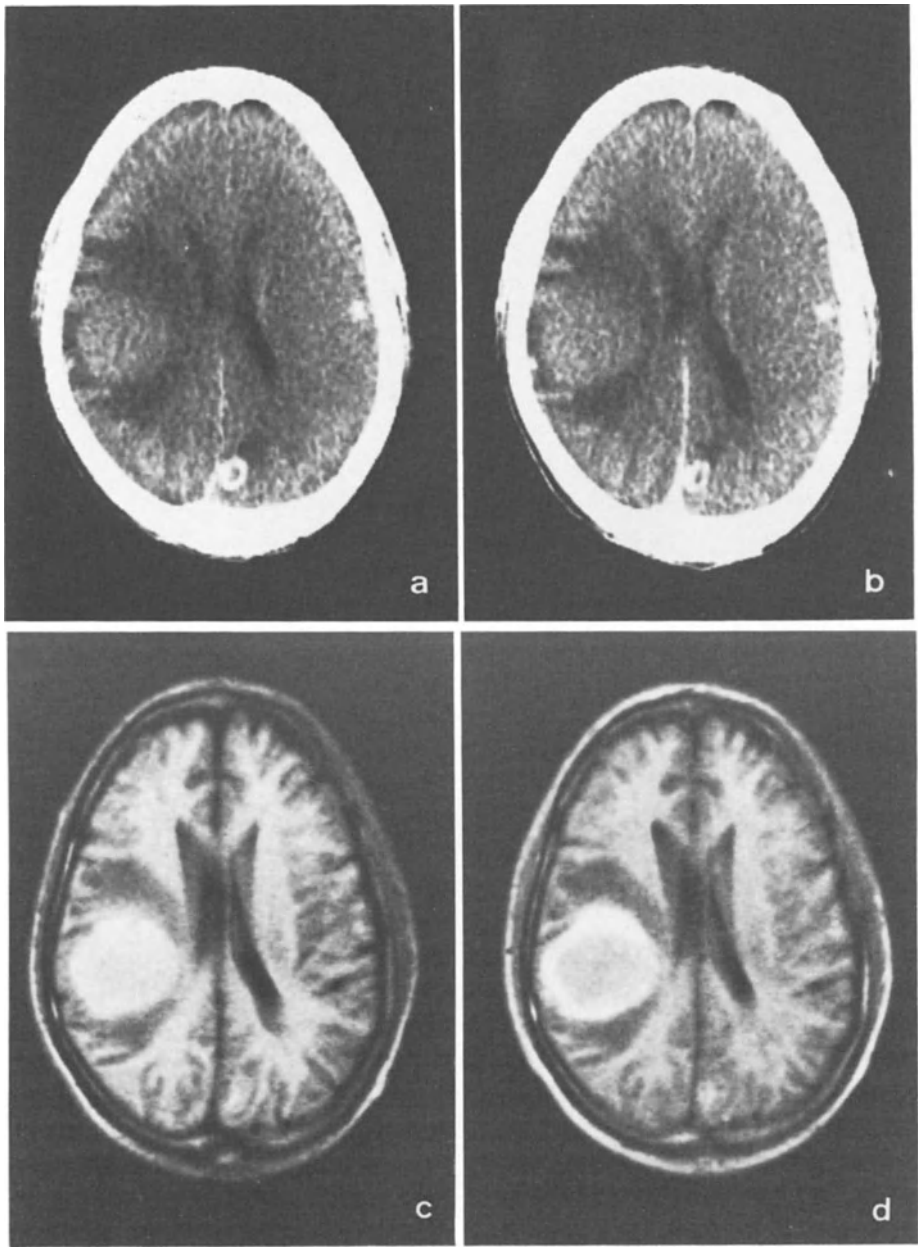

Fig. 12. Metastatic malignant melanoma. a Nonenhanced CT scan. b Contrast-enhanced CT scan. c $IR_{1500/500/44}$ scan precontrast. Tumour demonstrates short T_1 with surrounding oedema. d $IR_{1500/500/44}$ post-paramagnetic contrast agent. [Gadolinium diethylene triamine penta-acetic acid (Gd-DTPA)]. Ring enhancement of tumour is seen with NMR but not with CT

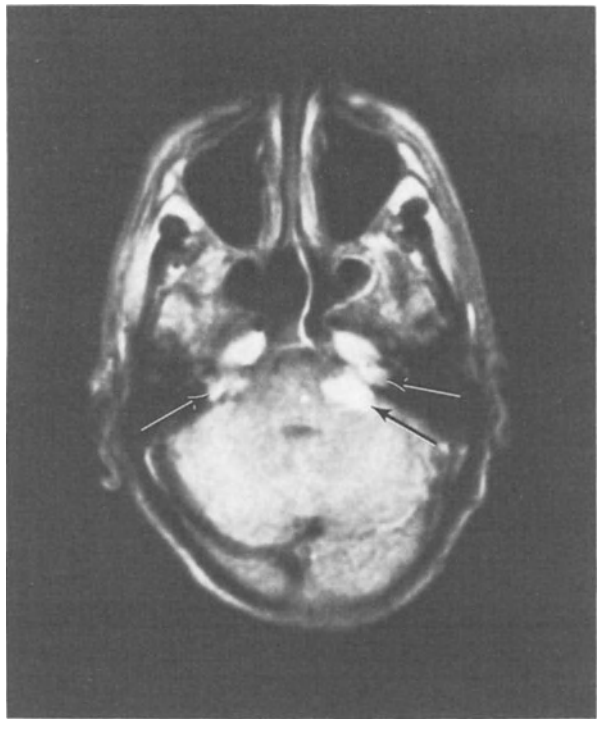

Fig. 13. Acoustic neuroma: $SE_{1580/80}$ scan. The tumours are recognized by expansion of the nerve and increased T_2 (arrows)

distinction to be made. As well, oedema is usually confined to white matter, whereas long T_2 of tumour is confluent. Contrast agents which demonstrate breakdown of the blood-brain barrier may be very useful in distinguishing tumour from oedema by NMR[33] (Figs. 11 and 12).

More subtle mass effects can be identified on IR scans than on CT as there are a greater number of grey-white matter interfaces to assess. Sagittal and coronal images can allow better demonstration of anatomical relationships, particularly of midline and deep-seated lesions. The ability to recognize flowing blood allows distinction of juxtasellar aneurysms from pituitary and juxtasellar tumours.

The lack of bone artefact is especially important in demonstrating tumours of the posterior fossa and NMR has been shown to be more sensitive than CT. The assessment of intra-axial versus extra-axial location, a feature of great importance in patient management, is more readily made using NMR. As with supratentorial tumours, mass effects and extent of tumour are more readily determined using NMR. Small acoustic neuromas have been demonstrated (Fig. 13).

Because of the lack of signal from cortical bone on NMR, bony erosion is better identified on CT. Tumour invasion in seen on SE sequences where long T_2 of tumour within bone contrasts with the dark area of bone.

Differentiation of tumour recurrence from change secondary to radiotherapy is difficult. Initially radiation therapy can result in increased T_1 and T_2 of tumour along with increased cerebral oedema. Later, regions of increased T_2 in the distribution of the radiation field can be seen within cerebral white matter, especially adjacent to the ventricular system and adjacent to the tumour. These changes are frequently more extensive than those seen with CT.

Thus, NMR is sensitive in detection of tumour and differentiation between tumour types and other space-occupying lesions is currently made in a similar was to CT by considering the patient's age, site of the lesion, and associated features such as oedema. A great deal of interest surrounds the possibility that NMR may provide more specific information than CT. Although benign and malignant tumour and other space-occupying processes show overlap of T_1 and T_2 values, it is possible that combinations of T_1 and T_2 parameters and multiexponential analysis of relaxation curves may be more specific than single T_1 and T_2 values.

Paediatric Neurological Diseases

It has long been known from pathological studies that myelination, which follows an orderly sequence, is not complete at birth, but begins in midgestation and has a rapid initial phase and then continues more slowly into adult life[34].

The high level of grey-white matter contrast available with IR sequences allows this normal process of myelination to be visualized in vivo for the first time (Fig. 14). By comparison with IR scans in normal age-matched controls, delays or deficits in myelination have been recognized in cases of previous intraventricular haemorrhage, cerebral palsy, aqueduct stenosis, neurodegenerative disorder, Hurler's syndrome, and probable rubella embroypathy (Fig. 15).

Areas of long T_1, more prominent than those seen in a normal control, were identified in the periventricular regions of infants who had suffered ischemic anoxic encephalopathy. The significance of these areas remains uncertain at present and requires further study.

Areas of long T_2 in the anterior periventricular regions of an infant with spastic diplegia have also been identified. Haemorrhage, infarction, leukomalacia, hydrocephalus, porencephalic cysts, tumours, and white matter disease have also been recognized on NMR studies in patients in the paediatric age group.

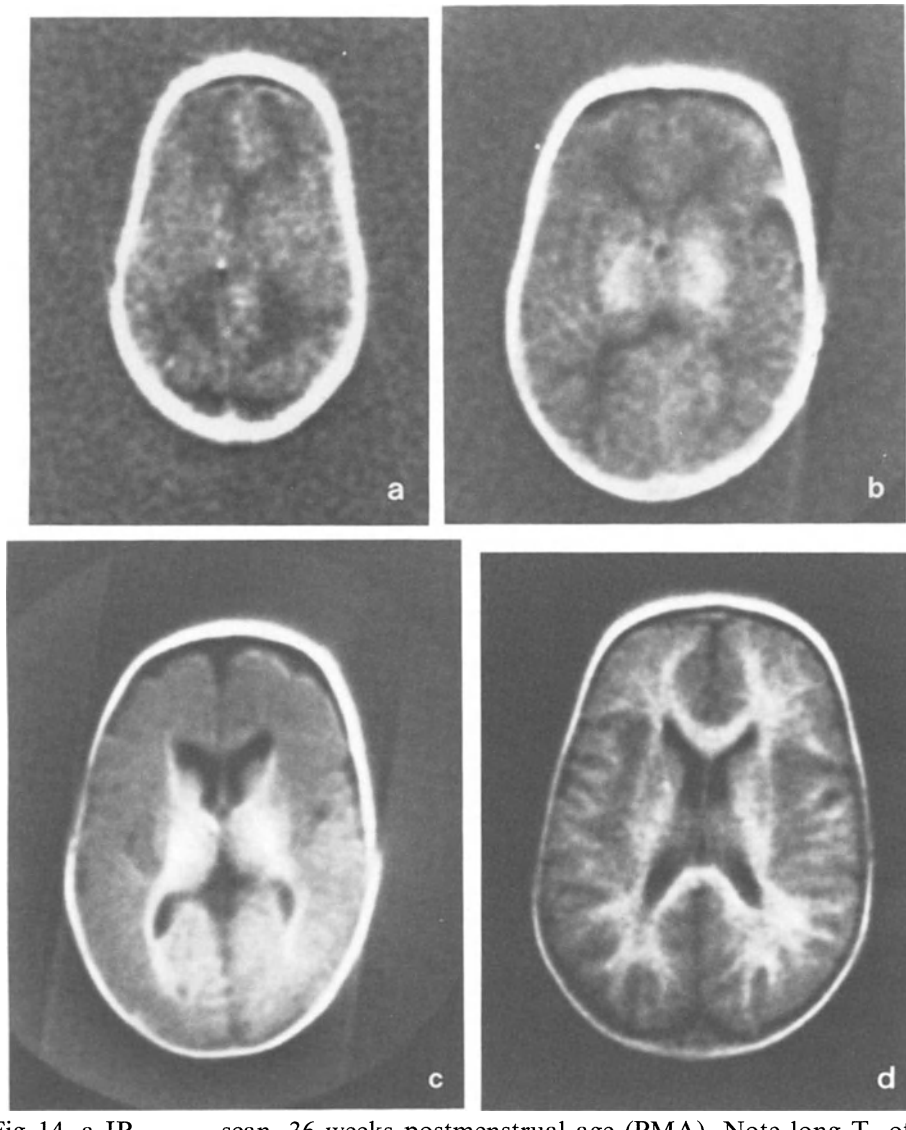

Fig. 14. a $IR_{1800/600}$ scan. 36 weeks postmenstrual age (PMA). Note long T_1 of periventricular regions. b $IR_{1800/600}$ scan—42 weeks postmenstrual age. The long T_1 in periventricular regions is less pronounced. Early myelination in the thalami and posterior internal capsule demonstrates short T_1. c $IR_{1800/600}$ scan—6 months age. Further myelination. d $IR_{1800/600}$ scan—20 months age. Further myelination

Spine

The craniovertebral structures, spinal cord, CSF, annulus fibrosis, nucleus pulposus, and vertebral bodies are visualized on NMR without the need for intrathecal contrast media. Unlike CT, the cord is directly

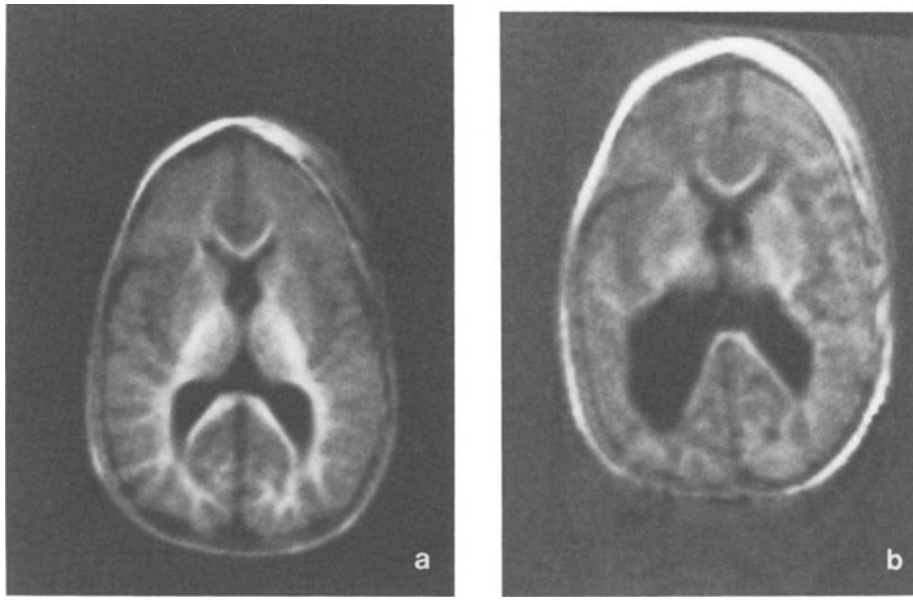

Fig. 15. a $IR_{1800/600}$ scan in normal triplet-age 9 months PMA. b $IR_{1800/600}$ scan in triplet with delayed development at 9 months PMA-note delay or deficit in myelination relative to (a)

visualized free from bone artefacts and evaluation of intramedullary spinal cord lesions appears to be the best application of NMR imaging of the spine. The normal nucleus pulposus usually has a higher signal intensity than the surrounding annulus fibrosis. Marrow within the cancellous bone of the vertebral body results in high signal intensity surrounded by very low signal intensity of cortical bone. Sagittal images are particularly useful in evaluation of the spinal canal and its contents and in determining the craniocaudal extent of lesions[35—40].

The cystic central cavity of syringomyelia is recognized by its long T_1 and low signal intensity on SR, IR, and short TE, SE scans. Extention into the medulla and the craniocaudal extent are defined on sagittal images. Associated Arnold-Chiari malformations have been recognized.

Spinal cord tumours generally demonstrate increased T_1 and T_2 as well as associated cord expansion on IR and SE scans (Fig. 16). Extent of lesions is determined on sagittal images. Separation of long T_1 of tumour from surrounding CSF can be difficult on IR scans, but this distinction can usually be made on short TE SE scans. Fat-containing tumours, such as lipomas and teratomas, are recognized by their high signal intensity due to high ρ and short T_1. Tethered cord in association with lipoma and dural ectasia have been recognized.

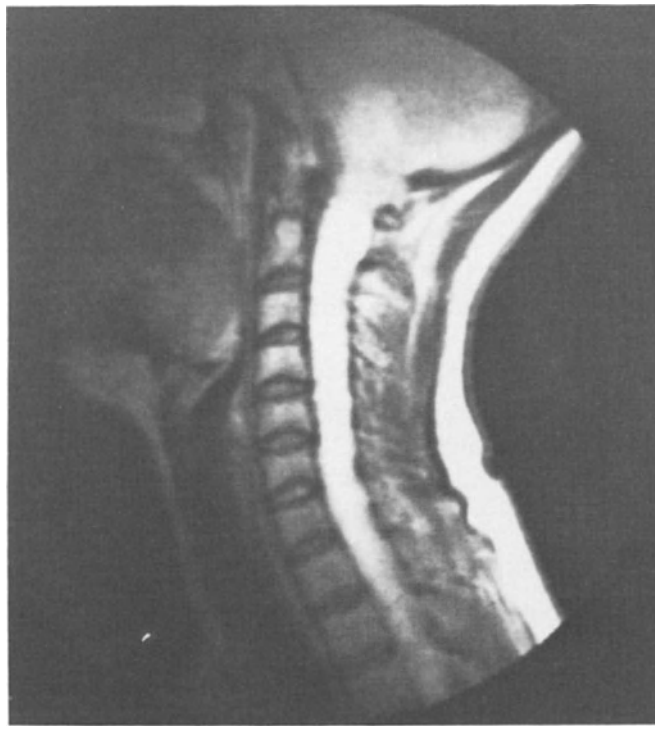

Fig. 16. Spinal cord haemangioblastoma. $SE_{1580/80}$ scan. Extensive tumour is demonstrated

Vertebral body abnormalities such as fractures and tumours have been studied. Associated vertebral collapse and soft tissue masses can be identified.

Atlantoaxial subluxation has been demonstrated on flexion and extension sagittal NMR images, with superior definition of the narrowing of the neural canal in the foramen magnum. Soft tissue mass posterior to the dense has also been described.

Herniated disks have been recognized by visualization of the protruding disk effacing epidural fat or displacing the thecal sac. The ruptured disks as well as degenerated disks without herniation may display lower than normal signal intensity with loss of contrast between nucleus pulposus and annulus fibrosis. Unlike degenerated disks, a case of acute disk space infection demonstrated long T_2 and increased signal of the disk and adjacent vertebra on long TE SE scans. Postoperative fibrosis has been recognized as areas of intermediate to high signal intensity adjacent to the thecal sac on SE images suggesting that it may be possible to distinguish this from protruded disk, a distinction which is difficult on CT.

Spectroscopy

NMR spectroscopy relies on information provided by chemical shifts. The magnetic field around nuclei in a chemically complex environment is altered due to "shielding" currents that are associated with the electron distribution around adjacent atoms.

These alterations in the magnetic field cause shifts in the resonance frequency, *i.e.*, chemical shifts, and thus allow differentiation between the same nuclei in different chemical environments.

In 1974, Hoult *et al.* demonstrated that ^{31}P NMR spectroscopy could be utilized to measure the concentration of ATP, phosphocreatine, and inorganic phosphate, as well as the intracellular pH in muscle[41]. Since then, ^{31}P spectroscopy has been utilized for metabolic studies in patients with a variety of muscle diseases and abnormalities have been seen in patients with Mc Ardle disease[42], mitochondral myopathy[43], and Duchenne muscular dystrophy[44]. It has also been used to study ischaemic conditions and metabolism in tumours.

The clinical use of ^{31}P-NMR spectroscopy has previously been limited to small objects such as limbs and small animals because of magnet size. Recently, ^{31}P NMR spectroscopy has been utilized to study neonatal brain and abnormalities were identified in birth asphyxia, brain atrophy, and perencephalic cysts[45]. Large whole-body spectroscopy machines are becoming available with the potential to study human adults[46].

Conclusions

NMR imaging has proved to be sensitive to a wide variety of pathological change in the brain although a great deal of work remains to determine its efficacy relative to currently available imaging techniques.

Improvements in image quality will continue but there is likely to be more emphasis on speed, ease of operation, reliability, and cost effectiveness than in the initial phase of NMR development.

The more widespread use of paramagnetic contrast agents is likely to add significantly to the value of NMR imaging although difficulty in detection of calcification is likely to remain.

The clinical role of NMR spectroscopy in examination of the central nervous system is yet to be determined. Should spectroscopy prove useful in routine practice it will have a major influence on the design of NMR machines. While it is possible to obtain useful images over a wide range of magnetic fields spectroscopy is only possible at high fields. If NMR machines are to be used for both spectroscopy and imaging they will of necessity be high field which will increase their expense, complexity, and siting difficulty.

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Glossary of Terms

Spin

Individual protons and neutrons can be considered as being in orbit around the nucleus just like electrons. They also rotate about their axis and thus have a spin. Pairs of neutrons or protons align so that their spins cancel out. A nucleus with an odd number of neutrons and/or protons has a net rotational component characterized by a quantum number called the spin of the nucleus.

Magnetic Moment

The small magnetic field produced by rotation or spin of charged nuclei with an odd number of protons or neutrons.

Paramagnetic Atoms (or Ions)

Atoms or ions that slightly increase a magnetic field when placed within it. They usually have an odd number of electrons and a partially filled inner shell.

Proton Density

The number of hydrogen nuclei participating in the NMR process within a unit volume.

Spin-lattice (T_1) Relaxation Time

The exponential time constant at which the component of magnetization parallel to the external fields decays *i.e.* reaches equilibrium. This results from the interaction of a nucleus with its surroundings, hence the name spin-lattice.

Spin-spin (T_2) Relaxation Time

The exponential time constant at which the component of magnetization perpendicular to the external field decays. It results from the interaction of a spinning nucleus with the spin of an identical nucleus pointing in opposite direction, hence the name spin-spin.

Pulse Sequence

A series of short magnetic field pulses oscillating at the nuclear spin frequency (*i.e.*, radiofrequency for protons) which are used to rotate the patients proton magnetization typically through 90° or 180°. The commonly used examples are saturation-recovery inversion-recovery and spin-echo. The names are derived from classical NMR spectroscopy.

Saturation Recovery

A pulse sequence in which the patients nuclear magnetization is rotated through 90° and allowed to decay back to equilibrium. Contrast in images produced with this sequence largely reflect changes in proton density with some dependence on T_1.

Inversion Recovery

A pulse sequence involving rotation of the patients magnetization through 180° then 90°. Contrast in these images largely depends on differences in T_1.

Spin-Echo

A pulse sequence involving rotation of the patients magnetization through 90° then 180°. Contrast in the resultant image is mainly dependent on T_2.

Static Magnetic Field (Bo)

The principal magnetic field which aligns nuclear spins in the same direction.

Magnetic Field Gradients

Magnetic fields which vary linearly with distance and position of hydrogen nuclei to be defined.

Projection-Reconstruction and Two Dimensional Fourier Transformation

Two methods of reconstructing images by computer. Projection-reconstruction is the technique used in X-ray CT while two dimensional Fourier transformation is unique to NMR.

Update and Trends in Venous (VDSA) and Arterial (ADSA) Digital Subtraction Angiography in Neuroradiology

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With 6 Figures

Contents

Summary

After presentation of the principles and technology of digital subtraction angiography the results of 649 investigations with the peripheral venous method are presented and first experiences are discussed critically. In conclusion the method of venous digital subtraction angiography can be stated to be useful for adequate diagnosis of the extracranial brain vessels. Diagnostic statements about the intracranial vessels by venous injection are limited, and therefore a combined investigation including normal angiography remains necessary. In the near future neuroradiological investigations cannot abandon selective arterial techniques. One may expect however that normal angiographic investigations will subsequently be replaced by digital subtraction angiography with arterial injection of contrast medium.

Index Terms

Venous DSA (VDSA), arterial DSA (ADSA), spatial resolution, contrast resolution, contrast medium concentration in the arteries, image quality, trends.

Introduction

Within the scope of modern radiological imaging procedures, digital image processing is increasingly gaining ground. A prerequisite for digital radiography has been the further development and adaptation of computerized techniques to the requirements of radiology. In this respect, *digital subtraction angiography* (DSA) represents a young, but promising development. This modality is based on the principle that by computer-assisted automatic subtraction of the pre-contrast from the post-contrast image the vascular contrast is enhanced to such an extent that extensive diagnostic information is obtained from the subtracted image with an extremely low contrast medium concentration. Digital subtraction angiography consists of electronic breakup, storage, and subtraction of the video data from a high-resolution television fluoroscopy installation[7]. With respect to the site of contrast agent injection, we distinguish between *indirect venous (VDSA)* and *direct arterial (ADSA)* subtraction angiography.

Material and Method

The experience we have gained with DSA originates from the study of a total of 855 patients (from July 1982 to August 1983). The breakdown of the examinations by vascular regions is shown in Table 1.

An overwhelming number of patients (808) was studied following peripheral venous contrast medium injection. 47 patients underwent arterial DSA.

The following comments mainly refer to the display of the brachiocephalic vessels following peripheral venous contrast medium injection (VDSA) into the extracranial and intracranial region in 649 patients.

Imaging System

For the examinations, we used the Siemens Angiotron DSA system in conjunction with a Siregraph 2 fluoroscopy unit provided with remote and tableside control. The fluoroscopy examination table features a stable, vibration-free wooden cradle for patient positioning. In this cradle, the patient can be rotated isocentrically by motordrive about the longitudinal axis. The table is also provided with an angle indicator.

The overtable tube of the Siregraph 2 installation can be tilted in the longitudinal direction of the table; moreover, tube and image-intensifier television system can be moved in the longitudinal direction of the examination unit, independent of the table top shift lengthwise and crosswise. At the table, a focusfilm distance (SID) of 95 cm and 115 cm can be chosen. Parallax compensation of the

image intensifier for an oblique projection of up to 40° is provided. The television system is made up of a Videomed N unit, 625 lines, and a high-resolution Hivicon N TV camera tube. The high-resolution image intensifier tube Sirecon 30 H—triplex can be switched over to the 33, 23, and 17 cm input field.

The digital image processing system Angiotron, which functions in conjunction with the X-ray installation, has 3 digital semiconductor memories using a matrix of 512 × 512 pixels. The analog TV image with 625 lines and 25 frames/sec is digitized via an analog-digital con verter (ADC) at a frequency of 20 MHz and an 8 bit information depth. The Sirecord X/AN which is capable of archiving up to 337 scenes serves as an image store.

Image reproduction with separate setting of the upper and lower window limit is archived via a digital analog converter with 256 gray steps and 8 bit information depth.

For documentation on transparent film, a multiformat camera is available, which facilitates subdivision into 8 different fields including a slide format.

Table 1. *Breakdown of the VDSA Studies*

Supra-aortal branches and skull	649
Pelvis and legs	150
Kidneys	52
Others	14
Total number	855

The Examination Method

For visualization of the brachiocephalic vessels, the patient is positioned in the stable, vibration-free, motorized and rotary wooden cradle, and, after selection of the angular setting, can be rotated isocentrically about the longitudinal axis. The use of a rotary cradle offers the advantage that the whole patient can be moved with reproducible settings, thus the so-called "screw effect", *i.e.,* faulty rotations of the head with respect to the neck and trunk movement, is avoided.

An important goal to be achieved during the study is to ensure as much object homogeneity as possible. In the head and neck area, this is attained by the careful placement of homogenizers (Mondamin, rice-flour) of different size and thickness. Moreover, accurate field collimation by the use of lead plates close to the image intensifier is very important. Careful arrangement of such lead plates on the examination table tends to decrease the strong noise at the edges of the confined collimator field.

Correct patient positioning calls for radiological intuition. Optimum positioning of the measuring chamber as well as selection of an adequate angulation for oblique projections have a considerable bearing on the result of the study.

The following standard program for the examination of the brachiocephalic vessels has become firmly established at our institution:

1. Series in frontal patient position from the aorta as far as the base of the skull, with image intensifier 33 cm input field.

2. Series for the visualization of the cervical arteries in left anterior patient position, with rotation through 45° with respect to the mediosagittal plane, with the image intensifier 23 cm field.

3. Series in right anterior patient position for the display of the cervical arteries and the siphon of the carotid arteries, rotation through 45° with respect to the mediosagittal plane to the right side, with image intensifier 23 cm field.

4. Frontal semi-axial scene of the intracranial vessels with a cranially coned tube tilt from 15 to 18°.

Depending on the clinical objective and the course of examination, the standard program is supplemented by oblique projections of the aortic branch segments, by further oblique projections of the cervical region as well as by lateral or oblique projections of the cranial area.

The examination is carried out under continuous ECG control. Drop infusion and the availability of all facilities required for emergencies are mandatory, but with modern contrast media sensitivity reactions are extremely uncommon.

Injection

In VDSA the contrast medium is invariably injected into a peripheral vessel, *i.e.*, the right or left cubital vein. For ADSA the contrast medium is injected by way of the counterflow overpressure technique into one or into both brachial arteries or the contrast solution is administered via an arterial catheter.

For studies of the peripheral veins, a Braun cannula with an external teflon sheath and sharply ground mandrin is used. The puncture cannula has an outside diameter of 1.7 mm.

42 to 47 ml Telebrix 380 (about 0.7 ml/kg body weight) are injected per series at a flow rate of 12 to 15 ml/sec. For injections, the microprocessor-controlled high-pressure Simtrac C syringe is used. This injector is provided with a recorder to register all data such as patient identification code, examination data, injection number, effective and rated injection data, errors in operation and/or errors of the apparatus. The course of the injection is recorded graphically.

Via a Y-shaped connector, 20 to 30 ml NaCl solution is injected through a second injector electronically coupled to the contrast medium syringe. This injection immediately following the contrast medium injection is triggered automatically via one and the same control and the solution is administered at the same flow rate as the contrast medium. Releasing the triggering button stops the saline injection in any desired phase.

Factors Influencing the Image Quality

In DSA, the image quality, *i.e.*, the ability to assess the vessels, is influenced by the following criteria:

Spatial resolution of the system
Diameter of the image intensifier input field
Noise
Thickness of the homogenizer
Interference by overlying vessels

Artefacts attributable to:
- Patient motion or instability of the imaging system
- Laryngeal movements caused by swallowing
- Intestinal peristalsis
- Metallic foreign bodies
- Overexposure of the pulmonary apices
- Contrast medium in the brachiocephalic veins on the injection side

Circulatory, respiratory, and psychic situation of the patient ("cooperation")
Contrast medium concentration in the arteries (contrast density)

Results

Based on a visual assessment of the image quality, 649 peripheral venous digital subtraction angiograms of the brachiocephalic region were systematically evaluated by two independent examiners. 72% of the views had an image quality which fully measured up to the diagnostic objective. In 26% of the examinations, the image quality was inconsistent, but still permitted a diagnosis. 2% of the views were of inadequate image quality, so no diagnosis could be established.

The average age of the patients examined, both male and female, was 53 years. 22% of the patients presented with unremarkable conditions, 24% had stenoses, occlusions, plaque formation, 40% were affected by diffuse arteriosclerotic vascular lesions, while 14% suffered from different vascular diseases (Figs. 1 and 2).

Arterial digital subtraction angiographies were carried out only in isolated cases to clarify special diagnostic problems. This involved a heterogeneous patient group, which was not included in the systematic analysis.

In DSA, the quality of the clinical results greatly depends on the experience of the examining physician as well as on his ability to interprete the findings. Two months after commissioning the unit, we contrasted, over a period of 9 months, the diagnostically conclusive results with the unreliable ones. It was found that only after a 4-month period was it possible to obtain consistent results by improving the method and by gaining more experience. The diagnostic results were considerably and constantly improved when 9 months after commissioning the DSA installation, a saline solution was separately and automatically injected via a second high-pressure syringe. In contrast to the "layering technique", an optimum bolus effect is obtained by means of the subsequent injection of the saline solution, which enhances the contrast density in the arterial vessels and avoids obscuring effects caused by contrast-filled brachiocephalic veins in the region of the upper thoracic aperture and in the neck on the injection side.

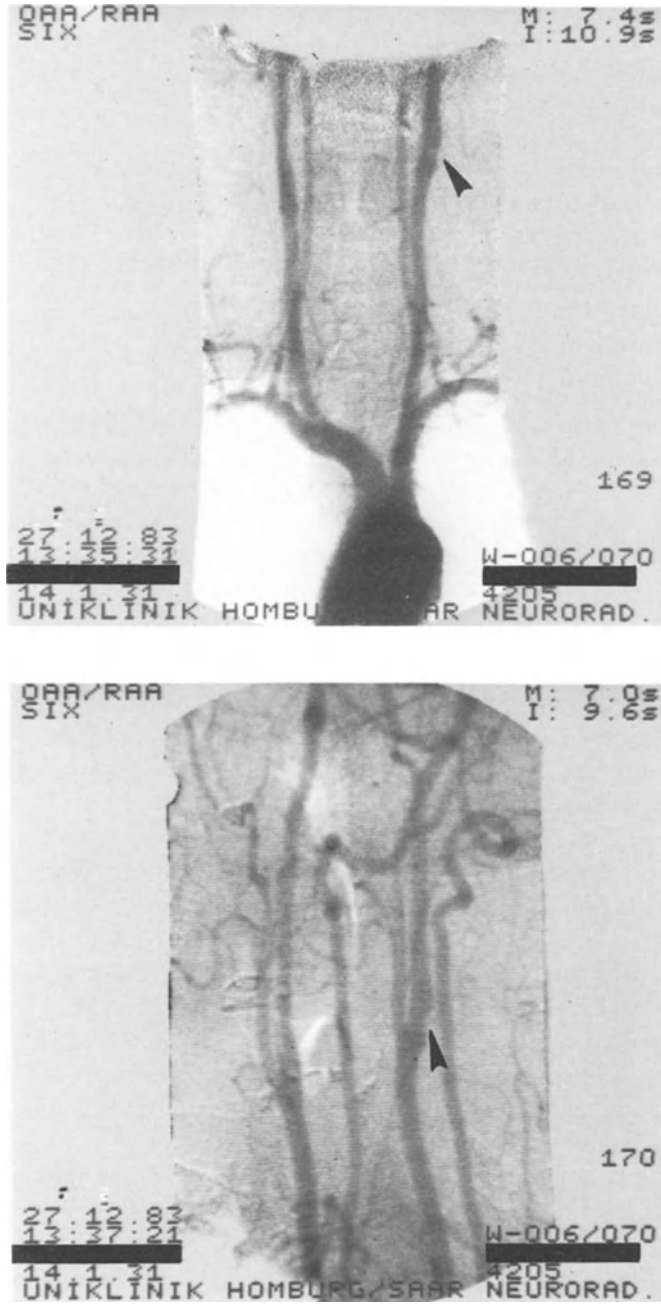

Fig. 1. 53-year-old patient with left cerebral transient ischemic attacks, VDSA demonstrates atheromatous plaques at the origin of the left internal carotid artery(→). Perfusion of the intracranial arteries is symmetrical

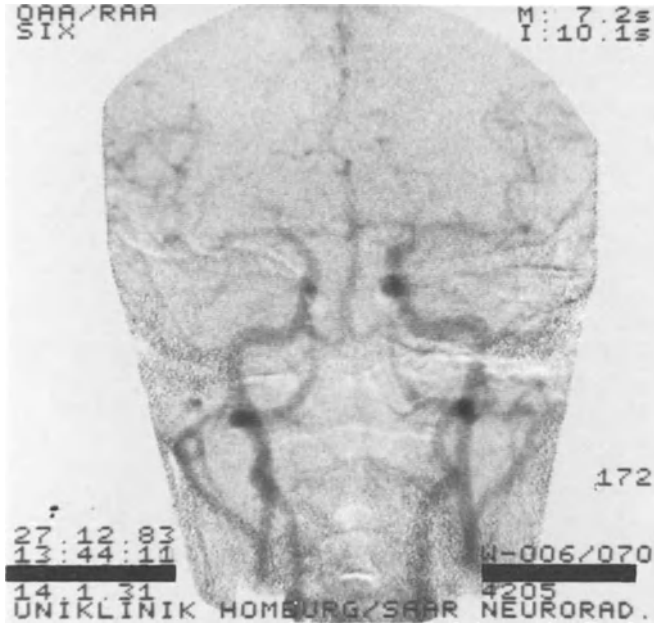

Fig. 1 (III)

As far as the intracranial region is concerned, VDSA has not been found suitable without restriction; this is because the contrast medium concentration in the branches of the cerebral arteries is insufficient. However, despite the limited contrast density, information is also obtained for the intracranial region which largely answers a series of diagnostic questions. A case in point is the differences in perfusion of the anterior, middle, and posterior cerebral arteries, which, as a rule, can be reliably identified or ruled out.

In addition, sinus and venous thromboses are well diagnosed. Moreover, sufficient information is obtained postoperatively following the exclusion of aneurysms and angiomas (Figs. 3 and 4).

Vascular conditions in the neighbourhood of hypophyseal adenomas can be outlined and differentiated from large basal arterial aneurysms. Meningiomas and arterio-venous malformations are visualized, and here the comprehensive coverage of the arterial supply and the venous drainage will prove particularly advantageous. However, very detailed evaluation of the intracranial vessels to reliably exclude pathological processes is not possible.

The safety and ready implementation of VDSA are particularly valuable features. At our institution, VDSA is predominantly performed on outpatients. A skilled and well-trained team is expected to carry out an examination within 15 to 20 minutes on average.

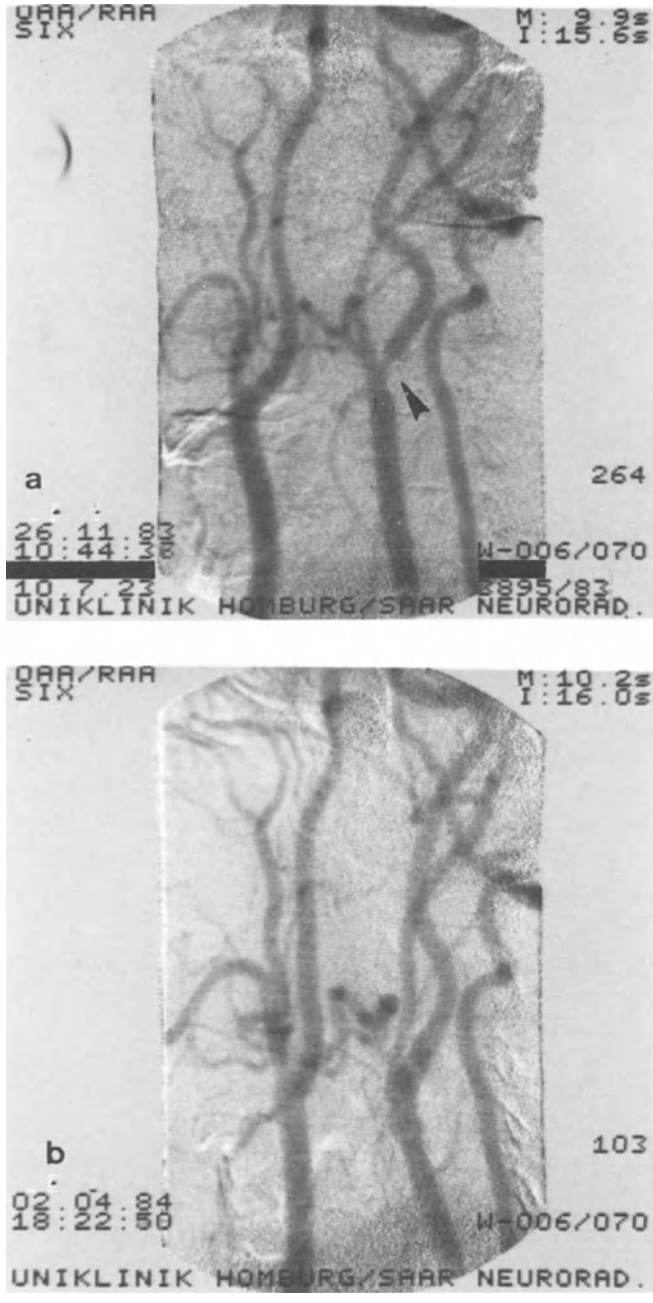

Fig. 2. VDSA: severe stenosis of the left internal carotid artery at its origin (→) before (a) and after (b) endarterectomy

The following side effects and complications were observed:

— Rupture of the veins with local hematoma	9
— Thrombophlebitis	2
— Mild allergic reaction (urtication, sneezing, and itching)	10
— Nausea	5

Discussion and Conclusions

The basic drawback of the available DSA systems is the clearly lower *spatial resolution* compared with direct conventional large-format cutfilm angiography. Whilst in the conventional technique roughly 5 line pairs/mm are resolved, the DSA installations in common use resolve between 1.4 line pairs/mm for the 25 cm image intensifier input field, and 2.0 line pairs/mm for the 17 cm input field[4,8,11].

The low spatial resultion has to be set against the considerably higher *contrast resolution* compared with conventional angiography brought about by the electronic facilities of subtractions, summation, and signal amplification in digital image processing[4,5,9]. The high contrast resolution of DSA is of the order of 1%[5,12].

The favourable contrast resolution in DSA is somewhat counterbalanced by the problem that via the transvenous route only a limited contrast concentration is obtained in the arteries, insufficient for visualizing ramifications of the second and third order. It is apparently not so important whether the contrast medium is injected into the peripheral veins or into central veins near the heart[1]. The resulting contrast concentrations in the arterial system are the same, the only difference being that with central vein injection a lower contrast volume suffices to obtain the maximum arterial contrast concentration. Investigations carried out by Fischer and Schultz[3] and our own measurements have shown that after taking blood samples from the femoral artery, the maximum arterial concentration obtainable in the cervical arteries and in those of the extremities amounts to a maximum of 10 ± 5 mgI/ml following venous contrast injection.

As was demonstrated by Fischer and Schultz[3] and Schultz and Fischer[10], in DSA image quality, vascular lumen and contrast concentration in the arterial blood follow an inverted pattern. The contrast medium concentrations required for adequate image quality depend, according to Fischer and Schultz, clearly on the thickness of the homogenizer and amount to

2–6 mgI/ml in a vessel with 8 mm lumen
10–20 mgI/ml in a vessel with 2 mm lumen
20–37 mgI/ml in a vessel with 1 mm lumen

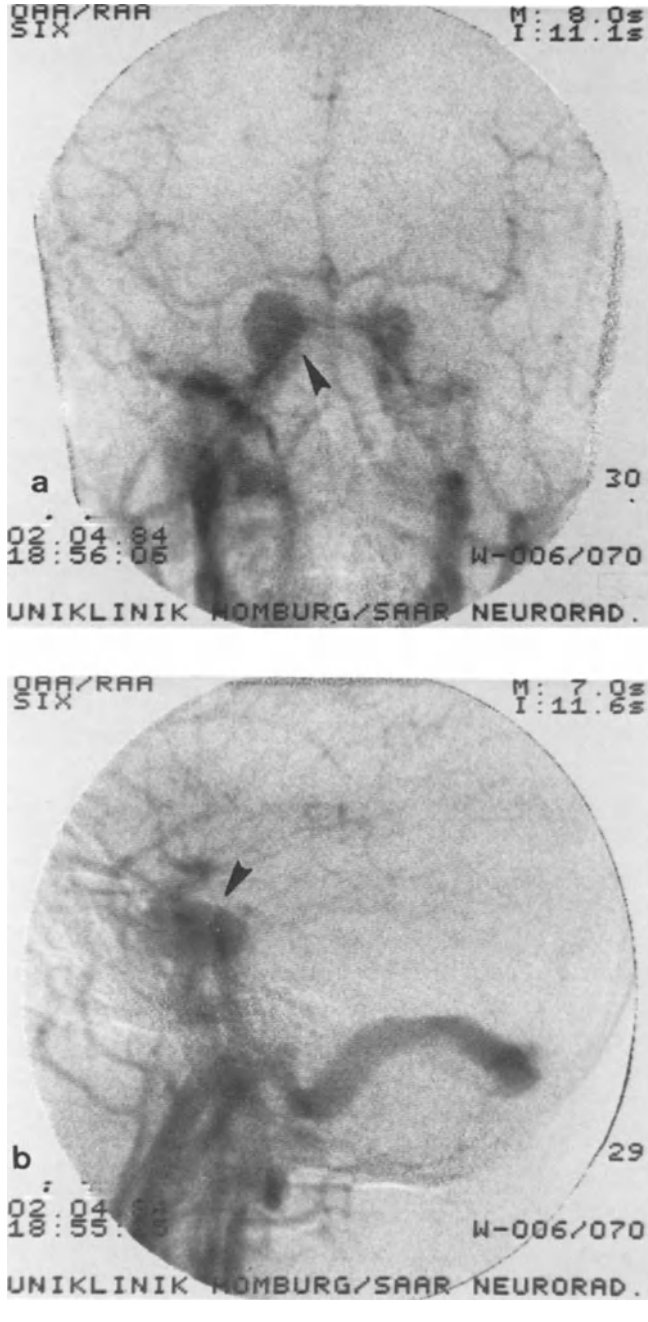

Fig. 3. VDSA: Right-sided traumatic carotid cavernous sinus fistula before (a and b) and after (c and d) balloon occlusion (→)

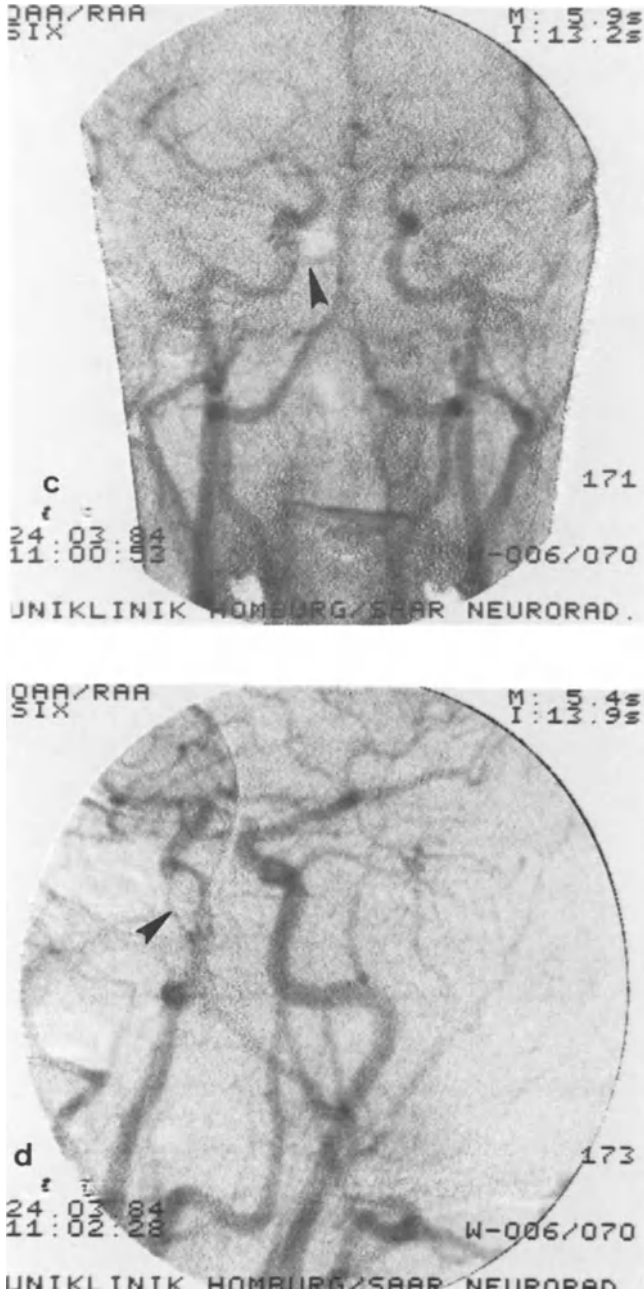
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Fig. 3c–d

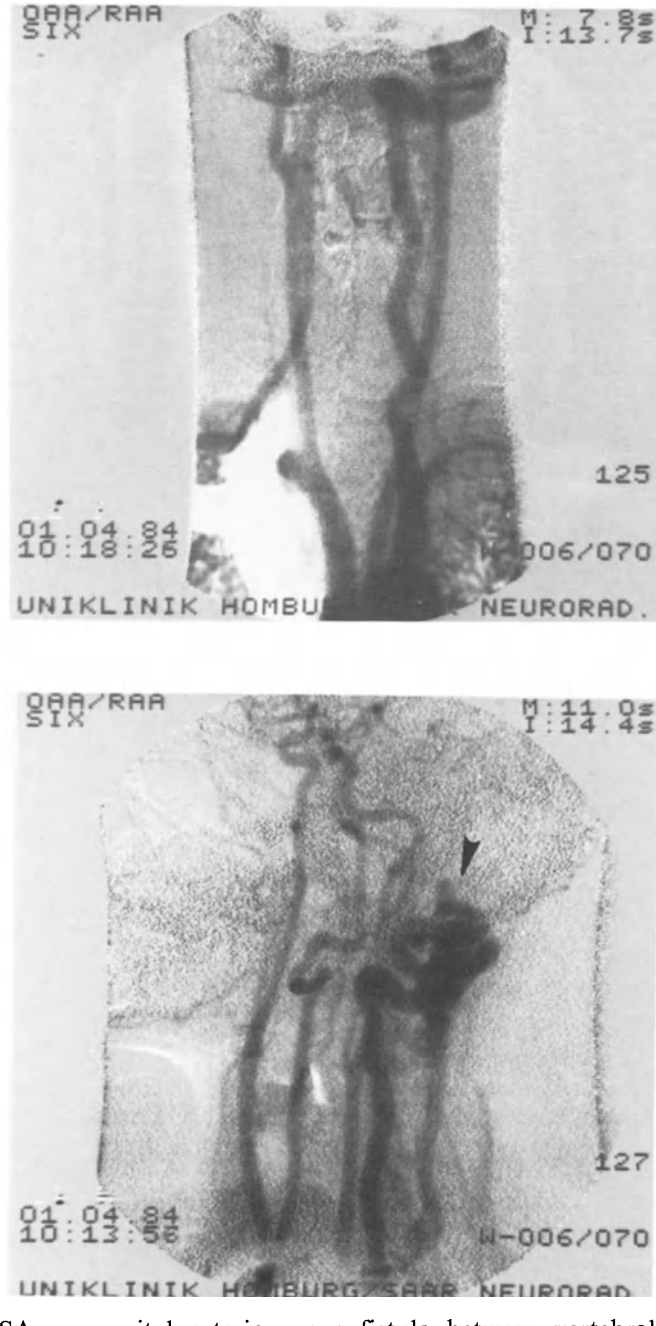

Fig. 4. VDSA: congenital arteriovenous fistula between vertebral artery and external jugular vein on the left (→). The arterial supply and venous drainage by the enlarged vessels is seen clearly

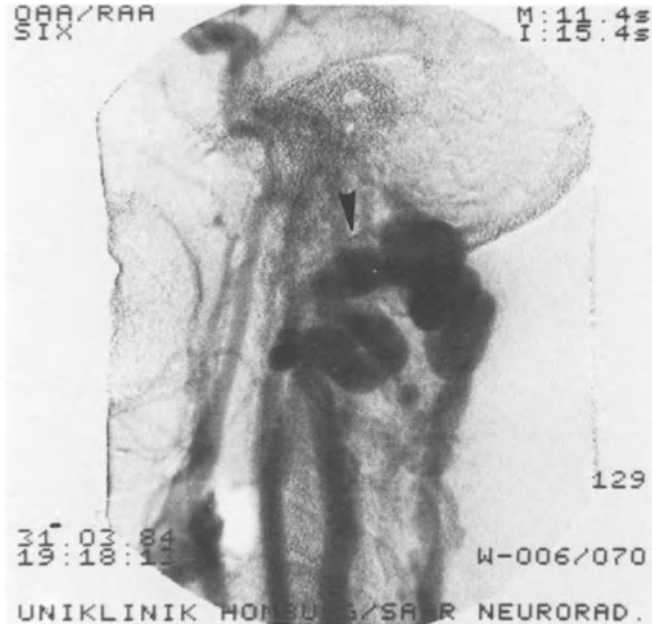

Fig. 4

For the display of vessels with a minimum lumen of 0.5 mm, contrast concentrations above 10% (37 mgI/ml) are required. If the image quality is not expected to meet stringent requirements, *e.g.*, when the diagnostician puts up with the finding "vessel is present or vessel is patent", a concentration of 18.5 mgI/ml is sufficient for the visualization of a 1 mm lumen vessel[3].

Huber[6] had already demonstrated by densitometric methods that in conventional angiography the maximum contrast concentration is normally 41.6% ± 3.6 in the carotid siphon. Christenson and workers[2], who used the video subtraction system, found that the cervical and the trunk sections of the intracranial arteries can be outlined with an extremely low contrast medium concentration of 2% to 3%. In agreement with the measurements carried out by Huber, we have realized that an intraarterial contrast concentration of 40% to 50% is needed to obtain a similar contrast density in conventional standard angiography. Even under the most favourable examination conditions and with a homogenizer of minimum dimensions and with kinetic artefacts completely missing, the arterial contrast concentration obtainable transvenously proves insufficient for the evaluation of arteries with a caliber below 2 mm. In practice, this can be illustrated by transvenous digital subtraction angiography of the hand, where the palmar arches can still be displayed, but not the digital arteries[3].

If one wants to visualize vessels with a lumen of less than 2 mm, even in DSA the contrast medium must be injected *arterially* despite the high density resolution—this is what in theory is to be expected. Vessels having a lumen of as low as 0.1 mm can only be visualized when the contrast medium is injected into an artery[4,7].

After *intravenous* administration of the contrast agent, digital subtraction angiography faces certain restrictions as a result of the physiological circulatory conditions, which cannot be offset at all or only to a limited extent by technical improvements of the imaging systems. Nevertheless, the lower invasiveness as well as the practicability, flexibility, and rapid implementation of the examination justify consideration of the procedure as being suitable for the visualization of the extracranial segments of the cerebral arteries, and capable of answering most of the clinical questions regarding this area with sufficient dependability. On comparing VDSA with large-format conventional angiography, one must take into account that a number of drawbacks, such as the problem of disturbing superimpositions by vessels, also apply to conventional techniques. Strictly speaking, only the conventional *global* procedures such as the display of the brachiocephalic arteries by transthoracic survey aortography and—with some reservation—by retrograde counterflow techniques are comparable to VDSA. A critical comparative assessment also presupposes that the conventionl angiograms are subtracted photographically. It is inaccurate and misleading to compare, say, selective conventional angiographies of the common carotid artery with panangiographic transvenous images generated by DSA.

Conventional panangiography of the brachiocephalic arteries entails the following disadvantages:

— Reciprocal superimposition of the brachiocephalic and cervical arteries
— Contrast agent limit (2, 3 injections at most)
— Intracranial vessels can only be assessed as a whole
— Expensive subtraction
— High film consumption
— Unnecessary multiple documentation of normal and pathological findings
— Higher invasiveness
— Catheterization in elderly patients difficult or impracticable
— Frequently inadequate contrast of the carotid bifurcation
— Increased catheterization risk

The disadvantages of conventional survey aortography can be lessened by rotating the patient or the imaging system during the injection or by using medium-sized formats with a cutfilm camera to keep the film consumption low.

VDSA affords the following advantages over global conventional angiographic procedures:

— Lower invasiveness
— Catheterization is not attended by problems or hazards
— Visualization of the vessels at the base of the skull is markedly better
— Selective documentation of the examination results without waiting time
— Minimum film consumption
— Automatic instantaneous subtraction (no additional expenditure of time and film)
— Also possible as an outpatient procedure
— Higher number of projections
— Considerably simplified archiving
— Generation of slides with multiformat camera
— Lower dose to the patient
— Less time-consuming

By contrast, there are also some disadvantages to be considered:
— Superimposition of veins on the injection side
— Overexposure of the pulmonary apices, and as a result, impaired visualization of the aortic arterial branches
— Further selective arteriography usually only possible in a second session
— Higher dependence on the patient's "cooperation", circulatory and respiratory conditions and anatomy
— Contrast density in the cervical region is not always considered adequate

If we analyse the merits and demerits of conventional *and* digital-venous global angiography under common aspects, we find that the following restrictions apply *to all panangiographic methods*:
— Only a coarse evaluation of the intracranial vessels is possible
— In the cervical region, contrast medium concentration is not always adequate
— Reciprocal superimposition of supra-aortic and cervical vessels cannot always be reasonably avoided
— Total contrast volume too high

From these aspects, we can conclude that with respect to both conventional and digital angiographic procedures, extended *selective arterial* angiographic techniques cannot be dispensed with, if global visualization fails to resolve the clinical problem adequately. This holds good particularly when the intracranial vessels have to be evaluated as well.

It is obvious, as future developments will in all probability confirm, that this extended procedure will become the domain of *arterial* DSA because of the main advantages it offers, *i.e.,* lower invasiveness compared with conventional angiography with reduced neurotoxicity as well as simplification and improvement of image subtraction. There are considered to be decisive in vascular diagnosis in the entire central nervous system. The decreased spatial resolution of DSA is of negligible importance for practical

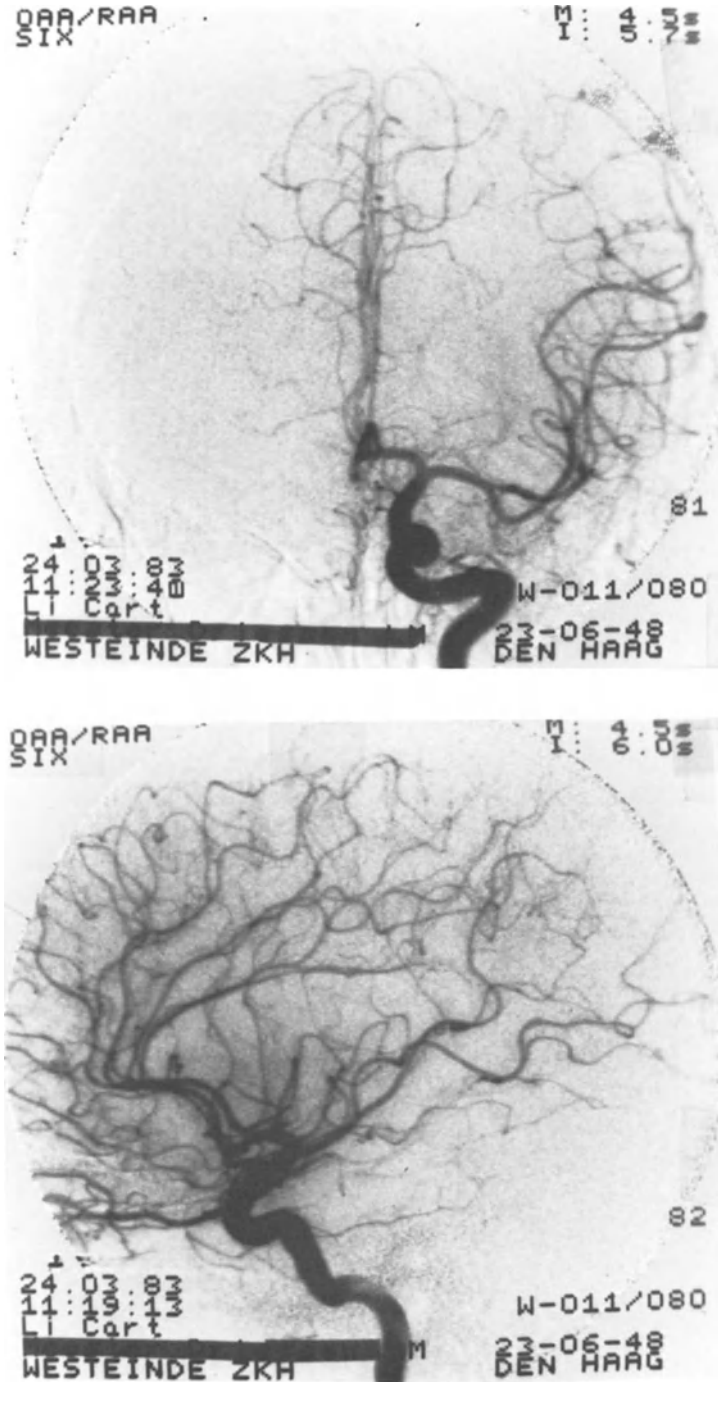

Fig. 5. ADSA: left internal carotid angiography performed by catheter technique. Normal study (Dr. Hoogland, Den Haag)

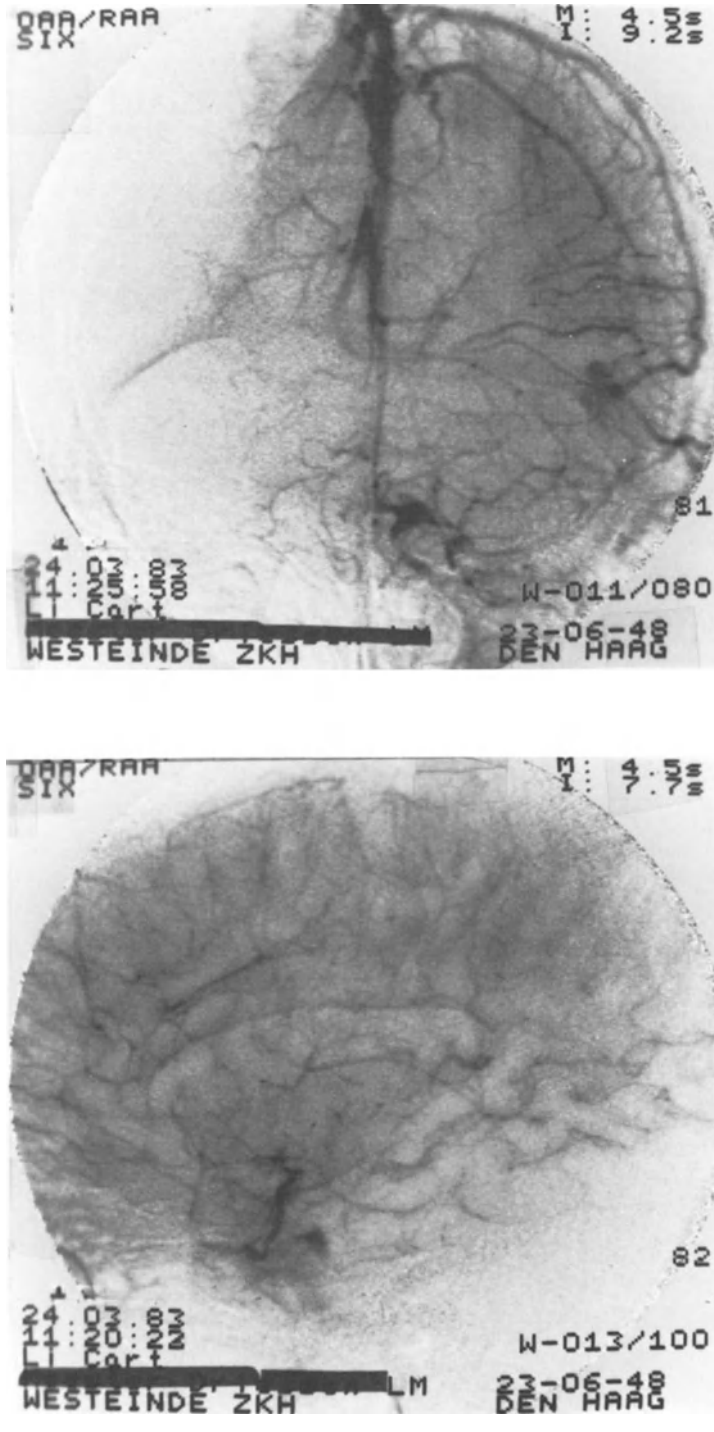
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Fig. 5

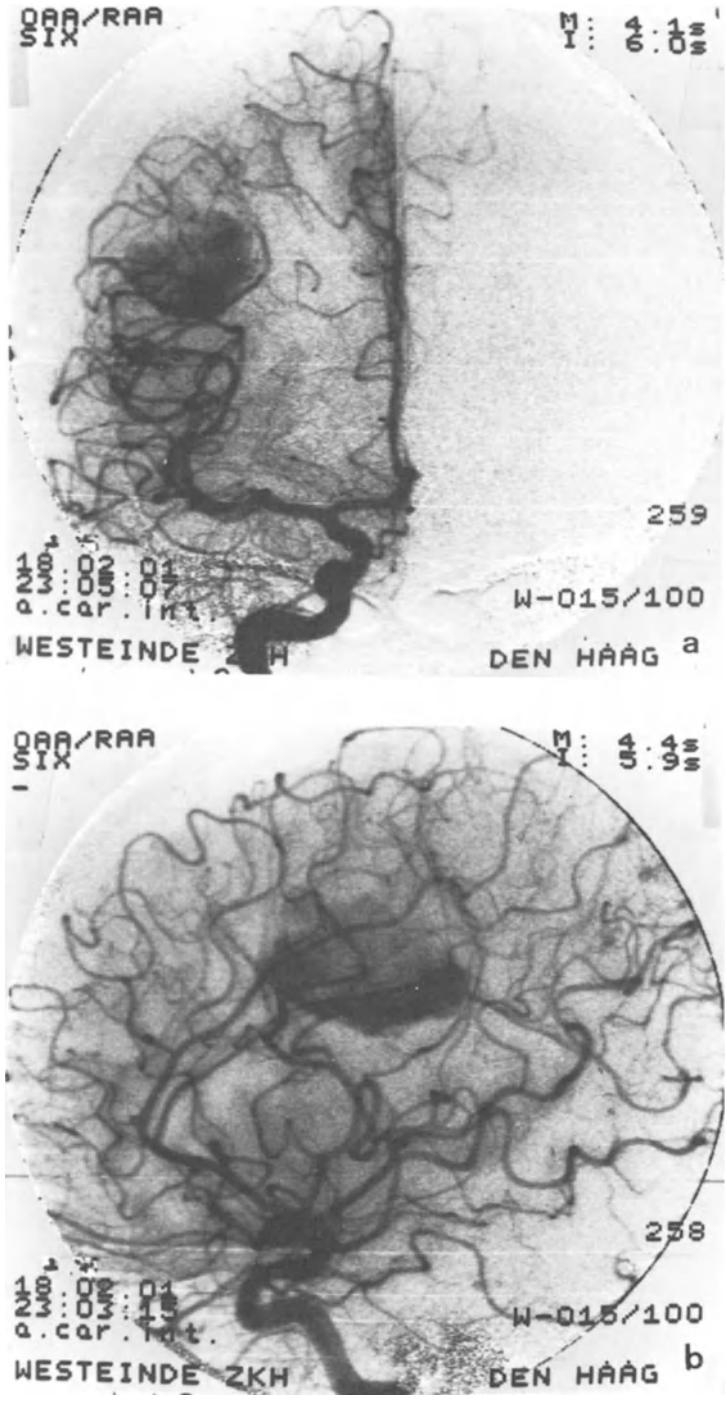

Fig. 6. ADSA: parietal meningeoma on the right visualized by selective catheter angiography of the right internal (a and b) and external (c) carotid artery (Dr. Hoogland, Den Haag)

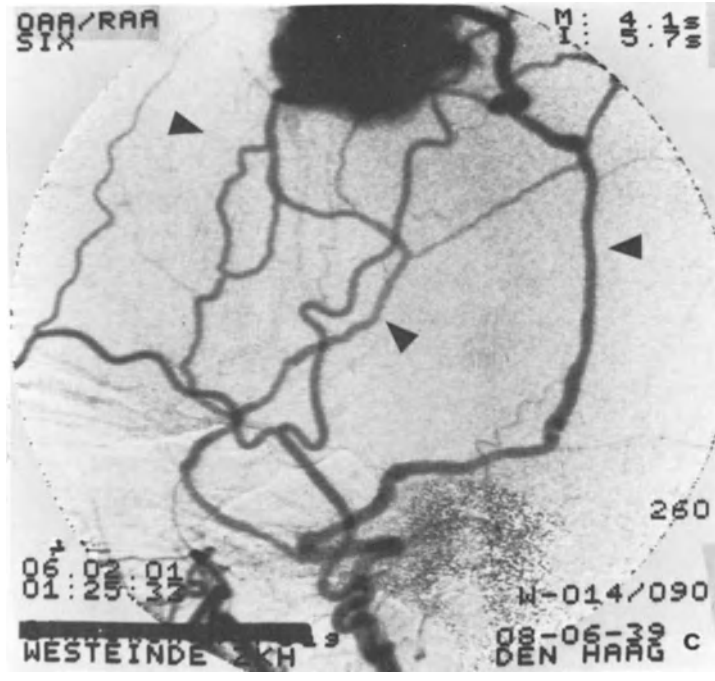

Fig. 6c

clinical diagnoses when the contrast material is administered via the arterial route.

When analyzing selective arterial DSA images (Figs. 5 and 6), it becomes apparent that the higher arterial contrast concentration in comparison with venous application, combined with a marked increase in contrast resolution, plays a decisive role. There is no doubt that following arterial contrast administration, all vascular regions of diagnostic interest can be adequately visualized and assessed.

In summary, *ADSA* affords the following advantages over conventional angiography:

— Low single contrast dose and therefore the possibility of extending the angiographic examination without added risk
— Survey aortography sufficient for the evaluation of the aortic and cervical region as well as for global display of the intracranial vessels
— Selective and superselective angiography of the cerebral vessels with a sufficient variety of projections (search for aneurysms, vascular lesions)
— Angiography can be extended to include extracranial examinations in the same session (*e.g.*, renal arteriography in the case of hypertension)

— Arteriography of the pelvis and legs in patients with generalized vascular pathology, search for primary tumor with cerebral metastasis
— Low film consumption with selective documentation of findings
— Automatic instantaneous subtraction without loss of time and additional film consumption
— Interventional catheterization through instantaneous subtraction with low expenditure of time and added flexibility
— Documentation in a clear and easy-to-handle manner
— Archiving is simplified and less expensive

These days, neuroangiography is at its best when it comes to identifying or ruling out primary vascular processes, *i.e.*, obliterative vascular diseases, arterial aneurysms, arteriovenous malformations. The use of angiography for the exclusion of space-occupying lesions has greatly diminished since the advent of computed tomography. In terms of frequency and volume of the contrast doses administered, the brain and the spinal cord are much more sensitive than the kidneys, the liver, pancreas, the extremities, and other organs. The risk attending cerebral angiography in patients with vascular diseases is increased by 30% to 50% as compared with other pathologies. It is however especially the vascular patients who usually require a rather comprehensive angiographic examination to meet the diagnostic requirements. In such instances, a largely complete diagnosis of the extracranial and intracranial vessels must be established. This frequently proves impracticable in a single session using conventional angiography. Quite often, important projections or vascular segments are overlooked. A similar situation is encountered in the diagnosis of aneurysms, where answers to numerous and specific questions are required.

In the diagnosis of occlusive pathology of the cerebral and spinal vessels, digital angiographic procedures must definitely be preferred as they permit a considerable number of single injections of minimum individual and total contrast doses, while the risks associated with catheterization are noticeably reduced. In order to achieve this goal, the combined use of VDSA and ADSA with conventional angiography offers the best prerequisites for success, especially when the present state-of-the-art of these techniques is considered. On the grounds of theoretical and practical considerations, we must expect that in cerebral and spinal angiography conventional examination techniques will be increasingly replaced by digital angiography with arterial administration of contrast agents.

Acknowledgement

We extend our thanks to Dr. Hoogland, Den Haag, for providing us with images illustrating examples of arterial DSA (Figs. 5 and 6).

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B. Technical Standards

Arteriovenous Malformations of the Spinal Cord

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The Arterial and Venous Anatomy of the Spinal Cord

Arterial Supply

The arterial blood supply of the spinal cord has been well and extensively described (Kadyi 1889, Clemens *et al.* 1957, Adamkiewicz 1881). The dorsal rami of the segmental intercostal and lumbar arteries give rise to a spinal ramus which amongst its other branches sends a neural branch to supply roots, spinal ganglia and, in a number of instances, to go on as either an anterior or posterior radicular artery to the cord. The number of neural branches that actually reach the spinal cord on average is twenty-four. The dominant blood supply to the cord is by a series of usually no more than six to eight anterior radicular arteries which together with descending branches from the vertebral arteries, contribute blood supply to the anterior spinal artery. The dominant contributors to the blood supply of the spinal cord are the vertebral vessels in the upper cervical cord, branches from the vertebral vessels in the lower cervical cord, and the artery of Adamkiewicz or arteria radicularis magna found on the left side accompanying any anterior root from D 6 to L 4 (Adamkiewicz 1881). Most frequently, however, it occurs on roots between D 8 and D 12. The general surgical experience is that the division of no single radicular artery is likely to impair the blood supply of the spinal cord with the exception of the arteria radicularis magna, but that extensive disruption of intercostal arteries of supply as, for example, by dissecting thoracic aneurysm, are likely to affect the cord as high as the critical watershed between the relatively poorly arterialized thoracic cord and the cervical enlargement.

The Venous Drainage of the Spinal Cord

The venous drainage of the spinal cord has been subjected to much less study. The classic paper of Gillilan (1970) however, not only codified the anatomical description of spinal veins, but also foretold the influence of extradural arteriovenous malformations, at that time unknown, as a cause of the necrotizing myelopathy which pathologists were then becoming aware of as possibly due to chronic venous distension (Foix and Alajouanine 1926, Lhermitte *et al.* 1931, Greenfield and Turner 1939, Mair and Folkerts 1953, Antoni 1962).

Gillilan's description showed the spinal cord draining by two principal groups of intrinsic veins, a central group collecting from the anterior horns and associated white matter and draining into central veins in the anterior median fissure which in turn converged to an anterior median spinal vein, and a second radial group arising from a capillary plexus at the periphery of the dorsal or lateral white matter and draining white and gray matter towards the surface to join the coronal plexus of veins (Fig. 1). On the

surface of the spinal cord the anterior median spinal vein, which is frequently a double structure, lies in the anterior median fissure superficial to the accompanying artery and extends the length of the spinal cord. Into it drain the central veins of the anterior intrinsic group and branches of the coronal plexus on the anterior and ventrolateral parts of the cord. The coronal plexus is irregularly distributed around the cord but tends to align in irregular columns, particularly over the posterior median sulcus, the

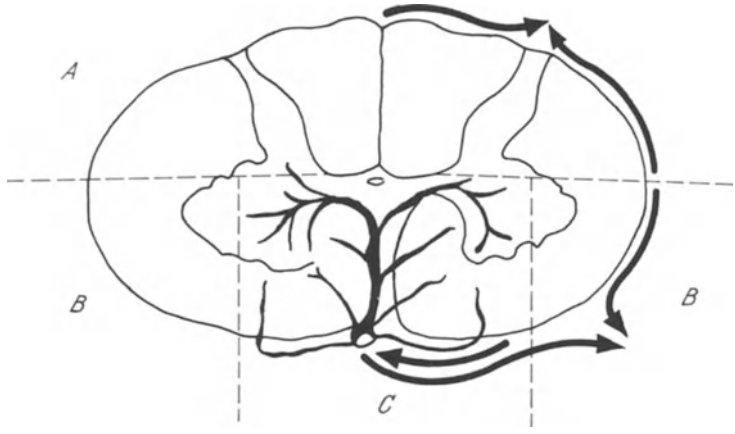


Fig. 1. The venous drainage of the spinal cord. Zones *A* and *B* drain predominantly into the coronal plexus, zone *C* drains into the central veins in the anterior median fissure. Both groups converge on veins draining into the extradural space along anterior or posterior nerve roots (after Gillilan)

posterior intermediate sulcus or along the line of entrance of the posterior nerve roots. Gillilan points out that the posterior plexus is usually much more massive and becomes more dilated as age advances. It can be very highly convoluted and particularly dense over the enlargements.

The superficial veins of the cord drain through medullary veins accompanying certain of the nerve roots and anteriorly there are about as many medullary veins as arteries, 8 to 12 or 14. They usually arise in the anterior median spinal vein, they are asymmetrical in their location not necessarily travelling with the medullary arteries, and a particularly large vein leaving from the upper part of the lumbar enlargement, usually on the left side has been known as the great anterior medullary vein. It descends in the roots of the cauda equina. Posterior medullary veins are more numerous, particularly in the cervical enlargement where there is usually one large vein accompanying the posterior roots in each segment. They are less frequent in the thoracic region but again, slightly more frequent in the lumbar region. There may be one sufficiently large to be titled "great posterior medullary vein", although

this is not invariable. Medullary veins drain into the plexus around the nerve roots in the intervertebral foramen and in turn into the paravertebral or epidural venous plexus. It in turn communicates with the venae cavae, azygos and hemi-zygos veins, and above with the sinuses of the dura mater and cerebral veins. Clemens (1961) has described the presence of valves in the medullary veins at their junction with the vertebral plexus, which would tend to prevent reflux of blood from the paravertebral plexus into the veins of the spinal cord itself.

From this excellent anatomical description Gillilan went on to point out the presence in the clinical literature of a syndrome of sensory and motor loss together with sphincter disturbance often described as subacute necrotizing myelitis and agreed by a large variety of authors as due to pathological changes in the blood vessels.

Classification of Spinal Arteriovenous Malformations

Progressive improvements in techniques of selective spinal angiography have led to a better understanding of both normal and abnormal vascular anatomy of the cord. However, the anatomical configuration, pathophysiology, and even the siting of spinal arteriovenous malformations remain incompletely understood and a diversity of views has developed in terms of their classification and optimum means of treatment.

Since the early reviews of Sargent (1925) and Wyburn-Mason (1943) there have been many papers describing the various morphological types of spinal AVM, each series having its own preponderance of intradural and extradural lesions (Ommaya *et al.* 1969, Di Chiro *et al.* 1971, Pia 1973, Aminoff and Logue 1974, Houdart *et al.* 1978, Symon *et al.* 1984). The commonest lesion would seem to be a single coiled or tangled AVM lying on the dorsum of the cord and supplied by multiple small arterial feeders at the level of each dorsal root. A second type comprises dilated tortuous draining veins lying principally on the dorsal aspect of the cord and supplied by only a single or very few arteries, the nidus (shunt) lying within the dura at the level of the dorsal root. Ommaya (1969) described a glomus malformation usually supplied by only a few major feeders and despite its mainly dorsal position, being fed often by ventral segmental arteries. These glomus lesions may lie partially within the cord when they receive a supply from the anterior spinal artery. Malis (1982) differentiated two types of cervical intramedullary lesion with the entirely intramedullary glomus angioma being more common than the dorsally placed lesion found at the cervico-medullary junction. Di Chiro *et al.* (1971) described a juvenile AVM, a curaisse of feeding vessels both ventral and dorsal to the cord with intramedullary extensions. This type may be associated with other vascular anomalies.

The recorded incidence of significant penetration of the cord by the different types of spinal AVM varies, occurring in 60% of a series of 150 cases (Hurth, Houdart, Djindjian *et al.* 1978; Houdart, Rey, Djindjian and Djindjian 1978) but in only six of seventeen cases (35%) described by Cogen and Stein (1983).

The importance of considering AVMs principally in relation to vertical and transverse levels of the spinal cord and in terms of their vascular supply has been emphasized by Hurth, Houdart and Djindjian (Hurth *et al.* 1978, Houdart *et al.* 1978, Djindjian 1976) and by Lazorthes (1978). This concept is very relevant to the intramedullary angioma both in respect of the natural history and treatment of the lesion.

Whichever classification is adopted, two features of the spinal AVMs appear to be reasonably well established:

1. Most extramedullary spinal AVMs lie on the dorsal or dorsolateral aspect of the cord in the thoracic or thoracolumbar region. They may be supplied by a single or several feeding vessels derived from segmental arteries, they are relatively well differentiated from the normal cord vasculature, and the "shunt" is in the dura not the cord.

2. AVMs with significant intramedullary extension and those which appear to lie entirely within the cord differ from the extramedullary spinal AVMs in their clinical presentations, pathophysiology, and vascular supply. The intramedullary lesions are frequently found in the cervical region and at the conus and derive, at least in part, a blood supply from the anterior spinal artery.

Dural Arteriovenous Malformations

Pathology

It is only with the recognition of the true pathological nature of the dural arteriovenous fistula or malformation that much of the confusion relating to spinal cord arteriovenous malformations has been resolved. Early topographical attempts to classify spinal angiomas were based upon the relationship of the lesion to the various compartments of the spinal canal, thus lesions were classified as either intradural, intermedullary, extramedullary, extradural or vertebral. The development of selective spinal angiography (Di Chiro *et al.* 1971, Di Chiro *et al.* 1967, Djindjian 1972, Doppmann *et al.* 1969, Doppmann *et al.* 1983, Kendall and Logue 1977) demonstrated that the most common type of arteriovenous malformation in adults was indeed extramedullary and that the true arteriovenous communication lay not within the cord at all but on the surface of the dura, usually in an intervertebral foramen. Our accumulated experience (Symon *et al.* 1984) indicated that in adults the dural type of lesion is the commonest spinal

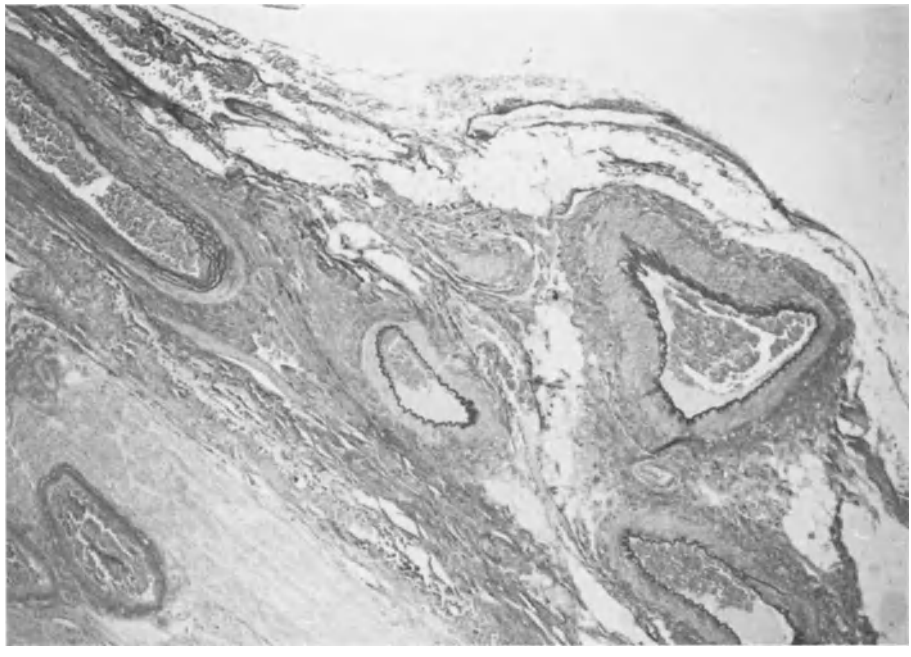


Fig. 2. Closely related arterial vessels (right) and venous vessels (left) in the substance of the dura in a typical dural arteriovenous malformation or fistula. (Slides prepared by Dr. Scaravilli from a case of Mr. R. D. Illingworth)

arteriovenous malformation producing a characteristic clinical picture hinted at in Gillilan's analysis and without true intrinsic pathology in the spinal cord at all. Di Chiro *et al.* (1967), and subsequently Kendall and Logue (1977) demonstrated that the pathology of the extradural arteriovenous fistula or malformation lay usually in the dura in the intervertebral foramen. They and Merland *et al.* (1980) showed that the arterial supply came usually from the radicular artery in the region of the intervertebral foramen sometimes associated with descending branches or ascending branches in the dura from neighbouring radicular vessels and sometimes even from vessels crossing from radicular vessels of the opposite side. As a rule, however, the radicular feeding arteries to the AVM or fistula were limited in number. From the AVM or fistula a single, draining vein quite distinct from the medullary veins which lie in association with nerve roots, pierces the dura a few millimeters from the associated nerve root either above or below it, and passes to the coronal venous plexus.

The exact nature of the abnormality in the dura is still to some extent open to debate. In a number of our own cases where it has been excised, it has been thought to be an arteriovenous malformation proper (Symon *et al.* 1984). Merland *et al.* (1980) regard these lesions as invariably arteriovenous

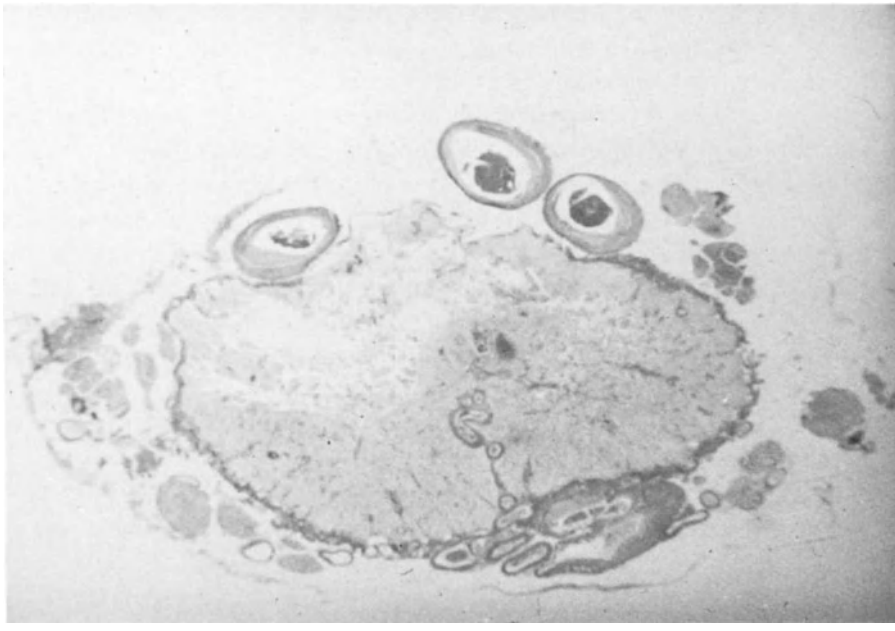


Fig. 3. A spinal cord from a case of dural arteriovenous malformation unoperated, discovered at autopsy. The dorsal columns are particularly affected by a necrosis, with preservation of the anterior columns as described by Gillilan (photographs courtesy of Dr. R. O. Barnard)

fistulae, and a specimen recently taken by a colleague (courtesy of Mr. R. D. Illingworth, Fig. 2), suggests that, in a number of instances, the coiled vessels extradurally are no more than elaborations of the venous plexus associated with a single fistula; it is likely that the basic pathology, in many cases, may well be that of a single AV fistula with redundant veins associated in the dura. At all events, there is no disagreement as to the subsequent course of a communicating vein to the coronal plexus, nor the fact that there is actually no arteriovenous pathology within the spinal cord itself. The classification of "single coiled vessel malformation", "long dorsal arteriovenous malformation" or "type I arteriovenous malformation" is irrelevant. The vast dilatation of the coronal venous plexus which is demonstrable principally on the posterior aspect where the coronal plexus is, at any rate, biggest, is secondary to its dilatation by high pressure venous blood emerging from a fistula or malformation outside the cord itself.

A new light is, therefore, thrown upon the pathological changes seen in advanced cases within the spinal cord. These have been described by a variety of pathologists (Greenfield and Turner 1939, Mair and Folkerts 1953, Antoni 1962) and an example of pathological material of this type is shown in the accompanying figure (courtesy of Dr. R. O. Barnard, Fig. 3).

The particularly interesting feature of this pathology was, quite apart from the degenerative necrosis evident in all aspects of the cord, its distribution particularly involving the lateral corticospinal tract and spreading gradually into the adjacent portions of the white matter of the lateral funiculus and thence progressively into the anterior gray and posterior columns also. As Gillilan (1970) has pointed out the area of the cord consistently spared in these lesions is the antero-median segment. In addition to the necrotic changes however, as Barnard has observed, a typical feature is the appearance of many new capillaries. These have been thought from time to time to indicate arteriovenous angiomata (Antoni 1962), but it now seems likely that the neovascularization is the result of prolonged ischaemic hypoxia secondary to venous congestion (see below).

Pathophysiology

While no clear appreciation of the exact pathology of these lesions existed, a variety of explanations were produced to account for the clinical picture of a very slowly progressive, mixed motor and sensory myelopathy. After the myelographic recognition of considerable dilatation of the coronal venous plexus the initial suggestion was that the spinal cord was simply being compressed by venous bulk. The association between gross venous distension and thickening of the pachymeninges led some to attribute the cord compression to arachnoiditis and secondary vascular changes (Fay 1937, Spiller 1911); in the minds of some investigators, this appeared to be due to small haemorrhages such as occur in cerebral angiomas. The comparative rarity of subarachnoid haemorrhage in this type of arteriovenous malformation of the cord will be referred to later.

The entire clinical picuture, reversible in its early stages, irreversible in its later stages and associated with typical neuropathology of necrotizing myelopathy, can be explained on the basis of chronic venous congestion (Aminoff *et al.* 1974). High pressure venous blood is brought to the coronal plexus by the draining vein of the malformation on the dura. This communicating vein may have a very long intradural course. Several of our cases had a dural malformation in the sacral dura and one a dural AV malformation at the foramen magnum. Distension of the coronal plexus may extend from the lumbar enlargement as high as the cervical cord and tends to become progressive the longer the fistula has been present and the more severe its symptoms. The raised pressure in the coronal venous plexus communicates itself to the veins within the spinal cord, and the area of permanent disruption shown in cases proceeding to advanced cord pathology corresponds to regions drained by the postero-lateral radial veins. It seems likely that the relative preservation of the central veins and of the anterior median spinal vein may be due to no more than an accident of

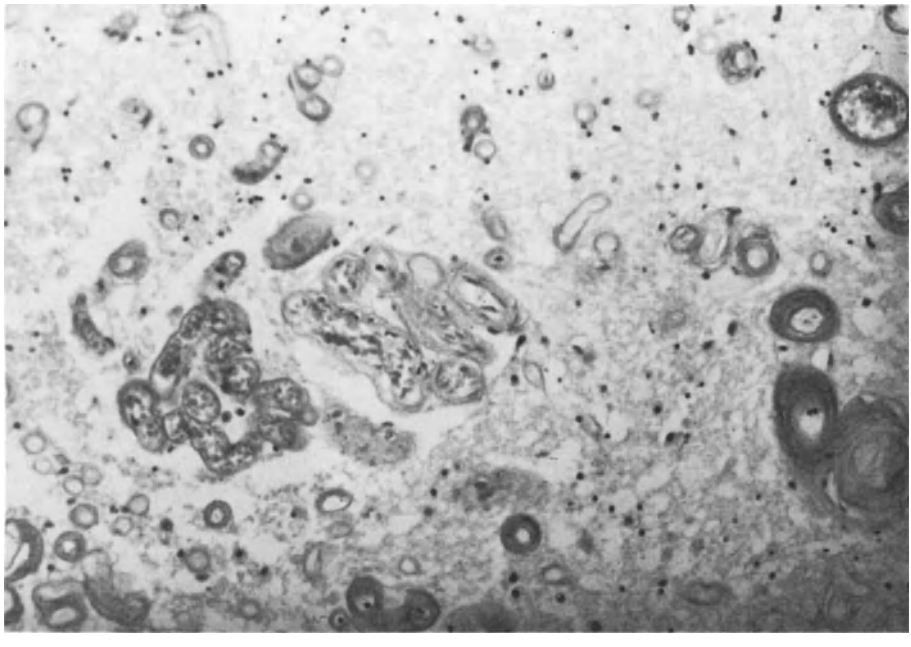


Fig. 4. Haematoxylin eosin stained preparation of neovascularization in ischaemic dorsal columns (courtesy of Dr. R. O. Barnard)

anatomy in which the posterior coronal plexus already meandering and dilated in the elderly, is earlier and more directly involved by this accessory medullary vein draining blood into, instead of out of, the cord. The result of grossly raised venous pressure its, of course, to diminish the arteriovenous pressure gradient and to embarrass perfusion of the spinal cord. Spinal cord blood flow is therefore likely to be reduced as a result of the increased intra-medullary venous pressure. Raised venous pressure will in turn lead to intra-medullary vasodilatation, and possibly to progressive exhaustion of autoregulatory capacity in the spinal cord in the affected areas. Progressive vascular dilatation in this uncontrolled fashion communicating itself to capillaries will result in the transmission of undamped pulsation to the cord, increased tissue pressure, and progressive formation of oedema with, in the most advanced cases, extensive ischaemic loss of cord tissue. The first area of the cord to be affected is the postero-lateral white matter and the lateral cortico-spinal tract. Progressive extension of the process gradually involves the lateral spinothalamic tract, both spinocerebellar tracts and the white matter of the posterior columns.

Extensive ischaemic anoxia is a strong stimulus to neo-capillary formation, and the appearance of these abnormal capillaries may therefore

be explained as the culmination of an advancing pathophysiological process, and not as evidence of true intramedullary arteriovenous malformation (Fig. 4).

Clinical Features of Arteriovenous Malformations

Dural Arteriovenous Malformations

A series of fifty-five cases of arteriovenous malformation of this type in which the lesion was arteriographically and surgically verified to be in the dura has been treated in the National Hospital over the past fifteen years and has formed the basis for a series of reports (Kendall and Logue 1977, Aminoff and Logue 1974 a, Aminoff and Logue 1974 b, Symon *et al.* 1984).

Table 1. *Age and Sex in 60 Patients with Confirmed Spinal Arteriovenous Malformations* (from Symon, Kuyama and Kendall: J. Neurosurg. 60)

Sex	Location of lesion	0–9 yrs	10–19 yrs	20–29 yrs	30–39 yrs	40–49 yrs	50–59 yrs	60–69 yrs	70–79 yrs	Total
Female										
	dura					2	1	2	1	6
	spinal cord				1					1
Male	dura			1	1	7	19	19	2	49
	spinal cord	1	1				1	1		4

Age and Sex Incidence

In our recent series, which we believe to be the largest angiographically verified series of dural arteriovenous malformations, there were forty-nine males and six females. In contrast to other types of arteriovenous malformation of the spinal cord, the maximum incidence of these lesions is in the two decades from fifty to sixty-nine. The age and sex incidence is shown in Table 1.

Presenting Symptoms

Some form of pain was the presenting feature in 39% of these cases, sometimes a fairly general, non-specific complaint of back and buttock pain, sometimes specifically localized as back pain and less commonly actual radicular pain. In some cases pain would persist for several months before gradually progressive leg weakness or sensory disturbance the next most common presenting signs, were evident. Weakness of the legs occurred

as the first symptom in nearly 30% and sensory symptoms other than pain in 24%.

Another fairly frequent initial complaint was disturbance of micturition, defecation or sexual dysfunction usually taking the initial form of impotence.

These initial symptoms are in no way specific for dural arteriovenous malformations, and indeed they are similar to those of cord compression from any slowly progressive cause.

The Progression of Symptoms

In about 80% of cases the clinical history of a dural arteriovenous malformation is slowly progressive. After the initial complaint, the general picture is that of the development of a progressive myelopathy with a combination of motor, sensory (quite often posterior column) and sphincter disturbances. By the time of admission and diagnosis, some degree of leg weakness with pyramidal findings and a lower motor neurone disturbance was almost always present, being evident in 95% of our own cases, while sensory disturbance particularly in the buttocks and saddle area was evident in 90%. By this time almost a similar proportion showed disturbance of micturition or defecation and almost a third of the patients had disturbance of sexual function. Some form of pain persisted in 90% of the cases, again either back, root or remote pain. A fairly typical history is appended below, and the features pointing to the diagnosis are the slow progression of a mixed upper and lower motor neurone deficit, appreciable buttock wasting with saddle sensory loss, and some sphincter disturbance, in a middle aged male. Such a picture should prompt the differential diagnosis of a dural arteriovenous malformation.

It is worthy of note that in all cases recorded in the large series reported by Symon *et al.* (1984), symptoms were clearly referable to the lumbar enlargement despite the fact that the site of the AVM varied from the foramen magnum to the sacral hiatus. It seems likely therefore, that the maximum dilatation of the coronal venous plexus affects the plentiful posterior coronal plexus in the region of the cauda equina wherever the high input venous leak may be, and that posture may have some part to play in the determination of this (Jellinger and Neumayer 1972).

The mixture of upper and lower motor neurone features is often demonstrated by the presence of increased tendon reflexes in the legs despite appreciable buttock or ankle weakness. Thus, thirty-one of thirty-five cases in our own series showed some degree of muscle wasting, usually in the buttocks or calves, a feature which must be fairly carefully looked for in relation to the habitus of the body as a whole since elderly people frequently show some relative atrophy of the buttocks and lower limbs. However, this

is frequently quite a striking sign; an elderly well-covered individual, with well-built shoulders will show quite striking wasting of the buttocks and this, associated with a typical sensory loss, should be a clinical clue.

Increase in tone in the lower limbs was present in thirty-one of our fifty-five cases and decrease in tone in thirteen. There was thus the potential for a mixture of upper and lower motor neurone signs, loss of tendon reflexes being present only where weakness had become fairly advanced. Tone was thought to be normal in only eleven cases. Plantar responses were generally extensor where tone in the lower limb was increased.

Sensory change of some description was almost invariable. Some detectable band of superficial sensory disturbance was present in every case except two. This was frequently a small band of hyperaesthesia in the region of one segment, often lower to mid-dorsal or in the sacral and buttock area. Joint position sense was impaired or lost in all save ten cases and vibration sense lost at the toes in all save five.

Findings are those, therefore, of a lesion which could partly be attributed to the conus, but which partly is clearly spinal cord above the level of the conus. This mixed medullary and conal picture is almost pathognomonic of dural arteriovenous malformation.

Site

The site of the shunt is characteristic. Only four of the cases in our series of fifty-five showed a fistula outside the area T 3 to L 3, only eight were lower than L 1 and only seven higher than T 6. The vast majority, therefore, lie in the lower thoracic or upper lumbar spine. The great majority verified by selective angiography drained by a single vein into the coronal plexus. Thus, of the fifty cases verified by selective angiography, twenty-nine showed a single draining vein communicating with the coronal plexus. In a further eight cases two communicating veins were evident and in the remainder, the shunt having been identified, it was not possible to attribute exactly the number of communicating veins, and exploration was carried out on the site of the shunt only.

Intramedullary Arteriovenous Malformations

Houdart and Djindjian have described four types of intramedullary AVM based on operative and angiographic studies (Djindjian 1976, Hurth *et al.* 1978, Houdart *et al.* 1978). They discussed the surgical approach they felt most appropriate for each type and presented their results with radical surgery.

In order to rationalize the operative techniques described below the clinical data and results in those patients with intramedullary AVMs operated upon in Zürich are briefly documented. Whilst it must be

acknowledged that some might, today, have been treated by embolization, others certainly could not and the surgical principles involved in their radical excision are similar in each case.

Clinical Data

Between 1967 and 1983, seventy cases of intradural spinal AVM (and two of epidural AVM) have been operated upon using microtechniques by MGY at the University Hospital, Zürich. Forty-one (58.6%) had a significant intramedullary component (10 = 100%, 23 = 40%–90%, 8 = 20%). Nineteen of the forty-one patients (46%) had lesions in the cervical region and eighteen (44%) were thoracolumbar.

Table 2

Age	Number of cases
1–10	2
11–15	5
16–20	3
21–30	12
31–40	9
41–50	8
51–60	1
61–70	1

Most previous series have recorded a higher incidence of extramedullary ("retromedullary", dural, or epidural) AVM (Wyburn-Mason 1943, Houdart and Djindjian 1969, Aminoff *et al.* 1974, Pia 1978, Symon *et al.* 1984). Our present figures more closely resemble those of Hurth, Houdart and Djindjian (1978) and probably reflect an early interest in these two centers in the microsurgical treatment of such lesions.

In their series of 150 cases Hurth *et al.* (1978) demonstrated 23% cervical, 25% dorsal, and 52% thoracolumbar intramedullary lesions. Of the "entirely" intramedullary lesions in the present series (24%) half were in the cervical and half in the thoracolumbar region (at the conus) and the rarity of extramedullary forms in the cervical region has been noted before (Yaşargil 1976).

The age distribution of these forty-one cases at operation is shown in Table 2. There were twenty-five (61%) male and sixteen female patients. Eight (20%) were children under sixteen years (five male) and thirty-three were adults (20 male).

Back and limb pains, weakness of one or more limbs, sensory disturbances, meningism, bladder, bowel, and potency difficulties are common to all types of spinal AVM but some features perhaps more typical of intramedullary lesions were noted in the present series.

The early symptoms were invariably those due to intramedullary or subarachnoid haemorrhage with or without neurological deficit (24 cases), pain (13), and/or progressive weakness/numbness of one or more limbs (12). Eight patients presented with pain as their overriding symptom.

Although the duration of symptoms (mean 7.6 years) before diagnosis may reflect more the medical facilities available at the time, the age of onset was characteristically lower than that for the extradural AVM. 70% of our patients were symptomatic before the age of 40 years and 34% had their first symptoms in childhood. We are still largely ignorant of the extension and growth of these lesions with time but many intramedullary cervical and conus AVMs are found to have varices and aneurysmal dilatations.

The striking feature of the clinical presentation in this group of patients is that of the extraordinarily high incidence of subarachnoid (SAH) or intramedullary haemorrhage (IMH) frequently associated with a worsening of the clinical condition. 76% of patients had a bleed at some stage of their clinical course with 24% having severe associated neurological deficits. The haemorrhage was the first symptom of the disease in 77% of those who bled and in half the children. These figures are much higher than those usually quoted for extramedullary lesions (10–30%; Aminoff *et al.* 1974, Djindjian *et al.* 1970) and higher than the 49% quoted for intramedullary lesions by Houdart *et al.* (1978). Intraspinal haemorrhage was most common in those patients with cervical lesions (58%) although many cases with aneurysmal dilatations or varicoceles within the conus had also bled.

The term intraspinal haemorrhage (ISH) is used to cover both SAH (confirmed by lumbar puncture in 40% of cases) and IMH—or a combination of the two. Intramedullary haemorrhage, with stretching of the pia accounting for pain and meningism, may have been the cause of acute symptoms compatible with SAH in those cases in whom the CSF was clear. SAH rather than IMH is probably commoner in those cases with extramedullary/intramedullary lesions whereas the reverse might be true for the entirely intramedullary AVMs.

At operation yellowing of the cord, arachnoiditis or staining of the arachnoid was found in almost every case in which bleeding was thought to have occurred. Only one case was found which could be lexened to acute transverse necrotizing myelitis (Foix-Alajouanine syndrome).

This high incidence of ISH in the intramedullary AVMs may be partly due to the lesions being higher pressure systems than their purely extramedullary counterparts, particularly the long dorsal AVMs (Malis 1982). They are also frequently associated with thin-walled aneurysmal

dilatations and varices within the cord and have feeding vessels from both dorsally and ventrally derived arteries. Several of the large varicose/aneurysmal AVMs in this series found at the conus may be more properly regarded as the juvenile type (Di Chiro *et al.* 1971) which have a known tendency to recurrent haemorrhage.

Clinical Deterioration

Although a clear history was not always obtainable it seemed that neurological deficit was stepwise following ISH in at least 40% of cases. Houdart *et al.* (1978) noted a relapsing course in 80% of their patients. The average interval between onset of symptoms and surgery in the present series was 7.6 years emphasizing the need for earlier diagnosis. Common misdiagnoses included syringomyelia, peripheral neuropathy, intermittent claudication, idiopathic sclerosis, multiple sclerosis, disc disease, and idiopathic SAH. Myelography is not always helpful (in this series it demonstrated combined AVMs in 72%, and 60% of intramedullary AVMs were shown as cord swelling). More refined CT scanning may lead to quicker diagnosis and earlier treatment of these lesions thereby avoiding neurological deterioration and occasional death from the massive bleeds which they sometimes produce.

There are several theories as to why spinal AVMs cause neurological deterioration. Ischaemic cord changes due to compression by the AVM bulk, pulsatile water-hammer effects of dilated vessels, thrombosis within the AVM bulk, arachnoiditis and increase in venous pressure have all been put forward (Malis 1982).

For the extramedullary AVM, neurological deterioration seems most likely to be linked with increased venous pressure (Aminoff *et al.* 1974, Kendall and Logue 1977, Symon *et al.* 1984). This may be partly true for the combined intra/extramedullary AVMs (Hurth *et al.* 1978) although steal from the anterior spinal artery and thrombosis within varicose dilatations may also be important.

Hurth *et al.* (1978) and Houdart *et al.* (1978) related deterioration to the vertical level of intramedullary AVMs within the cord finding midthoracic lesions to have the poorest prognosis. This may be due to the more tenuous blood supply to the cord in this region and the tendency they noted of lesions here to develop intramedullary pouches. Intra/extramedullary lesions at the conus were thought to exert mass effects and more ventrally placed AVMs to be associated with acute deterioration due to spasm of the anterior spinal artery. Intramedullary AVMs in the cervical region were felt to carry the most favourable prognosis as they generally showed only a slow progression of neurological signs. This has not been found in our cases in whom there was a high incidence of intraspinal haemorrhage (58%) and associated acute deterioration in the patients with cervical AVMs.

Sphincter Disturbance

Twenty-two patients (54%) had evidence of difficulty with urinary sphincter control (10 cervical, 1 thoracic, 11 thoracolumbar).

Table 3

Grade	Description	Number of patients
0	No symptoms other than pain	4
I	Minor sensory symptoms distal to the AVM	4
II	Sensory symptoms and Pyramidal signs No functional disability	7 (3)
III	Moderate sensory and Motor symptoms/signs Independent/working	5 (2)
IV	Severe sensory and Motor disturbance Gross functional disability	14 (11)
V	Total loss of sensation distal to AVM Para/tetra/triplegic	7 (6)

( ) denotes sphincter disturbance.

Previous Surgery

A total of 10 cases had been previously operated upon for their neurological symptoms either in Zürich or elsewhere. The operations described were:

Ligation of feeding vessels	3
Laminectomy for presumed disk prolapse	1
Laminectomy for presumed tumor	1
Laminectomy for AVM, few feeders eliminated	1
Laminectomy for AVM—no attempt at excision	4

One patient received radiation therapy before transfer to Zürich. There were no cases of preoperative partial transvascular occlusion.

In order to obtain a reasonably accurate objective assessment of outcome following surgery it has been found useful to employ a grading system of clinical condition similar to that devised for cases of SAH from ruptured intracranial aneurysm. The classification used for the spinal AVM patients and the numbers of patients in each subgroup are shown in Table 3.

Investigation

CSF Investigations

CSF sampling of the cases seen in Zürich was incomplete. Certainly, not every case of suspected subarachnoid haemorrhage was verified by lumbar puncture. In some cases of acute deterioration in which intramedullary haemorrhage may have occurred there were no changes in the CSF. Plain spinal films were generally unremarkable in our cases but Hurth *et al.* (1978) have described the value of plain X-rays in cases shown to have intramedullary AVMs.

Myelography

The classical investigation which led to the diagnosis of spinal arteriovenous malformations in most instances until quite recently was myelography (Fig. 5). The abnormal mass of sinuous, turgid, pial veins in the posterior cord surface, typically below the mid-thoracic region was easily identifiable myelographically, and as Wyburn-Mason (1943) pointed out, this type made up 75% of the hundred and ten cases in his report of spinal AVM, what was not recognized at this time was that the myelographic demonstration of dilated veins was purely an epiphenomenon.

In the Zürich series Myelography, carried out as an initial investigation demonstrated the presence of abnormal vessels in sixteen cases (72%) and in the "entirely" intramedullary lesions showed cord swelling or a block in six. Normal myelography cannot exclude the presence of an intramedullary AVM and myelographic block may be well removed from the site of the intramedullary component in mixed lesions.

Spinal Angiography

The breakthrough in the investigation of this type of spinal angioma undoubtedly arose with the investigations of Di Chiro and his colleagues in the National Insitute of Health in the early sixties. Doppman's classical publication in 1971 summarized the concepts arising from the work in the N.I.H. over the preceding years as a result of spinal angiography and the philosophy was further amplified in the publication of Kendall and Logue in 1977. It is now clear that the only satisfactory way to diagnose and effectively anatomize these lesions is by selective spinal angiography, and series have been reported from France, Great Britain, and the United States in which this investigation has led to appropriate localization of the fistula for treatment.

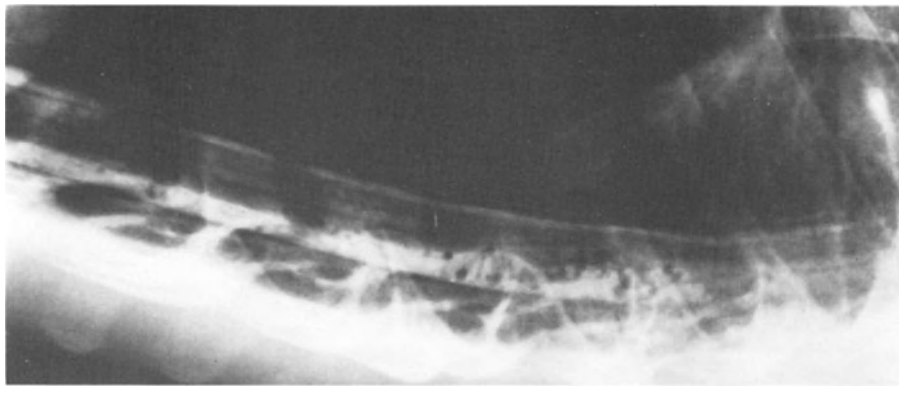


Fig. 5. Supine myelogram in a case of midthoracic arteriovenous malformation. The contrast (Iothexyl) lies posteriorly and the shadows of a group of parthologically distended veins are visibly behind the actual cord shadow

Selective spinal angiography is a technique requiring skill and practice. It requires the detailed catheterization of first the thoracic (Fig. 6) and lumbar arteries on each side and then if the picture is clinically suggestive and no lesion has been found, it may be necessary to extend the investigation to the vertebral arteries (Fig. 7), the remainder of the spinal radicular vessels, and the sacral branches of the internal iliac vessels. Only when the entire potential dura in which such an arteriovenous malformation has been discovered has been opacified and shown to be free can the diagnosis be dismissed. In 82% of a recent series the shunt was projected lateral to the spinal cord and in 30% of these instances the nidus encroached into the intervertebral foramen. It is however, evident that in a number of instances, the opacification of a radicular branch from more than one intercostal artery will result in opacification of the fistula, since the anterior radiculomedullary artery at more than one level may send a branch down along the dura to join the fistula in a neighbouring intervertebral foramen or close by. It has even been recorded for a vessel to cross behind or in front of the theca to join the fistula from the opposite side though this is excessively rare, having been seen in only one of our most recent cases.

Most recently Doppman *et al.* (1969), pioneers in the field, have suggested that intra-arterial digital subtraction angiography may permit more accurate localization of the precise area over which selective spinal arteriography is required. This thus limits the longitudinal extent of the selective catheterizations and vastly simplifies the procedure. In their view however, opacification of the fistula itself was not clear on DSA and the study alone did not allow one to distinguish a spinal dural arteriovenous fistula draining intradurally from a true spinal cord AVM. Selective spinal angiography remains therefore, the ultimate investigation. It should be

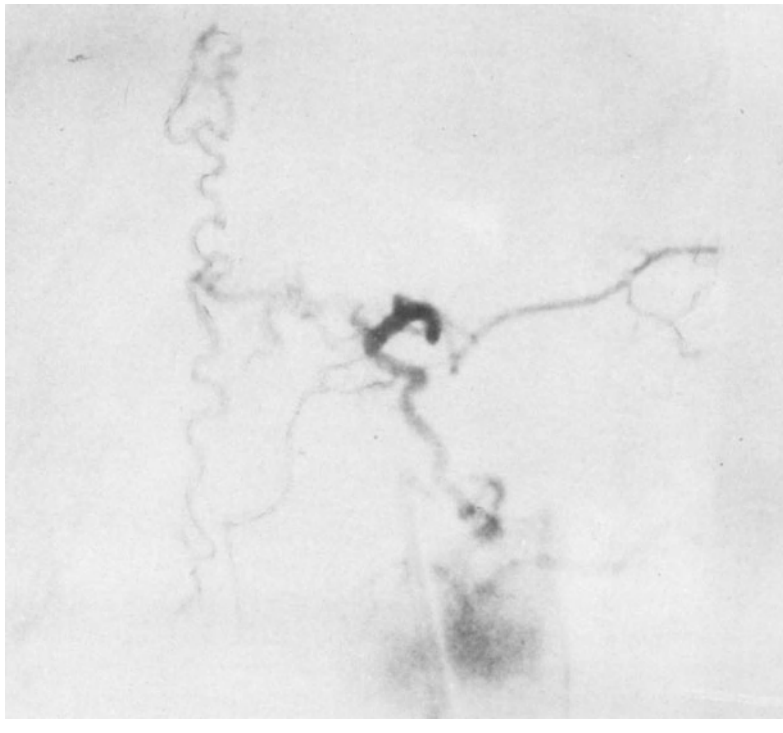


Fig. 6. Left six intercostal angiogram showing an arteriovenous malformation (arrowed) situated laterally in the spinal canal in the left T 6–7 vertebral foramen. The arterial supply is from two small branches of the posterior division of the intercostal artery, the drainage by a single vein into the coronal venous plexus (from Symon, Kuyama and Kendall: J. Neurosurg. *60*, 1984 with permission)

performed under general anaesthetic and one of the less toxic iodine compounds used such as Conray. While in the past the procedure has been associated with some risk, deterioration and even paraplegia having followed the selective angiography, with less toxic contrast materials these complications are fortunately now virtually unknown. Spinal jactitations which have been recorded from time to time following selective injection are best suppressed with Diazepam, but again, are much less frequent than heretofore.

The value of CT scanning (as an adjunct to surgery, rather than in confirming the initial diagnosis) has so far been rather disappointing in our cases. Scans have not correlated well with operative findings in respect of failing to demonstrate associated cysts and haematomas within the cord. CT scan may prove useful diagnostically in those cases in whom a spinal AVM is suspected but myelography is normal.

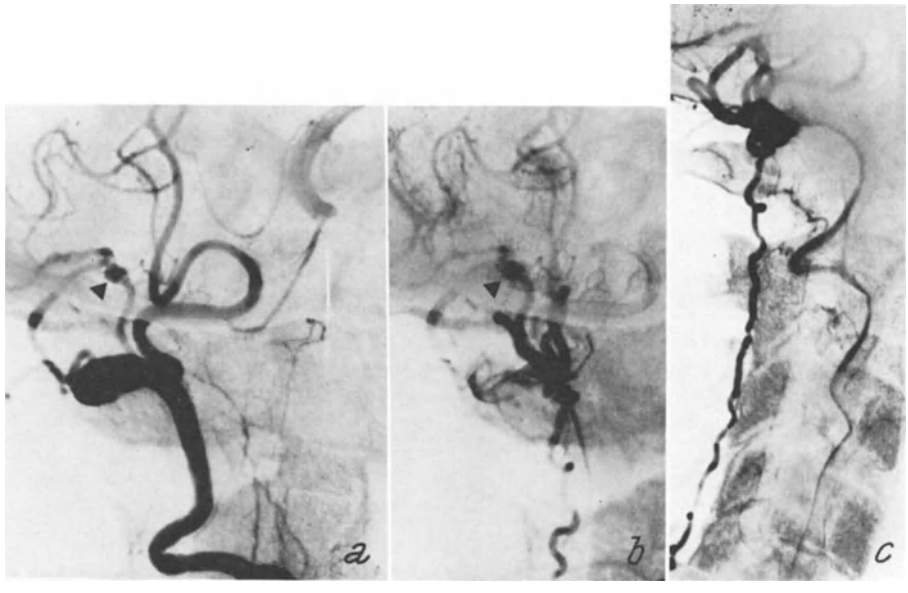


Fig. 7. Right vertebral angiograms, lateral projection showing a dural arteriovenous malformation on the right side of the foramen magnum (marker). The supply is from an enlarged posterior meningeal branch of the vertebral artery (a and b). The drainage through a single large vein into the posterior coronal plexus filling caudally (from Symon, Kuyama and Kendall: J. Neurosurg. *60*, 1984 with permission)

Treatment of Arteriovenous Malformations

The preferred treatment of any spinal AVM will be dictated by the location of the angioma itself, the size and source of the arterial supply, and the site of any AV shunt demonstrated on angiography. The choice of treatment lies between some form of transvascular occlusion and partial or radical surgical excision of the lesion or elimination of the shunt. Although several workers express concern as to its advisability (Lazorthes 1978, Pia 1978) and long term results will certainly need careful analysis, transvascular occlusion now seems set to provide a less traumatic and effective alternative to surgery in the treatment of increasing numbers of spinal AVMs. The attention of neurosurgeons must thereofre be directed more towards treatment of those lesions which are likely to remain particularly resistant to obliteration by such means. This group will include those AVMs fed not only by a few large afferent vessels but also by many much smaller arteries which cannot be demonstrated readily by angiography and which could be expected to hypertrophy if only the main feeding vessels are embolized. It will also include a number of intramedullary AVMs especially

those found in the upper cervical region. Such lesions may seem daunting from the surgical point of view, but the generally progressive downhill clinical course of intramedullary spinal angiomas does not justify undue procrastination in their treatment.

The Treatment of Dural Arteriovenous Malformations

Di Chiro and his associates reported in 1971 that their selective spinal cord arteriography had demonstrated 80% of spinal AVMs to be located on the dorsal cord surface. Most occupied an entirely extramedullary position. In our own series of fifty-nine verified arteriovenous malformations of the spinal cord, fifty-five were dural AVM, and only four true intermedullary or what we would regard as juvenile AVMs. These have been respectively classified by Oldfield as juvenile or glomus lesions.

It is thus of no more than historical interest to detail the variety of procedures which have been employed in an attempt to treat these conditions since all of them save the logical one of the disconnection of the fistula from the coronal plexus of the spinal cord, are clearly fruitless. Initial attempts confined to laminectomy and decompression such as we employed early in our own series were quite useless and in no way modified the progress of the condition.

Shephard was one of the first to point out that a very long laminectomy and excision of the coronal plexus over its entire dilated course could in some instances be followed by amelioration of the patient's symptoms. This of course was due to accidental disconnection of the coronal plexus with division of the afferent feeding vein at some point in the course of a long and tedious excision. This procedure however, was certainly not without its difficulties; it was tedious, inaccurate, associated with a high morbidity, and indeed sometimes promptly followed by paraplegia. The reason of course is not far to seek, disconnection of the coronal plexus from its radial draining vein depended entirely on the cord having a sufficient intrinsic collateral supply through the cord to convey all the blood to the anterior plexus, and as Gillilan (1970) has pointed out, anastomoses within the cord are notoriously fickle and unreliable. The more effective and radical the surgery therefore, the more likely the patient was to suffer damage.

Embolization

The two methods favored for the management of these lesions at the present time are either obliteration of the fistula by selective embolization (Merland *et al.* 1980) or excision coagulation of the fistula associated with division of the communicating vein (Symon *et al.* 1984, Oldfield *et al.* 1983).

Merland and his associates (1980) have made a strong case for the arteriovenous malformation being in fact a fistula with a single point of

communication between an artery and vein, the coiled vasculature in the epidural space being simply dilated veins, and this coiled nidus giving rise to a single vessel communicating with the cord. If the arterial supply can be accurately identified and if one of its vessels is sufficiently large to carry a single or multiple emboli direct to the fistula then it is possible to occlude the fistula completely by a nonoperative technique. Regrettably, a proportion of these cases have more than one small artery leading to the same fistula or arteriovenous malformation, and it may be extremely difficult to embolize more than the feeding artery so that the fistula or arteriovenous malformation itself remains unblocked. Merland and his associates have suggested that the injection of isobutyl cyanoacrylate or some such mass will be the most effective method of achieving embolization since this is likely to filter more effectively to the fistula site than will particulate embolic matter. Such liquid setting masses however, carry the considerable disadvantages that they may spread rather further than one wishes, and one of nine patients treated by this method in a recent report developed paraplegia. The proponents of this technique regard it as unsuitable for cases in which the artery or arteries of supply to the fistula also contribute blood supply to the spinal cord. In common with any of the other embolizing techniques involving isobutyl cyanoacrylate, there are considerable hazards in the use of the material and it should not be employed except by those with considerable experience in its management.

Direct Surgical Management

Direct surgical separation of the coronal venous plexus from the fistula has seemed to us and others (Symon *et al.* 1984, Oldfield *et al.* 1983) the simplest and most effective method of treatment.

Surgical Technique

The patient is prepared for anaesthesia with appropriate pre-medication and in our clinic is covered with Flucloxacillin in a dose of 250 mg six hourly for twenty-four hours before and three days after operation. Steroids are not routinely employed but if the patient is already showing considerable signs of cord damage Dexamethasone in the standard dose (4 mg, six hourly) should be started forty-eight hours before operation and continued for five days afterwards in diminishing doses.

The most important pre-operative requisite is accurate spinal marking. This should be performed by the neuroradiologist at the time of opacification of the fistula. The fistula is commonly in the region of an intervertebral foramen, and the most satisfactory marker is undoubtedly a metallic marker such as a solid needle, sunk into the spinous process or head of the appropriate rib. The more accurate the marking the less extensive need be

the laminectomy and ideally hemilaminectomy of 1½ vertebrae is all that is required.

The patient is positioned supine on the operating table, care being taken to ensure that the abdomen is free, to diminish venous congestion, and a small four inch incision centered on the site of the arteriovenous fistula marked out. With experience a unilateral clearance of the muscles from two adjacent spines and laminae will be sufficient, with the removal of 1½ laminae using an instrument such as the Hall drill to enable the laminectomy to be carried well laterally towards the intervertebral foramen. The larger part of the laminectomy should be performed above the fistula since the direction of the feeding vein is almost invariably upward. If it is not of course, the procedure may be modified. The small channel in the bone having been thus prepared, the nerve root at the appropriate region will be identified, and the dura opened just off the mid-line under the operating microscope. If the arachnoid can be preserved, this will enable inspection of the situation through the closed arachnoid the dural edges having been held back, without complication of floods of CSF. It will usually be possible to demonstrate the large vein emerging from the dura either just above or just below the nerve root in question and passing to the coronal venous plexus. It has been our usual practice thereafter to open the arachnoid, doubly clip and excise the feeding vein, and then to look both inside and outside the dura. In some instances we have endeavoured to circumscribe the dura and excise the whole area of abnormality, in others we have simply coagulated a knot of abnormal blood vessels visible either on the inner aspect or on the outer aspect of the dura. The abnormality on the inner aspect of the dura is frequently no more than a discoloration, while on the outer aspect of the dura the identification of the tortuous veins in the presence of the usual clutter of epidural veins may not be as easy as one might imagine. We have had no evidence that coagulation of the dura in these circumstances and division of the feeding vein has ever been associated with recurrence, but a recent report from Oldfield and his colleagues (1983) would indicate that such recurrence is possible. They reported a case in which selective spinal cord arteriography in 1966 had demonstrated a feeding vessel from an intercostal foramen which was exposed and clamped intradurally. There was virtually complete neurological recovery which remained stable for thirteen years when the typical progressive disturbance, mixed motor sensory and bladder recurred and the patient was re-investigated. Re-exploration followed re-investigation, and the previous clip was found 2 cm from the dural entry point of the vessel, the vessel itself having split into numerous other small vessels to join the coronal plexus and bypass the previously placed clip. Considerable neurological improvement followed gradually after the division of this vessel and the excision of abnormal vessels in the intervertebral foramen.

It appears however that provided the communicating vessel is divided at its point of entry and a segment of it excised, such recurrences must be rare. In our own series with a follow-up of over five years a number of instances of sacral AVM where the original nidus itself has not been approached, have been successfully dealt with simply by division of a large communicating vein ascending on the roots of the cauda equina. Time alone will tell whether the excision of a segment of this vein alone will be sufficient to prevent further recurrence, or whether it will be necessary in such cases to approach the frequently obscurely placed sacral AVM itself to ensure permanent cure. One of our further cases with a small arteriovenous fistula at the margin of the foramen magnum was treated by foramen magnum exploration and coagulation of the appropriate fistula which fed directly from the vertebral artery by a small communication, where excision was scarcely practicable. The feeding vein was once more excised from the point of exit from the fistula over a segment of one inch in length.

Treatment of Intramedullary Arteriovenous Malformations

Embolization

Thoracic and Thoracolumbar AVMs

Angiomas in these regions may derive a blood supply from dorsal or both dorsal and ventral radicular arteries, from dorsal segmental vessels but with a principal supply from the arteriy of Adamkiewicz, or from both dorsal and ventral vessels plus a contribution from the iliosacral vessels. This holds true for both extramedullary and intramedullary lesions and superselective angiography is therefore mandatory properly to demonstrate vascular anatomy, and to assess suitability for embolization or direct surgery.

Lazorthes (1978) advises that embolization is dangerous and Pia (1978) feels that embolization of the anterior spinal system is contraindicated. Nevertheless, retromedullary thoracolumbar AVMs fed by one or more posterior spinal arteries are now increasingly treated by embolization alone. Intramedullary lesions may also be safely embolized if the feeders are from dorsal, dorsolateral, and ventrolateral branches of the radicular arteries with the possible exception of the artery of Adamkiewicz as this may be the chief source of supply to the normal cord.

When contemplating embolization (or ligation or temporary clipping) of the artery of Adamkiewicz, perfect selective angiography is necessary to estimate the size of the parent vessels, to ascertain whether or not the ascending and descending trunks are involved as feeding vessels, and to demonstrate possible collateral supplies to the normal cord structures.

The suitability of intramedullary AVMs supplied by the artery of Adamkiewicz for treatment by embolization may be evaluated by temporary balloon or coagulum occlusion (Riché *et al.* 1983). The applicability of permanent occlusion in such cases will be determined by the anatomy of the vascular supply and by the size of the feeding arteries. When the feeding arteries are of fine calibre it may not be possible to reach the nidus of the AVM by catheter, nor to be sure that balloon or coagulum occlusion will spare more proximal branches to the normal cord.

Intramedullary AVMs supplied by the anterior spinal artery plus branches of the posterior spinal artery may be treated by embolization of the latter prior to a direct surgical approach to the lesion, if temporary occlusion of the artery of Adamkiewicz produces an unfavorable result.

Cervical AVMs

Lesions of the upper cord and cervicomedullary junction may derive their blood supply from a number of sources—from anterior and posterior spinal arteries arising from the intracranial vertebral arteries and PICAs, from radicular branches arising from the extracranial vertebral arteries and by external carotid branches via the occipital artery. In the midcervical region the supply may also be derived from the intratransverse vertebral artery, the ascending cervical artery and the thyrocervical trunk. Lower cervical and upper thoracic lesions may be supplied directly from the costocervical trunk, from the deep cervical artery, from direct branches of the vertebral artery and its origin, and in rare instances, from branches of the subclavian artery. Even lower thoracic ventral radicular arteries can be very large and play the role of main feeding vessels. In one case described above the main feeder was from a T 6 segmental artery.

A particular difficulty in treatment may be presented by the cervical intramedullary AVM which is supplied, as is commonly the case, by an anterior spinal artery derived from both vertebral arteries. In this instance transvascular occlusion is not safe, and a direct surgical approach is to be preferred. Those AVMs supplied by branches of the deep cervical or thyrocervical vessels lend themselves much more favorably to embolization techniques.

Effective embolization requires complete elimination of the AV shunt. Occlusion of the feeding vessels may reduce flow sufficiently to allow thrombosis of the lesion but will be safe only if there is sufficient collateral spinal vascular supply to prevent cord damage by ischaemia. Hurth *et al.* (1978) reported 21 cases of intramedullary AVM treated by embolization of which only 10 could be completely eliminated without significant neurological sequelae. However, these cases did largely represent complex lesions not well suited to surgery. Embolization for intramedullary lesions is indicated

if the afferent feeding vessel is short, there are large sulcocommissural vessels, good collaterals for the anterior spinal artery above and below the AVM, and if the lesion has limited venous drainage. Riché *et al.* (1983) suggests that embolization is now the initial treatment of choice for most spinal AVMs with surgery being reserved for those cases in which it has failed. Transvascular occlusion may also increasingly be used to partially eliminate lesions prior to surgery.

Surgical Treatment

The distribution of AVMs within the cord has been elegantly illustrated in terms of vascular supply, particularly in relation to the anterior spinal artery, by Djindjian (1976). He described three basic types of intramedullary lesion with several subdivisions. One form was that of the ventrally placed lesion which should be excised by posterior midline myelotomy only if the lesion did not extend anteriorly beyond the cord and was not adherent to the anterior spinal vessels. A second type often showed dorsal or posterior intramedullary components with the main mass lying anteriorly within the cord—the indications for operation being similar to the first group except that in a particularly diffusely scattered variant, surgery would also be contraindicated. The third group were mixed extra- and intramedullary lesions usually lying laterally and almost always amenable to surgery by dissection along vessels entering the cord substance. Djindjian suggested that surgery is best reserved for those cases in which the intramedullary AVM is focal, midline, supplied by long sulcocommissural arteries, partly ectatic, shorter than two vertebral levels and having only minor elements of dorsal venous drainage. This approach was confirmed in the later publication (1978) of Houdart *et al.* Pia (1978) commented on the hazards of subtotal excision of intramedullary AVMs and of operation on ventral lesions.

Observations in our own patients suggested a simplified plan (probably oversimplified) of anatomical distribution of AVMs within the cord and of their vascular supply which is of importance to the surgeon in planning his approach. The mixed lesions may be dorsal, dorsolateral, lateral or ventrolateral. Pure ventral lesions have not been seen (due to inability to mobilize the cord sufficiently for inspection) but probably exist. The arrangement of their vascular supply is shown diagrammatically in Figs. 8 a and b. The "pure" dorsal lesion has not been observed to receive a supply from the sulcocommissural artery. The "pure" intramedullary lesions were observed in the cervical region and the conus, but not in the thoracic region. Such angiomas are found to lie ventrocentrally and may even bisect the cord almost completely (Figs. 9 a and b).

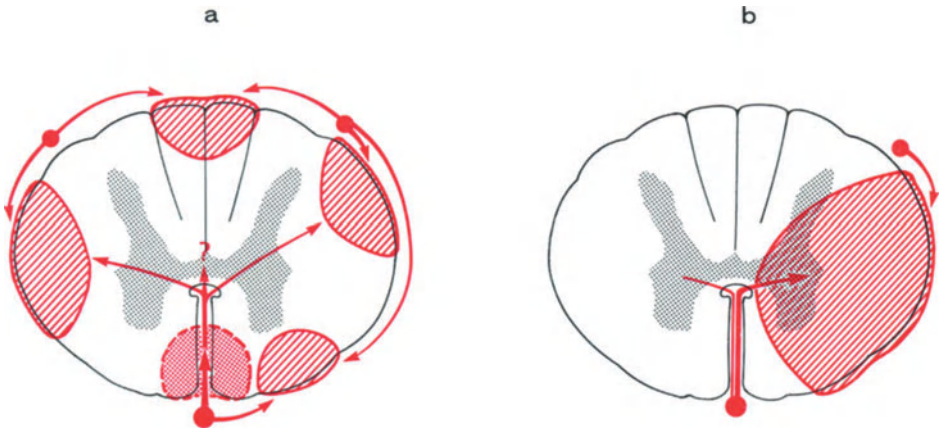


Fig. 8 a. Dorsal, dorsolateral, lateral, ventrolateral, and (?ventral) mixed AVMs and their potential blood supply from posterior and anterior spinal arteries. We have not seen a true dorsal lesion supplied by the anterior spinal artery

Fig. 8 b. Ventrolateral/lateral, extra/intramedullary AVM deriving arterial supply from dorsal segmental vessels and sulcocommissural artery (anterior spinal supply)

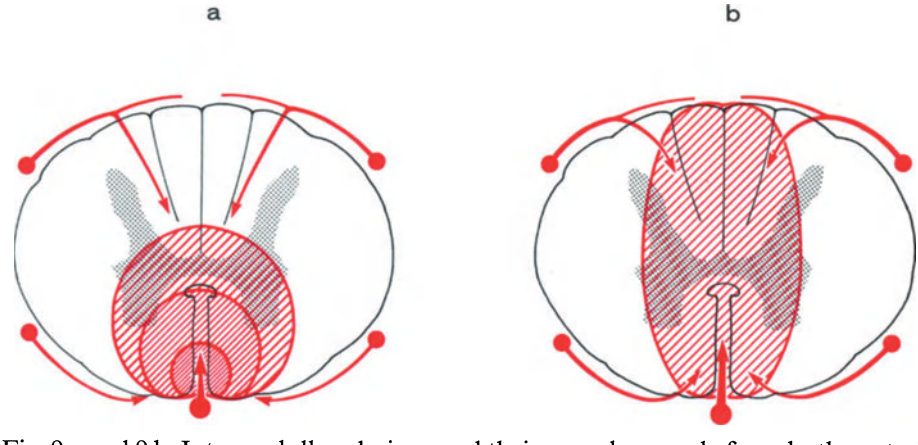


Fig. 9 a and 9 b. Intramedullary lesions and their vascular supply from both ventral and dorsal spinal arteries. Type 11 b virtually bisects the cord, is most commonly found at the conus and may be associated with extensive extramedullary vascular anomalies

Surgical Technique—General Principles of Radical Excision

Microsurgical techniques have proved invaluable in the operative treatment of all spinal AVMs. Under the operating microscope, using bipolar coagulation, small feeding arteries may be followed and divided close to their entrance into the malformation and normal spinal cord

arteries preserved. Arachnoid and pial cleavage planes can be followed and a precise excision of intramedullary components of the lesion carried out.

Operation is performed in the sitting position for cervical lesions extending caudally as far as the third thoracic level and in the prone position over a frame for thoracic and lumbar lesions when suitably arranged padding is used to prevent abdominal compression which could result in venous congestion. Patients operated upon in the sitting position are fixed in a Mayfield pinned headrest and have Doppler monitoring for detection of air embolism. Operations have normally taken three to four hours and it has not been found necessary to use supplementary heating. A midline skin incision extends the length of the suspected underlying malformation. Following retraction of the paraspinal muscles, the spinous processes are removed, and preserved in antiseptic solution. A groove is then cut along the lateral aspect of each lamina with a high-speed electric drill and the several laminae attached to each other by the ligamentum flavum are elevated as one piece and preserved for replacement at the end of the procedure.

A small slit is made in the dura with a microscalpel taking care to avoid opening the arachnoid. The dura is opened by gentle traction with two forceps. A small dissector is used to separate the dura from the arachnoid, dividing the delicate trabeculae running through the arachnoid to attach to the dura. The dura is retracted and fixed to the musles with clips or sutures. This provides maximum exposure, reduces bleeding from epidural veins and keeping the arachnoid intact prevents inadvertent damage to the AVM. Inspection of the surface AVM at this point will allow extension of the laminectomy if necessary. The retromedullary portion of an extra/intramedullary AVM can be seen to lie in the subarachnoid space. The arachnoid is next opened by incising it just off the midline with fine microcissors and undercutting the perpendicularly directed dorsal medial septum. Care must be exercised here as the septum is not always running in a straight line but may be vermicular owing to distortion by the abnormal vessels themselves. The arachnoid may then be elevated from the lesion and the cord retracted and fixed to the dura with microclips. When dealing with AVMs which are predominantly extramedullary (lying on the dorsum of the cord) this will leave the lesion largely exposed on the pia and greatly facilitates dissection. In areas where the arachnoid is very adherent to the lesion no attempt is made to separate it initially.

From the surgical and pathological point of view, the spinal AVM is a subarachnoid lesion with variable extensions into the spinal cord. Uncommonly the AVM is supplied by one artery which when clipped allows the entire malformation to collapse. More often the dilated vessels are coiled on the dorsal surface of the cord and are fixed near every first or second nerve root by feeding arteries from the supplying radicular arteries. Radicular

arteries supply both normal vessels to the spinal cord and feeding vessels to the malformation. One may be a branch of the other. It is therefore difficult to predict the course of a small artery when first seen in the subarachnoid space.

Combined Extramedullary/Intramedullary Lesions

The main radicular feeding arteries will generally be found laterally on the spinal cord and frequently run longitudinally for some distance before suddenly turning into the malformation. These feeding arteries are usually slightly larger in calibre than normal spinal cord arteries and are somewhat redder in color because of their thinner walls. However, a definite distinction cannot be made and it is important to follow an artery until it joins the malformation to avoid interrupting normal blood supply. Temporary clips may be used to assess the contribution of an artery to the malformation. Dissection of the arachnoid round these vessels should be carried out using fine pointed microscissors, avoiding blunt dissection which may rupture small communicating vessels to the normal cord. The feeding vessel should be carefully dissected from the adjacent nerve root then coagulated using fine bipolar forceps at a low setting and with copious irrigation. Section of the dentate ligaments may facilitate examination of ventral feeders, small vessels in the dentate ligaments may require coagulation first.

If all feeding arteries have been clipped, the malformation will collapse and turn blue and dissection of the malformation from the spinal cord may begin. Almost invariably, some feeding arteries will be beneath any intramedullary extension and will not be accessible until some dissection of the lesion has been performed. The surface component is removed by dissecting free its small arachnoid attachments to the pia using microscissors. When the intramedullary component is small and superficial it is easy to follow the extramedullary vessels into the cord although pial incision and wider exposure may be needed for all but the simplest lesions. Dissection is kept close to the malformation which is separated from the surrounding gliotic cord tissue by gently spreading the tips of the bipolar forceps alongside the pathological vessel and using a small bore sucker at minimal setting. Small arterial and venous attachments to the malformation are coagulated and divided as they are encountered. It is important to coagulate these thin-walled vessels over 3 or 4 mm before half dividing, recoagulating and fully dividing them to prevent their retraction and rebleeding. Vessels larger than 0.2–0.3 mm diameter require vascular clips on the proximal side. If there is doubt regarding identification of a vessel as either a draining vein or feeder a temporary clip applied to the vessel may help.

When the glomus of vessels has been excised such that no more feeding vessels are entering it and the draining veins are blue and collapsed, a clip is placed across the veins and the lesion excised. Any small bleeding points are controlled by bipolar coagulation. It will not be necessary to resect the draining veins over their entire course as long as all connections with the malformation have been severed.

Intramedullary AVMs

Malformations which lie within the substance of the spinal cord are invariably at least partially supplied by feeding arteries from the anterior spinal artery and frequently demonstrate aneurysmal dilatations or varices. The malformation does not usually include normally functioning nervous tissue within the glomus of pathological vessels and an apparently ventrally derived arachnoid extension separates the pathological vessels from the surrounding gliotic cord. Consequently a dissection plane can be developed between the lesion and the remaining cord that allows preservation of function despite complete excision of the malformation. Maintaining this plane and isolating the feeding arteries close to the lesion are the critical operative requirements. Dorsal and ventral radicular feeders should first be identified and the dorsal elements eliminated. Sectioning the dentate ligaments may help identify and prepare ventral feeders but the cord is often swollen and cannot be rotated. When in doubt regarding the course of a ventral "feeding" vessel it should be left until proper identification can be made.

If the malformation is entirely intramedullary or is ventrally placed, ventral feeding vessels will be more safely eliminated after dissection of the AVM within the cord. The pia is carefully incised with a round-bladed tenotome in the posterior midline or slightly to one side if this area is obviously thinned. It is gently separated by spreading apart with two sponges rather like opening the segments of an orange. Frequently, large convoluted pathological vessels lie over the midline of the cord making incision at this point difficult. It will normally be possible to open a plane just under/marginally lateral to such vessels by displacing them gently from the midline but occasionally they may have to be coagulated and divided to facilitate dissection. Dissection in a vertical plane is continued using two small sponges held in position one at the sucker tip and the other in the bipolar forceps. At this stage cutting and coagulation are kept to a minimum to avoid damage to normal cord tissue. The plane is developed by gentle spreading rather like a cerebral sulcus.

The pia is now gently sutured to the laterally fixed dura on each side. This will minimize manipulation of the cord and help to develop the plane of dissection and exposure still further.

When an element of the AVM is present at the surface it may be followed toward the bulk of the intramedullary component. Otherwise, dissection continues in the midline until the most superficial part of the lesion is reached.

Then, keeping in the plane between the AVM and normal cord tissue the dissection is developed dorsolaterally to both left and right. This, like the remainder of the dissection is carried out using gentle compression of the AVM with a fine sucker tip over a wet cottonoid sponge whilst the plane is developed by gentle spreading with the fine bipolar forceps. There is no place, particularly when operating on cervical lesions, for fixed retraction or for an assistant using hand-held retractors.

Retrograde dissection of the AVM after coagulation and dividing a large draining vein (Malis 1982) can only be performed with safety if one can demonstrate by temporary occlusion that there are other large venous channels still patent.

Bleeding from ruptured AVM varices is usually controlled by applying a small piece of muscle and holding it in place with gentle suction over a sponge. Intramedullary AVMs are generally high pressure systems and aneurysmal dilatations are frequently encountered. Ventral feeders often run into the lesion at such points. Dissection may be facilitated, and underlying vessels eliminated with less cord dissection, by carefully shrinking the aneurysm sac with bipolar coagulation. Bleeding from rupture of the aneurysm may sometimes by controlled by gentle pressure but more often by temporary clipping of the sac or main feeding vessels. On occasion, the aneurysm may have to be opened (after applying clips to the main feeding vessels close to the AVM) and ventrally placed feeders identified and eliminated as they enter the aneurysm.

The dissection is advanced dorsolaterally and ventrolaterally then carried to the cranial and caudal poles to identify and prepare feeding vessels at these points and to isolate the poles. It may be necessary to coagulate and divide occasional lateral feeding vessels to the AVM. By keeping strictly within the subarachnoid plane, dissection and identification of the feeding vessels is facilitated at any level within the cord or medulla oblongata.

When the lesion is centrally located the main feeders together with the principal draining veins will be found ventrally. Although the major vessels from which these arise must be demonstrated, dissected and prepared for temporary clipping, it is important to avoid application of temporary clips to them except in absolute emergencies.

In cases in which the AVM lies eccentrically to either right or left it is normally found that there is a much greater input from feeders derived from radicular arteries. It is often possible for these feeding arteries to be embolized preoperatively thereby greatly facilitating surgical removal of the

lesion. When this is not possible, such branches must be meticulously dissected, coagulated and divided—sometimes without knowing the overall picture of the arterial supply. Excessive, prolonged coagulation of any vessel leads to sticking of the bipolar forceps and tearing. An artery which is inadequately coagulated before dividing or is torn and then retracts into the cord substance can be difficult to relocate and to seal and this leads to unnecessary manipulation of the cord.

High cervical lesions are frequently supplied in part by branches from the vertebral artery coming off distal to the anterior spinal artery. In such cases early elimination of these feeders after temporary clipping may make the remaining dissection easier.

Following haemostasis, the pial sutures are divided and the cut pial edges gently brought together and sutured with 8/0 nylon. The arachnoid is then closed either by microsuture or by using gentle intermittent bipolar coagulation at several points along the exposure. The dura is closed with a continuous watertight nylon suture and drill holes made in the bony margins and in the removed laminae so that the laminae may be held in their original position by means of 2/0 silk sutures. If gaps between laminae and margin remain they may be filled in with bone chips from the spinous processes. The soft tissues are closed in layers with interrupted sutures and the wound drained with a low-suction drain if it extends more than two or three segments.

With intramedullary AVMs total surgical excision, by means of elimination of all arterial feeders, provides the best chance of cure. However, caution must be exercised in over-zealous clipping of ventral arterial feeders—particularly if their relationship to the anterior spinal artery cannot be clearly demonstrated. This is especially true of those cases in which feeding vessels (not well seen angiographically) run cranially from deep among the roots of the cauda equina to an AVM in the conus. Safe dissection of these vessels is often difficult and incautious clipping may be disastrous.

Results of Treatment

Dural Arteriovenous Malformations

In common with most disturbances of CNS function, the results to be expected even after uncomplicated surgical management are determined by the degree of disability which has been allowed to develop before treatment commences. It can however, be confidently stated that these lesions can be treated without increasing the neurological deficit. We have used the assessment of functional capacity described by Aminoff and Logue (1974 b) in the following grades:

Grade I — Disturbance of gait, occurrence of leg weakness, abnormal stance or gait, no restriction of activity
Grade II — Restricted activity
Grade III — Requires one stick or some other support for walking
Grade IV — Requires crutches or two sticks for walking
Grade V — Unable to stand, confined to bed or wheelchair

Disturbances of micturition were classified by them as follows:

Grade I — Hesitancy, urgency, or frequency
Grade II — Occasional urinary incontinence or retention
Grade III — Total urinary incontinence or persistant retention

In fifty-five cases only seven showed deterioration after surgery and these were early in the series when the surgeon found it impossible to restrain himself from some excision of the coronal plexus in association with division of the feeding vein. In 65% of thirty-one severely disabled patients and in 80% of fifteen moderately disabled patients there was appreciable improvement of gait. Improvement of bladder function was even more striking, several patients returning from episodic or even total urinary incontinence to normal. There were no deaths in this series.

Intramedullary and Mixed Arteriovenous Malformations

A summary of the findings at operation in terms of the site, size, and description of the intramedullary component of each lesion is given in Table 4.

The mean length of hospital stay was 33 days and the mean follow-up time was 2.9 years. Two patients were lost to follow-up and one patient underwent only exploratory surgery in Zürich and later had further angiography and definitive surgery in Paris. Postoperatively, one of the two patients lost to follow-up had marginally deteriorated (having developed a mild weakness of a lower limb) and the other had remained unchanged.

Eleven lesions (27%) were incompletely removed as judged at the time of surgery (2 cervical, 1 thoracic, 8 thoracolumbar). Three cases of suspected/known incomplete removal were confirmed angiographically, two of them having the remnant apparently successfully dealt with by embolization or a second open procedure elsewhere without further neurological deficit. The third patient refused reoperation despite a deteriorating neurological condition.

One patient with a conus AVM (juvenile type) in Grade V was operated upon solely to relieve pain and to prevent further subarachnoid haemorrhage. The lesion was found totally unresectable. Another patient explored for a spinal tumor (without angiography) and found to have an AVM had a rare blood group of which only a limited quantity was available and radical surgery was not attempted. Radical excision was not felt

Table 4

Case no	Morphology of principal intramedullary component and extent of AVM	Size of i/med component as % of whole AVM
1	Olive sized nodule; partially thrombosed, T 7[9]	90%
2	Massive displacement with penetration of cord mainly at T 10–L 1 by leash of vessels	30–40%
3	Large varix C 1–5	60%
4	2 cm lesion at T 11–12 totally within cord, remainder extramedullary, T 10–L 3	30–40%
5	Angioma C 2–T 1 "entirely" intramedullary	100%
6	Nodule C 1–2; massive feeding vessels C 2–3	80%
7	Intramedullary vessels T 10–L 2; extramedullary vessels T 7/8	60%
8	"Totally" intramedullary C 1–3 AVM	100%
9	C 3–T 4 intramedullary vessels; anterior spinal artery aneurysm (6 mm)	80%
10	T 11–12; exploration discontinued because of blood loss	?
11	T 9–L 4 with two large varices over 3 cm diam	69%
12	Midline, predominately intramedullary T 3–4; aneurysm, small portion ventral	90%
13	Ventral; small part intramedullary T 9–L 1	20%
14	Large intramedullary haematoma (1.5 × 1 cm) intramedullary AVM C 4–5	100%
15	C 3–T 1 part intramedullary	40%
16	Mainly intramedullary at C 3–T 1 more on left; three nodules extramedullary	60%
17	A few pathological vessels on surface but remainder entirely intramed C 3–4	100%
18	Intramed aneurysm above conus T 8–L 1 with convoluted branches; large varix	50%
19	Massive compression by varices, partly intramedullary; T 9–L 1	20%
20	2 × 1 cm ventrally placed nodule C 4–6 intramed; single nodule extramed on left	90%
21	Massive intra- and extramedullary convulsions, C 2–T 1	60%
22	"Entirely" intramed lesions C 4–7	100%
23	Intramedullary penetration of otherwise extramedullary AVM	20–40%
24	Predominantly intramed T 6–7	80%
25	Intramedullary haematoma running caudally for 25 cm; AVM C 5–7 (intramed)	60%

Table 4 (continued)

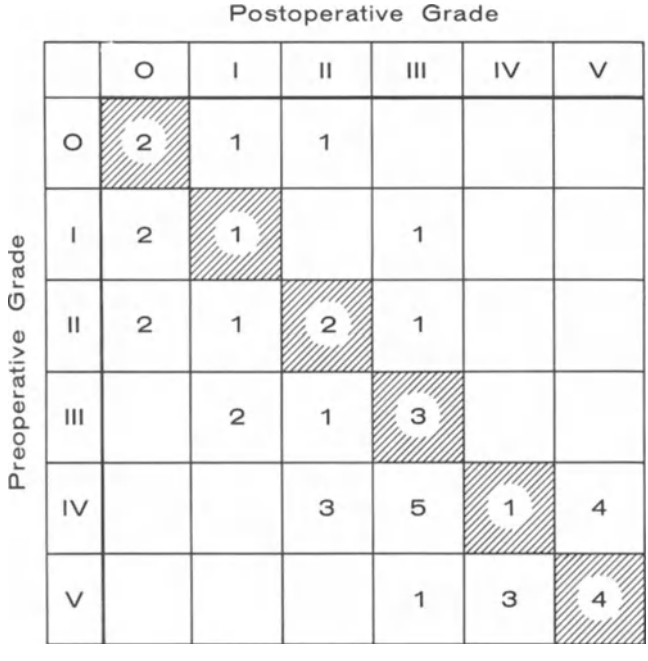
Case no	Morphology of principal intramedullary component and extent of AVM	Size of i/med component as % of whole AVM
26	"Totally" intramed; AVM at C 2	100%
27	Nodule 3 × 1.5 cm laterally on left with intramed haematoma and AVM at C 2–3	20–30%
28	Predominantly lateral with intramedullary penetration by mainly aneurysmal dilatations at C 2–3	20–30%
29	Calamus scriptorius—C 3; mainly intramed 3 × 2 × 2 cm	90%
30	C 3–T 5 retromedullary; large vascular knots with ventral feeders at C 7, T 1, and T 3	60%
31	Three large varices in conus and cauda equina with massively dilated feeders T 10, T 12, L 3	100%
32	Whole cord replaced by AVM T 8–L 2	100%
33	Angioma predominantly at T 11/12; bilobular aneurysm arising from T 12 feeder with dense adhesions to cauda equina T 8–L 3	80%
34	Dorsal extension into cord with large varix in conus/cauda equina T 11–L 1	90–100%
35	Dorsolateral extension into cord T 8–L 1	40%
36	Two elements T 8–9, T 12–L 1	40%
37	Dorsal element with intramedullary varix in conus; massive extramedullary component also T 9–L 2	80%
38	Large intramedullary component at conus with 2 varices T 9–12	100%
39	Mainly intramed; coiled varicose vessels T 8–11	100%
40	Dorsal and ventrolateral; feeders plunging into cord T 2–7	50%
41	Feeding vessels passing into cord	30%

appropriate in one patient with no clinical signs and a large ventrally placed intramedullary lesion at the conus. Most of the patients in whom complete removal could not be achieved had large ventral feeding vessels found at surgery but not demonstrated on preoperative angiography. These could not be adequately dissected from other branches of the anterior spinal artery without undue risk to the arterial supply of the normal cord.

Recurrent subarachnoid haemorrhage occurred in two patients after operation, both known to have had incomplete removal of their AVM. Ten patients (77%) of the thirteen who had severe preoperative pain were

Table 5. *Graded Outcome Following Surgery*

Numbers to the left of hatched area = cases improved after operation (20)
Numbers to the right of hatched area = cases deteriorating after operation (8)
Numbers in hatched area = cases unchanged neurologically by operation (13)



relieved of this after operation although three patients with no preoperative pain developed chronic pain in the postoperative period. Of the twenty-two patients with preoperative sphincter disturbances, six (27%) were improved after surgery and this appeared relatively independent of their motor/sensory grade. Only one patient previously continent of urine was rendered incontinent after operation.

Changes in overall clinical grade after operation are shown in Table 5. Altogether, twenty patients (48.4%) were improved by operation, thirteen (31.7%) remained unchanged, and eight (19.5%) deteriorated after surgery. Fifteen of those who improved had cervical lesions (15/19), two had thoracic lesions (2/4), but only three/seventeen patients with thoracolumbar lesions benefitted neurologically. Eight of these patients with thoracolumbar lesions were in very poor initial grades (two being totally paraplegic).

There were two deaths among the forty-one patients. One patient, initially Grade 5 with a tetraplagia from a C 5/6 AVM and intramedullary haematoma made a modest improvement after radical surgery but died two

years later from a urinary tract infection and septicemia. A second patient developed a bacterial meningitis after complete removal of a C 1–3 intramedullary AVM (see Yaşargil 1976) and died two weeks after operation.

Of the thirty patients (73%) whose lesions were thought to have been totally removed, eighteen (60%) showed an improvement in their postoperative neurological state, seven (23.3%) were unchanged, and five (16.6%) deteriorated.

Again, the greatest number of improvements was seen in the group with cervical AVMs (14/18) and those patients with mild or moderate preoperative deficits. Only one patient has shown a delayed deterioration (10 years after operation) which might be attributable to residual angioma. However, he had originally presented in Grade 5, had made only a modest improvement in the interim, and has not had further spinal angiography. Despite the lack of angiographic evidence, it would appear that in effect, true radical surgical excision has been obtained in each of those cases in whom it was judged to be complete at operation. These patients should thus have no further risk of deterioration or death from intraspinal haemorrhages.

Excluding those patients in whom it would not have been possible to demonstrate neurological deterioration postoperatively if it were present, and the patient in whom no excision was performed for lack of available blood, it was possible to obtain complete removal of the AVM in twenty-three out of thirty-seven (62.5%) cases with improvement or at least no deterioration in their clinical state.

Osteoplastic laminectomy was carried out in 8 cases and the numbers are too small to be able to comment on the postoperative occurrence or prevention of angular deformities of the spine at the operation site.

The experience of operating upon the forty-one cases described and a comparison with existing literature emphasizes several specific points regarding the management of intramedullary AVMs.

There is an absolute need for a sound knowledge of the normal vascular supply to the spinal cord and for perfect superselective spinal angiography before attempting operative intervention. Even with this facility, however, at operation one almost invariably finds and must be prepared to deal with sources of arterial supply not demonstrated angiographically. The simplified relationship of the spinal AVM to its vascular supply shown in Fig. 1 and 2 has been adopted in preference to that depicted by Djindjian because in our own hands the relationship to the anterior spinal artery was properly demonstrated only at surgery despite very high quality preoperative angiography. Our operative findings do, however, correspond closely with those of Houdart *et al.* (1978) although the extra/intramedullary lesions were not found to predominate on the anterolateral aspect of the cord.

The intramedullary lesions are frequently associated with aneurysmal and varicose swellings and fistulae between the anterior spinal artery and venous system. The varices are occasionally thrombosed but more often are patent and being under considerable pressure will bleed vigorously if ruptured.

Dorsolateral, ventrolateral, and lateral lesions with intramedullary components have probably been approached incorrectly on several occasions in this series. It may well be preferable to risk immediate myelotomy and intramedullary dissection in these cases to eliminate feeders from the anterior spinal artery and to see if one can spare ventral radicular branches which might otherwise be taken early in the dissection. Certainly, better results have been obtained in those several cases in which intramedullary dissection was carried out initially when no ventral radicular input could initially be demonstrated. The operative approaches found most appropriate in this series have otherwise been similar to those described by Houdart *et al.* (1978) using dorsal commissurotomy to explore the ventrocentral AVMs and subarachnoid dissection alongside the abnormal vessels in the mixed group.

In some cases, the ventral radicular branches which were participating in the AVM were altogether too substantial to eliminate. If a much earlier diagnosis could be made when the lesion was, perhaps, much smaller one might be able to excise the AVM more readily. This could involve operation in a patient with a ventral or ventrocentral AVM an minimal symptoms which Pia (1978) feels is inappropriate.

Embolization may prove to have a place in the palliation or inhibition of growth of lesions in such cases. However, we would agree with the view that even in these borderline cases, their natural history is such that early radical microsurgery is the treatment of choice (Houdart *et al.* 1978) but feel also that this concept should be extended to included the cervical intramedullary lesions.

Late surgery for patients with fixed, severe neurological deficits (mainly conus lesions in this series) is rarely beneficialy in terms of neurological recovery but can eliminate the risk of life threatening recurrent haemorrhage and prevent pain.

Pia (1978) has commented on the dangers of subtotal removal in intramedullary angiomas but suggests it may be preferable in late cases with mixed lesions. 12% of patients in this series (all but one with mixed conus lesions) appear to have gained long term improvement in their symptoms and freedom from recurrent haemorrhage after subtotal removal.

Summary

The operative experience in Zürich of forty-one cases of spinal AVM with major intramedullary components showed that it was possible, with

the aid of precise microsurgical techniques, to remove completely 60% of these lesions with improvement, or, at least, without deterioration in neurological condition. A further 12% could be apparently effectively palliated by subtotal removal. Radical surgery may be justified in patients with irreversible neurological deficits to treat pain and to prevent fatal SAH. The best results have generally been obtained in patients with less severe neurological deficits and with lesions in the cervical region rather than the thoracolumbar region. The natural history of intramedullary spinal AVMs—that of deterioration after recurrent haemorrhage—is analogous to that of intracranial aneurysms—and the need for earlier diagnosis and for early preventive surgery is the same for both.

It would, perhaps, be preferable to treat all cases of spinal AVM by transvascular occlusion to obviate the risk of open surgery and of spinal deformity, but some AVMs will remain impossible to treat by this means and the long term results of embolization still require full analysis before it can be accepted as definitive treatment. Comprehensive and exact super-selective spinal angiography is a mandatory prerequisite to surgery and preoperative partial embolization may facilitate operation considerably in the future. However, even the most careful angiographic studies do not always totally define the lesion and the surgeon must be prepared to find unexpected vascular relationships at operation. A simple classification of intramedullary and mixed extra/intramedullary lesions is described.

The experiences with dural arteriovenous malformations in Queen Square again show that the best results are obtained in patients who have mild or moderate neurological deficit preoperatively. There is no doubt that progressive neurological deficits finally become irreversible and it is therefore clear that once the diagnosis is suspected, it should be definitively established and operation should follow immediately. The prime, indeed the only, necessary investigation is selective spinal angiography, which demands a high degree of radiological skill and experience, but given these prerequisites, may be performed with little hazard. While embolization of these lesions is possible, the simple surgical disconnection of the nidus of the shunt from the coronal venous plexus is effective in most cases, apparently permanently, and is substantially without risk.

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Tumors of the Lateral Ventricles

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With 38 Figures

Contents

Introduction

Since the earliest anatomical descriptions of intra-ventricular tumors, neurosurgeons have shown a high degree of interest in the management of these neoplasms, perhaps because these tumors grow freely in an existing cavity with minimal damage to the underlying brain. As a result, they usually show symptoms related to ventricular enlargement rather than to compression of the adjacent brain. Their surgical removal is facilitated by their particular situation and by the fact that they often present as benign encapsulated tumors.

Only tumors arising within the lateral ventricles and growing inside the ventricles (primary ventricular tumors) are here considered; tumors which arise from brain tissue and involve the ventricular cavities secondarily (secondary ventricular tumors), are excluded.

With the advent of newer diagnostic procedures, intra ventricular tumors may be disclosed at a time when they are still small and produce insignificant clinical manifestations.

1. Etiology—Anatomy

Primary tumors of the lateral ventricles may arise from the septum pellucidum, the walls of the ventricle, or from the choroid plexus. They may be located in any part of the ventricles: frontal horn, foramen of Monro, body, atrium, occipital horn, temporal horn.

Many types of lateral ventricular tumors have been described in the literature. It is possible to assess for each type of tumor the incidence of ventricular localization compared to all intracranial cases, or of the lateral ventricle site compared to the entire ventricular system. However, it seems almost impossible to obtain a clear idea of the incidence of any given type of intraventricular tumor.

Meningiomas, papillomas, ependymomas, sub-ependymomas are the most frequently reported tumors[28,56]. Reports of other histological types, are scattered through the literature.

Meningiomas

The incidence of lateral ventricle meningiomas is variously reported in the literature, between 0.5% to 2% of all intracranial meningiomas, at least in adults[28,40,56,94,110]. However, the incidence appears much greater in children and young patients (15–17% according to Vassilouthis[110], and 30% according to Janisch[56], although Delandsheer[28] found the highest incidence (27%) between 30 and 40 years of age. Regardless the age group, females are more commonly affected than males, by a ratio of 2 : 1[28,94]. For some unexplained reason, intraventricular meningiomas occur more frequently in the left ventricle than in the right one (about 58–60%[28,76,94]). Most of these meningiomas arise from the posterior part of the ventricle (Atrium, Trigone).

According to Mani[76], intraventricular meningiomas are thought to arise from the arachnoid tissue, carried with the choroid plexus as the ventricular system invaginates[64]. Others think that intraventricular meningiomas are derived directly from the stroma of the choroid plexus, which arises from the same cell layers as the meninges[114]. Fornari *et al.*[40] emphasized that in their 18 cases, there is nothing to support or refute the distinction of Cushing and Eisenhardt between "true plexus meningiomas" and "lateral meningiomas of the velum". This differentiation was accepted by Delandsheer[27] but has been criticized by others. The blood supply may come from both posterior and anterior choroidal arteries; the lateral posterior choroidal arteries are the main source of blood supply to the body and posterior horn of the lateral ventricle. Usually the macroscopic aspect of the tumor is the same[27]. The meningioma is "encapsulated", of a red-grey colour, with a few vessels on its surface, with an ovoid shape corresponding to the ventricular cavity; in several cases, the choroid plexus may lie on the surface of the

tumor, which is thus separated in two lobes. The meningioma is of hard texture and may reach considerable size, isolating a ventricular horn (most frequently the occipital or the temporal horn).

Papillomas

The incidence of choroid plexus papillomas is generally agreed to be less than 0.5% of all intracranial tumors[106]. However, the incidence is much higher in children. Amongst the different figures reported, we found 3.9% of all tumors in children under 12 years of age [79], 20% of papillomas occur in infancy and 48% in the first decade of life[106]. The tumor is reported more frequently in males (65%, Janisch[56]) than in females.

Concerning the location within the ventricular system, the decreasing order of incidence for choroid plexus papillomas[16] is the fourth ventricle, the lateral ventricle and lastly the third ventricle. However, the favoured site appears to be different in adults and in children. In adults, the fourth ventricle seems to be the preferred location and for children occurrence in the lateral ventricle is more common (43% of all choroid plexus papillomas of the new-born and infants[56]. Some have reported a higher incidence in the left lateral ventricle than in the right[16]. In 5.8% of cases bilateral tumors were found[56].

Histologically, choroid plexus papillomas arise from the choroid plexus epithelium and can be differentiated from choroid plexus meningioma, which arises from the stroma. They are frequently located posteriorly in the ventricle. Macroscopically, the tumor is a red grey mass possibly with cystic cavitation; the area of origin may be large or with a thin pedicle. They may be well delineated from the underlying parenchyma but in some cases may infiltrate underlying white matter[56].

Choroid plexus papillomas may bleed, producing intraventricular hemorrhage. Other tumors produce hydrocephalus. Several mechanism for production of hydrocephalus have been described in such cases. The tumor may produce a complete obstruction of the lateral ventricle at the foramen of Monro, with unilateral hydrocephalus[42]. Communicating hydrocephalus may be caused by over-production of cerebrospinal fluid associated with the papilloma. It has been well established that choroid plexus papillomas can secrete large amounts of fluid; this over-production of CSF reverts to normal after removal of the tumor[6].

Ependymomas

Ependymomas arise from the cells lining the ventricular system.

Supratentorial ependymomas are, however, most commonly found outside the ventricular system[63]. The incidence of this tumor inside the

lateral ventricles has been variously quoted as 6.5%[50], 17.5%[46], 18.6%[63], or 23.3%[7] of all intracranial ependymomas (Table 1).

Patients with supratentorial ependymomas seem to be older than those with infratentorial tumors, with an average age of 27.8 years compared to 18.7 years[39], or 18.8 years compared to 15.4 years[63]. No clear-cut sex difference has been noted.

According to Kernohan[61] ependymomas within the intracranial cavity may be found in the fourth, lateral, or third ventricle in that order of frequency. There is no favored site of predilection within the supratentorial ventricular system[16,63].

Table 1

	Lateral ventricle	Supra-tentorial	Intra-cranial	All locations together
Kricheff[63]	13	18	70	
Barone[7]	11		47	74
Fokes[39]	4	32		133
Hahn[50]	3	20	46	
Goutelle				
Fischer[46]	33	102	188	322

Macroscopically[61], those in the lateral ventricle may have a slightly different appearance from ependymomas within the fourth ventricle; they may be soft, friable, granular, and pink-grey, and partially or wholly within the ventricle or surrounding white matter. They are relative sharply delineated from surrounding non neoplastic tissue. Cysts may be present, containing xanthochromic or clear yellow fluid. Cyst formation is more common in supratentorial ependymomas than in ependymomas of the fourth ventricle.

Sub-Ependymomas

Sub-ependymoma is a rare, relatively benign tumor of the central nervous system, with an origin variously postulated as astrocytic, ependymal, or mixed[73]; these various components account for the assortment of names which are given to this tumor: sub-ependymal glioma, sub-ependymal glomerate astrocytoma, sub-ependymoma.

Actually, the majority of sub-ependymomas are small, asymptomatic, fourth ventricle tumors, found incidentally at autopsy, often in elderly men; most of them go unreported. However, they may also arise in the septum

pellucidum, the walls of the third and lateral ventricle, occasionnally associated with congenital malformations or other primary neoplasms of the central nervous system[73].

Scheithauer[96] gives a good account of this tumor. He reports a series of 95 cases, 48 from the literature and 47 additional personal cases. Of these 95 cases, 43 were asymptomatic. Additional cases have been reported[20, 73] since then. In this series, the mean age was 49 years, with 80% of males. 116 tumors were found in the 95 cases. Of these, 27% were supra-tentorial, 71% were infra-tentorial, and 2% located in the cervico-thoracic region. Of supra-tentorial tumors, 24 (21%) originated from the wall of the lateral ventricle, 6 (5%) from the septum pellucidum.

Tumors originating in the wall of the lateral ventricles greatly exceed tumors of the septum pellucidum in a ratio of 4 : 1. The foramen of Monro was frequently obstructed.

Scheithauer[96] pointed out that the presence of symptoms was directly correlated with the size of the tumor. Symptomatic neoplasms averaged between 4 to 5 cm in greatest dimension, while asymptomatic neoplasms averaged 0.8 cm in their greatest dimension.

Unlike the well described asymptomatic incidental tumors, large sub-ependymomas may have several sites of secondary attachment to the ventricular wall, and more frequently demonstrate a soft consistency, cyst formation, focal calcification, and hemorrhage. Growth into the ventricular lumen and sharp demarcation from the underlying brain are characteristic of all sub-ependymomas. Some tumors of the lateral ventricle may be pedunculated and attached to the ventricular wall by a narrow vascular pedicle[73]. In addition to mass symptoms, sub-ependymomas of the lateral ventricle may bleed, producing sub-arachnoid hemorrhage.

Sub-Ependymal Giant-Cell Astrocytomas

Sub-ependymal giant-cell astrocytoma is a rare cerebral glioma that occurs mainly in patients with tuberose sclerosis[113]. Exceptionally, cases may be reported in patients showing no evidence of this condition[49].

Cooper[22], reviewing previous reports of this tumor, accepted 33 cases and noted that in only one case reported since 1946 was tuberose sclerosis not recognized. Tuberose sclerosis is widely accepted as a hamartomatous disease, characterized by multiple focal tumor-like malformations of various organs, which are represented in the brain by cortical tubers and ependymal nodules. Sub-ependymal giant-cell astrocytomas occurring in cases of tuberose sclerosis have been considered to be either large examples of sub-ependymal nodules or neoplasms arising from these nodules. Most of these tumors are typically situated near the Foramen of Monro[113], obstructing the foramen and resulting in hydrocephalus; they usually grow

slowly, and malignant changes are exceptional. Hemorrhage coming from giant-cell astrocytomas seems to be exceptional[113].

Malignant Tumors of the Choroid Plexus

Carcinoma

Malignant choroid plexus papilloma, or choroid plexus carcinoma, is the malignant counterpart of choroid plexus papilloma; it is an extremely rare neoplasm[16,33,108]. Dohrmann and Collias[33] could find only 22 well documented examples. This tumor which is typically located in the lateral ventricles of young children, is generally associated with a very poor prognosis. It can metastasize widely in the neuraxis. Two additional cases have been reported by Valladares[108] and Carpenter[16].

Melanoma

One case of malignant melanoma of the choroid plexus epithelium was reported by Beatly[9]. This was an eight-year-old boy with a tumor arising from the choroid plexus of the atrium. In view of the embryology of the choroid plexus one would not be surprised to find pigment in the stroma of the choroid plexus. This patient however had pigment in the epithelium. Melanin, apparently has not been described in this location. The possibility that this tumor represented a metastasis was considered, but not confirmed at autopsy.

Miscellaneous

Oligodendroglioma

Oligodendrogliomas, usually found in the cerebral hemispheres, may invade the ventricles; however, more rarely sub-ependymal oligodendrogliomas, arising near the ventricles, may grow only in the ventricular cavities. These primary intraventricular oligodendrogliomas are rarely reported. Markwalder *et al.*[77], reporting two cases, found only 31 reported cases of the fourth, third, and lateral ventricles. Some additional cases were reported by Maiuri[75], Geuna[45], Page[86], Laine[65].

Xanthogranuloma

Choroid plexus xanthogranulomas are frequently encountered at post mortem examination. They are usually asymptomatic and very few cases have been reported with surgical intervention. Terao *et al.*[103] reported a case of a child with bilateral xanthogranuloma arising from both choroid plexuses. The tumor was described as an ovoid, smooth-surfaced tumor in the trigone, attached to the choroid glomus; small arteries and veins from

the choroid plexus supplied the mass; there was no adhesion and no invasion of the ventricular wall. According to these authors, the paucity of clinical report of choroid plexus xanthogranulomas is due to the fact that most of these tumors are too small to produce clinical symptoms.

Teratocarcinoma

This is a rare teratoid tumor. Marshall *et al.* [78] reporting 3 cases of children with teratocarcinoma of the lateral ventricle (arising from the wall), found less than 100 cases in the literature, only four of them occurring in the lateral ventricular system.

Hemangioma

Hemangiomas of the choroid plexus are uncommon. Towfichi *et al.* [105] reporting one case of bilateral choroid plexus hemangioma, found only 28 cases including their own. The ages of the patients varied from 2 days to 74 years; 61% occurred in the first two decades of life, and females had a slightly higher incidence (59%) than males. In all cases, except two, choroid plexus hemangiomas produced symptoms and were associated with intraventricular hemorrhage; they were bilateral in four instances.

Hemangioblastoma

Diehl and Symon[32] reported a case of hemangioblastoma arising from the choroid plexus of the lateral ventricle, in a patient with Von Hippel-Lindau disease, presenting with isolated temporal horn enlargement. Reviewing 62 cases of supra-tentorial hemangioblastomas in the literature, they were unable to find any other case of symptomatic intraventricular hemangioblastoma.

Epidermoid Tumor

In their general review of intracranial epidermoid tumors, Lepoire and Pertuiset[70] found 20 intraventricular cases out of a series of 100 patients. With additional series of the literature, they found an incidence of 15% (59 cases out of 341). The sites most frequently encountered were, in order, the temporal horn, the fourth ventricle, the trigone. Out of their 10 cases located in the lateral ventricles, 7 were in the temporal horn and 3 in the trigone. More recently Higashi *et al.*[53] described a case of epidermoid tumor located in the frontal horn.

Cyst of the Choroid Plexus

Symptomatic cysts of the choroid plexus have been rarely reported. Andreussi *et al.* [2] collected five cases in children including of their own.

Dempsey and Chandler[30] added one more adult case. Small cysts of the choroid plexus of small dimensions are found in all the cerebral ventricles, but rarely produce clinical symptoms. These cysts contain clear fluid with all the characteristics of cerebro-spinal fluid. Their wall has a structure similar to the choroid plexus; thus these cysts differ from colloid cysts; they are attached to the choroid plexus at only one point, by a narrow pedicle, so that they may float freely within the ventricle and intermittently obstruct the circulation of the ventricular fluid; they may also enlarge to the point of total obstruction.

Cysticercosis

Cysticercosis is the larval stage of taenia solium. Apuzzo *et al.*[3] reviewing a series of 45 cases of intraventricular cysticercosis found only 5 cases located in the lateral ventricle. They emphasize the potential for cyst migration due to postural changes during the pre-operative period. This migration may take place inside a given ventricle, or from one ventricular cavity to another.

Metastases

Metastases in the choroid plexuses are infrequently encountered. Janisch *et al.*[56] reported several cases in the lateral ventricle, coming from primary neoplasms of stomach, oesophagus, and breast. These metastatic tumors may be single or multiple, and associated with metastases in other locations. Isolated examples have been described in the wall of the lateral ventricles.

Various Other Types of Tumors

Other tumors reported infrequently in the literature include cryptococcal granuloma[82], dermoid tumor, hydatic cysts[4], spongioblastoma, neuroblastoma, reticulo-sarcoma, melanoblastoma, seeded pinealoma[67], astrocytoma[65], lipoma, chondroma[56].

2. Symptoms and Signs

Due to their development in non-evocative areas of the brain, tumors involving the lateral ventricles usually produce non-specific manifestations resulting either from obstructive hydrocephalus or from compression of the adjacent brain tissue. Acute presentations may also be encountered associated with intra-ventricular bleeding.

Clinical syndromes can be correlated grossly with the location of the mass within the ventricular lumen. On the other hand, no clear correlation between the clinical course and the pathological type or the size of tumor

can be discerned in many cases. A tiny mass occluding the Foramen of Monro may produce severe symptomatic intracranial hypertension, whereas a huge tumor in the trigone is likely to remain asymptomatic for a long period.

Presenting Symptoms

Headaches are the most common presenting symptom. Patients complain of progressive permanent headaches sometimes worse during exertion and during the night.

Paroxysmal headaches as an early symptom of intra-ventricular tumor were originally described by Bruns[14] in 1902. Since Dandy suggested the "ball valve" mechanism some authors have stressed the role of postural changes and head movements in producing the headache. Debruyne *et al.*[26] have recently described migraine-like paroxysmal headaches leading to early detection of intra-ventricular tumors.

Persistent vomiting and nausea may however be the only complaint, especially in childhood and infancy.

Progressive head enlargement is also common in the pediatric group. Infants frequently show widened sutures and bulging of the fontanelle.

Visual disturbances are usually associated with other symptoms of raised intra-cranial pressure. They include blurring of vision, visual field defects, and diplopia.

Various types of *seizures* may be observed: grand mal seizures, focal seizures or absence attacks. Branch and Dyken[13] reported a child with infantile spasm syndrome and EEG hypsarrhythmic pattern in whom the removal of a ventricular plexus papilloma was followed by clinical recovery.

Hemisyndromes are unusual and are proportionally more frequent in adults and in glial tumors than in childhood and in meningiomas or choroid plexus papillomas. Progressive hemiparesis, speech disturbance, hemianesthesia are most frequently observed in patients with tumors causing brain edema or involving the adjacent brain tissue. Hemisyndromes usually cause mild disability and may be disclosed only on a thorough physical examination.

Psychiatric disorders may mimic low-pressure hydrocephalus and include gait disturbances, abnormalities of behaviour and of micturition. These symptoms may be caused by tumorous involvement of the frontal lobes, by obstructive hydrocephalus or both.

A few patients show *memory loss, unsteady gait, vertigo*, and *endocrine disturbances* as the initial manifestations.

Acute Presentation

As pointed out in several reports, intra-ventricular tumors are seldom manifested by massive *intra-ventricular hemorrhage*[1,37,80,91].

Before the use of the CT scan, the origin of bleeding could be overlooked or identified only during surgical operation.

When ventriculography was routinely performed in patients with intra-ventricular tumors, ventricular tap often revealed that the cerebro-spinal fluid was slightly hemorrhagic or xanthochromic, indicative of repeated asymptomatic bleeding. Undoubtedly, this should be considered as an additional factor of hydrocephalus in such patients.

Patients with intra-ventricular tumors rarely show symptoms of *tentorial herniation* requiring emergency treatment.

Paraparesis and back pain due to spinal metastases of intra-ventricular glioma have also been reported[60].

Physical Findings

The physical examination if often unrewarding. Papilledema is the most common physical finding. Visual field defects, especially lateral hemianopia, may be noted. Asymmetry of reflexes, ataxia of gait, intellectual or mental changes may be apparent. Manifest stigmata of tuberose sclerosis or of neurofibromatosis are present in less than 5% of the patients.

Clinical Syndromes

They vary greatly according to the patient's age, the location, and to a lesser degree, the pathological type.

In infancy, macrocephaly is always present. Before the routine use of CT evaluation, head enlargement was often ascribed to a non-tumorous condition and led to ineffective and hazardous shunt surgery. *In childhood*, signs of raised intra-cranial pressure are predominant and can misleadingly suggest a posterior fossa tumor, especially when ataxia is associated. In this age range, persistent vomiting is sometimes initially mistaken for a systemic condition, such as a digestive or urinary tract infection. In the *adult*, intra-cranial hypertension may be absent. Seizures and hemisyndromes more commonly lead to admission. In the *elderly,* intra-ventricular tumors are likely to produce mental changes and bladder dysfunction. A diagnosis of low-pressure hydrocephalus may be initially considered in error.

Tumors originating from the *frontal horn* cause early obstruction of the Foramen of Monro. Their clinical presentation is typically that of acute intra-cranial hypertension including paroxysmal headaches, sometimes relieved by head movements. Masses involving the *septum pellucidum* and the anterior fornix may in a similar manner occlude both foramina of Monro. Memory impairment may also be encountered with tumors in this location. Patients with *trigonal tumors* often show a homonymous

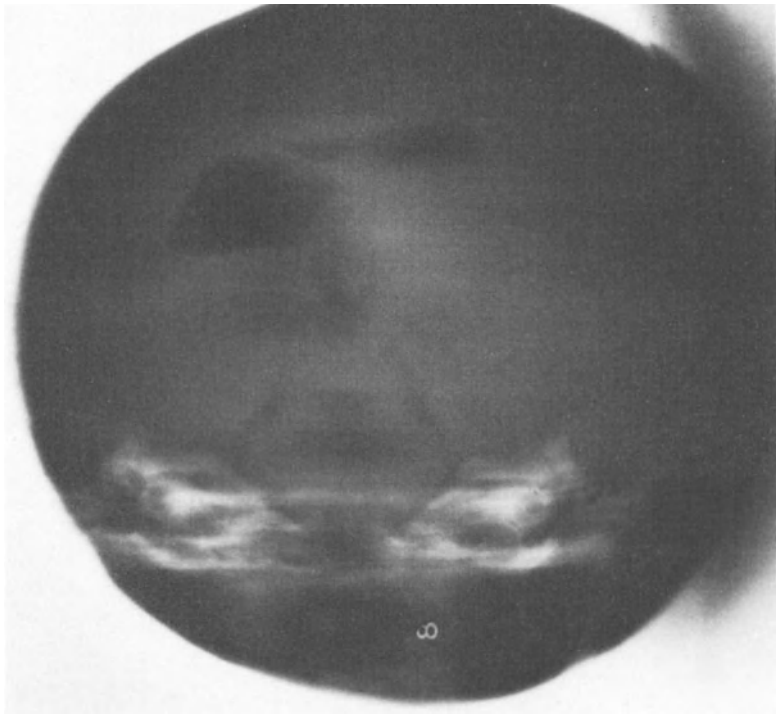


Fig. 1. Air encephalography, frontal tomogram showing sub-total obliteration of the left ventricle by a irregular mass. Pathology: grade I oligodendroglioma

hemianopia associated with papilledema. Tumors originating from the *temporal horn* usually show no difference from parenchymal tumors and produce temporal seizures, speech disturbances, and visual field defects.

Choroid plexus tumors are mainly found in pediatric patients, and their clinical presentation reflects the young age of the patients. It is generally assumed that choroid plexus papillomas are associated with communicating hydrocephalus due to an overproduction of cerebrospinal fluid[106, 16, 42. 35, 83, 6]. Head enlargement may be asymmetric[60]. The incidence of intra-ventricular hemorrhage seems higher in this pathological type.

In the *other pathological varieties,* the clinical manifestations are non-specific and are predominantly related to their site of development and the changes occurring within the parenchyma.

3. Radiographic Diagnosis
(Figs. 1 to 32)

Patients with intra-ventricular tumors are accurately evaluated by computed tomography (CT). There is no need to stress the value of this

procedure which has obviated the necessity for positive contrast or air ventriculography and for air encephalography, in most cases. However, angiographic evaluation is still widely performed to document preoperatively the arterial supply and venous drainage of tumors arising in the lateral ventricles.

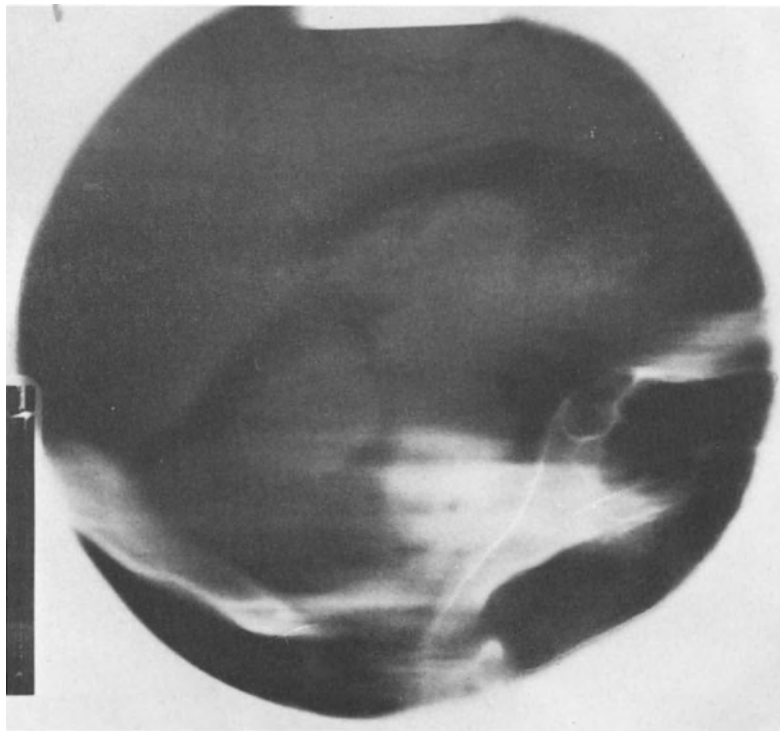


Fig. 2. Air encephalography, sagittal tomogram displaying a mass lying in the frontal horn. Pathology: grade I ependymoma

Plain Skull Films

Skull roentgenograms may be unremarkable in many cases. However, signs of raised intra-cranial pressure are common in infants and children, especially those with plexus papilloma: the macrocephaly may be asymmetric[60]. Opened sutures, cerebriform skull, and erosion of the sella turcica may also be noted. Intra-cranial calcification may occur in any type of tumor, but oligodendrogliomas and low-grade astrocytomas seem more likely to produce calcification than other neoplasms.

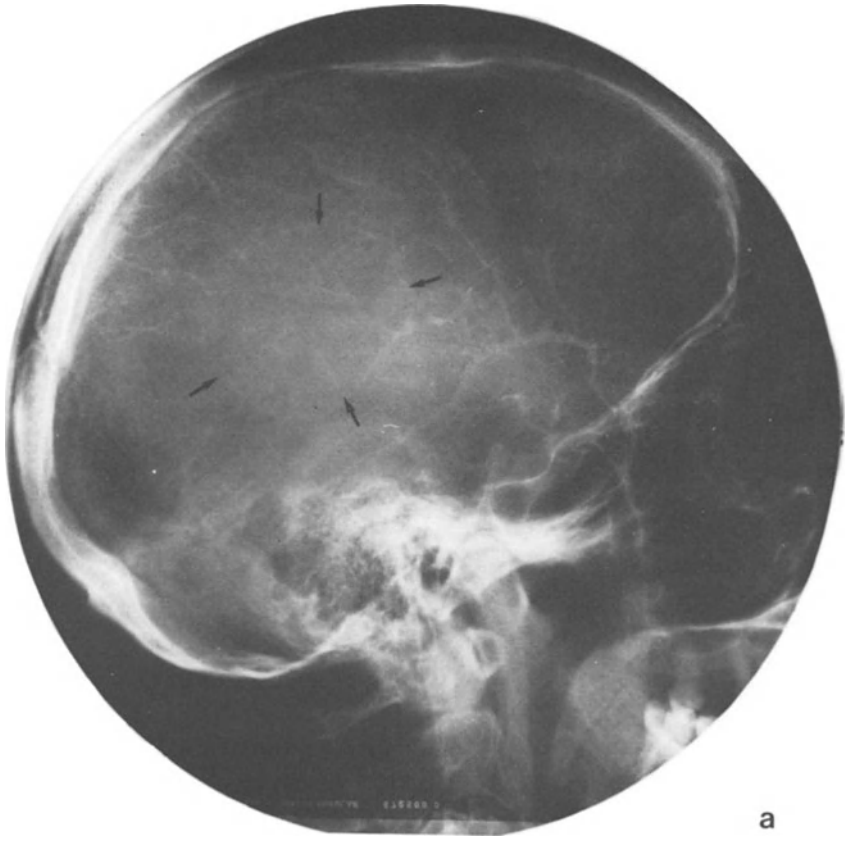


Fig. 3. Left trigonal meningioma. a) Carotid angiography, venous phase. Homogeneous capillary blush delineating the mass (arrows). b) Vertebral angiography: supply from the postero-lateral choroidal arteries (arrows). Note the curvated course of the postero-medial choroidal artery (arrowhead)

Positive Contrast and Air Ventriculography

These procedures have become obsolete since CT has been used routinely in the evaluation of these patients. They provided valuable information about the site and the delineation of the mass, as well as about the associated ventricular enlargement. Despite the significant risks of intra-ventricular bleeding, ventriculography was considered more reliable than air encephalography when papilledema was present.

Air Encephalography
(Figs. 1 and 2)

The advent of CT has significantly decreased the number of pneumoencephalograms performed for the diagnosis of intra-ventricular tumors.

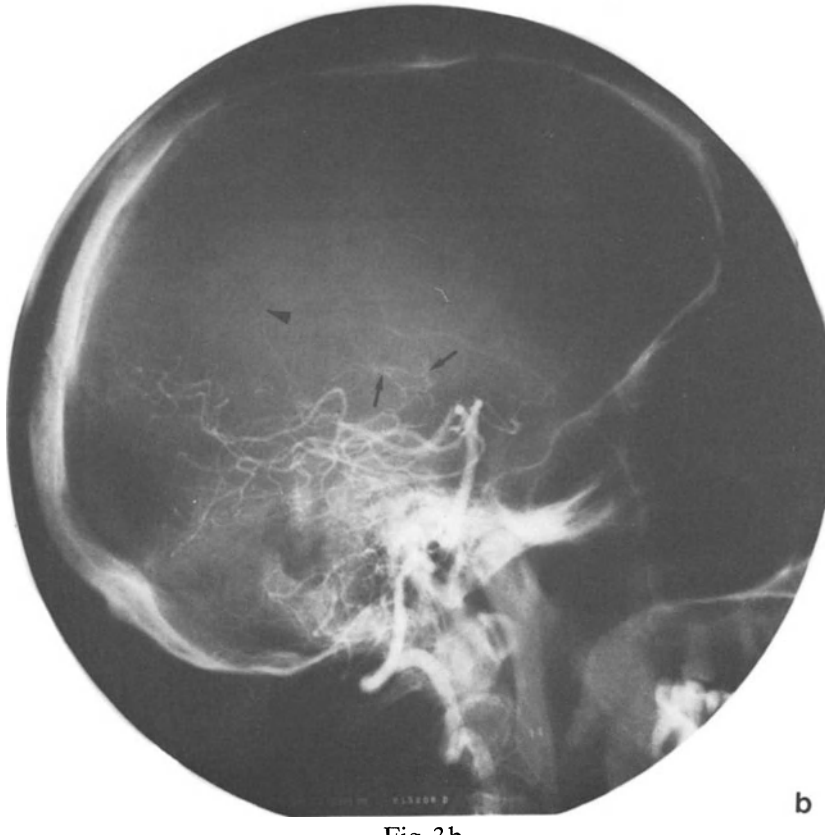


Fig. 3b

However, as pointed out in 1979 by Smith *et al.*[100], a few situations remain in which the diagnosis will be clarified by air studies performed subsequent to CT. This report documented the fact that when a massive dilatation of the temporal horn is associated with a ventricular or a juxta-ventricular mass, this trapped horn may be confused on CT with a cystic portion of the tumor. Pneumoencephalography has proved helpful in identifying the trapped temporal horn and thus in avoiding incorrect surgical or radiation therapy.

Angiographic Studies

Carotid and vertebral angiography provides two groups of information:

Changes in the course of main vessels. The wider sweep of the pericallosal artery and the elevated course of the middle cerebral artery are indicative of underlying ventricular enlargement. Similarly, depression of the internal cerebral vein reflects depression of the third ventricle roof due to hydrocephalus. These features are greatly variable since the ventricular enlargement can be total, asymmetric or confined to one ventricular horn.

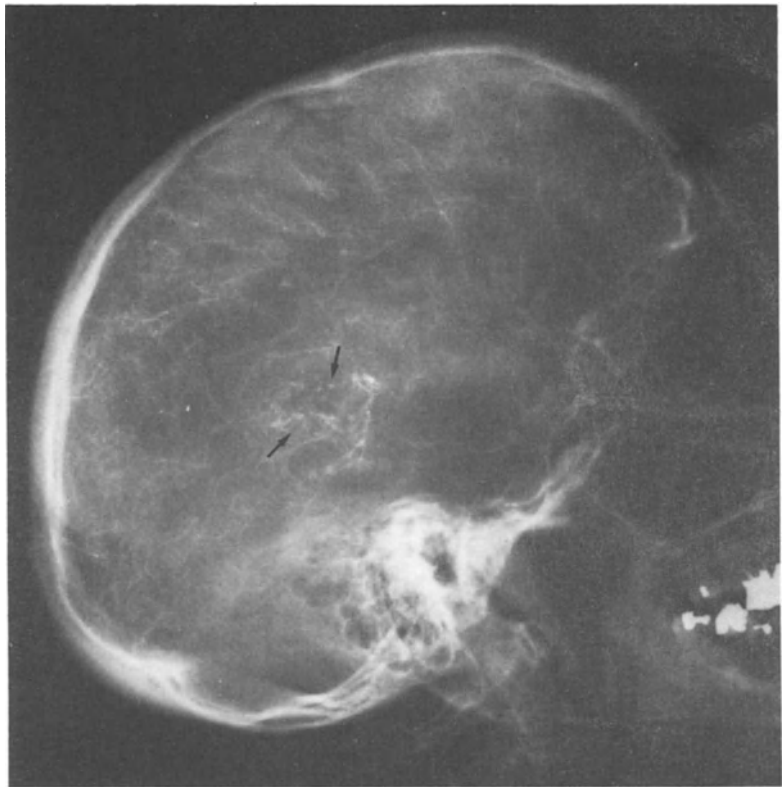


Fig. 4. Left trigonal meningioma. Carotid angiography. Capillary blush (arrows)

AP views often demonstrate shift of the internal cerebral vein away from the side of the tumor.

Information about the vascular supply of intra-ventricular tumors is obtained from angiographic studies. Typically, the main blood supply arises from choroidal vessels, regardless of the histological type. Anterior choroidal arteries supply most of the tumors originating from the trigone. Medial posterior choroidal arteries supply the majority of the tumors involving the septum pellucidum. Angiography shows the dislocated course and the enlarged appearance of both anterior and posterior choroidal arteries on the side of the lesion. In addition, a supply from lenticulostriate arteries or from perforating branches may occur in tumors originating from the floor or the wall of the lateral ventricles. The demonstration of tumor pathological circulation depends on the histological type and will be further discussed in this study.

Most ventricular tumors demonstrate venous drainage towards the deep cerebral veins via dilated subependymal branches.

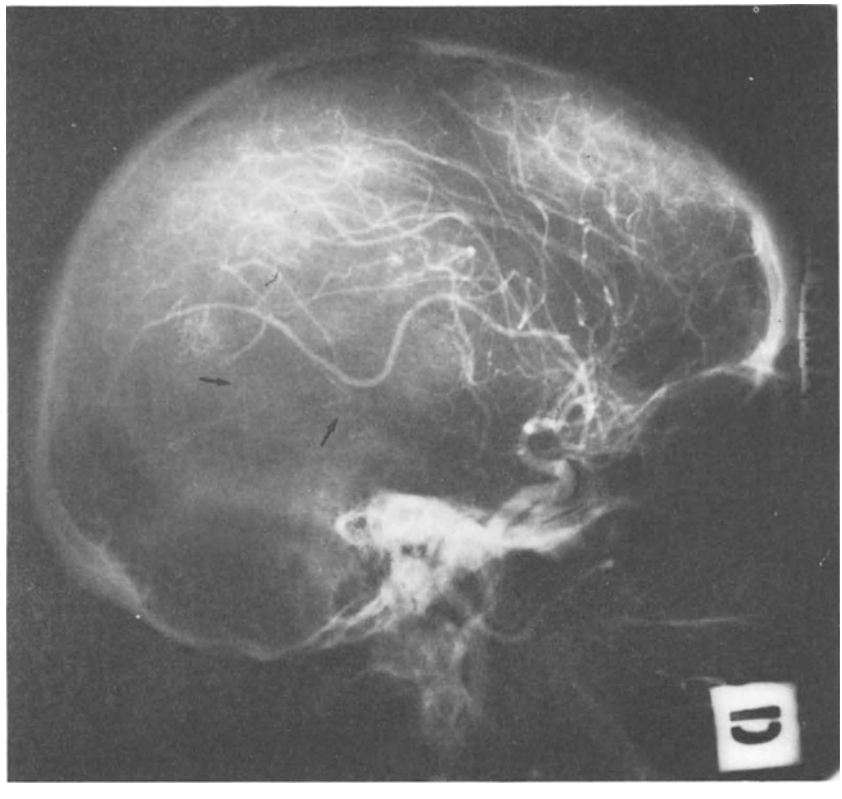


Fig. 5. Right trigonal meningioma. Carotid angiography. The supply is from the anterior choroidal artery. Note the spiral arteries (arrows)

General CT Appearance of Intra-Ventricular Tumors

Intra-ventricular masses may cause important changes in the normal ventricular system and in the surrounding brain tissue, regardless of their pathological variety. These changes are accurately documented by CT scanning:

Hydrocephalus (Figs. 11 a and b, 12) may present as a symmetric generalized ventricular widening in patients involved with midline masses or with tumors occluding both foramina of Monro. Usually it appears predominantly unilateral since the ipsilateral ventricular lumen is obliterated by the mass. Focal dilatation of the ventricle around the mass may be noted.

Trapped horns (Figs. 16, 17 a and b, 18) are due to cerebrospinal fluid production upstream to the neoplastic obstruction. If large enough, the trapped horn can cause a mass effect and may be confused with a juxtatumoral cyst when its density is higher than that of normal cerebrospinal

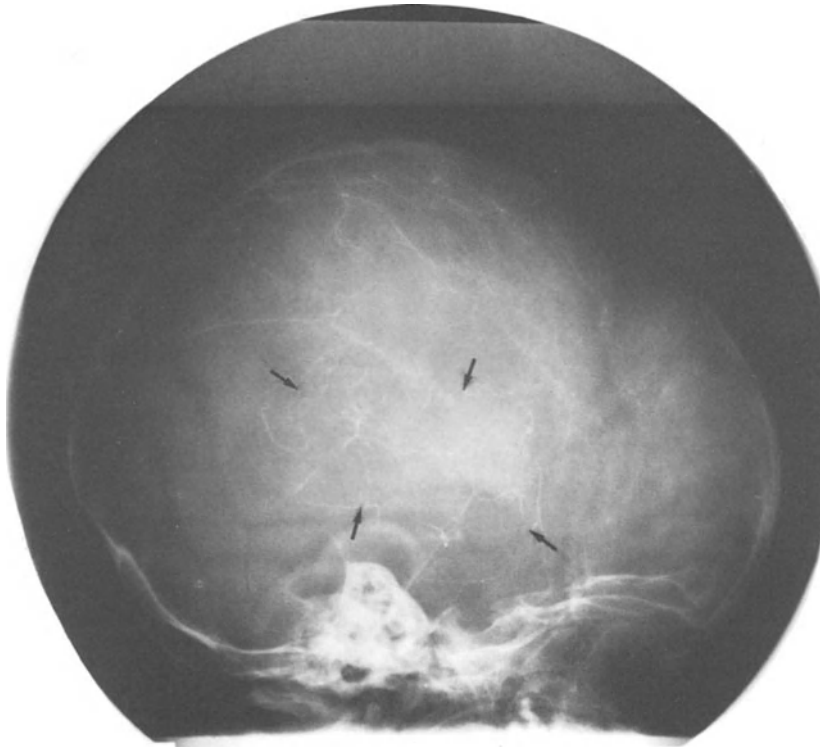


Fig. 6. Grade II ependymoma. Carotid angiography (late phase). Neovascularity (arrows)

fluid. This cyst-like appearance is related to an increased CSF protein concentration in the dilated region.

Changes in the adjacent white matter (Figs. 13, 14 a, and b). Periventricular lucencies are commonly associated with massive ventricular enlargement. Their CT appearance is that of low density areas in the brain tissue adjacent to the frontal horns. Unilateral low density area spreading in the white matter may mimic the more common brain edema and may be consistent with a tumor of parenchymal origin. Nevertheless such hypodensity may be observed in "true" ventricular tumors, with no clear correlation with vascularity or malignancy of the neoplasm. Disruption of the ependymal wall by the tumor may be an important factor.

Stigmata of intra-ventricular bleeding may be disclosed by CT evaluation. They appear as high density areas lying in the occipital horns.

Occasionally, CT may also show *intra-tumoral calcification* unvisualized on plain films (Figs. 15 a and b). In patients with tuberose sclerosis, CT may show calcification remote from the actual site of development of the neoplasm.

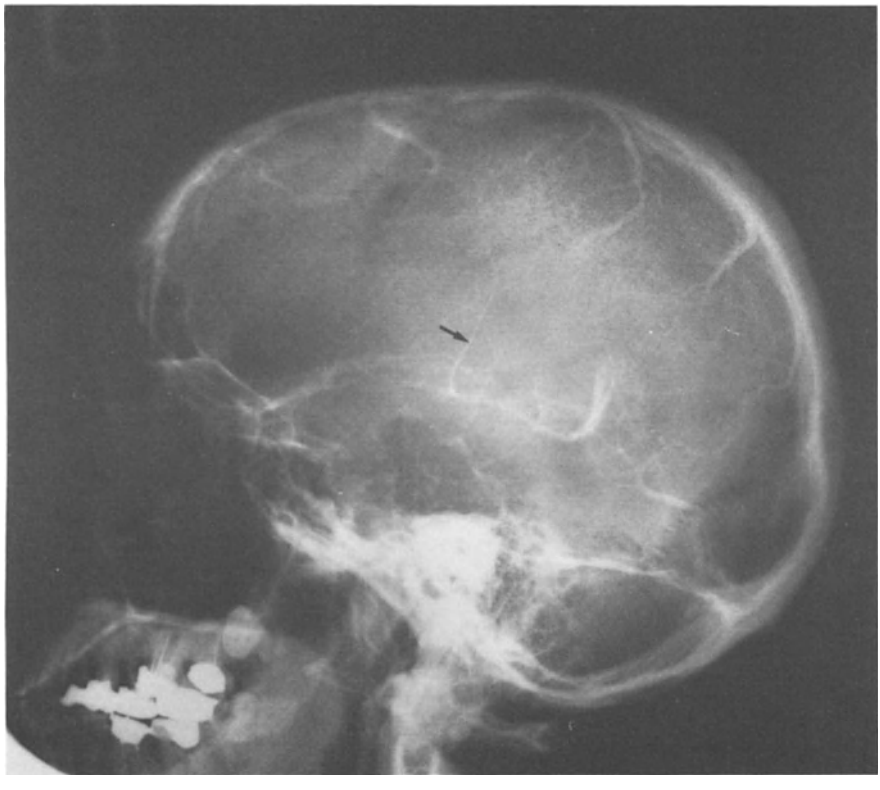


Fig. 7. Septal grade I astrocytoma. The course of the thalamo-striate vein is more vertical than normal (arrow)

The CT characteristics of the mass itself, its density, the way it enhances, its location, shape, and size are to be correlated with the histology. This is the purpose of the next paragraph.

Angiographic and CT Appearance of the Main Pathological Types

Choroid plexus tumors (Figs. 19 a and b): Papillomas and carcinomas are both located mainly in the trigone, extending into the bodies of the lateral ventricles. CT usually exhibits a higher density than the brain, and almost invariably enhances, but isodense choroid plexus papillomas may be encountered. Inhomogeneous density may suggest malignancy. Associated hydrocephalus is generally present, involvement of the whole ventricular system, including the third and the fourth ventricles may suggest a communicating type.

Arterial supply is from the choroidal arteries. The capillary blush is inhomogeneous. Early draining veins are useful signs in detecting malignancy[5,16].

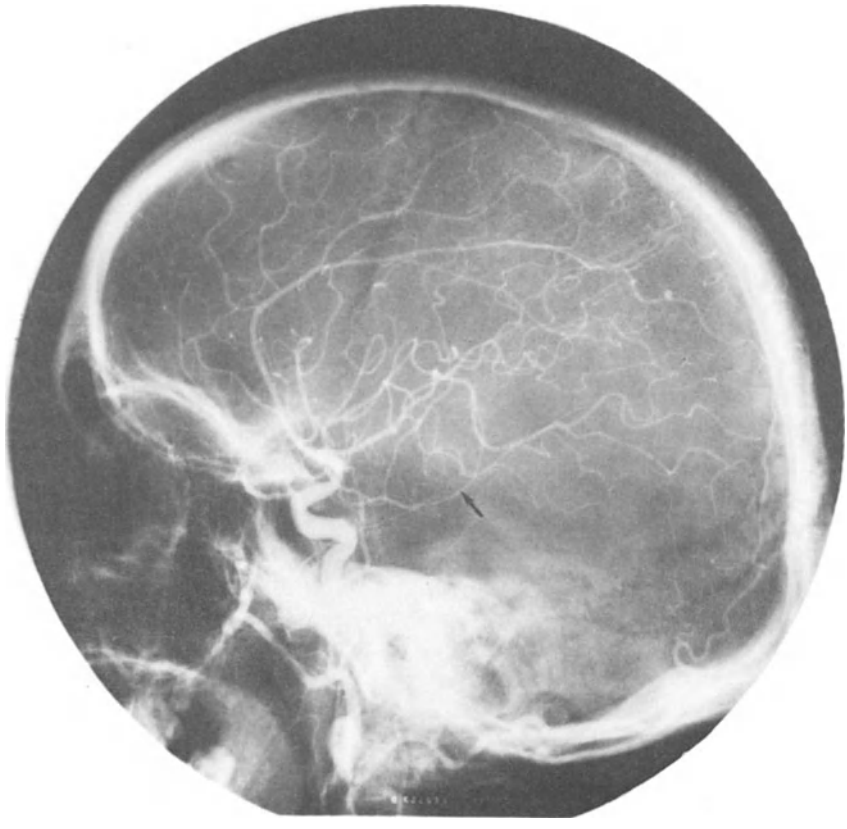


Fig. 8. Grade IV astrocytoma of the left atrium. Note the curved course of the anterior choroidal artery (arrow)

Intra-ventricular meningiomas (Figs. 20 a and b): These often show a similar pattern. Most meningiomas arise in the trigone. The tumor is of increased density; however, a few meningiomas may have the same attenuation coefficient as surrounding brain. The margins of the mass are smooth and it enhances markedly after contrast infusion. Surrounding brain edema may be noted. Angiographically, the anterior choroidal artery is often enlarged and displaced on the side of the tumor, but the lateral posterior choroidal arteries may be the only vascular supply to meningiomas. The angiograms display small areas of neovascularity or an intense homogeneous vascular blush (Figs. 3 a and b, 4, 5).

Ependymal Tumors

Ependymomas are mainly located in the ventricular body often extending into the frontal horn. CT shows a mass of mixed density. Enhancement

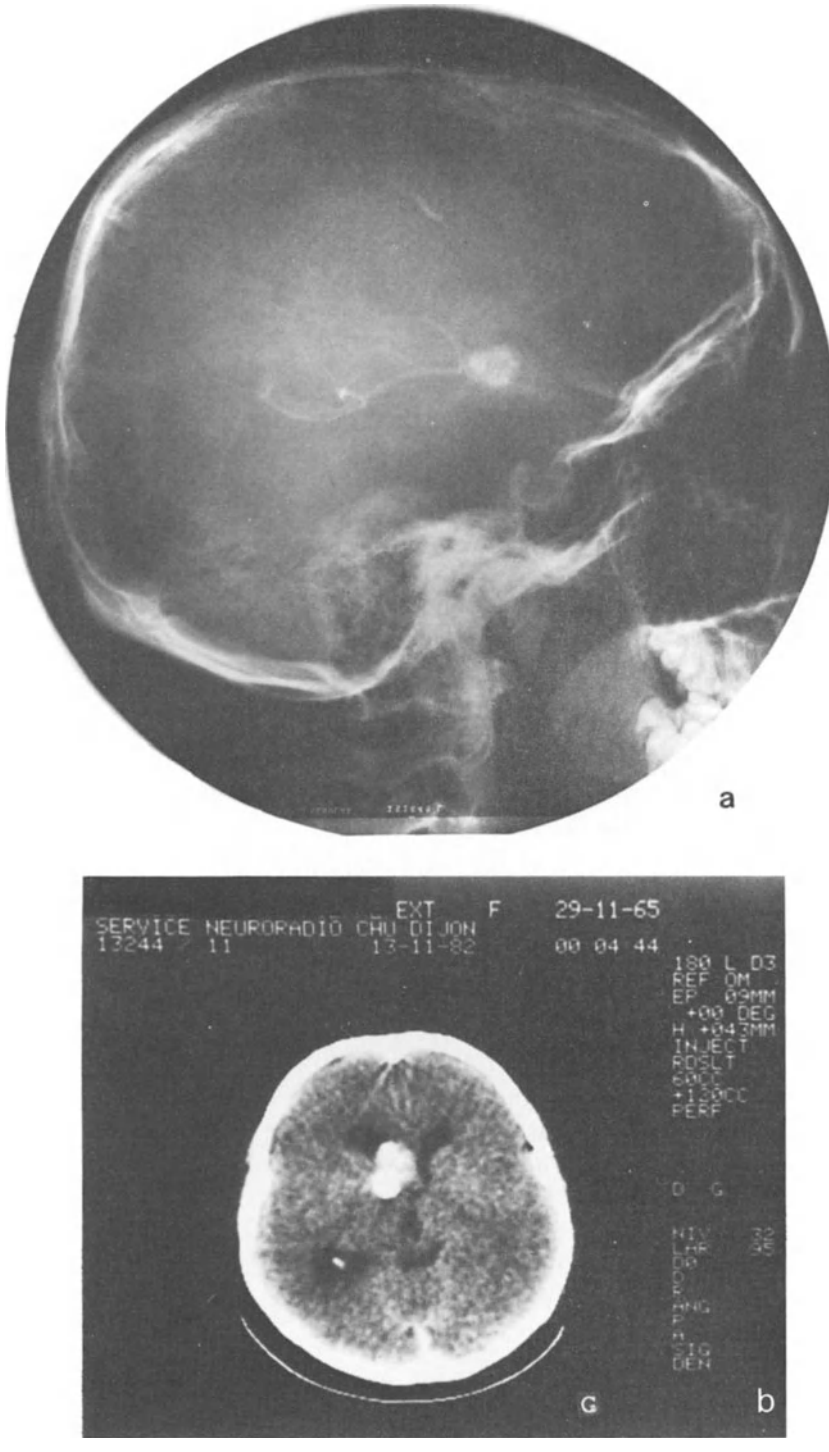


Fig. 9. Grade I astrocytoma. a) Carotid angiography. Venous phase. Note the calcified mass projecting in the region of the Foramen of Monro. Lengthened septal veins are consistent with a dilated frontal horn. b) Enhanced CT in the same patient. Calcified enhancing mass originating in the right frontal horn and producing asymmetric hydrocephalus

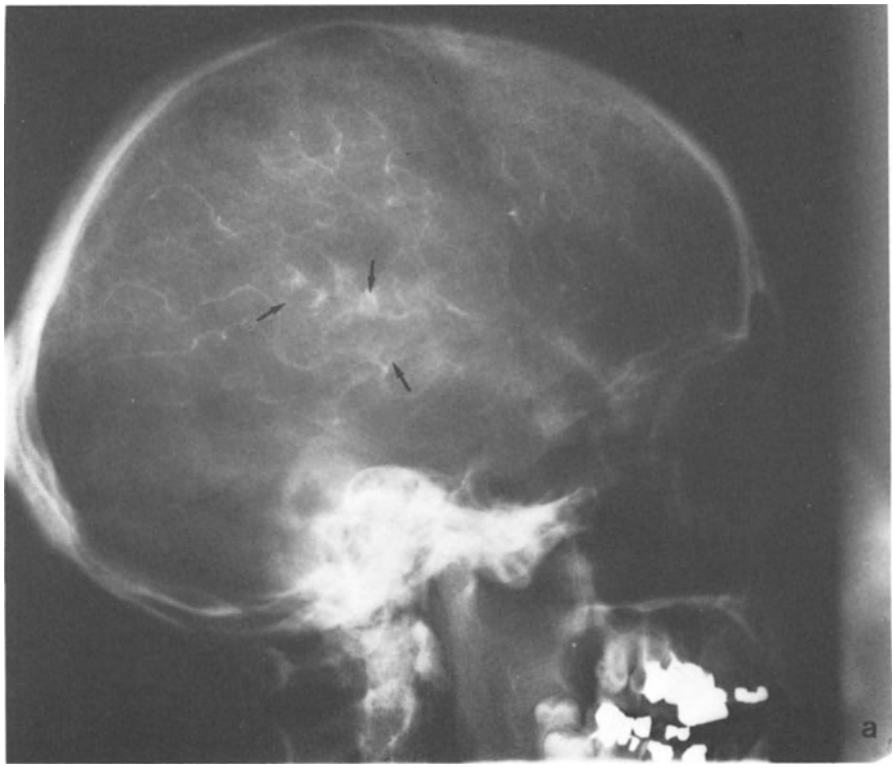


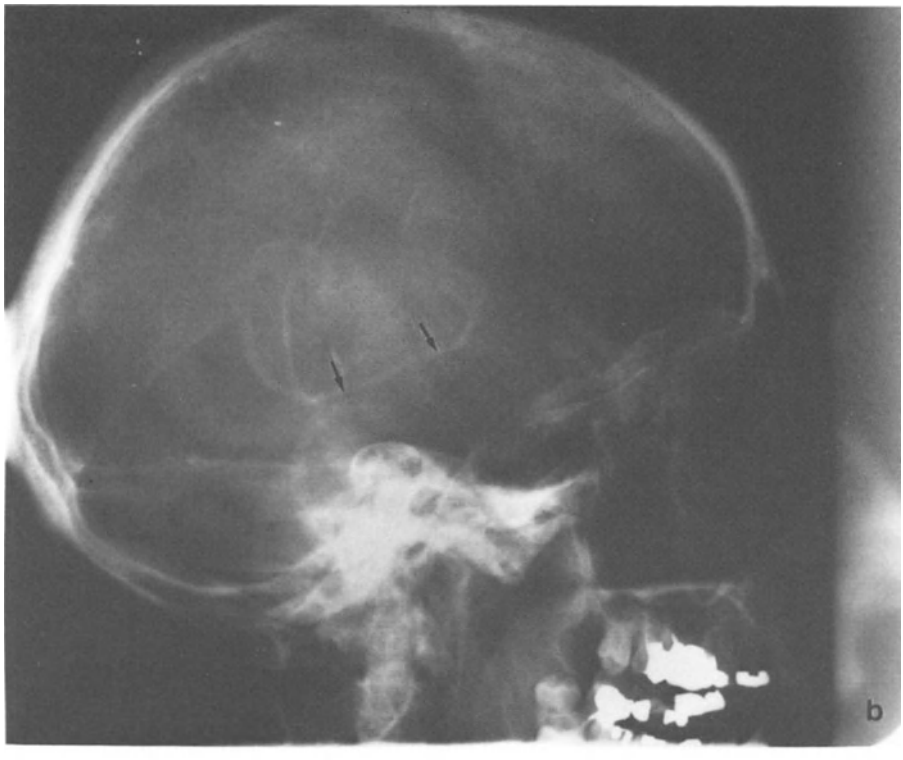
Fig. 10. Intra-ventricular oligodendroglioma. a) Left carotid angiography (capillary phase): inhomogeneous vascularity (arrows). b) Left carotid angiography (venous phase): note the massive lowering of deep veins (arrows). c) Unenhanced CT in the same patient. d) Enhanced CT in the same patient

is usual, but frequently not pronounced. Infantile ependymomas may present as huge hemispheric masses including areas of increased density and cyst-like non-enhancing areas. Associated hydrocephalus is predominantly contralateral (Figs. 21 a and b, 22 a and b, 23, 24 a and b).

Angiograms show displacement of the anterior choroidal arteries, but the main blood supply is from the lenticulo-striate arteries. Displacement of subependymal veins (septal and thalamo-striate veins) is consistent with dilatation of the ventricular body or the frontal horn (Fig. 6).

Subependymomas

In the single report on the CT appearance of subependymomas[73], the mass did not produce hydrocephalus and was composed of an enhancing solid part and two large cystic cavities. Angiographically these tumors are avascular.



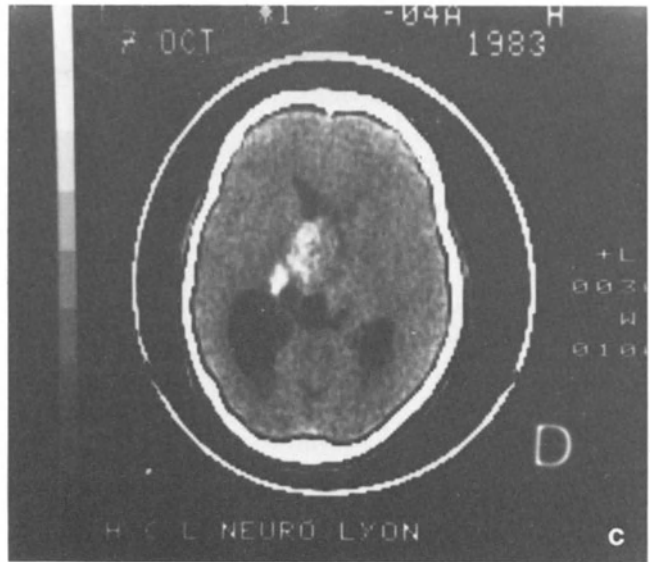


Fig. 10b–c

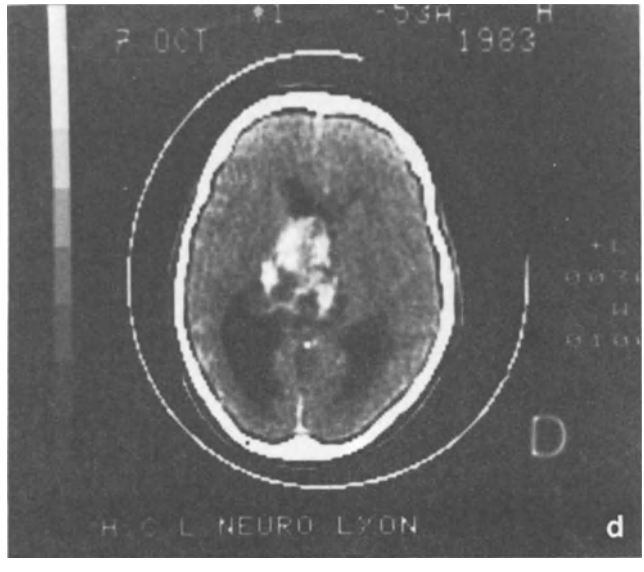


Fig. 10d

Astrocytomas

Low grade astrocytomas are generally of mixed density and not infrequently of low density. They enhance slightly on post-contrast CT, and may be associated with thickening of the septum pellucidum[60] (Figs. 25 a and b, 26 a and b). Subependymal giant-cell astrocytomas associated with tuberose sclerosis typically arise from the head of the caudate nucleus. In this condition, subependymal calcification is often noted. Blood supply is from the lenticulo-striate arteries, but usually the angiogram exhibits no pathological vascularity (Figs. 7, 8, 9 a, and b).

Oligodendrogliomas (Figs. 10 a, b, c, and d, 27 a and b). Only a few reports have been published relating the CT appearance of intra-ventricular oligodendrogliomas[45,75,77] Our experience is consistent with the description given by previous authors: CT reveals a large hyperdense mass lying in the midline and causing symmetric hydrocephalus. Calcification was present in 2 of 4 reported cases. Angiographically, a faint blush may be visualized in the late venous phase.

Miscellaneous (Figs. 28 a and b, 29 a and b, 30 a and b, 31 a and b, 32 a and b).

Other histological varieties are too rare to describe typical CT and angiographic appearance. *Intra-ventricular cysticerci* cause low density masses and may show ring enhancement. *Epidermoids* cause cauliflower-

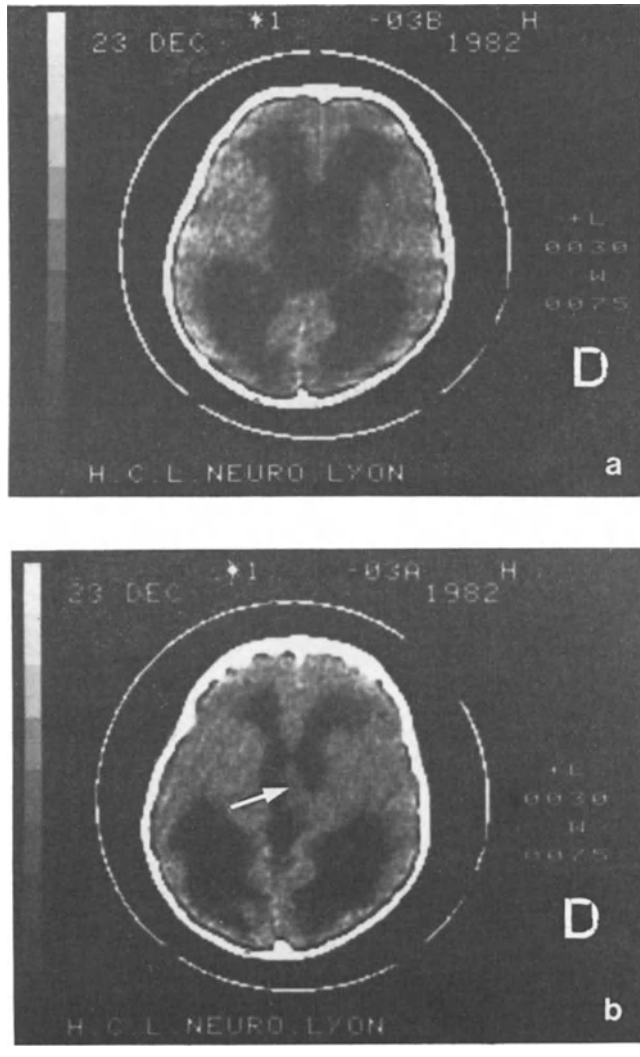


Fig. 11. a) Massive symmetric hydrocephalus with periventricular lucencies. Enlargement of both frontal horns and of the III^{d} ventricle is featuring the "Mickey Mouse" Syndrome. b) Small isodense mass originating from the septum (arrow)

like very low density masses within the ventricles similar to those noted in epidermoids of any situation[53]. *Metastatic tumors* are likely to cause bilateral trigonal enhancing masses[52]. No information is available today relating the CT appearance of *blood vessel tumors, teratocarcinomas, xanthogranulomas. Seeding medulloblastomas* may cause intra-ventricular iso or hypodense enhancing masses with associated involvement of the

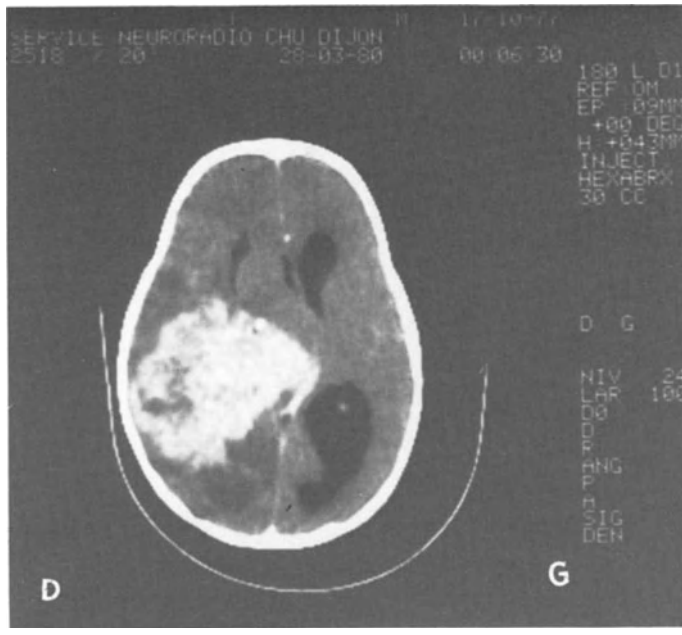


Fig. 12. Enhanced CT appearance of a choroid plexus carcinoma with unilateral hydrocephalus

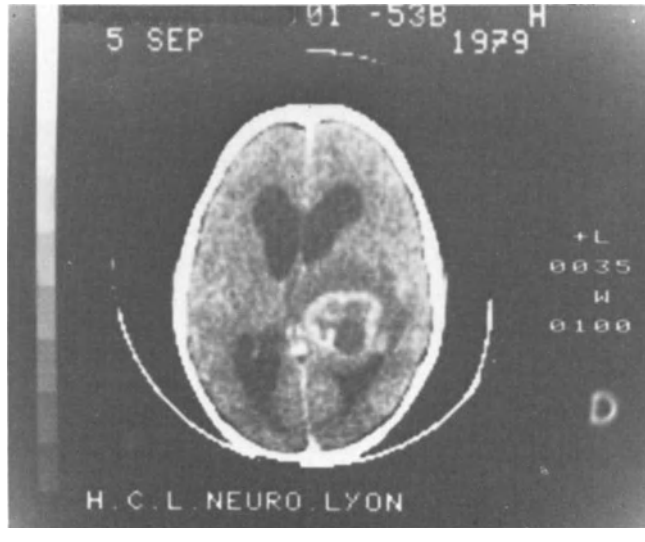


Fig. 13. Grade IV ependymoma of the right atrium. Note the asymmetric hydrocephalus and the edema surrounding the mass

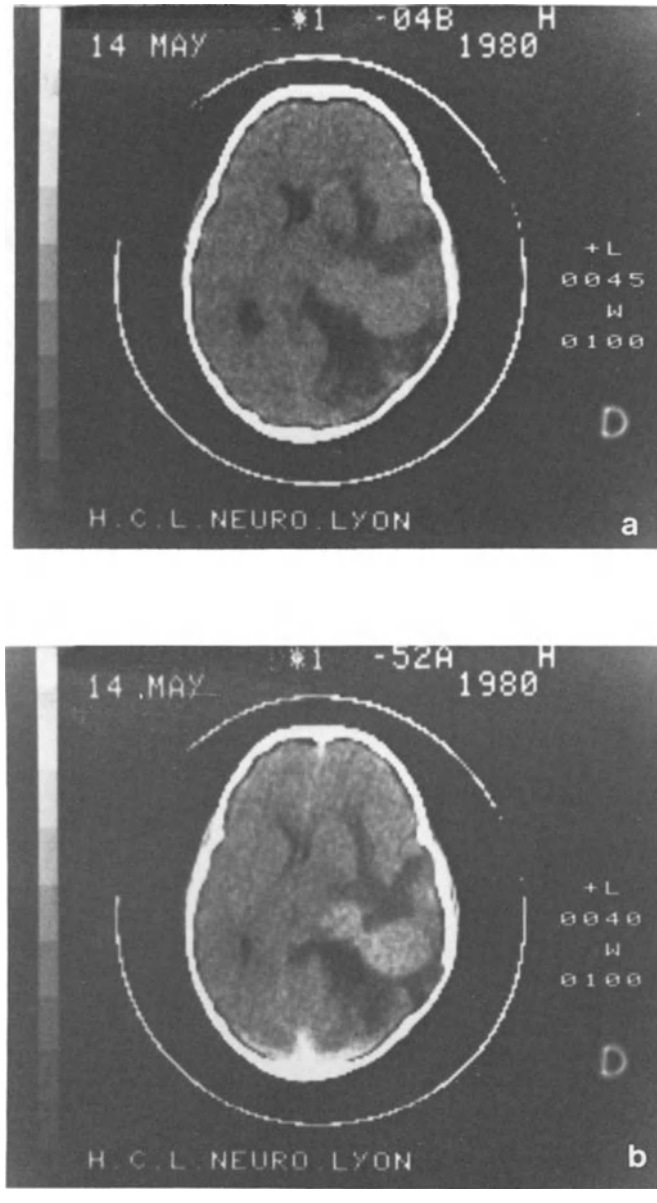


Fig. 14. Right atrial meningioma. a) Pre-contrast CT. No hydrocephalus is present. Note the edema surrounding the tumor. b) Post-contrast CT

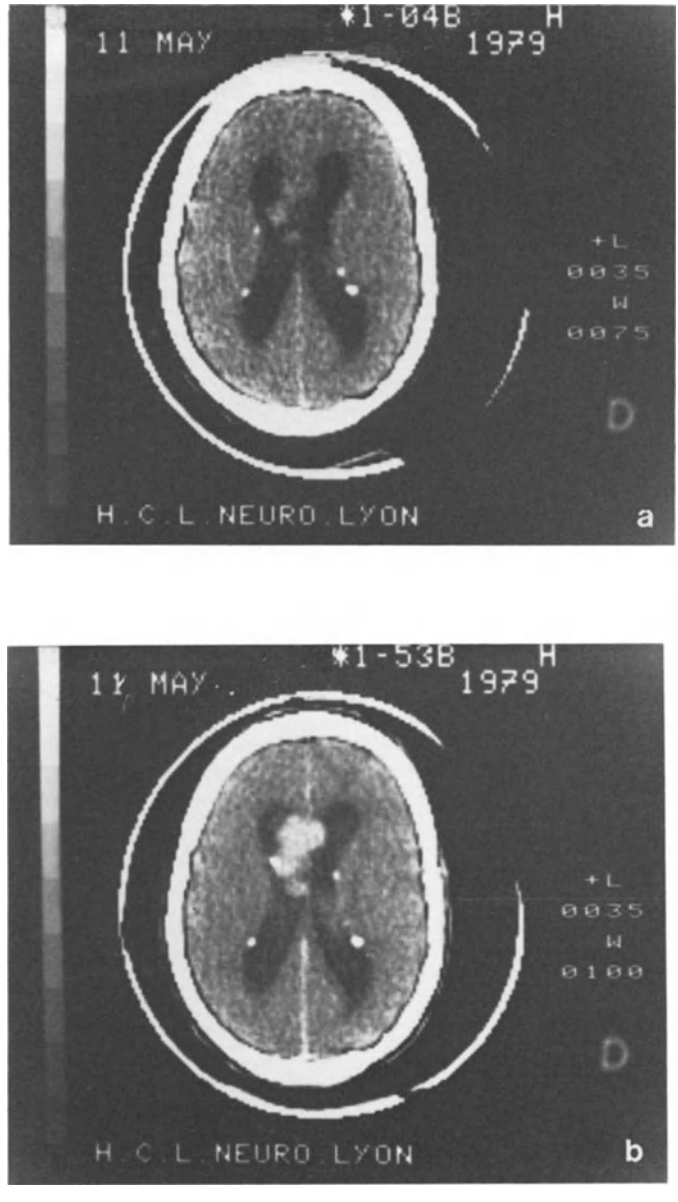


Fig. 15. Grade I ependymoma in a patient with tuberous sclerosis. Note the calcifications remote from the neoplasm. a) Pre-contrast CT. b) Post-contrast CT

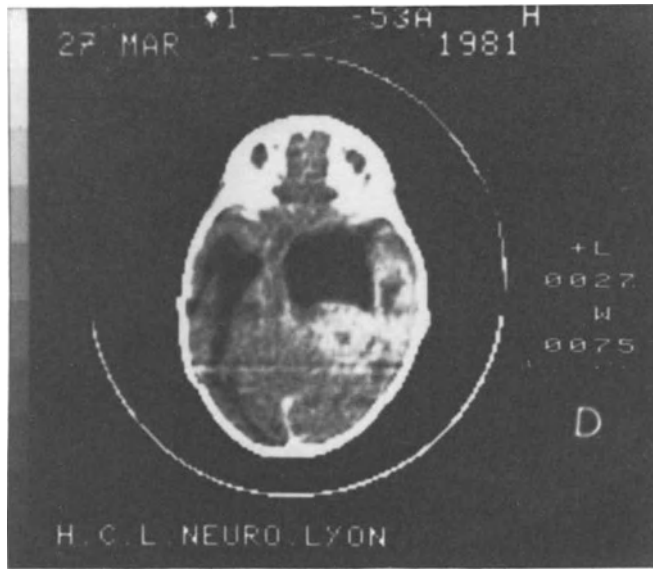


Fig. 16. Enhanced CT in an infant with a grade IV ependymoma. Massive dilatation of the right temporal horn with mass effect

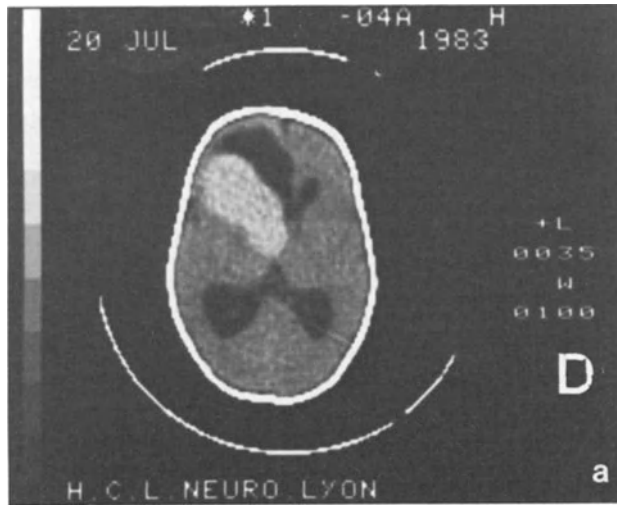


Fig. 17. Grade III infantile ependymoma with trapped frontal horn. a) Pre-contrast CT. b) Post-contrast CT

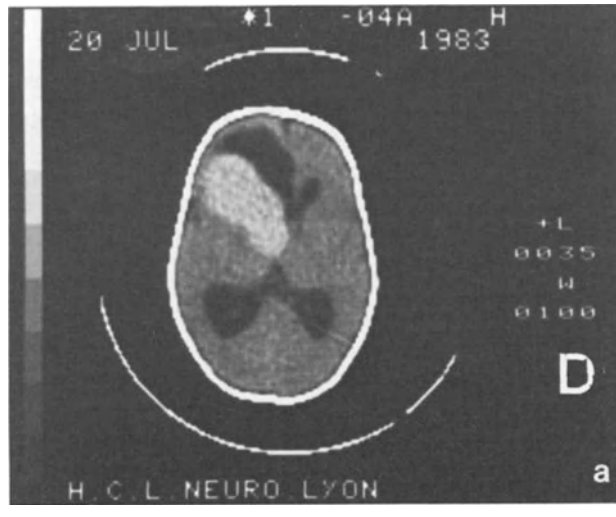


Fig. 17b

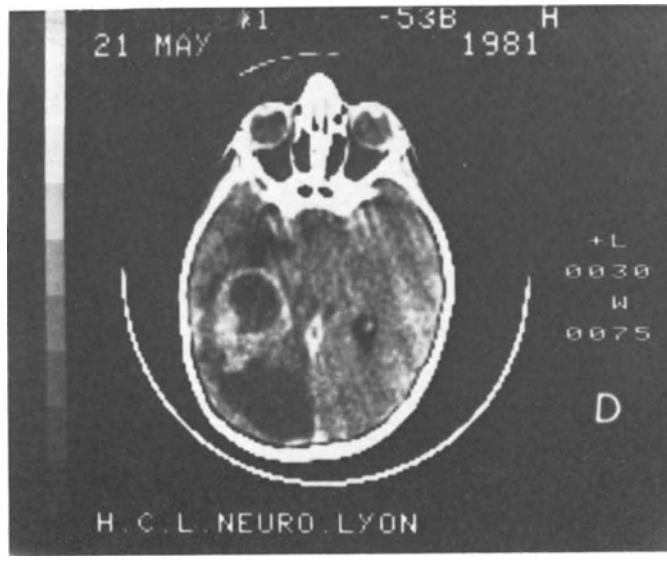


Fig. 18. Enhanced CT appearance of a grade II ependymoma with a cyst-like trapped occipital horn

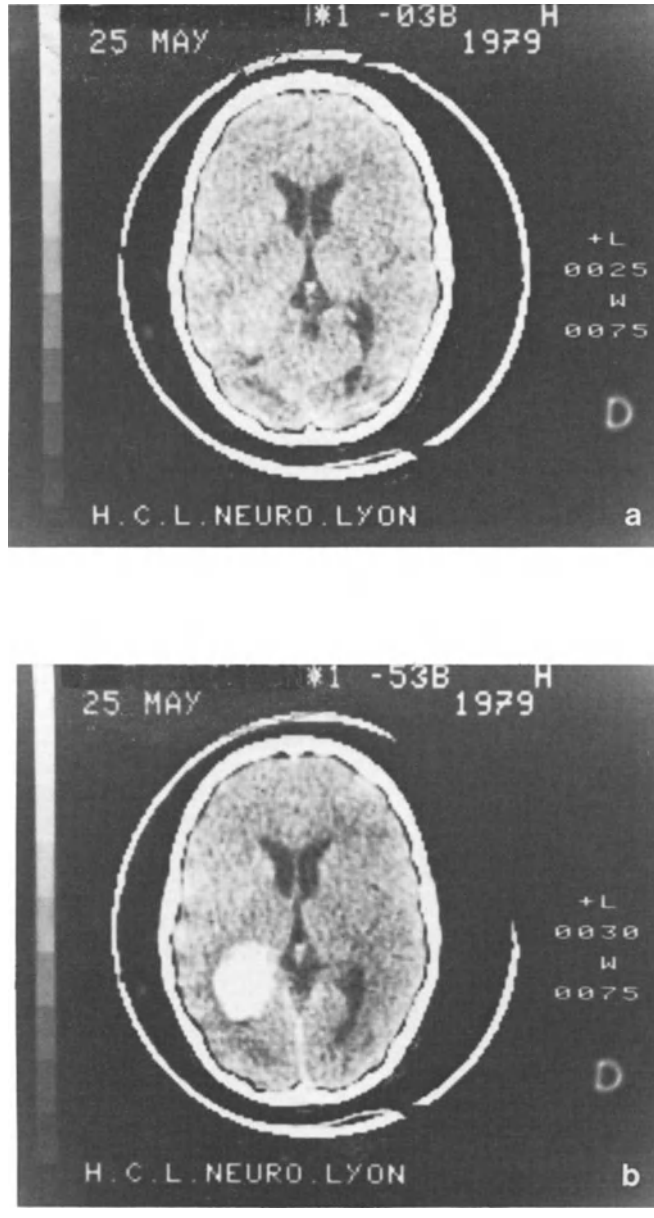


Fig. 19. Left atrial choroid plexus papilloma. a) Pre-contrast CT: isodense mass. b) Post-contrast CT: enhancing mass

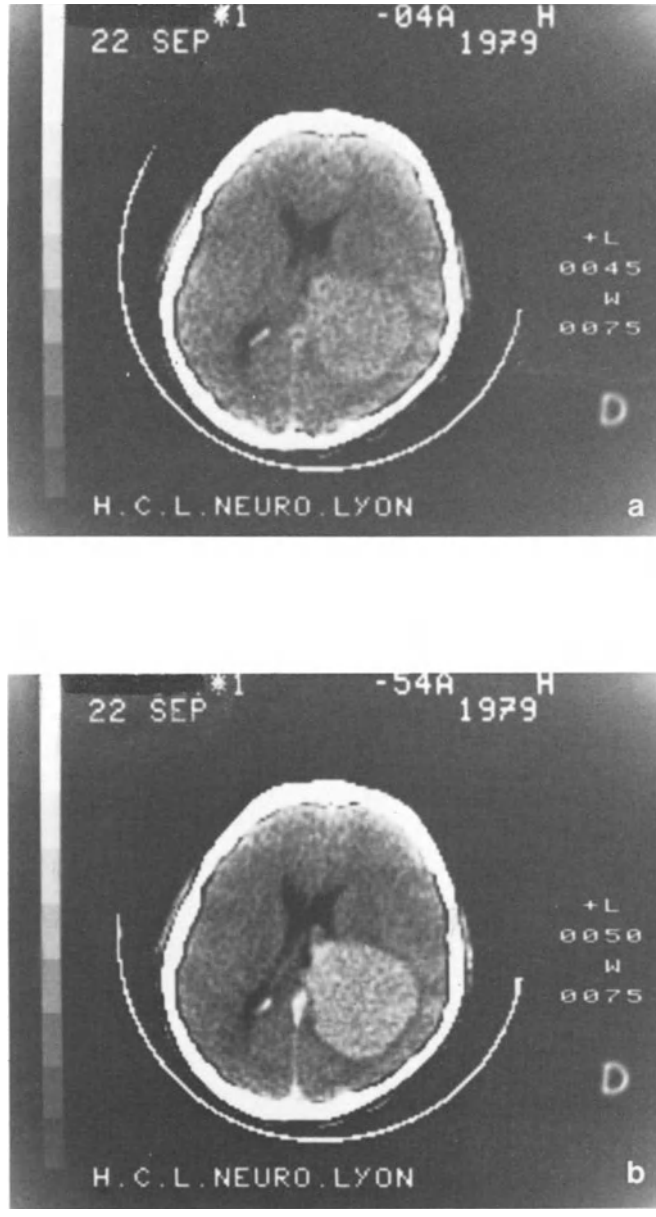


Fig. 20. Right atrial meningioma. a) Pre-contrast CT displays a large mass with slightly increased density. b) Post-contrast CT: marked enhancement

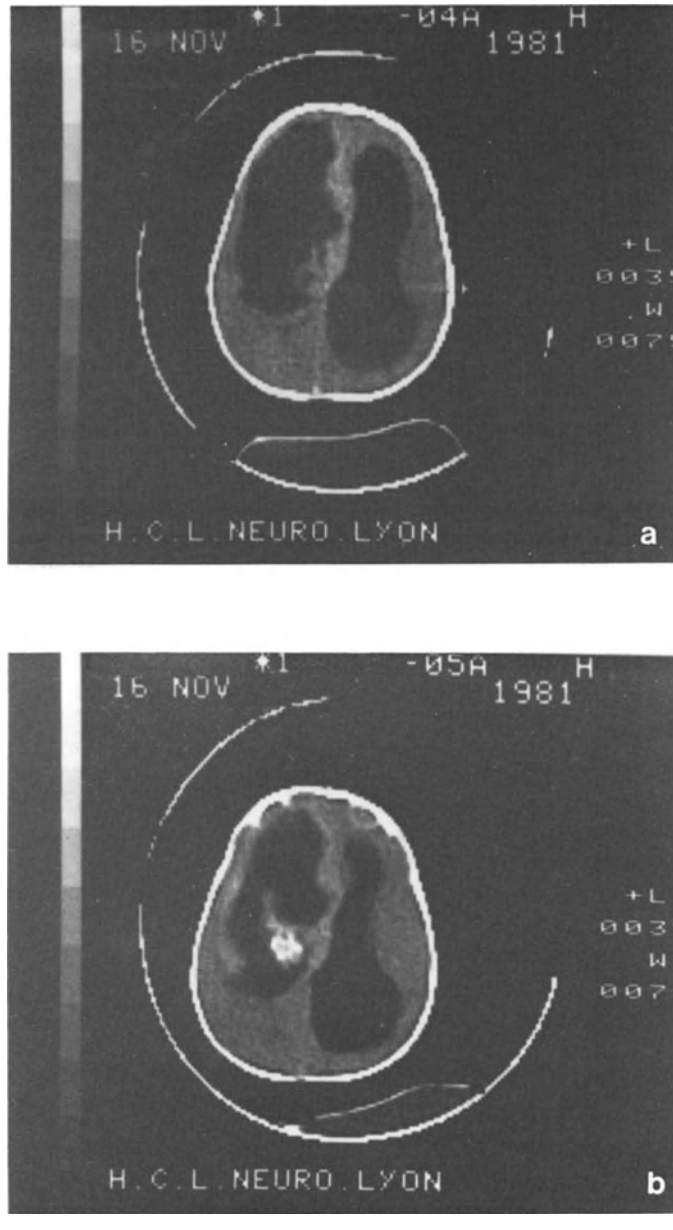


Fig. 21. Grade IV ependymoma. a) Pre-contrast CT: asymmetric hydrocephalus. No mass is visualized. b) Post-contrast CT is documenting enhancement of a small septal mass

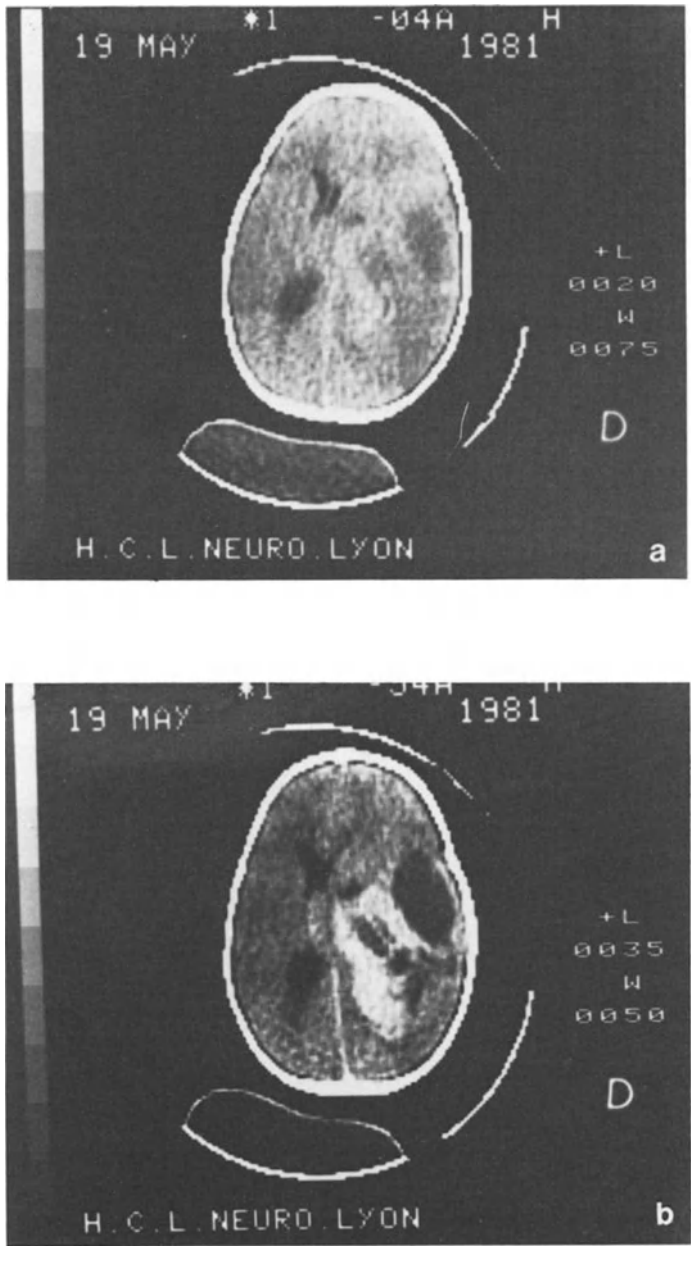


Fig. 22. Grade II infantile ependymoma. a) Pre-contrast CT. b) Post-contrast CT exhibits associated areas of enhancement and of necrosis

subarachnoid spaces[36]. The CT appearance of other rare intra-ventricular tumors is displayed in the figures: *spongioblastic astrocytoma, neuroblastoma, angioglioma*, and *malignant lymphoma.*

4. Surgery

Introduction

Tumors located in the various areas of the lateral ventricle each have a specific surgical approach. The choice between different approaches depends on the size of the tumor, where it is attached, whether in the

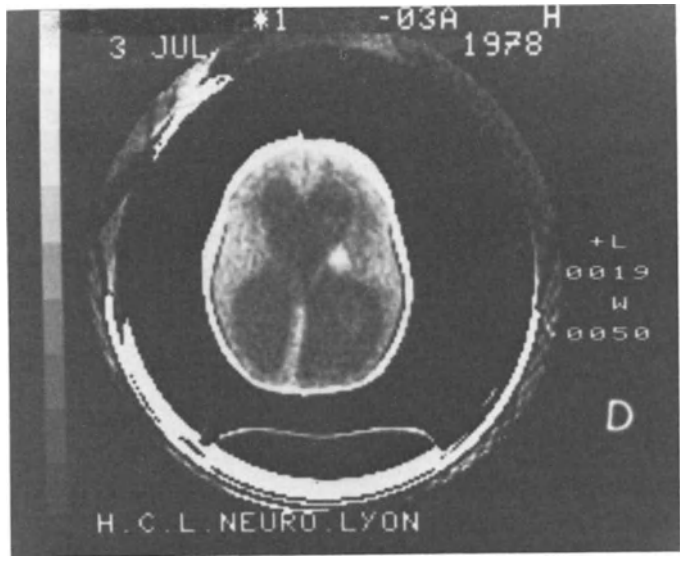


Fig. 23. Grade I ependymoma causing congenital hydrocephalus. Unenhanced CT showing a hyperdense tumor arising from the outer wall of the ventricle. Huge symmetric hydrocephalus is present

dominant or non dominant hemisphere location, the size of the ventricles, vascularity, where the arterial feeders originate, the venous drainage, and the relationship between the tumor, the choroid plexus and the internal cerebral veins. Each of these factors is evaluated prior to choice of the specific surgical approach.

When performing surgery, some structures must be preserved, as the Rolandic cortical motor area, the speech area on the left side, the fornix at least on one side, the internal cerebral veins and the great vein of Galen. The pericallosal arteries and their branches, specially the calloso-marginal

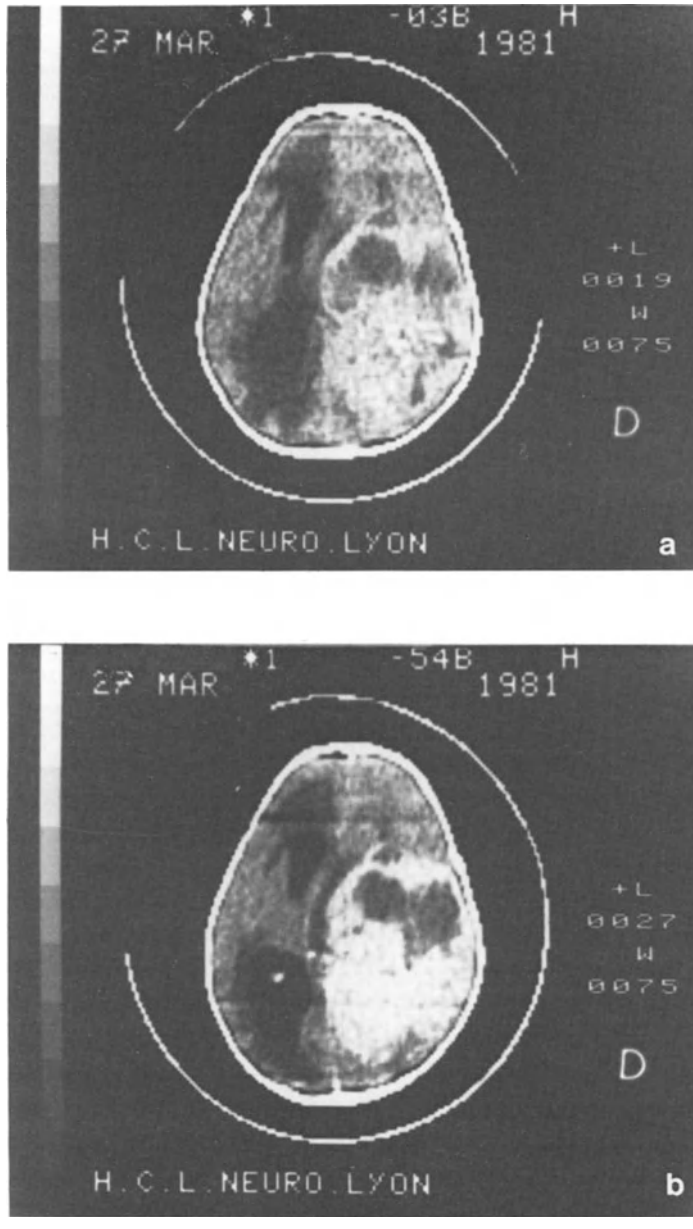


Fig. 24. Infantile grade IV ependymoma: huge hemispheric tumor including solid and necrotic areas. a) Pre-contrast CT. b) Post-contrast CT

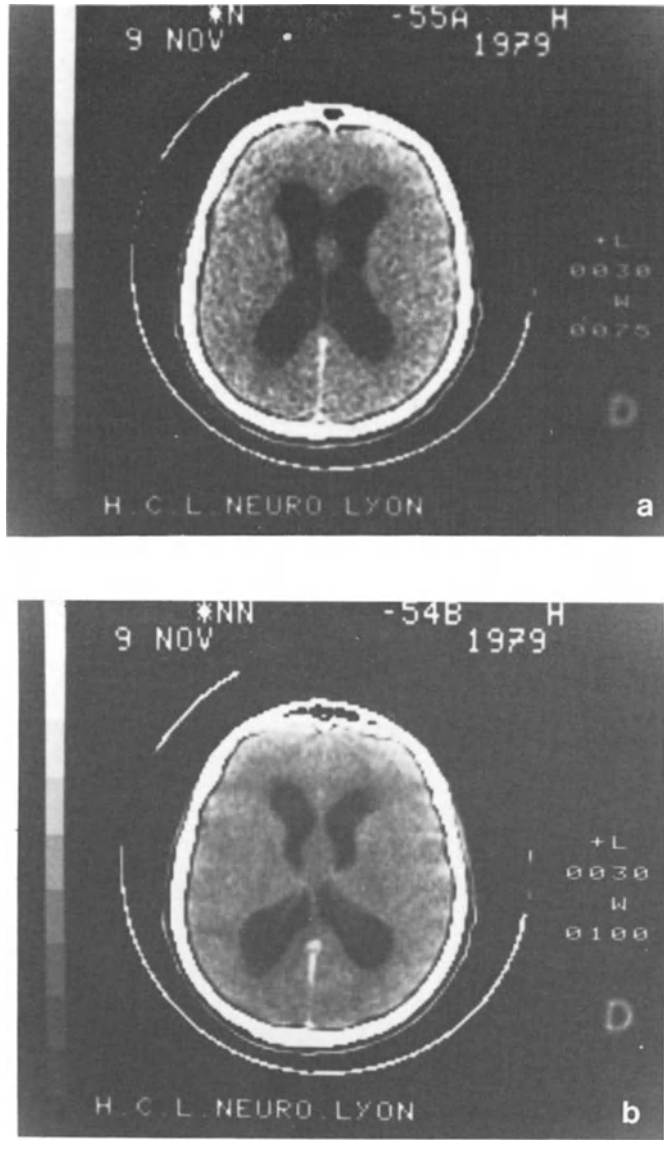


Fig. 25. Grade I septum pellucidum astrocytoma causing symmetric hydrocephalus. Both figures a and b are post-contrast CT studies

arteries, must be spared during a parasagittal approach. These recommendations apply for all neurosurgical procedures.

It is possible safely to divide, open or resect some structures during the approach or within the lateral ventricles. These are described later.

Finally, if the choice of the surgical approach is based on the location of the tumor and its extension, the decision during surgery depends on which

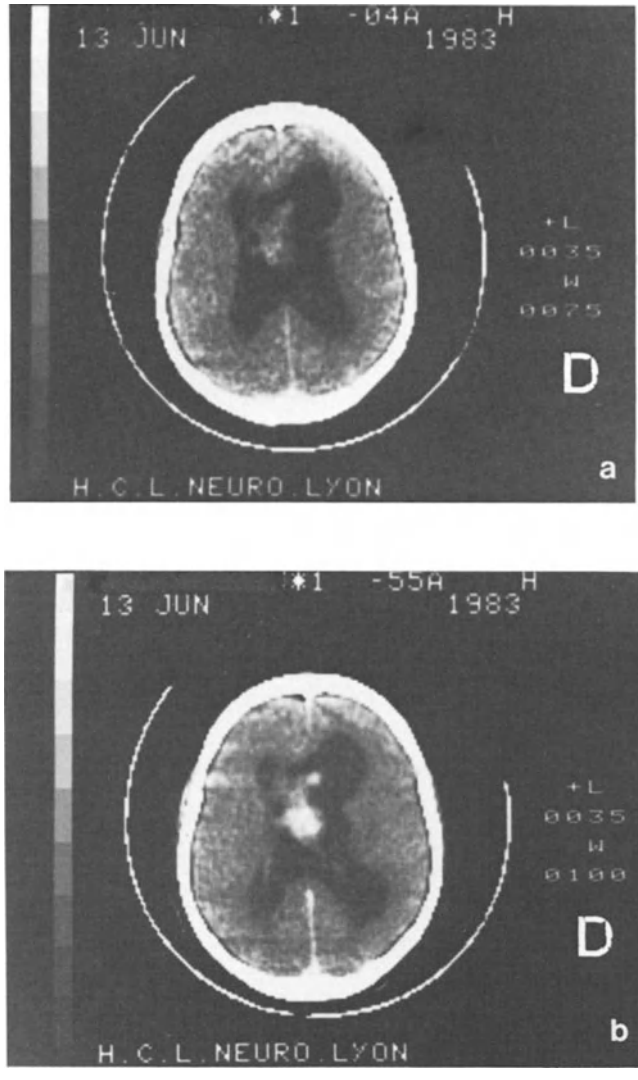


Fig. 26. Grade I septal astrocytoma. a) Pre-contrast CT. b) Post-contrast CT

nervous structures must be preserved. It depends also on the preoperative neurological deficit, as well as what postoperative deficit is acceptable. The best example of this problem is the prediction of a homonymods hemianopia in some conditions.

We shall describe the different approaches with their advantages and disadvantages, and indicate, from our experience, the best choice for each tumor location.

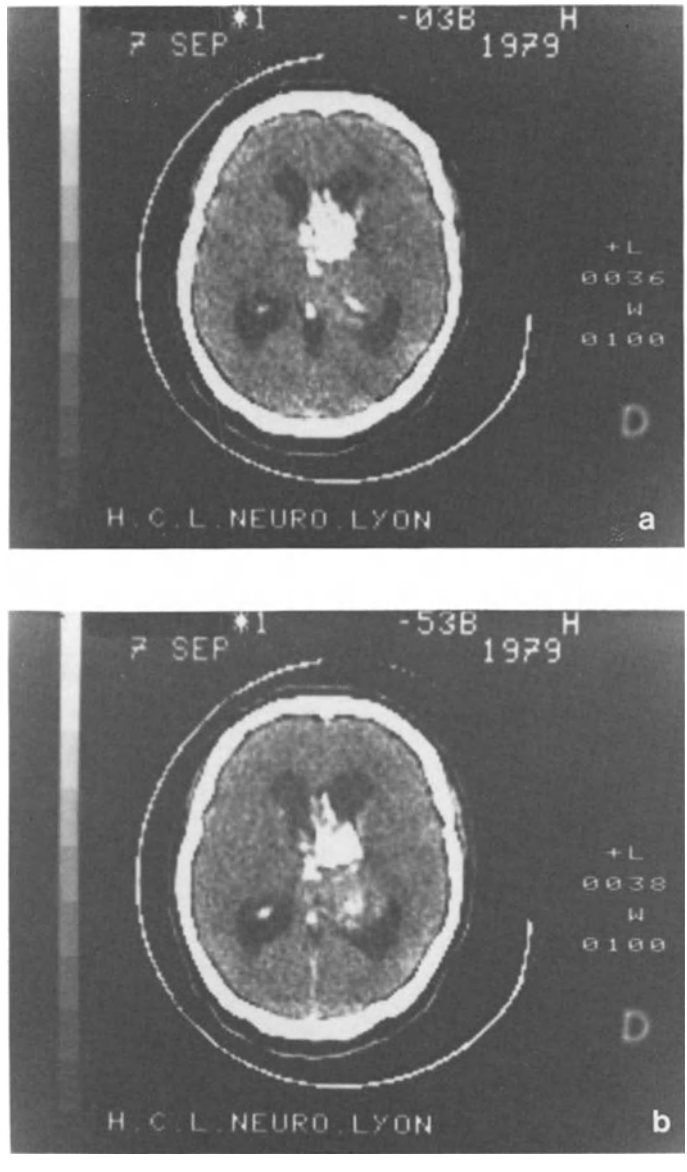


Fig. 27. Calcified oligodendroglioma. a) Pre-contrast CT. b) Post-contrast CT: slight enhancement

The Frontal Transcortical Approach (Fig. 33)

The most frequently used approach for tumors of the anterior part of the lateral ventricles is through the frontal lobe. The head is turned toward the contralateral side. The coronal suture, the bregma, and the sagittal suture are the landmarks for the bone flap. It is outlined near the midline, in front

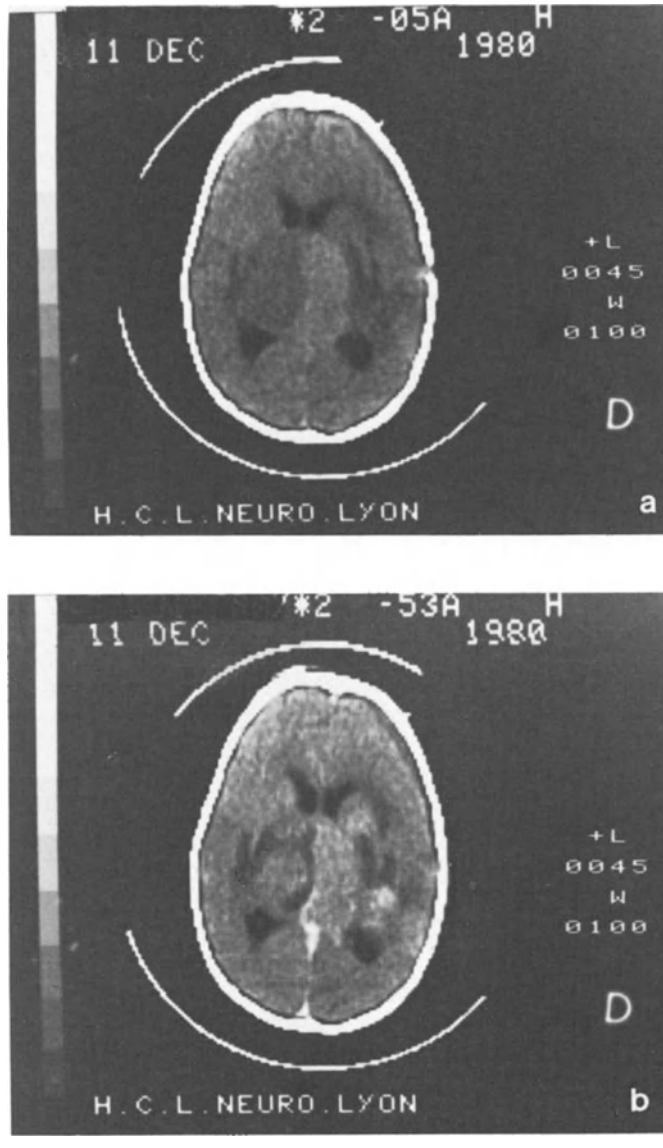


Fig. 28. Intra-ventricular neuroblastoma. a) Pre-contrast CT. b) Post-contrast CT

of the coronal suture or extending to each side of the suture, twice as much anteriorly as posteriorly. Although near the midline, it is not necessary to reach the sagittal sinus. The dural flap is reflected toward the midline. Before opening the cortex it is safer to retract the base of the frontal lobe to see exactly where the sphenoid ridge is. A line drawn between the upper part of the coronal suture and the edge of the lesser wing of the sphenoid gives the

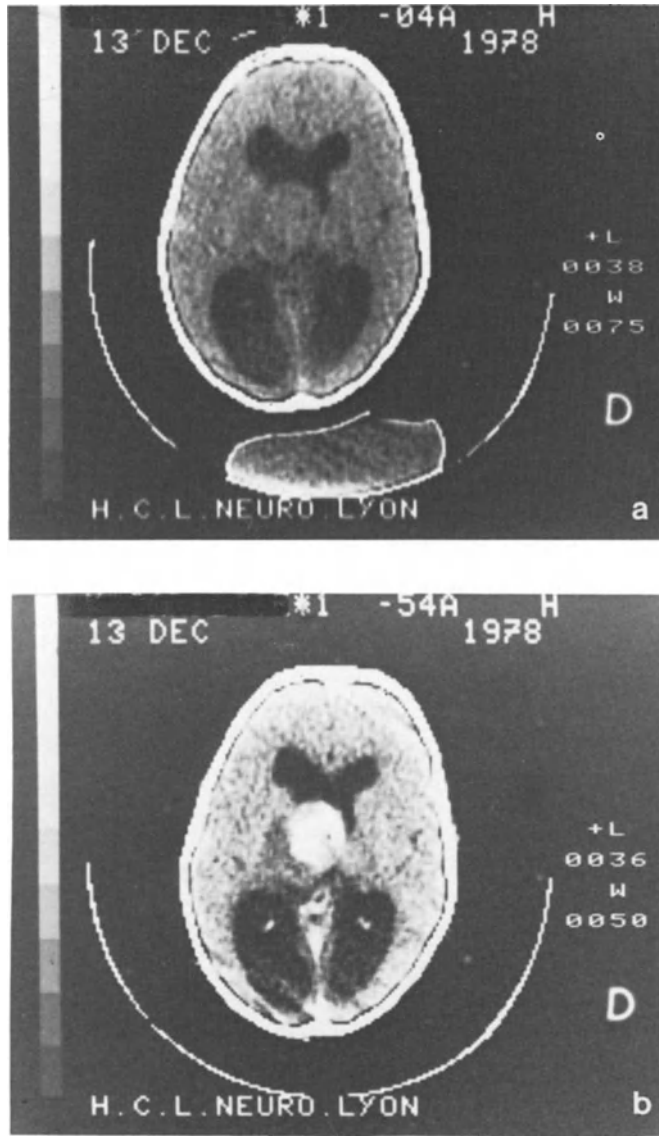


Fig. 29. Spongioblastic astrocytoma. a) Pre-contrast CT: isodense mass. b) Post-contrast CT: massive enhancement

posterior limit of the cortical incision and avoids any risk of injury to the motor cortex. This line is a less suitable landmark on the inferior aspect of the frontal lobe, especially on the dominant hemisphere. At this level a cortical margin of 1.5 cm in front of the sphenoidal ridge must be preserved. The cortical incision is made through area 9 of Brodman in the prefrontal lobe. A parasagittal incision is preferable to a coronal one, because, if

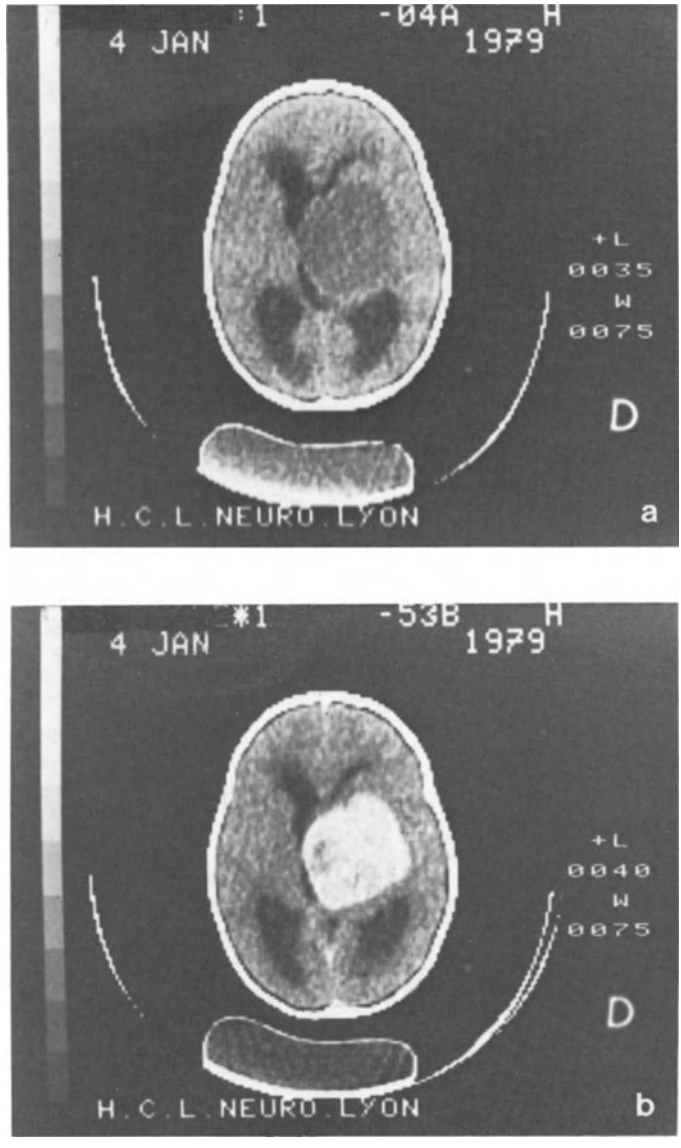


Fig. 30. Spongioblastic astrocytoma. a) Pre-contrast CT: hypodense mass. b) Post-contrast CT: massive enhancement

necessary, it is easily extended anteriorly and because it is parallel to the long axis of the ventricle. This incision is situated 2.5 cm from the midsagittal plane. Approaching the midline the danger is to go in the wrong direction toward the pericallosal artery. Inferiorly the danger is in approaching the supra-Sylvian Gyri and the speech area. In elderly patients, to avoid dangerous retraction of the cortex, it is preferable to resect a frontal cortical

area rather than to use a linear incision. McKissock[74] recommended that tumors of the lateral ventricle or the third ventricle should be exposed by a frontal transcortical approach, through an aperture made by excising a conical block of cerebral tissue.

This approach leads to the supero-lateral angle of the frontal horn at the junction of the ventricular body. It is suitable for all tumors implanted near the head of the caudate nucleus or the anterior thalamus and for tumors of the septum lucidum, especially when the lateral ventricle is enlarged. It is useful for all tumors extending laterally. Exposure is extensive and easy in all directions; the anterior part and even the body of the lateral ventricle are exposed. When the lateral ventricle is narrow, spontaneously or after CSF shunting, this approach becomes more difficult. The ventricle is sometimes difficult to find, particularly when it is displaced by tumor. In these cases, it is important to locate the foramen of Monro first to avoid injury to the fornix. The foramen sometimes is hidden behind the bulk of the tumor or the bulging head of the caudate nucleus compressed by tumor. To find the foramen, it is necessary to follow the veins to the venous confluence situated behind it, where the septal vein joins the thalamo-striate vein to form the internal cerebral vein or lesser vein of Galen. Anteriorly the septal vein is near the midline. It is followed backwards along the septum, turning above the foramen. Posteriorly, the thalamo-striate vein is recognized by its relationship to the choroid plexus and its oblique direction across the floor of the lateral ventricle, it is followed forward. Sometimes a direct collateral to the internal cerebral vein (the direct lateral vein), crosses the floor of the lateral ventricle 1 or 2 cm behind the thalamo-striate vein. This direct lateral vein must be clearly differentiated from the thalamo-striate vein because it can erroneously lead the surgeon to look for the foramen too posteriorly, under the body of the fornix, through the Velum interpositum.

For tumors developing posteriorly, mainly in the body of the lateral ventricle, this approach is not as convenient, because a part of the venous drainage of these tumors flow posteriorly and goes directly itnto the internal cerebral vein. With the frontal trans-cortical anterior approach, these posterior veins are difficult to control, especially when in addition, the ventricle is small.

The incidence of epilepsy after a frontal transcortical approach in published series (McKissock[74]), is not high, but it must be considered as a risk of this procedure as some of the seizures are very disabling.

The Anterior Transcallosal Approach (Figs. 34–37, 38 a and b)

The patient is placed in the supine position, the head fixed in a vertical direction. The skin flap is bilateral or unilateral but always across the midline. The bone flap is turned slightly more posteriorly than for the

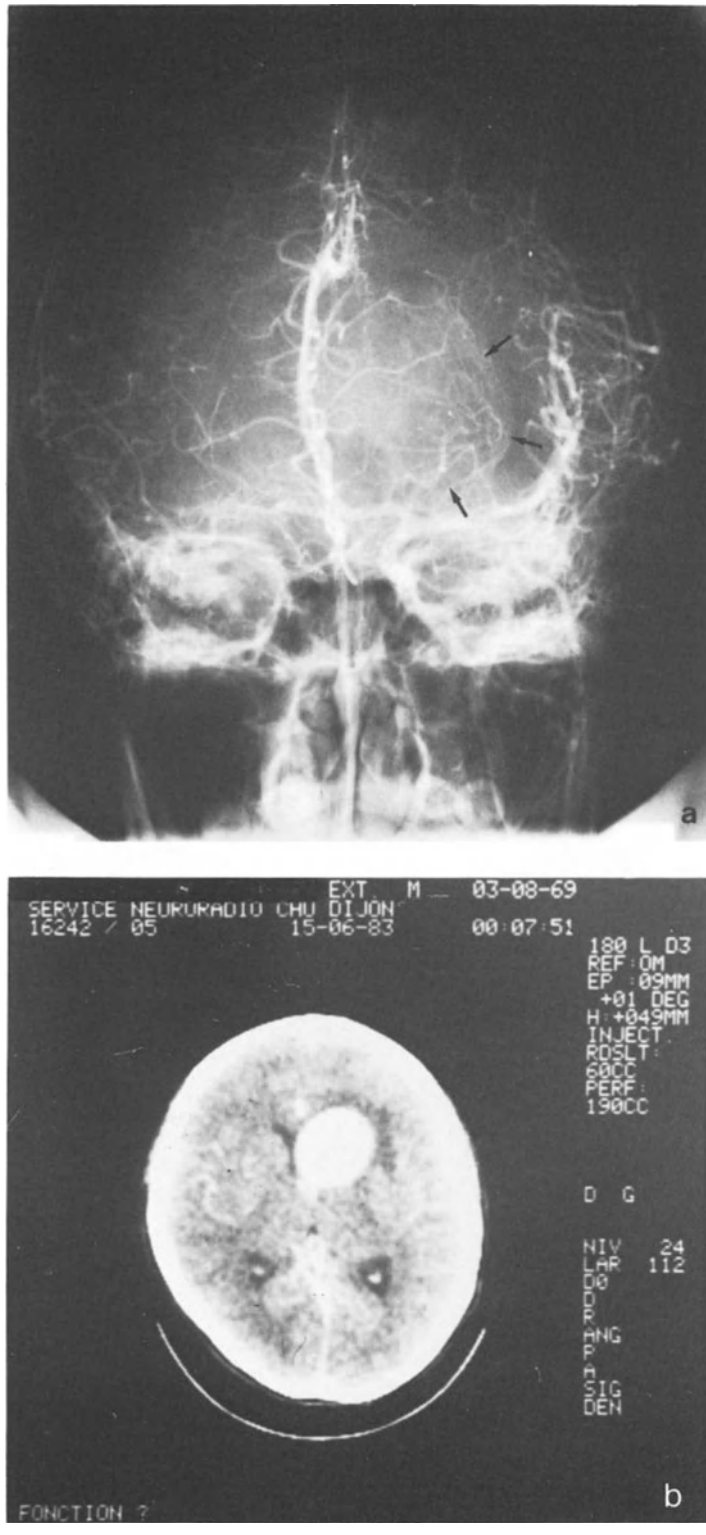
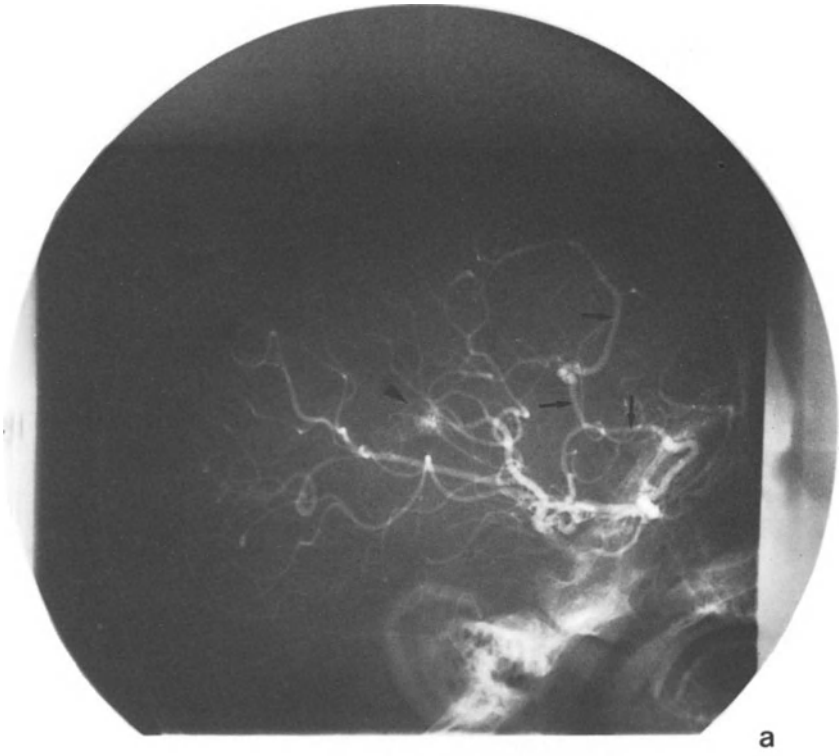


Fig. 31. Malignant lymphoma of the left frontal horn. a) Carotid angiography, AP view. Shift of the pericallosal artery. Lenticulostriate arteries are displaced and enlarged (arrows). b) Enhanced CT appearance



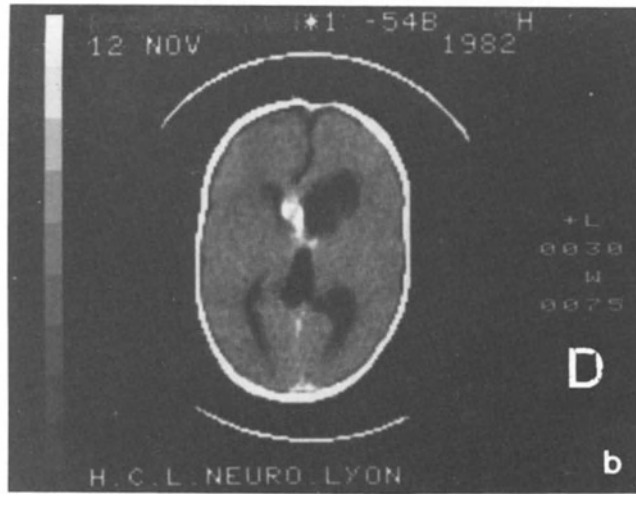


Fig. 32. Septal ganglioglioma. a) Carotid angiography. Note the area of neovascularity (arrowhead) and the atypical course of the pericallosal artery (arrows). b) Post-contrast CT: calcified enhancing mass

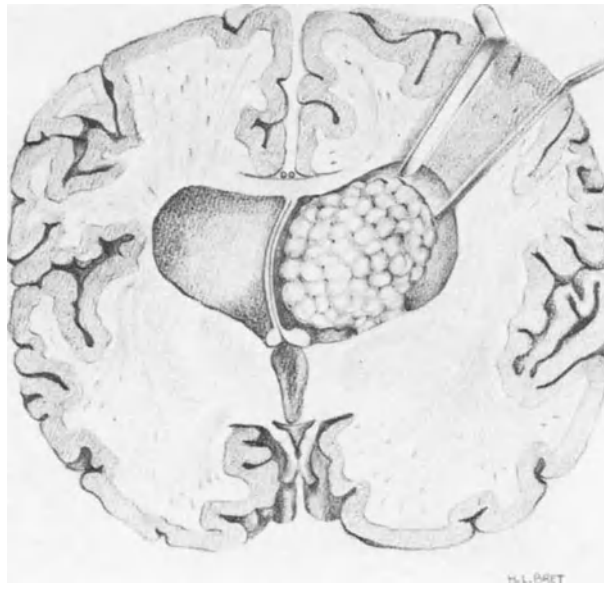


Fig. 33. Anatomical representation of the transcortical approach for a right ventricular meningioma. Operative approach is through the right frontal cortex

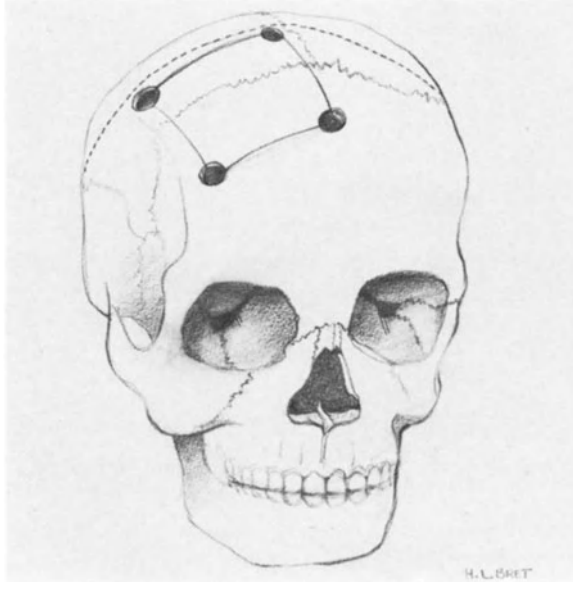


Fig. 34. Transcallosal approach. Skin incision (dotted line) and craniotomy for a transcallosal approach to the right ventricle

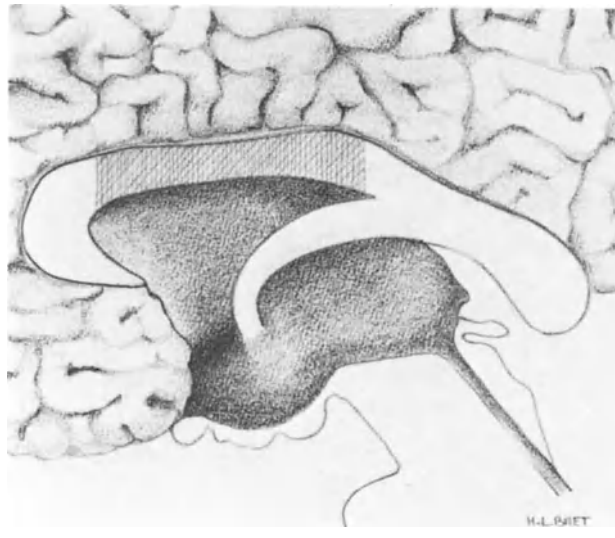


Fig. 35. Transcallosal approach. Sagittal section through the corpus callosum. The shaded area is indicating the portion of corpus callosum which has to be incised to provide exposure of the frontal horn and of the ventricular body

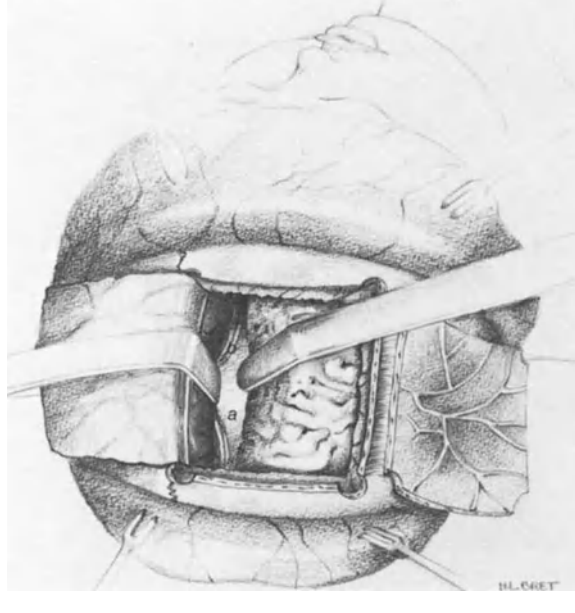


Fig. 36. Transcallosal approach. *a* Corpus callosum exposed through the interhemispheric fissure. *b* Both pericallosal arteries are preserved

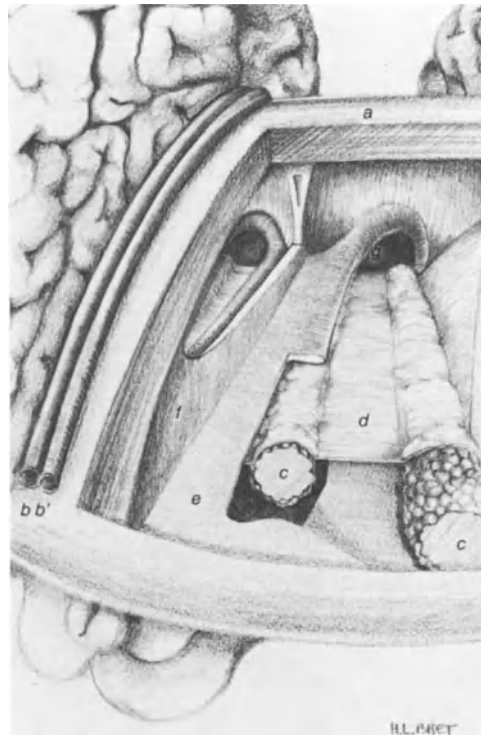


Fig. 37. Transcallosal approach. Anatomical presentation of the normal right lateral ventricle following incision of the corpus callosum. *a* Corpus callosum (cut edge). *b b'* Pericallosal arteries. *c* Choroid plexus. *d* Velum. *e* Trigone. *f* Septum pellucidum (incised). *g* Foramen of Monro

frontal transcortical approach; it is centered on the coronal suture. It is cut exactly to the midline. Often the superior sagittal sinus is partially uncovered. Before opening the dura, it is attached to the bone along the midline with 3/0 sutures to avoid tearing the wall of the sinus. The dural flap is reflected toward the midline.

The bridging veins are divided along the exposed midline as necessary.

Throughout the procedures, we must take care to preserve the bridging veins of the motor cortex. These veins are situated more posteriorly and often concealed posterior to the bone opening. It is useful to wrap these veins with a piece of surgicel. This coating reinforces their wall and prevents them from being torn.

The frontal lobe is retracted laterally. Going downward progressively some small bridging veins from the medial cortex to the falx or to the inferior sagittal sinus are discovered and divided. Before reaching the corpus callosum, the calloso-marginal artery is located under the arachnoid, followed by the pericallosal arteries. In the beginning, it is difficult to know

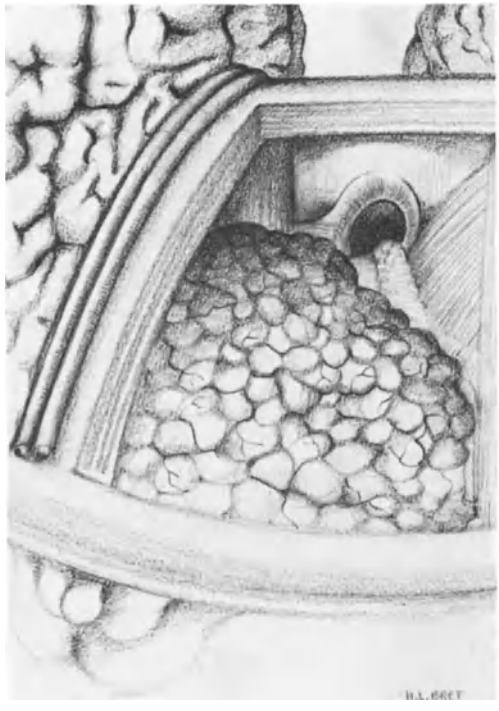


Fig. 38a. Anatomical representation of a choroid plexus papilloma as exposed by the transcallosal approach

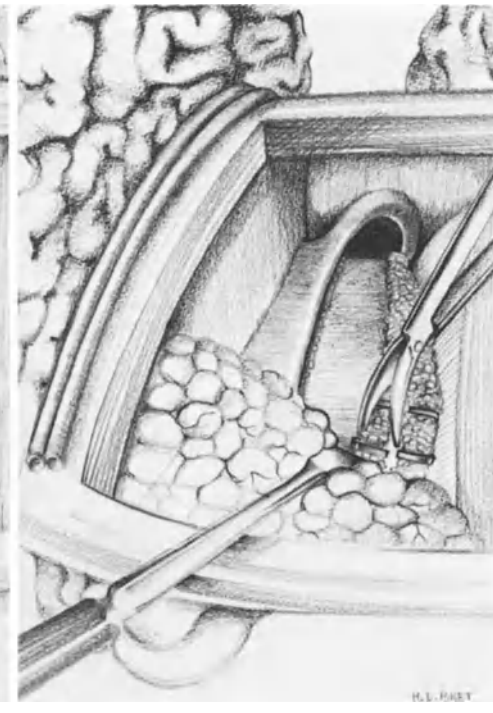


Fig. 38b. The mass is gently retracted with a spatula to expose the normal choroid plexus. Choroid plexus are clipped and divided

exactly where the right and the left artery are. It is useful to dissect the arteries for a few centimeters and to see some of their branches.

When there has been no preoperative angiography, we must be aware of some anomalies like a single pericallosal artery sending branches to both sides. The peri-callosal arteries may either be separated or retracted after microsurgical dissection. Sometimes it may be necessary to abandon a transcallosal approach and to resort to a transcortical procedure. This change in approach might be necessary if the anatomy of the draining veins or the pericallosal vessels is unfavourable or if there are dense inter-hemispheric adhesions.

It is important to preserve the pericallosal gyrus or cingulate gyrus bilaterally, especially when the section of one fornix is foreseen. It is important to dissect the pericallosal sulcus as far as possible laterally to open the roof of the appropriate lateral ventricle. Opening the corpus callosum near the midline, the danger is to enter the wrong lateral ventricle or through the septum, especially when a tumor has displaced these

structures. Working through the coronal bone flap it is possible to dissect the greater part of the corpus callosum: the genu and the body. It is not advisable to open the corpus callosum too far anteriorly or posteriorly.

Section of the corpus callosum is begun with a fine spatula and completed with the sucker tip. Generally an opening of 5 cm in length is enough to get a satisfactory exposure of the ipsilateral frontal horn, the foramen of Monro, and the body of the lateral ventricle. Even when the ventricle is collapsed, it is easily exposed.

This approach has the advantage of avoiding a cortical incision and lessens the danger of epilepsy. It is particularly useful for tumors of the body of the lateral ventricle and of the midline especially when the ventricle is small.

It has the disadvantage of sacrifice of a significant part of the corpus callosum which may be functionally valuable. Many authors have reported use of this approach without neurological sequelae: Greenblatt[47] advocated the transcallosal approach in preference to the transcortical route; more recently Long and Chou have used this route without neurological complications. Gordon reported on the absence of the so-called disconnection syndrome after commissurotomies involving the genu and anterior 50 mm of the corpus callosum and also the anterior commissure. In contrast, Jeeves[57] reported three patients who underwent transcallosal surgery years earlier, tested by procedures designed to demonstrate interhermispheric transfer of information. They were all found to have defects in transfer of tactile data, but not of information obtained visually. One patient had a deficit in short term acquisition of new information (repeated digit and spatial sequences). The patients were not aware of the inability and did not appear to suffer much, if any, inconvenience from this subtle incapacity. Risse[57] observed a lack of transfer of tactile information, a deficit in finger differentiation and localization of touch, predominant on the left side, but the patients did not appear inconvenienced by this inability.

After a more complete and anterior lesion of the corpus callosum, Zaidel and Speery[57] observed marked impairment of recent memory, perhaps reflecting the additional section of the callosal genu, the anterior commissure and the hippocampal commissure.

When the commissurotomy extended posteriorly, Dimond noted severe disturbances of somatic and visuospatial transfer. The patients reported a deficit in finger differentiation, transient impairment of short term verbal memory, no impairment of visual memory but impairment of memory of the patient's own actions (auto-pragmatic amnesia).

More posteriorly, complete section of the splenium of the corpus callosum in a right handed patient who already has a right homonymous hemianopsia will always lead to postoperative alexia (Greenblatt[47]).

In conclusion, the transcallosal route, when restricted to the anterior half

of the body of the corpus callosum seems convenient; the neurological sequelae seem acceptable and the patients do not appear inconvenienced by them. Anteriorly the section must spare the genu of the corpus callosum especially near the rostrum and the anterior commissure. Posteriorly it is not advisable to go beyond the interparietal commissure, the splenium must be preserved.

The Parietal Transcortical Approach

This is the most convenient approach for tumors of the posterior part of the body of the lateral ventricle or trigone. The patient lies in the lateral position, the head turned and slightly elevated. The skin flap is centered just behind the transition between the second and last third of the cranial vault, on its most apparent convexity, clearly behind the inter-aural plane, and in front of the lambdoid suture. It is centered 4 cm above the general direction of the middle cerebral artery on the left; on the right side, it is possible to go lower. The skin flap should be large enough to see the sagittal suture so as to know exactly how far from the midline is the cortical incision. We recommend 2 to 3 cm. It is not necessary to expose the superior sagittal sinus. Generally, it is useful to turn a large bone flap, allowing retraction of the cortex and avoiding compression of the brain between the cranial vault and the retractor.

The cortex is opened between the main veins through a linear incision or a small cortical resection. The operative route descends obliquely through the cerebral mantle, inferiorly medially and anteriorly, directed toward the middle of the baseline between the external canthus and the external auditory meatus. Sometimes, when looking for the ventricular cavity, it is useful to tap it to find where it is displaced by the tumor. After tapping, the needle is left in place, as a guide to help the dissection. The atrium of the lateral ventricle is opened at its postero-superior border.

Tumors developing in this part of the ventricle, completely fill the cavity. The main arterial feeders of these tumors come from branches of the anterior and posterior choroidal arteries, near the hilum of the choroid plexus or along its insertion. Often the choroid plexus itself adheres to, or includes the tumor. This means that the piece-meal resection of the tumor is performed prior to controlling the arterial pedicle on the posterior angle of the thalamus; it is dangerous to pull on the tumor, because of the risk of tearing the vascular pedicle.

The venous drainage of these tumors is in three main directions:

— directly through the medial cortex and the velum interpositum to the vein of Galen;

— anteriorly along the ependymal wall of the body of the ventricle to the venous confluence near the posterior border of the foramen of Monro;

— inferiorly to the medial part of the temporal horn and the basal vein of Rosenthal.

Thus the only venous danger is at the posterior border of the thalamus, near the midline, especially when the tumor has a short venous attachment to the vein of Galen.

This approach is most frequently used for posterior tumors of the lateral ventricle. It is seldom complicated by neurological deficit. It is performed behind the parietal somato-sensory area and not normally complicated by astereognosia. It is far from the optic radiations to avoid hemianopsia. It is situated above the inferior parietal lobule and the angular and supra marginal gyri where intermodal associations are carried out for visual auditory language connections.

Castaigne[18,19] has however reported a case of visual apraxia after an approach to the right trigone through the parieto-occipital junction. The patient experienced visual motor dissociation on the other side without hemianopsia due to a loss of visual control of the fore limb after interruption of the ipsilateral occipito-frontal or cortico-cortical connections. These connections support the mechanism of visual guidance of fore limb movements or more precisely for independent hand and finger movements.

The parietal transcortical approach is preferable to the posterior transcallosal approach, because this does not give access to the lateral ventricle, but to the roof of the third ventricle through the internal cerebral veins in the membrana tectoria, as shown by Dandy[23]. At this level the lateral ventricle turns laterally and is too far from the midline to be easily approached through the corpus callosum. In addition section of the posterior corpus callosum is sometimes complicated by neurological deficit, especially in transference of visual information. The transcallosal posterior approach is used only as a complement of the transparietal approach to achieve complete removal or to control the venous pedicle of large tumors extending near the midline.

The Temporal Transcortical Approach

The head is turned 30° toward the other side. After a lateral fronto-temporal skin flap, elevation of the temporal muscle, a free bone flap is delineated from the pterion to a point 2 or 3 cm behind the external auditory meatus, near the base of the skull. It is preferable to saw the four borders of the bone flap rather than to break the lower one, because adhesions are sometimes found between the dura containing the lateral sinus and the bone at the level of the venous angle.

When the temporal lobe is exposed, four landmarks are noted: the superficial Sylvian vein, the vein of Labbe, the temporal veins between the inferior part of the temporal lobe and the petrous bone, the projection of the

external auditory meatus. The superficial Sylvian vein shows the upper-limit of the temporal lobe.

The three remaining landmarks indicate approximately the posterior limit of temporal resection to avoid homonymous hemianopia or speech disturbances. They are not exactly in the same plane. The vein of Labbé is the most posteriorly situated but not far behind the external auditory meatus; the temporal veins are slightly more anterior. Concerning the danger of hemianopia, the vein of Labbé is a reliable landmark, but it is not easy to avoid speech disturbance. On the dominant hemisphere it is preferable to limit the posterior resection of the temporal lobe to the level of the temporal veins. The anterior third of the temporal lobe is also a valuable guide.

Tumors developing in the temporal horn are removed through a cortical incision on the T 2 or T 3 gyrus, between the Sylvian fissure and the base of the skull, limited posteriorly by the temporal veins. Often these tumors extend to the medial structures of the temporal lobe. It is easier to perform a resection of the anterior third of the temporal lobe than to continue deeply through a simple linear incision. The resection opens the tip of the temporal horn and shows the exact medial limits of the deep structures, avoiding injury to the upper brain stem. The free edge of the tentorium is also a good indicator of this medial limit. Posteriorly, resection of the deep structures (hippocampus and amygdala) is possibly safer than removal of the superficial cortex on the dominant side.

Above the tentorial notch the choroidal, posterior communicating, and posterior cerebral arteries are visible through the arachnoid of the basal cistern. They may send direct branches to intraventricular tumors, in addition to the arterial pedicles descending from the middle cerebral artery. Venous drainage is partly to the superficial veins, and partly to the basal vein of Rosenthal.

Although tumors of the anterior part of the temporal horn are easy to remove by this approach, the problem is more difficult for tumors of the posterior part of the temporal horn, which extend partly into the trigone. On the right side it is possible to extend the temporal incision posteriorly or to resect the cortex with the only consequence a left hemianopia, but on the left side the speech area is involved. Posterior removal has to be performed under the cortical speech area, by elevating it with retractors. Sometimes it may be useful to approach the tumor by two routes (a transtemporal and a transparietal to the trigone) to achieve good hemostasis and total removal.

Technical Variants

When a tumor extends into the third ventricle, especially the anterior part, neurosurgeons have usually enlarged the foramen of Monro by sectioning the ipsilateral column of the fornix anteriorly and superiorly.

Whether this can result in disabling loss of short term memory is a controversial matter. Memory impairment is more likely, if either the tumor or the operation also compromises the contralateral fornix. In 1951, Cairns and Mosberg reported 7 out of 8 surving patients after unilateral fornicotomy without apparent memory dysfunction. In 1974, Little and Mc Carty reported 5 out 6 patients operated by dividing the anterior pillar of the fornix with no memory or personality disturbances. The memory deficit produced by unilateral anterior fornicotomy may, however, be subtle. Because the majority of patients reported did not undergo psychometric testing to discern the difference between intelligence quotient (IQ) and memory quotient (MQ), we are unable to substantiate the claim that unilateral interruption of the fornix is never associated with a permanent, albeit small, memory deficit (Lavyne)[69].

Concerning bilateral fornicotomy, evidence is accumulating to suggest that it is frequently, if not always, complicated by memory deficit. In 1954, Garcia-Benchoa reported on the effect of 12 bilateral fornicotomies with no unfavourable neurologic or psychiatric sequelae. A similar experience was described by Umbach in 1961, who reported 5 bilateral fornicotomies for epilepsy without lasting disturbances of memory. Sweet *et al.* in 1959, however, were the first to document the short term memory deficits created by bilateral interruption of the columns of the fornix. Their patients developed a differential between IQ and MQ. The same experience was reported by others (Talairach, Pecker[89, 90, 91]).

It is generally agreed at the present time that section of both columns of the fornix or of one column when the contralateral temporal lobe, contralateral pericallosal gyrus, or any part of the limbic system on the other side is injured, is to be avoided.

The same experience has been reported after a bilateral posterior fornicotomy. Posterior interruption of the fornices results in a permanent amnestic state with an IQ-MQ disparity of at least 30 points (Heilman and Sypert).

After transcortical or transcallosal exposure of the anterior horn of the lateral ventricle when a tumor extends into the third ventricle, especially its medial part, it is possible to use an alternative to anterior fornicotomy. This second option is to enlarge the foramen of Monro posteriorly. This route was described by Hirsch as the "interthalamo-trigonal approach" and later by Lavyne and Patterson as the "subchoroidal transvelum interpositum approach". This approach is conducted behind the foramen of Monro after coagulation and section of the thalamo-striate vein. A blunt spatula is introduced into the foramen of Monro and directed backwards under the choroid plexus. This approach is not apparently associated with any sequelae[54—69]), it is simple and gives a good view of the third ventricle.

In infancy, some malignant tumors (ependymoblastomas or choroid

plexus carcinomas) fill the ventricular cavity and have a particularly rich vascular component. They behave like sponges filled with blood. As the dura is opened, the cortex bulges and the blood pressure falls. This sudden and marked variation of blood pressure is related to the blood filling and enlarging the tumoral vascular bed. When this phenomenon is observed, a tumor removal is risky because of large, numerous dilated veins in and around the tumor. We observed operative deaths only in these cases. It would be preferable to perform biopsy only and delay tumor removal for a second stage.

When surgery is performed in the anterior part of the lateral ventricle and the Foramen of Monro is easily seen and opened, it is useful to block the foramen temporarily with a cotton pattie to prevent leakage of blood into the third ventricle and Aqueduct.

When the Foramen of Monro is not easily seen, a fenestration must be made through the septum lucidum to explore the opposite lateral ventricle and to mark the relationship to its foramen. The same fenestration of the septum lucidum, preexisting or surgically made, is useful when the Foramen of Monro is obstructed by an invasive tumor.

When the tumor is located near the Foramen of Monro or behind it, post-operative oedema can interrupt, at least temporarily, the flow of CSF through the inter-ventricular foramen, or blood can partially obstruct the aqueduct. If the frontal boneflap is large enough, it is possible to retract the frontal lobe, dissect the chiasma, and to open the lamina terminalis. This sometimes prevents post-operative intracranial hypertension.

When the tumor is responsible for marked hydrocephalus with a thin cerebral mantle, especially in childhood, it is necessary to support the cortex with malleable retractors to prevent collapse during surgery and tearing of the bridging veins, sometimes far from the operative field. At the end of the procedure, the ventricular cavity is refilled with fluid, and if possible, the cortical incision is approximated by one or two thick threads to prevent post-operative accumulation of subdural fluid. When the surgical approach can be delayed, a preoperative shunt can decrease ventriculomegaly and decrease the risk of post-operative subdural hematoma.

When hydrocephalus is not marked but ventricular drainage is needed, it is preferable to insert it after the surgical removal of the tumor. A shunt before surgery reduces the size of the ventricles and makes tumor removal more difficult. Post-operative external drainage is better than an internal shunt since blood in the CSF post-operatively can obstruct the catheter. When the approach is anterior, it is useful, at the beginning of the procedure, to prepare the skin for draping to enable a burr hole to be placed over the opposite frontal horn to insert a drain. A lumbar drain inserted through a needle is another alternative.

5. Results

Personal Experience

Case Material

Between 1967 and 1983 75 cases of tumors of the lateral ventricles have been operated upon. (Service Neurochirurgie B, Hôpital Neurologique, Lyon.) Some of these cases have previously been reported[67]; 52 were children (25 infants); 23 were adults (13 had more than 30 years). In childhood tumors of the lateral ventricle represent 9.1% of all intracranial tumors operated on in the Department, and 24.4% of supra-tentorial tumors. In adults they represent only 1.6% of all intracranial tumors. Pathology is given in Table 2.

Astrocytomas or ependymomas comprise the majority of these tumors. We found true papillomas of the lateral ventricles to be rare. Meningiomas are more frequent. A wide variety of other tumors were observed, each in small numbers.

Three cases were associated with Bourneville's syndrome (two giant cell astrocytomas, one ependymoma). No cases were observed with von Recklinghausen's syndrome.

Localization of these tumors is given in Table 3. Two thirds of the ependymomas (the majority malignant) were situated in the posterior part of the lateral ventricle; only one out of four ependymomas (the majority benign) was situated in the frontal horn. All the meningiomas were localized in the posterior part of the lateral ventricle. Among tumors of the septum, we found seven astrocytomas, one oligodendroglioma and one ganglioglioma.

Deaths

26 patients (35%) have died during a follow-up period ranging from 1 to 15 years. 2 patients died during surgery; both were infants with large malignant vascular tumors invading the greater part of the lateral ventricle. 5 patients died during the first post-operative month, 11 cases during the first year, and 8 cases after some years. 8 other cases have been lost for follow-up after 1 year.

10 out of 30 patients with ependymomas (33%) have died, but only 2 out of 15 with astrocytomas (13%). One of 7 operated meningiomas died in the early post-operative period.

Radiation Therapy

29 patients have been irradiated after surgery, the majority had a malignant ependymoma or glioma, but we have also irradiated, after partial

Table 2. *Pathology*

Ependymomas	30
Astrocytomas	15
Meningiomas	7
Malignant Gliomas	5
Oligodendrogliomas	4
Reticulosarcomas	4
Choroid plexus papillomas	3
Metastasis	3
Neuroblastomas	2
Germinoma	1
Ganglioglioma	1
	75 cases

Table 3. *Intraventricular Localization*

Frontal horn	16
Temporal horn	11
Atrium	30
Septum	9
Body of the ventricle	6
The entire lateral ventricle	3
	75 cases

surgical removal, 5 benign astrocytomas with good results. The average dose was 5,000 rads. 4 of these 5 patients are still alive 2 to 5 years later.

We have had rather bad experience of radiation therapy in children under two years of age, even with low doses, ranging between 3,000 and 4,000 rads. Of 4 infants irradiated immediately after surgery, one died 5 months later from extension of the tumor, one is alive and not handicapped after a follow-up of 4 years, but 2 other infants also surviving for some years after radiation (a neuroblastoma and a malignant ependymoma) are badly handicapped. In infancy, radiation therapy may be efficient to cure malignant tumors, but is also dangerous for the developing brain, even in low doses.

Considering this danger, we have decided to delay radiation therapy for one or two years in the treatment of 6 infants. 4 of these died from recurrence of the tumor between 6 to 18 months. One is alive well, without handicap, 2 years after the total removal of a malignant ependymoma. He never needed radiation. The last one had a recurrence of a malignant ependymoma one year after radical surgery, was submitted to a second

operation when only a partial removal was possible. Postoperative radiation therapy and chemotherapy were used after this second stage procedure but the patient died from extension of the tumor 2 years later.

Second Operations

9 tumor recurrences have been operated upon (2 astrocytomas, 4 ependymomas and 3 ependymoblastomas); 4 patients are still alive with a follow-up ranging from 1 to 5 years, 5 are dead. It seems advisable to consider second stage surgery in some favourable cases.

Results in the Literature

Ependymomas

Goutelle and Fischer[46] reported on 48 ependymomas of the lateral ventricles. They observed 13 deaths during the first two months, 11 deaths before 1 year and 9 deaths after 1 year; 33 patients of 48 intraventricular ependymomas (69% mortality). Compared to the intra-parenchymatous supratentorial ependymomas (28 patients dead out of 54 operated on 52%) intraventricular tumors appear to be a more serious surgical problem. Results are different whether or not radiation therapy is used: 20 dead out of 24 patients treated by surgery alone (83% mortality), 14 dead out of 24 patients treated by surgery and radiation therapy (58% mortality).

Obrador[85] reported 33 cases, representing 46.8% of tumors of the lateral ventricle. Total removal was possible in 12 cases. The early mortality was 42%.

Bartlett[8] noted that the five year survival varied in different series between 15% and 80%. Favourable factors appeared to be operation with radiotherapy but it seems certain that the character of the tumor also plays an important part. Cystic tumors and those in older patients are often associated with longer survival, contrasting with solid tumors of childhood where the prognosis is universally bad.

Meningiomas

According to the studies of Dandy (1934), Ladenheim (1963), Delandsheer (1965), Kobayashi (1971), and Obrador (1972), surgical mortality lies between 15% and 20%. Functional recovery appears to be independent of tumor size. Two thirds of patients can expect a good functional result. Dysphasia is usually the most serious handicap.

Choroid Plexus Tumors

Laurence[68] collected 74 cases reported in the literature and treated by radical removal, of which 52 were successful. In 18 cases only partial

removal was possible; 13 of these patients died within a short time of the operation. 5 patients who survived from 9 months to 8 years were severely handicapped or retarded. Concerning carcinomas of the choroid plexus only 6 cases out of 38 recorded by Laurence seem to have survived after surgery with follow-up ranging from one month to 11 years.

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Traumatic, Spontaneous and Postoperative CSF Rhinorrhea

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Contents

Introduction

The paper is based on the experiences which have been collected and analyzed from a total of 237 cases in the Neurosurgical Departments at the University Clinics La Pitié, Paris, and Homburg/Saar. The senior authors are the Heads of these Departments. Both Departments have different traditions according to the different history of French and German neurosurgery. As might be expected the senior authors did not agree on all points. The divergencies of opinions and policies have not been concealed but considered important and openly discussed in our paper in order to give an as large a view as possible. In the end we feel that with regard to rhinorrhea the old proverb remains true, more than one road leads to Rome.

The paper deals only with rhinorrhea. Otorrhea which also mostly occurs as a consequence of head injuries, only exceptionally needs neurosurgical operative treatment and is therefore not included in this chapter.

Rhinorrhea is the leakage of the cerebro-spinal fluid, colourless and transparent as water, from the basal cisterns or the frontal or rarely the temporal horn of the ventricle, into the nasal cavities through a defect of the frontal, ethmoidal or sphenoidal bones. Very occasionally CSF may gain entrance to the eustachian tube from a lesion of the petrous bone and, if the tympanic membrane is intact, drain to the nose. This rare phenomenon, called oto-rhinorrhea, will not be described in this paper.

Rhinorrhea in about 80% results from traumatic lesions. The rare non-traumatic rhinorrhea can be caused by tumours, malformations or infections of the skull base or occur as a consequence of the so-called empty sella syndrome. It can also be the consequence of neurosurgical or ENT operations.

Meningitis is the main risk of an open connection to the CSF spaces. To prevent this dangerous complication all those CSF fistulas which by experience are not likely to close spontaneously and permanently, have to be closed operatively.

Answers to the following questions are therefore of special importance:

How can the diagnosis and the precise location of a CSF fistula most effectively be made?

What cases have to be operated upon and what is the best timing for the operative treatment?

What is the best operative procedure for any given pathology?

What are the results and risks of operative treatment?

Our aim is to give practical advice to younger neurosurgeons, to outline the standard. In order to remain didactically clear and to be practical we do not intend to give a complete literature review.

Historical Notes

A well-documented history of CSF fistulas has been published by McGee in Volume 24 of the Handbook of Clinical Neurology in 1976.

Charles Miller was the first to demonstrate in an anatomical specimen that CSF might escape spontaneously through the nose. He stated in 1826: "The opening through which the water had distilled into the nostrils was a foramen above and to the right of the crista galli; it might have admitted from bristles and had a direct communication with the nasal cavity." Already in 1847 Robert showed that the chemical composition of the fluid which in certain skull fractures pours out was identical with that of CSF.

The first clinical report of a case of rhinorrhea was given in 1877 by Tillaux.

Dandy was probably the first to report in 1926 successful surgical closure of a CSF fistula using an autogenous fascia lata graft. The report of the first larger series of surgically treated cases (53 cases of the New York Hospital-Cornell Medical Center: Experiences from 1932 to 1967) was published as lately as 1969 by Ray and Bergland.

Causes of Rhinorrhea

About 80% of all CSF fistulas are caused by head injuries with skull base fractures (Tables 1 and 2). Only 3–4% are so-called spontaneous fistulas and about 16% are the result of operations within the nasal and paranasal cavities and the skull base.

A. Traumatic Rhinorrhea

The incidence of rhinorrhea varies in larger non-missile head injury series between 2% (Lewin 1954) and 9% (Raaf 1967). Approximately 150,000 traumatic rhinorrhea cases are yearly treated in the United States, which is about 5% of the estimated 3 millions head injuries per year. A lower percentage—only 3.5%—has been found in children by Einhorn and Mizrahi (1978).

It seems however that all these figures are too high and that in reality, corresponding with the report published by Cooper (1982) and with the experiences of our Departments in Paris and Homburg, the incidence lies around 2–3% of all head injuries and above 11% of patients with skull base fractures.

Fistula locations. A nasal CSF leakage can only occur when all tissue layers which separate the CSF spaces from the nasal respectively paranasal cavities—arachnoid, dura, bone and mucosa—are breached. Thinness of bone and adherence of the dura to the skull facilitate the formation of a fistula. The more frequent fistula sites are thus the cribriform plate, the

Table 1. *Operated Rhinorrhea Cases* (own material)

	Traumatic	Spontaneous	Postoperative
Homburg (1960–1983)	149	4	(numbers not available)
Paris (1975–1983)	68	3	13

Table 2. *Age and Sex in 237 Rhinorrhea Cases* (own material)

	Mean age	Male	Female
Homburg	31.4	78%	22%
Paris	29.6	80%	20%

Table 3. *Traumatic Rhinorrhea: Site of Bone Defect* (own material)

	Frontal and fronto-ethmoidal	Ethmoidal	Ethmoidal-sphenoidal
Homburg	47%	49%	4%
Paris	42%	42%	16%

posterior wall of the frontal sinus and, much less frequent, the jugum sphenoidale (Table 3), the sella floor or the great sphenoidal wing in cases with large lateral development of the sinus. In one single case of a young child a permanent fistula resulted from a fracture through the epiphyseal plate of the clivus. There is no predominance of frontal sinus fractures, in spite of the fact that such a predominance is often postulated in the literature.

The *kind of fracture* is of importance, whether linear or compound (Table 4). Over *compound fractures* the dura is mostly badly torn, often with actual loss of substance. Bone fragments may have penetrated into the frontal lobe, the sinuses or the nasal cavity. Dislocations of the crista galli and of the vomer are not rare findings. If in such cases early surgery is advised from one or another reason, then a conglomerate of dura, bone, mucosa and destroyed brain tissue is evident all admixed with blood clot. The large communication may result in profuse CSF leakage which may

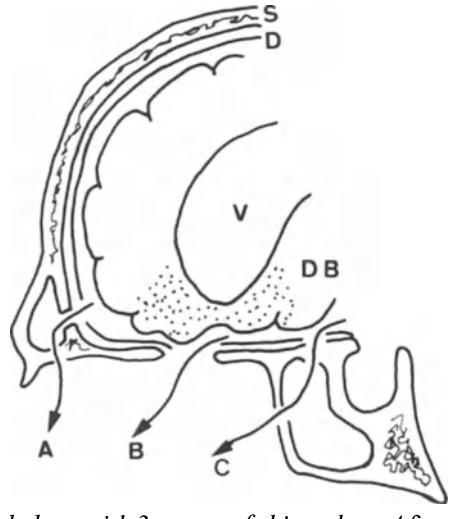

Fig. 1. *Sketch of a sagittal plane with 3 causes of rhinorrhea. A* frontal sinus fracture. *B* ethmoidal defect. *C* sphenoidal fracture. (*S* skull, *D* dura, *V* ventricle, *DB* damaged brain)

Table 4. *Type of Fracture in Traumatic Rhinorrhea Cases* (own material)

	Compound	Linear
Homburg	55%	45%
Paris	34%	66%

have disastrous consequences caused by severe intracranial hypotension or—in small children—electrolyte fluid balance disorders. Usually air enters the skull cavity through the large connection.

More often the mass of clot, bone and brain initially prevents leakage, which manifests itself only when the lysis of the clot begins after one to two weeks, unless arachnoiditis in the meantime has isolated the traumatized area from the cisterns. Even then leakage can develop or meningitis occur at a later period.

The *late appearance of a CSF fistula* can be provoked for two main reasons: by an elevation of the intracranial pressure—for instance development of a posttraumatic hydrocephalus—and by an enlargement of the frontal ventricular horn as consequence of a frontal lobe contusion. In such cases even a direct communication between the ventricular system and the nasal or paranasal cavities may be established.

Linear fractures may be the cause of CSF fistula if the dura is cut at the

same site as the bone. Because the dura is adherent to the bone its edges are not always able to join for spontaneous repair.

The *appearance of a CSF fistula* occurs in about 55% by the first or second posttraumatic day, in about 15% after one week when brain oedema diminishes, and in about 10% later, up to more than 25 years. In the remaining 20% CSF rhinorrhoea has never been apparent and meningitis is the first manifestation of pathological communication between the intracranial and extracranial spaces (Table 5).

Table 5. *Traumatic Rhinorrhea: First Clinical Symptoms*[1] (own material)

	Rhinorrhea R	Meningitis M	Aerocele A	R+M	R+M+A	R+A	A+M
Homburg	49	14	5	9	5	15	3
Paris	58	13	6	12	5	5	1

[1] Figures in %.

In three cases in the Paris Department, and in one case in the Homburg Department *bullet wounds* have been the cause of rhinorrhea. In such cases, as a rule, the bone is widely shattered, combined with a large dural defect.

B. So-Called Spontaneous Rhinorrhea

This term should be used for nasal CSF fistulas with no evidence of traumatic origin and which are not the result of operation. It also includes cases in which for no clear reason CSF escapes through or along the olfactory nerves (Andrioli *et al.* 1966, Coleman *et al.* 1974, Dandy 1944, Visot *et al.* 1979). Such are cases with high pressure hydrocephalus of markedly raised intracranial pressure from other reasons* (Rovit *et al.* 1969, Little *et al.* 1975), tumours of the base of the skull (Vigouroux 1971,

* In one of the cases of the Paris Department, the leakage occurred one month after removal of a convexity meningioma located in the left coronal area. Preoperatively the patient, a young female, had markedly rised intracranial pressure with papilloedema of 4 dioptries. At surgery in order to close the fistula the surgeon observed an extremely thin cribriform plate with a localized dehiscence of bone and dura. This leakage, in our opinion, was not related to the operation of the meningioma and therefore does not belong to the postoperative fistulas, but to the long-lasting non-hydrocephalic raised intracranial pressure.

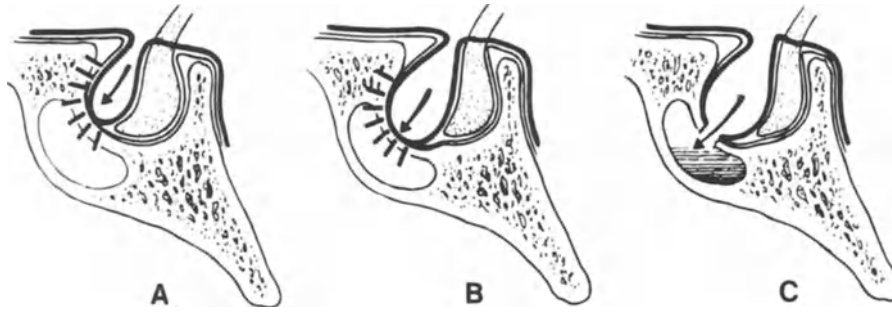

Fig. 2. *CSF fistula in empty sella syndrome.* Schematic drawing of CSF fistula formation with rhinorrhea, due to progressive cortical erosion of the sellar floor by a pulsatile arachnoid intrasellar diverticulum (empty sella syndrome). From de Divitiis *et al.* 1981

Table 6. *Causes of 7 Spontaneous CSF Fistulas with Rhinorrhea* (own material)

	Number of cases	Causes
Homburg	4	1 fistula through the olfactory nerves 3 through defects of the cribriform plate
Paris	3	1 meningocele 2 cases with raised intracranial pressure (1 posterior fossa tumour, 1 convexity meningioma)

Ommaya 1976), ethmoidal encephalomeningocele (Danoff *et al.* 1966) and other sphenoidal defects (Guegan *et al.* 1975, Hooper 1971). The empty sella syndrome (Fig. 2) according to Jordan *et al.* (1977) and de Divitiis *et al.* (1981) is accompanied or followed in about 10% by rhinorrhea, and exceptionally also cases of chronic paranasal sinus infections with bone and dura involvement may produce a CSF fistula (Nori *et al.* 1964) (Table 6).

C. Postoperative CSF Fistulas with Rhinorrhea

These most frequently result from the transsphenoidal approach to pituitary adenomas and are due to intraoperative damage of the pituitary tentorium diaphragma sellae in the attack on the upper tumour capsule. The incidence of this complication is decreasing thanks to better operative technique, the use of the operative microscope, specially designed instruments and intraoperative X-ray control.

Rhinorrhea also may follow the removal of tumours of the base of the skull, especially olfactory groove meningiomas when the cribriform plate has been extensively coagulated during surgery or when the tumour has infiltrated the cribriform plate and invaded the superior part of the nasal air spaces. Similarly and unavoidably a large dura and bone defect remains after removal of an aesthesioneuroblastoma of the anterior cranial fossa and the paranasal sinuses, which should be closed very carefully using a pedicled pericranial flap (Jakumeit 1971). Fistulas may also result from the removal of other benign (for instance osteoma) or malignant tumours of the skull base.

Fistulas less frequently occur during ENT operations within the nasal or paranasal cavities, performed by less experienced or less careful surgeons. When closing such a fistula by the transfrontal approach we have even found in one case a marked fronto-basal brain lesion together with displacement of the anterior cerebral artery into the basal dura and bone defect, fortunately without rupture of the artery (Homburg material).

Diagnosis and Location of CSF Fistula

The main symptom is rhinorrhea. It is easily detected if CSF trickles from the nostrils. Difficulties arise if only a few drops appear in the nose, similar to those of a vasomotor rhinitis, or if a comatose patient lying on his back swallows the leaking CSF but has no fluid coming from his nostrils. It may be very difficult indeed to obtain evidence of CSF fistula in a deeply comatose and intubated patient. An acute meningitis some days, weeks, months or even years after the head injury may be the very first symptom of an open connection to the CSF spaces (Table 5).

Detection of a Hidden CSF Leakage

In any *comatose* head injury patient it is mandatory to look for a hidden rhinorrhea by placing the patient in the lateral position and bending the face downwards.

In a *conscious* patient the possibility of a hidden fistula should be considered for example with regard to a fronto-basal fracture or in cases with meningitis and a history of head injury—a *pressure test* should then be done. The patient is asked to bend down or to lie flat on his face with his head down out of the bed. Pressure is then applied to the abdomen and/or the jugular veins. By this means a CSF leak can often be provoked even in previously latent fistulas. It must be stressed however that, according to Laun (1982), about 55% of traumatic rhinorrhea cases are obvious without any provocation within the first 48 hours, an additional 35% manifest themselves during the first three months and only 10% show a longer

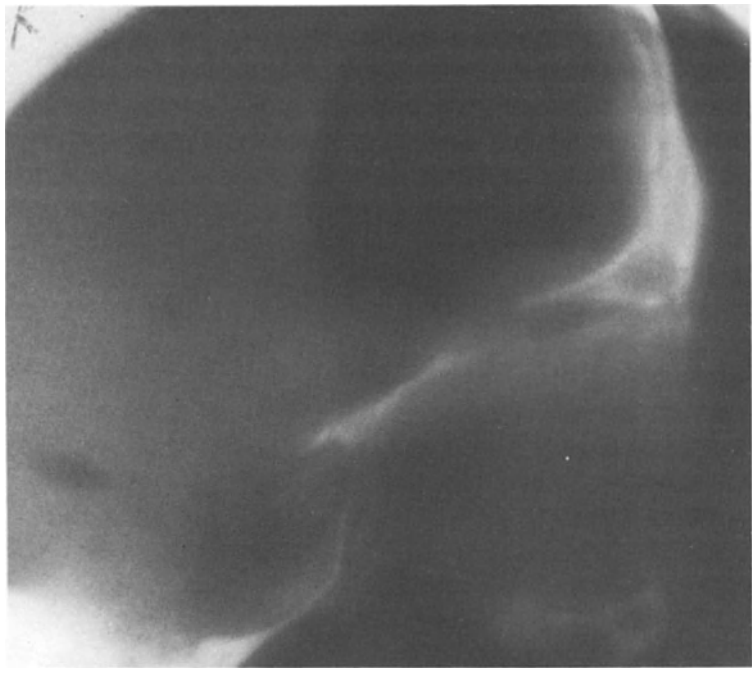

Fig. 3. *Polytomographic lateral view of an aerocele within a frontal lobe together with a bone defect of the cribriform plate*

interval. Laun also reports anosmia or hyposmia in about 60% of the posttraumatic rhinorrhea cases.

The X-ray finding of a pneumatocele sometimes reveals the existence of a fistula. Clinically in conscious patients an aerocele provokes long-lasting headache difficult to relieve adequately with analgesics.

Identification of CSF

Acute and marked rhinorrhea never needs special identification of the escaping fluid, but if only few drops of colourless, transparent fluid are detectable, identification is essential to exclude so-called vasomotor rhinitis.

Glucose oxidase test: This often recommended test, based on the use of a glucose test paper, is unreliable with as much as 75% false negative reactions (Gateholt 1964). It is therefore obsolete.

Immunoelectrophoretical identification of the double arc of transferrin (β_1 and β_2): This is peculiar to CSF; but unfortunately it is not only a painstaking procedure but also needs several milliliters of fluid. It is therefore practically not very useful since the diagnosis of such a profuse CSF leak is already certain without transferrin identification.

Identification using isotope tracers: This method is relatively reliable. It is used mostly in combination with isotope cisternography for precise localization of the fistula site. A radio-active tracer, for instance ^{99m}TC pertechnetate, is introduced into the CSF by lumbar or suboccipital puncture. In cases of fistula the tracer can be detected using cotton pledgets introduced into the nostrils. A silent fistula may sometimes be activated and

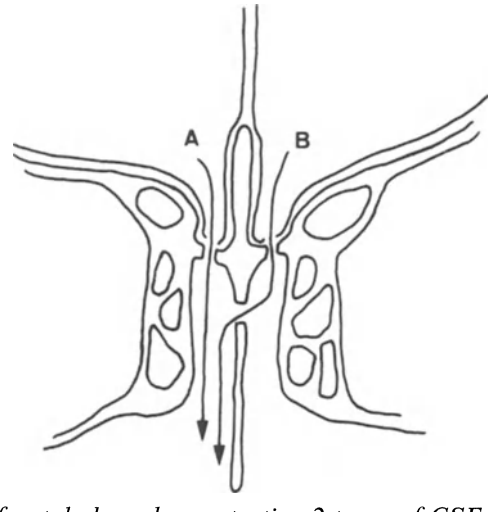

Fig. 4. *Sketch of a frontal plane demonstrating 2 types of CSF fistulas through the lamina cribriformis. A* the leakage is on the same side as the ethmoidal defect. *B* the CSF drops from the contralateral nostril because of a sagittal dislocation involving crista galli and vomer

then identified by increasing the CSF pressure (Spetzler *et al.* 1978) by intrathecal saline infusion to reach a pressure of 600 mm H_20 maintained for at least 15 minutes.

Location of the Fistula

1. From clinical findings: Bilateral rhinorrhea gives no clue to the site of the fistula, but when unilateral, it seems logical that the defect of the skull base should be on the same side. Usually this is true. Paradoxical rhinorrheas do occur however (Pertuiset and Metzger 1983) when the midline structures—crista galli and vomer—are dislocated and CSF can flow through the nostril opposite to the defect. Such a situation can best be predicted from a tomographic study.

2. Plain X-rays: The demonstration of a *linear fracture* of the sphenoidal base is often unhelpful with regard to the fistula site, because these fractures lie mostly in or across the midline.

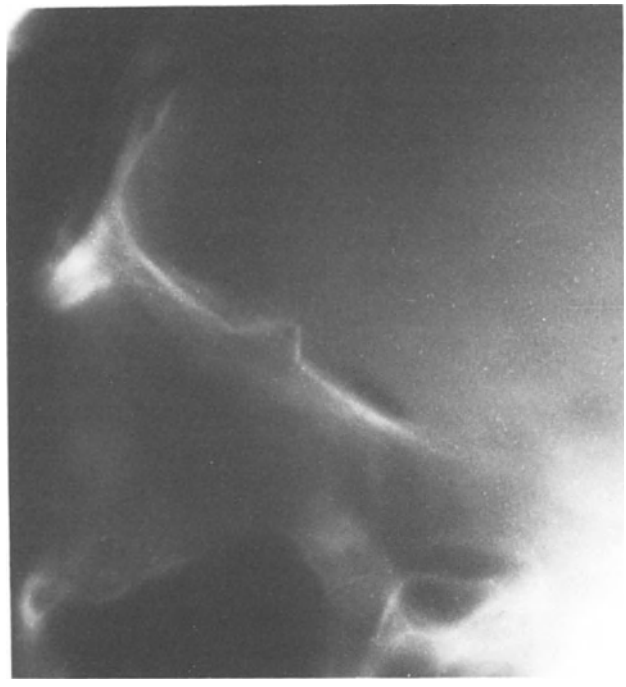

Fig. 5. *Polytomographic picture of a fracture of the cribriform plate with displacement of the bone fragments and epidural aerocele.* A frontal fracture, through the posterior wall of the frontal sinus, is also visible. Such displaced basal fractures are mostly accompanied by a dural tear and need operative repair even when the CSF rhinorrhea has stopped spontaneously. In this special case the epidural aerocele indicates that the dura may have remained intact

The demonstration of a *compound fracture* of the base of the anterior fossa is more helpful, but it has to be stressed that the images never show the exact extent of the damage.

The demonstration of air in the subarachnoid space or in the ventricles gives certain proof of pathological communication, but not always of its precise location. An epidural air accumulation indicates that the dura has probably remained intact (Fig. 3).

3. Polytomography: A complex movement tomograph should be used—if available—since it eliminates the artefacts which are regularly seen on linear tomography of the skull base region.

When there is a *compound fracture,* sagittal and coronal views give a perfect analysis of the fracture, its extent and displacement of fragments. Assessment of the area where the dura has been torn and the fistula is likely to be located is therefore relatively easy and reliable.

In *linear fractures* it is relatively easy to find fracture lines involving the walls of the frontal sinus or the sphenoid. Much more difficult is evaluation

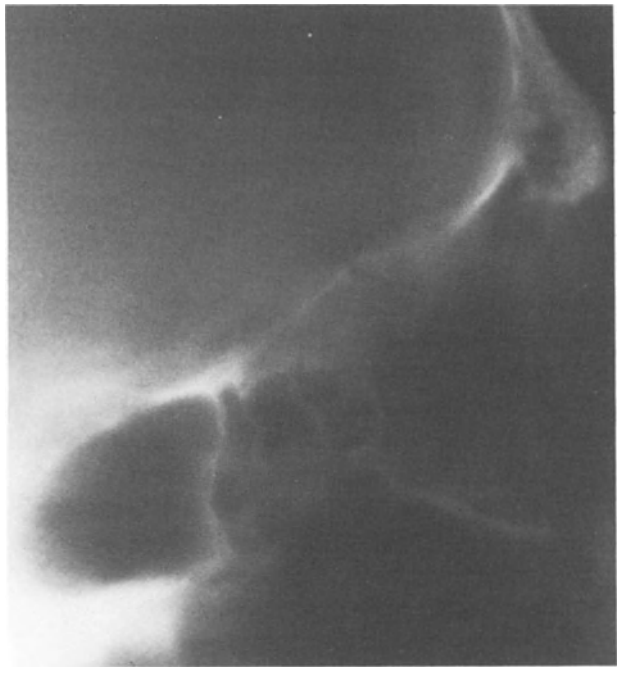

Fig. 6. *Polytomographic picture of a linear fracture of the ethmoidal region.* Such broad fracture lines indicate that a dural tear is most likely. As a rule such cases need operative treatment. An exception may be considered when the CSF fistula stops spontaneously within the first posttraumatic week and when olfactory function is not impaired

of the cribriform plate and the ethmoidal bone because of the complex bone structure of this region. Normal holes may give the wrong impression of fractures. Dislocations of crista galli and vomer on the sagittal plane may be helpful in these cases as well as an opacity of ethmoidal cells on one side only. In non-traumatic cases the poly-tomographic pictures may also give evidence of underlying pathology such as skull base tumour, sellar enlargement or bone destruction from other reasons.

4. Isotope cisternography was introduced in 1964 by di Chiro *et al.*, who gave further reports in 1966 and 1968. A radioactive tracer—for instance low protein RIHSA, ^{99m}TC human serum albumin-TC-HSA or ^{99m}TC pertechnetate— is injected into the CSF spaces. Its distribution is followed by serial scanning or scintiphotography of the head. A silent fistula sometimes can be activated by increasing the CSF pressure (see page 179). In most fistulas the leak can be proved by detecting tracer activity within the sinuses near the site of the fistula. The fistula track is often visible. Otherwise changes of the intracranial tracer distribution give a hint to the location of the leak (Oberson 1976).

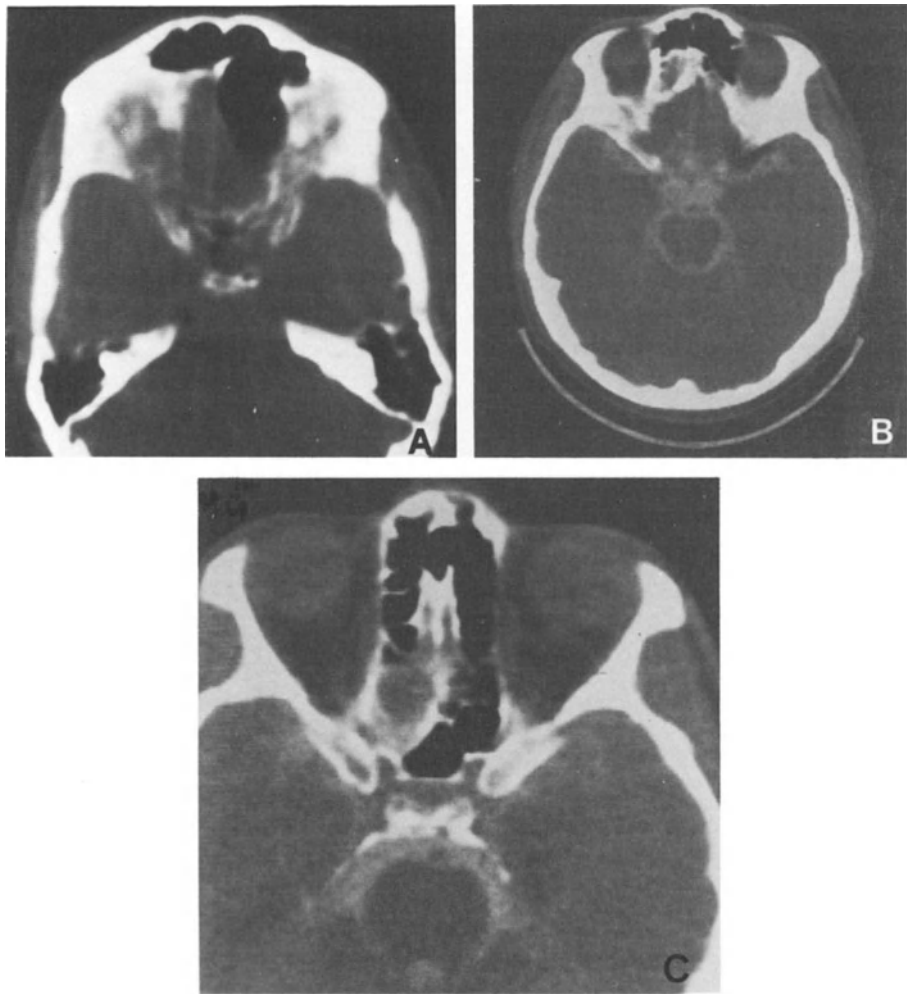

Fig. 7. *Right ethmoidal CSF fistula.* A) Unenhanced CT, showing a shadowing of the right ethmoidal cells, posteriorly also on the left side. B) and C) Metrizamide CT cisternography. The contrast medium fills the basal cisterns. A pathological accumulation, indicating the fistula, can be seen in the right posterior ethmoidal cells

5. CT investigation: This is the most important investigation and strongly recommended as the current mandatory standard.

Plain CT gives an evaluation of the brain's condition. It shows for instance if a frontal concussion is uni- or bilateral, if there is brain oedema, a haematoma or hydrocephalus. It does not demonstrate correctly damage to the skull base unless coronal sections can be done, which may be possible only in conscious, cooperative patients with flexible neck. More modern

scanners however, may enable sufficiently accurate reconstructions to be made in the coronal plane, to be of considerate value. In non-traumatic cases CT will reveal most tumours or other possible causative pathologies.

Above all CT enables *metrizamide CT cisternography* to be performed. This is in fact actually the best diagnostic technique if a sufficiently sophisticated CT is available, it is certainly the method of choice. The fistula will be seen in the sagittal plane after reconstruction.

It necessitates sophisticated CT with the possibility to visualize, on the console screen, the metrizamide at its passage through the fistula out of the skull. Metrizamide CT cisternography was first used by Manelfe *et al.* (1977) and by Drayer *et al.* (1977, 1978) for visualization of CSF fistulas and since then has proved to be very useful (Dohrmann *et al.* 1979, Naidich *et al.* 1980). Five to seven milliliter metrizamide (220 mg/ml) are introduced by lumbar or suboccipital puncture and then brought into the intracranial space by placing patient briefly in an 80° Trendelenburg position. Afterwards serial axial and/or coronal CT images are taken according to the suspected site of the fistula. Again for this method it can be useful to elevate the intracranial pressure by lumbar saline infusion.

6. Positron emission tomography with 68 Gy EDTA has been used by Bergstrand *et al.* (1982) with limited experience. It is certainly too early to evaluate the efficiency of NMR with regard to the location of CSF fistulas, this method, till now, is available at very few departments. Its ability to produce excellent sagittal plane images and the fact that most fistulas are located on or near the midline may lead us to hope that it will become a valuable help in future.

All other methods to identify fistulas by means of dyes or fluorescent substances as well as pneumencephalography and positive contrast cisternography without CT are outdated and belong to history.

Once more we must stress that the location of a CSF fistula in certain cases may be a very difficult task which should be solved before treatment, and still remains a real challenge to the neuroradiologist and neurosurgeon.

Treatment

Treatment policy depends mainly upon the cause and the location of the fistula and in traumatic cases also on the time of manifestation, whether early or late rhinorrhea.

A. Traumatic CSF Fistulas

Early and late traumatic rhinorrhea differ in several important aspects. Early CSF leakage can stop spontaneously and be cured for ever whereas late rhinorrhea almost never heals definitively without operative treatment.

Even if it stops spontaneously the dangers of recurrence and meningitis persist. In the early posttraumatic stage also other sequelae of the trauma may interfere with or have priority over the treatment of the fistula, for instance intracranial haematomas, brain oedema, or traumatic lesions of other organ systems. We have therefore to deal with the question of selection of those patients who do or do not require surgery and, if surgery is necessary, of its timing.

1. Selection of Patients

When rhinorrhea has been detected in a conscious patient on the day of injury (D 0) or the first posttraumatic day (D 1), it usually takes 3 to 5 days to stop spontaneously or under treatment. The most effective treatment is continuous lumbar CSF drainage with removal of about 150 ml CSF daily. Alternatively repeated lumbar punctures may be made. There is some theoretical objection that such treatment could enhance the risk of meningitis because decreasing CSF flow would allow bacteria to pass more easily through the fistula to the basal cisterns, but no evidence exists for this hypothesis. Such drainage should not be made in cases with marked brain oedema and a rised intracranial pressure, because of the risk of brain shifts and herniations.

Persistence of rhinorrhea for more than one week indicates that spontaneous cure is not likely. Additionally several rhinorrhea cases among those who stop within the first week have to be treated operatively because we know by experience that the risk of recurrence and meningitis continues (Jefferson *et al.* 1972, for elder literature see Dietz 1970). We do not follow the advice of Lewin (1951) to operate upon all CSF fistulas without regard to the kind of fracture or the duration of the leakage. A more eclectic selection is advisable taking into account the duration of leakage, the kind of fracture and the patient's olfactory function.

In general—all patients with rhinorrhea which persists longer than one week should be treated operatively.

When the leak has stopped within one week, no fracture or only a small linear fracture is detectable by X-ray study and the patient has no anosmia, operative treatment is probably not necessary. The patient should attend regularly for at least one year for review to ensure timely discovery of occult or recurrent rhinorrhea. Additionally the patient and his family have to be informed that meningitis could, if seldom, occur.

In a similar situation but with anosmia surgery should be discussed carefully and is probably better performed.

All cases with evidence of a compound fracture or a defect within the posterior wall of the frontal sinus or the cribriform plate and ethmoidal region should be operated upon even if rhinorrhea has stopped sponta-

neously within the first few days. Recurrence and the risk of meningitis are otherwise extremely high (for literature see Dietz 1970, Laun 1982). Tönnis (1948) and other authors have shown that thin bone fragments of the anterior skull base often shrink, bone defects enlarge due to resorption, and damaged brain tissue within the fistula often prevents a strong scar formation.

All bullet wounds of the anterior cranial fossa with rhinorrhea require operative treatment.

In fistulas arising from fractures of the sphenoid the decision has to be taken in regard to the pecularities of the single case. These cases are rare. Therefore experience is limited and no statistical data are available indicating a correlation between the kind of fracture, recurrence of the leak, and meningitis risk. It seems beyond question that persistent fistulas of the sphenoidal region should be closed operatively.

"Occult" rhinorrhea may go directly into the pharynx and can be detected by injection of radiotracers. In some cases a fluid level in the sphenoid sinus can be detected on tomography, indicating an active fistula. In these both situations operative treatment should be undertaken.

In sphenoidal fracture cases in which the liquorrhea stops spontaneously we advise an expectant attitude and operative treatment only when recurrence or meningitis appear.

2. Timing of Surgery

This depends upon the severity of the traumatic brain lesion and the importance of rhinorrhea.

In patients with severe brain lesions—unconsciousness, brain oedema, multiple contusions or haematoma—the operative closure of a CSF fistula should as a rule be postponed for at least three weeks, until the brain oedema has disappeared and the brain functions are stabilized. Exposure of the base of the anterior cranial fossa is much more difficult when the brain is swollen by oedema and the brain is more vulnerable during the early posttraumatic stage. Early operations for closure of a CSF fistula therefore have a much higher mortality and morbidity than delayed operations, outweighing by far the meningitis risk during the waiting period, as can be shown by the Homburg material. From 1960 till 1973 all rhinorrhea cases were operated upon during the first few posttrauma days from fear of meningititis. Since 1974 this policy has been changed and operation postponed for about three weeks. The mortality of the first group was 25%, of the latter only 3% (Table 7). Meningitis occurred only in two cases out of 61 (3%) of the second group during the waiting period and could easily be cured.

In less severely injured, conscious patients also, it is preferable to postpone the operative closure of a CSF fistula for about two weeks.

Table 7. *Mortality After Operative Treatment of Rhinorrhea* (own material)

	Number of operations	†
Homburg		
In trauma cases early operation (1960–1973)	56	13[1] = 25%
Delayed operation (1974–1983)	97	3[2] = 3%
Paris		
In trauma cases delayed operation	84	3[3] = 3.5%

[1] All have been traumatic cases and all of them died from the severity of brain trauma in combination with early operation.

[2] One died from meningitis, one from postoperative haemorrhage and one from pulmonary embolism.

[3] None of them were traumatic cases: two postoperative fistulas (one each frontal and sphenoidal), one spontaneous rhinorrhea. All of them died from infection.

Exceptions from this general policy should only be considered when rhinorrhea is so important that it results in severe intracranial hypotension and/or water and electrolyte disturbances. Such situations are extremely rare. They have never been seen at the Homburg Department in the almost 25 years of its existence.

Special cases should be discussed according to their pecularities. For instance if an acute intracranial haematoma is located at the fistula site it can be reasonable to close the fistula at the same operation after removal of the haematoma. The same is true if open cranio-cerebral trauma requires an operation with exposure of the region of the fistula.

3. Operative Treatment

Ethmoido-frontal and sphenoidal fistulas will be reviewed separately.

a) Ethmoido-Frontal Fistulas

The approach to these fistulas is possible through a unilateral or bilateral frontal craniotomy as described by Pertuiset (1974) in Volume I of this book series (Fig. 8).

The closure of frontal or ethmoidal fistulas from an ENT epidural approach is less reliable than through a craniotomy: the exposure is too small. Often in traumatic cases several frontal ethmoidal fistulas exist and

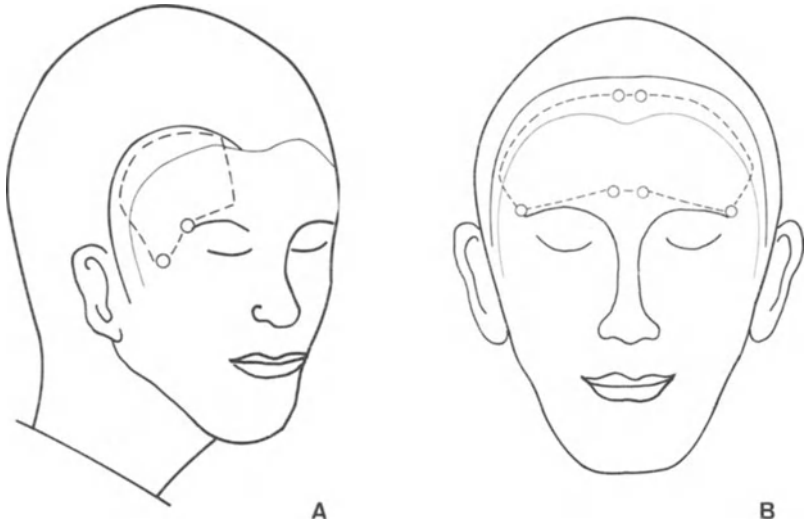

Fig. 8. *Sketch of unilateral and bilateral approaches.* A) Unilateral fronto-temporal approach. B) Bifrontal approach. —— Skin incision, – – – – The cutting of the bone flap

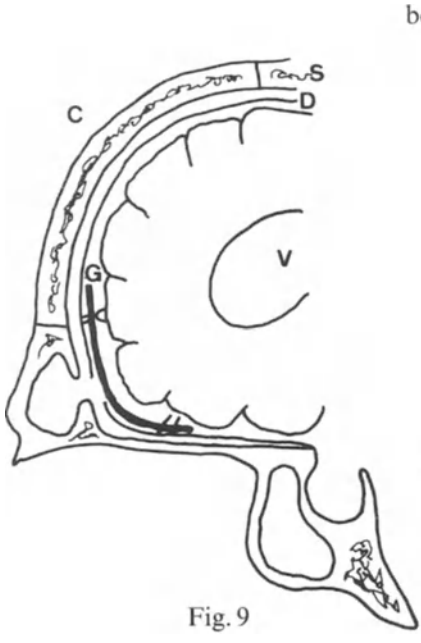

Fig. 9

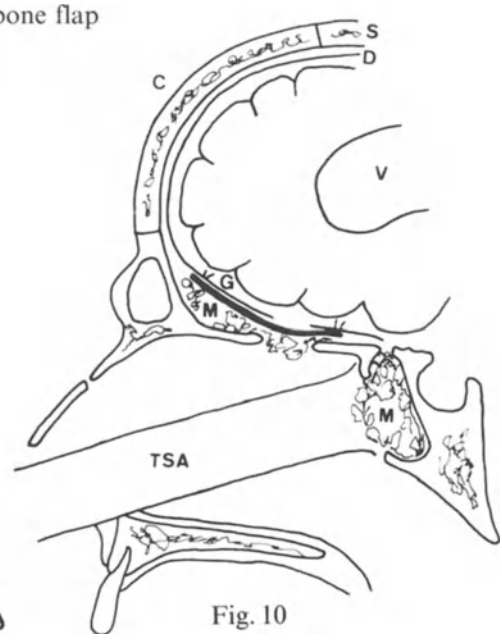

Fig. 10

Fig. 9. *Intradural approach through a craniotomy (C) for a frontal sinus defect.* The graft (*G*) is positioned and sutured intradurally. (*S* skull, *D* dura, *V* ventricle)

Fig. 10. *Epidural approach through a craniotomy (C) or transsphenoidally (TSA).* The graft (*G*) is positioned and sutured epidurally. In addition a transsphenoidal approach (TSA) is shown with insertion of muscle into the sphenoidal sinus for a leak coming from a fracture of the sphenoid. (*S* skull, *D* dura, *V* ventricle, *M* muscle)

may be overlooked using the very limited ENT approach. Fistulas which are produced unintentionally during an ENT operation can usually be closed from the same approach.

A unilateral approach is chosen when the site of the fistula is clearly known. When doubts remain concerning the site, a coronal scalp incision is advisable which allows an additional craniotomy on the other side when necessary. Proven or suspected bilateral fistulas are generally dealt with through a bifrontal approach, but an experienced neurosurgeon, using an operative microscope, can often complete the repair from a unilateral intradural approach. The unilateral approach increases the chance of preserving the contralateral olfactory nerve if it has not been destroyed by the primary impact.

Table 8. *Operative Procedures in 137 Rhinorrhea Cases* (own material)

	Homburg	Paris
Approach		
Frontal unilateral	93	31
Frontal bilateral	58	46
Transsphenoidal	2	7
Epidural	9	18
Intradural	142	—
Combined epi- and intradural	—	59
Graft material for fistula repair		
Pericranium	133	68
Fascia lata	17	16
Lyophilized dura	1	—

Once the craniotomy has been made, exposure and closure of the fistula can be performed epidurally, intradurally or by a combination of both (Table 8).

In the Paris Department as a rule the exposure begins epidurally and, if necessary, is enlarged to a combined epi- and intradural procedure. In the Homburg Department intradural exposure is preferred.

Arguments in favour of the epidural approach are, that the traumatized frontal lobe remains covered by dura during retraction, that it is not necessary to enlarge existing dural lesions in order to remove bone fragments, and above all that the arachnoidal adhesions which have formed after trauma and may be a kind of barrier against an extension of infection, are not destroyed.

Arguments in favour of the intradural approach are an undoubtedly better view and clearer identification of the fistula and easier access even to the

other side. It also can be avoided to create new dural tears indistinguishable from those caused primarily by the trauma, which often happen using the epidural route. These who prefer the intradural approach also argue that the adhesive barrier against infections mentioned above is no longer of importance after closing the fistula and that a good neurosurgeon should be able to handle the exposed brain carefully enough to avoid any additional damage compared to an epidural retraction.

At all events, a good neurosurgeon is not a dogmatic one; he should be flexible enough to treat his patients in the best possible way. Starting with the epidural approach it can be useful in some cases to check the intradural space, especially when the fistula does not appear clearly. Using the intradural approach it can be of advantage to procede partly also epidurally, for instance when the posterior wall of the frontal sinus has to be resected.

In *frontal sinus CSF fistulas* with loose and displaced fragmentation of its posterior wall it is advisable to remove the whole posterior wall together with the mucosa. For disinfection either a cotton pledget with iodine may be introduced into the sinus for several minutes or an antibiotic powder, for instance Nebacetin powder, may be applied.

If there are only linear fractures of the posterior sinus wall then they should not be enlarged ant the sinus not opened. We also do not advise removal of the anterior wall of the sinus even when there is a bone depression. Sometimes it is possible to correct such a depression primarily at the operation for closure of the fistula. Otherwise a cosmetic operation should be performed at a second, later stage.

In *ethmoidal fistulas* as little bone is removed as possible. We never remove the ethmoidal cells but leave them as they are even in the case of widespread bone fragmentation. A large opening of the nasal fossa should be avoided.

Careful investigation of the anterior fossa on the fistula side must be made to be sure that all openings have been uncovered. When the location of the fistula is questionable the surgeon can ask the anaesthetist to insufflate air into one nostril with a syringe, after packing and closing the other one. The operative field is filled with clear saline, bubbles of air then reveal the fistulous openings.

The repair of the dura can be done in some few cases by direct watertight suture, for instance with a linear fracture of the frontal sinus without dural defect. It is generally safer to add a graft since the fronto-basal dura is very thin, and of course, a graft is mandatory when a dural defect is present.

For *the choice of graft material* we strongly advise against the use of any artificial material and do not recommend devitalized tissue like lyophilized dura. Such devitalized tissue may indeed close a fistula in many cases, but it is unsafe and unreliable if an inflammation develops in its vicinity, a

situation which never can be ruled out in cases with destruction within the paranasal sinuses. Even if only a minor point, it also should be born in mind that all foreign materials are expensive whilst the patient delivers his own living tissues free of charge! Foreign substances like acrylate glue are more likely to prevent sound closure than to occlude a fistula safely and permanently.

We therefore recommend as graft materials only pericranium or autogenous fascia lata (Table 8).

Pericranium can be taken as a pedicled flap or a free transplant from the region of the skin flap. It is sometimes very thin and then must be handled with special care. Due to extensive skin lacerations it may happen in rare cases that insufficient pericranium is available. If available it has the advantage that it can be taken by the same exposure without an additional incision at the leg, but if not, *fascia lata* has to be taken from one thigh. This can be done during the cranial surgery when it becomes clear how much, if any, is required. A thin muscle layer may be left on the piece of fascia lata, which should be large enough to ensure that sutures can be placed in normal dura away from the defect. When an extradural exposure is used, the fascia lata graft is placed with its muscle layer on the bone, a pericranial flap or graft may be inserted between the dura and the skull base and sutured to the normal dura outside the defect. From an intradural approach the graft is placed on the inner surface of the dura and sutured in the same way. It can be difficult to place sutures around a defect close to the jugum sphenoidale where the dura is very adherent.

In small fistulas some neurosurgeons close the defect with a piece of muscle, taken from the temporalis, "gelitta" or other haemostatic sponge-like material and then use fibrin glue to keep them in place. Such procedures can be satisfactory but can also fail, and in our view, are less reliable than the procedures described above and are therefore not recommended by us.

Closure of bone defects: Frontal bone flaps as a rule are reinserted at the same operation, but special care has to be taken to cover and close safely all open paranasal sinuses to be sure that no connection persists between the sinuses and the bone flap.

If the bone flap cannot be used—for instance because it is too fragmented—different options are given:

to operate in two stages: at first only close the dural defect, and as a second step about one month later close the bone defect.

To close the defect immediately using autogenous bone, taken from ribs, tibia or ilium.

To close it immediately, but using methyl-methacrylate.

In the Paris Department the two stage procedure is preferred when there is no facio-cranial dislocation, but it is considered necessary to take bone grafts during operation, if such a dislocation is present. In the case of a large

defect over the nasal fossa or a large frontal sinus for example the defect is filled in order to avoid a meningocele, using a bone autograft as above. Foreign materials like methyl-methacrylate are considered to be unsafe because of possible infection from the sinuses with epidural abscess formation, and the possibility of rejection.

In the Homburg Department by contrast, the use of acrylate grafts even at the primary operation is not considered to be unsafe or dangerous, and bone autografts from ribs, tibia or ilium are not used.

The different experiences on which these two policies are based, may be explained by two facts: At the Homburg Department as a rule a pedicled pericranial flap is used to close a CSF fistula and at the same time to separate the opened paranasal sinuses from the graft layer, whilst at the Paris Department more oftenly fascia lata is used, especially when pericranium seemed to be not thick enough. Also, in Homburg a special preparation of methyl-methacrylate is used which contains gentamicin (Refobacin-Palacos®) which diffuses to the surrounding tissues very slowly during several weeks and thus gives additional protection against infection.

The policies of *wound closure and postoperative care* also show some differences between the Paris and Homburg Departments.

In the Paris Department the dura is attached very carefully especially at the anterior part of the craniotomy with non-resorbable suture material. When an epidural approach has been used this manœuvre at the same time adequately fixes the graft to the skull base. No suction drainage is used but a penrose drain is left in the epidural space for one day. Postoperatively the patients remain in bed for one week.

When the patient had developed *hydrocephalus,* which can be seen clearly on CT, external ventricular drainage (EVD) is advised to reduce the CSF pressure and the percentage of failures. The EVD is maintained for a week, then the reservoir is raised for two days and if the rhinorrhea has stopped the EVD will be removed. The drainage is placed in the frontal horn opposite to the skull flap before performing the craniotomy.

In the Homburg Department no sutures for attachment of the dura are considered necessary, because suction drainage is used. The epidural negative pressure of the suction expands the dural sac and apposes it to the cranial bone.

If the patient is conscious, and no other injuries or complications like hydrocephalus prevent him from doing so, he is allowed to stand and walk on the first postoperative day. In case of *hydrocephalus* lumbar CSF drainage is placed for some days.

Pneumatocele, pneumocephaly: Air may enter the skull cavities through the CSF fistula and reach the subarachnoid spaces (pneumatocele) and the ventricles (pneumocephaly). It may also accumulate within an area of

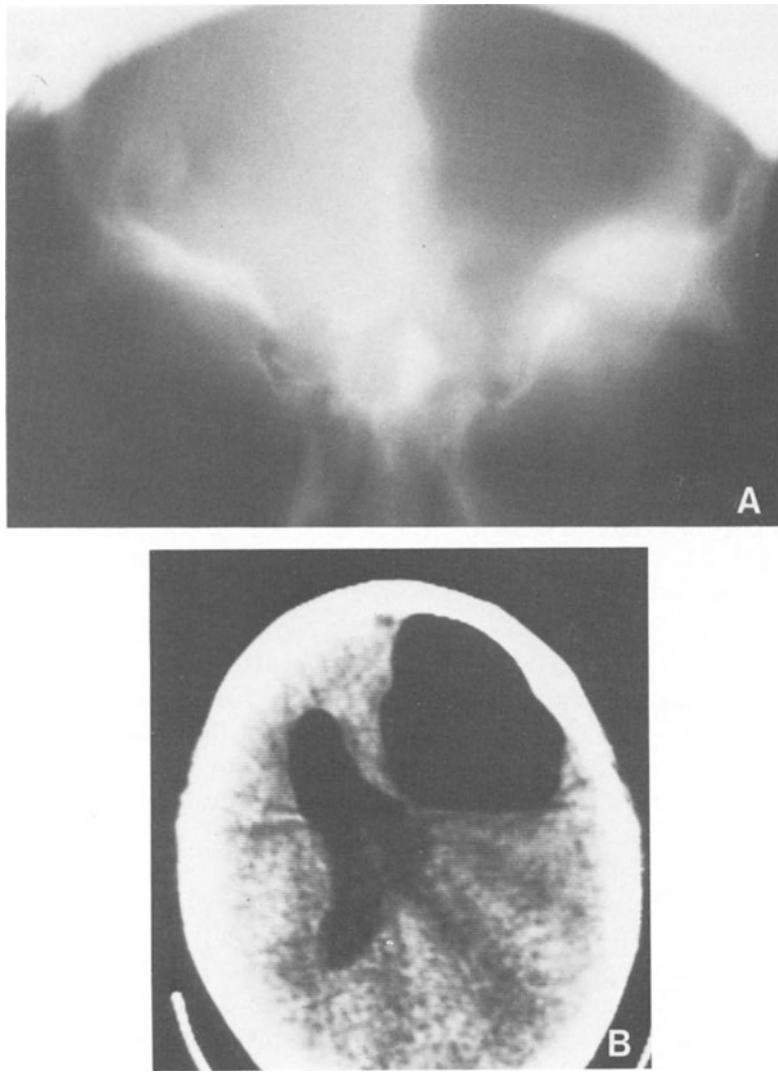

Fig. 11. *Traumatic space-occupying aerocele 7 years after the trauma.* A) Polytomographic a.-p. view. The large contusional defect of brain substance in the left frontal lobe is filled with air. A compound fracture of the fronto-ethmoidal region, including the orbital roof, is clearly visible. B) CT of the same case. The ventricles are partly filled with air. The shift to the right side indicates that the large pneumatocele within the left frontal lobe acts as a space-occupying lesion

contused brain, mostly in a frontal lobe. Air within the normal CSF spaces does not require special treatment. It will resorb spontaneously in one to three weeks. When air has accumulated within a contused area however, it can act as a space occupying lesion as the cavity expands by a valvular mechanism (Fig. 11). Such a cavity may transform into a brain abscess. It

therefore requires evacuation by puncture during surgery or through a preliminary a burr hole, if acting as a space occupying lesion and detected by CT.

When an abscess develops, which can be easily seen on CT, total resection is preferred in the Paris Department, and treatment by repeated aspiration with instillation of an appropriate antibiotic in the Homburg Department.

b) Sphenoidal Fistulas

Such fistulas almost never stop spontaneously.

The approach: They are approached through the sublabial transseptal route which is familiar to all neurosurgeons who perform transsphenoidal pituitary tumour operations. The approach has been described in detail by Landolt *et al.* in Volume 7 of this series (1980), using the operative microscope. It is especially important to avoid an opening of the mucosa when removing the nasal septum. The anterior wall of the sphenoidal sinuses is opened with its rostrum but not completely resected. The bony septum of the sinus should be removed to facilitate packing the cavity with muscle.

The fistula closure: In the beginning of the operation a piece of muscle the size of a finger tip is taken from one of the thighs. This muscle is placed in the sinus to pack it closely, not easy because muscle elasticity tends to make it slip out of the sinus. A useful trick is to mix the muscle with pieces of bone taken from the nasal septum and the anterior wall of the sphenoidal sinus.

In our experience it is not advisable to use any kind of glue, not even fibrin glue, which makes the muscle surface especially smooth, almost slippery, and appears likely to prevent rather than promote firm adhesion of the muscle. Because the dural opening cannot be sutured it is advisable to insert lumbar or ventricular CSF drainage for about one week. After suture of the sublabial incision the nasal cavities are tamponaded with vasoline cotton strips to bring both layers of septal mucosa together in the midline. After pituitary tumour operations they are usually removed on the second day. In CSF fistulas we recommend to renew them on the second day for another two days.

B. Spontaneous CSF Fistulas

These occur only rarely and are almost always located in the ethmoidal region. During more than 20 years there only 4 have been seen and operated upon in the Homburg Department and during approximately the same period only 3 in the Paris Department (Table 6). One of the seven cases was caused by a meningocele, two by raised intracranial pressure, three through defects of the cribriform plate and one by CSF leakage through the

olfactory nerve. We have not seen any CSF fistulas caused by the empty sella syndrome or by basal tumours.

In treatment it is always necessary to cover the fistulous region with a pericranial flap or fascia lata graft. *If no fistula opening can be identified* with certainty sacrifice of both olfactory nerves will be unavoidable and the whole ethmoidal region may then be covered with the graft resp. flap.

In *basal tumour cases* the treatment is closely related to the tumour removal as described on page 197.

When the fistula is caused by a *meningo- or encephalocele* it depends on its extent, whether a mere covering of the basal defect with pericranium or fascia lata is sufficient or whether in addition the bone defect has to be closed by a bone or acrylic graft.

If a spontaneous CSF fistula results from *raised intracranial pressure* the cause of the intracranial hypertension has to be treated at the first place. If the fistula persists after normalization of the intracranial pressure, the ethmoidal region must be exposed and the fistulous opening closed as described above.

Recently Sawicka and Trosser (1983) have reported a case of spontaneous rhinorrhea in a patient who was a *cocain sniffer* for the previous 19 years. During surgery it was noticed that bone and dura of the cribriform lamina were paperthin. In addition one olfactory nerve looked oedematous and was covered by inflammatory tissue. No defect was present, but the small perforations of the olfactory nerve endings were larger than usual. A graft of fascia lata was successfully applied.

In all cases with spontaneous rhinorrhea the patients should be informed beforehand about the likelihood of postoperative anosmia.

CSF fistula with *empty sella syndrome* are the result of destruction of the sellar floor caused by the pulsatile arachnoid diverticulum (Fig. 2). Surgical treatment consists in filling the empty parts of the sella, as a rule by the transsphenoidal approach, using muscle and bone as described for postoperative CSF fistulas after transsphenoidal pituitary tumour removal (see page 195). The transfrontal approach is only to be preferred when symptoms of invagination of the chiasma into the sella exist, as may happen after surgical or radiological treatment of pituitary adenomas (for literature and details see de Divitiis *et al.* 1981 in Volume 8 of this series).

C. Postoperative CSF Fistulas

The prevention of CSF fistula is most important, and at the same time easier than the difficult cure of postoperative rhinorrhea. We shall deal in this chapter therefore both with the avoidance and the treatment of such fistulas. They may be caused by operation on pituitary tumours, olfactory groove meningiomas and other tumours of the anterior skull base.

1. Pituitary Tumours

Frontal approach: Only exceptionally does rhinorrhea develop after the subfrontal removal of a pituitary tumour, unless it is an invasive type which has eroded or perforated the sellar floor. Mostly these are prolactinomas, which nowadays can be diagnosed endocrinologically in advance. They should first be treated with bromocryptin and some may avoid operation altogether. When such tumours have to be approached operatively, the surgeon should be cautious in evacuation of the sella with care to respect the mucosal barrier of the sphenoidal sinus.

Postoperative rhinorrhea following such an approach can be disastrous because the leak is always large. When it occurs, the patient must be reexplored transfrontally as soon as possible and the sella packed with muscle. Additional lumbar or ventricular CSF drainage is necessary.

Transsphenoidal approach: Most cases of rhinorrhea complicating pituitary surgery occur after a transsphenoidal approach although curiously, it is extremely difficult to find in the literature the precise incidence of this complication. Without doubt its occurrence has been markedly reduced with increasing experience, the use of the operative microscope, and specially designed instruments.

Prevention begins with a large opening of the anterior wall of the sphenoid sinus. This allows wide opening of the sella also, which is important in order to visualize the walls of the sella and especially the diaphragma sellae. The tumour should be dissected before removal. Pulling it out of the sella with forceps, before it has been completely dissected is absolutely contraindicated as it creates a risk of tearing the diaphragma. When the tumour has suprasellar extension and especially when the diaphragma is partly interposed between this extension and the sella, the surgeon has to be very cautious. Compression of the jugular veins or of the abdomen may result in spontaneous descent of the tumour.

Even when surgery has gone well we recommend, for safety reasons, to pack the sella with muscle, oxycel or fascia lata. The sphenoidal sinus should also be packed. The rostrum of the sphenoid can then be placed into the sinus opening as a bony barrier to prevent the muscle from slipping out.

When *intraoperative leakage* occurs and drops of CSF are evident, Landolt advises (Volume 7 of this series) to place a piece of fascia lata on the damaged diaphragma sella or the exposed arachnoid membrane. The sella is then filled with muscle and a second piece of fascia lata placed over its anterior dural opening before the sella is closed with a piece of bone from the nasal septum. Continuous lumbar CSF drainage may be used for two to five days.

There are two kinds of *postoperative CSF leakage:*

a) transient leakage of a few drops after removal of the nasal tamponade.

Such mild fistula may be the beginning of meningitis with oversecretion of CSF, and lumbar puncture for CSF examination is then necessary. Treatment of this kind of fistula consists of lumbar punctures and antibiotics (even when CSF culture remains sterile). The fistula will usually stop in a few days.

b) Permanent CSF leakages are those not cured within one week, in spite of the above treatment. The patient then has to be reexplored. Very careful packing of the sella and the sphenoidal sinus must be performed using muscle, fascia lata and bone, and continuous lumbar CSF drainage added.

Such permanent fistulas can remain occult, if leakage is minor and CSF escapes into the pharynx rather than through the nostrils. The patient must be carefully questioned. This probably explains the five cases with meningitis reported by Landolt (1980) in his series of 197 cases.

The danger that postoperative CSF leakage may become permanent and require operative treatment seems greater in cases with diabetes insipidus.

2. Olfactory Groove Meningiomas

CSF fistula can complicate the postoperative period for two reasons:

a) Extensive coagulation of origin of the tumour from the cribriform plate, from which the blood supply of the tumour comes, may have been necessary, after removal, to secure haemostasis. This necessary manœuvre can perforate the lamina or provoke its secondary perforation. Thus rhinorrhea can become apparent during the first postoperative days or even weeks later. It has to be treated like a traumatic fistula.

When it is already obvious during surgery that the plate has been perforated, a graft must be placed immediately.

b) There are olfactory groove meningiomas with infiltration of the cribriform plate and inferior extension into the superior part of the nasal cavities. They are not frequent and can nowadays easily be detected by CT. In the Paris Department such cases are operated upon in two stages. At the first one the intracranial tumour will be removed respecting the cribriform plate, which will be covered by a fascia lata graft sutured to the dura of both orbital roofs. At a second stage one month later, the nasal extension of the meningioma will be removed using a nasal approach. An exceptional meningioma, which developed only under the cribriform plate has been removed in the Paris Department. During the first operation a fascia lata graft was placed epidurally using a frontal approach. One month later a nasal approach allowed removal of the meningioma, followed by partial restoration of bilateral impairment of visual acuity.

In the Homburg Department the rule is to remove the whole tumour at once, going if necessary through the cribriform plate into the nasal cavities. This approach through the anterior skull base gives an excellent view and

allows removal even of tumours which have invaded the maxillary sinuses, as in one of the olfactory groove meningiomas in the Homburg material. It is self-evident that the resulting large opening into the nasal cavities has to be closed with special care by suturing a pedicled pericranial flap into the defect. Additionally a graft of fascia lata may be placed over the thin pericranial graft on its inner surface in order to reinforce it. Continuous lumbar drainage should be used to lower the CSF pressure for at least one week.

3. Other Skull Base Tumours

CSF fistulas caused by tumour destruction of the skull base may lead to the diagnosis of the underlaying disease and be cured in connection with the removal of the tumour. Even large basal defects resulting from tumour removal or from removal of an encephalo-meningocele can be safely closed using percranium or fascia lata.

Similar operative policies to those described above for the treatment of olfactory groove meningiomas with invasion of the skull base can also be applied to the removal of other basal tumours either in two stages or by a single stage step procedure.

We must emphasize that bone wax sealing of opened air cells is not a safe protection against CSF fistula development and does not replace the necessity to close the dura. It should also be realized that the removal of large skull base tumours needs special experience. These are long-lasting operations which should not be tried at all neurosurgical departments.

4. CSF Fistulas Resulting from ENT Operations

Accidental dural injury may occur during an ENT operation on the frontal or ethmoidal sinuses and a CSF leak result. Immediate repair using the same approach is often possible in frontal and anterior ethmoidal locations by suturing a patch of muscle or fascia lata into the dural defect. Direct suture of a dural laceration can only exceptionally be made since as a rule a defect exists. Posterior ethmoidal lesions are better closed via a transfrontal intradural approach. The same is mandatory if additional brain damage with the risk of intracranial haemorrhage has been produced (see page 177).

D. Antibiotic Prophylaxis and Therapy

Since the main complication of a CSF fistula is meningitis it seems reasonable to give antibiotics prophylactically without regard to cause and intended treatment. But after examination of the literature and the policies de facto used in most neurosurgical departments one will be puzzled at once by contradictory reports and attitudes.

We shall briefly review the literature and then deal with the more frequent situations in which a decision has to be taken.

Literature Reports About the Effectiveness of Antibiotic Prophylaxis

In a double blind study, using penicillin, Klastersky *et al.* (1976 and 1979) did not find any proof of usefulness, as stated previously by Mincy (1966). Even worse, infections with unusual germs which are more difficult to cure, may be provoked, as reported by Price *et al.* (1970). Only penicillin however had been tested by double blind studies. A useful effect of other antibiotics or combinations of antibiotics therefore cannot be ruled out. For this reason Brawley and Kelly (1967) had suggested either chloramphenicol or a combination of penicillin and streptomycin. According to Ignelzi *et al.* (1975) neither ampicillin and cephalothin prophylaxis was effective. McGee *et al.* (1970) reviewed 402 literature cases. Out of 325 who received antibiotic prophylaxis 46 cases (14%) got meningitis, compared to only 4 (5%) out of 77 cases without antibiotics. Landolt (1980) had 5 meningitis cases (4.5%) out of 113 patients operated upon for pituitary tumours, who received a daily dose of 2 g chloramphenicol, starting one day before and lasting four days after operation, but no meningitis occurred among a group of 84 comparable cases operated upon without systemic but with local application of antibiotics.

Even if no statistically significant conclusions can be drawn from the available data, the impression prevails that antibiotic prophylaxis is at least ineffective and may even be dangerous. We therefore advise against routine antibiotic prophylaxis. Exceptions may be reasonable in cases with higher meningitis risk, for instance caused by pre-existing sinusitis.

Patients Waiting for Operative Treatment

As explained on page 185 it is our general policy to postpone the operative closure of a posttraumatic CSF fistula for several weeks. Following the above reviewed literature reports, no routine antibiotic prophylaxis should be given during this waiting period. However in the Paris Department all patients with severe head trauma, especially with compound fractures of the skull base, waiting for the fistula closure on D 21, received prophylactically one or two broad spectrum antibiotics. The same was true at the Homburg Department until recently. Only since the preparation of this manuscript, impressed by the literature reports, have we abandoned such a general routine prophylaxis, till now without adverse results. Even under this new policy some patients with multiple trauma have to be treated with antibiotics because of other sequelae of the trauma.

Patients with Meningitis

When meningitis is the reason for admission to our Departments—after taking CSF by lumbar puncture for culture and sensitivity test—the patients receive immediately two broad spectrum antibiotics intravenously, in the Homburg Department also 2–5 mg gentamicin (Refobacin-L®) intrathecally. These antibiotics may be changed later according to the result of the sensitivity tests. In addition to the CSF examination blood cultures should be taken immediately, before the first antibiotics are given.

The operative treatment of a CSF fistula or of the recurrence of a fistula has to be postponed until the meningitis is cured. The recurrence of a CSF fistula may be the first symptom of meningitis, due to a CSF oversecretion.

According to the literature meningitis which is caused by a persistent CSF fistula has a very high mortality of about 50%. The severity of this complication is the reason why a CSF fistula, when permanent, has to be occluded by all means.

Results

It is a general rule that the results and risks of any operative treatment have to be compared to the results and risks of the so-called natural, untreated course and of the conservative treatment. The failure rate has also to be considered. We shall therefore first deal with the unoperated rhinorrheas, then with operative mortality and morbidity and finally with the recurrence or failure rate of operative treatment.

A. Non-Operated Patients

Mincy (1966) reported that in 85% of his patients CSF leakage ceased spontaneously within one week and that the infection incidence during this time was 11%. Rousseaux and Scherpereel (1981) had a comparable meningitis incidence in a series of 102 cases. 82 of them were not operated upon because the leak stopped spontaneously during the first posttraumatic week. 80% of these patients were followed-up for three years. Eight of them (10%) developed meningitis and one died as a result. McGee *et al.* (1970) calculated a meningitis rate of 12% (50 of 402 reviewed literature cases). The short-term infection rate of the Homburg material—meningitis during the three-week waiting period between injury and operative closure of the fistula—was 2 of 61 = 3%. Without any doubt this percentage would have been higher if the operative closure of the fistula had been postponed longer or not been done at all.

In conclusion it can be stated that the meningitis risk during the first three weeks, until the operative treatment can reasonable be done, ranges between 3 and 10% and that the incidence of late meningitis in cases in

which the rhinorrhea had stopped spontaneously during the first post-traumatic week remains as high as about 10%. Some patients will die from this complication.

As usual statistics can give broad scientific information but leave the neurosurgeon without real help for the treatment of a single case. The patient's future may lie anywhere within the wide statistical variation of the figures.

The data in the literature are not consistent enough to advise strongly that all rhinorrheas should be operated upon even when the leakage has stopped before the end of the first week. It would however give more security to these patients, since unoperated they have to live with a certain meningitis risk, and possible fatality. Operation upon all rhinorrhea cases to repair the dura carefully would probably be acceptable if there was no risk of postoperative anosmia. Progress in this field awaits operative-technical developments which give the possibility to preserve at least one olfactory nerve.

B. Operative Mortality and Morbidity

1. Mortality: The mortality figures vary widely and depend in trauma cases mostly on the timing of the operation, in early operations that is upon the severity of the primary brain injury.

Laun (1982) reported one death in a series of 120 cases = 0.8%. Rousseaux and Scherpereel (1981) had two deaths in 20 operated cases = 10%. In older series with operative treatment immediately after the injury the mortality figures were about 30% (for older literature see Dietz 1970). The mortality of the Homburg (3%) and Paris (3.5%) material after delayed operations is almost identical (Table 7).

In conclusion it can be assumed that nowadays in those patients in which the primary brain trauma problems have been solved, the postoperative mortality ranges between 1 and 3%.

The cause of death is in most instances infection: meningitis or septicaemia. Postoperative haemorrhage or pulmonary embolism can also be to blame as was the case in the Homburg series.

2. Morbidity: Disturbance of olfactory function is the only morbidity which can be related to the surgical treatment of CSF rhinorrhea. Its occurrence rate can only be given from patients in whom olfactory function had been evaluated before surgery, which may be difficult or even impossible in trauma cases during the first posttraumatic period. All reported figures have thus an important element of uncertainty. The figures of the Paris (14%) and Homburg (18%) material are given in Table 9. Laun (1982) reported about 25% and Rousseaux and Scherpereel (1981) about 10% postoperative anosmia. As a whole it seems reasonable to assume that the already considerable posttraumatic rate of anosmias will be increased by

the operative treatment by about 15–25%. It is even more difficult and uncertain to evaluate the postoperative incidence of hyposmia. According to the figures of the Paris material (16.6%) it may range between 15–20%.

Anosmia interferes with several human activities. It deprives patients from many pleasures, for example the smell of ladies perfume, flowers and food. It can be a real handicap in special professions. Great efforts therefore should be taken to improve our operative technique in order to preserve at least one olfactory nerve when repairing a CSF fistula.

Table 9. *Postoperative Olfactory Disturbances in 237 Rhinorrhea Cases* (own material)

	Number of cases	Preoperative hyp- or anosmia	Postoperative anosmia	Increase in %
Homburg	153	45	74	18
Paris	84	31	43	14

C. Failures and Recurrences

In the literature as in our own material, it has not always been possible to differentiate with certainty between failures of operative treatment of rhinorrhea—when the CSF leakage continues—and recurrences, remanifestation after a period of apparent cure. As a rule both situations have been covered by the term "recurrence". The related figures vary widely. Cooper (1982) reported 25%, Laun (1982) 18%, Rousseaux and Scherpereel (1981) 6%. In the Paris material there were 8% and in the Homburg material 6% so-called recurrences (Table 10).

The main causes for failures and recurrences are:

a) The dural defect was not found at surgery and therefore not repaired.

b) More than one fistula existed but only one taken into consideration and closed.

c) The graft has not been sutured watertight or has been rejected.

Recurrence is more frequent after unilateral exploration, its percentage would probably decrease if a bilateral approach were performed in all cases, but this would increase the incidence of postoperative anosmia. Those patients with a leak within the anterior cranial fossa who certainly are anosmic already before the operation—when a reliable verification is possible—should be explored by bilateral approach. The same is true for patients with a compound fracture of the fronto-ethmoidal region.

Table 10. *Recurrences and Failures After Operative Treatment of Rhinorrhea* (own material)

	Number of operations	Number of failures and recurrences
Homburg	156	9 = 6%
Paris	84	7 = 8%

Eight patients of the Homburg material could be cured by reoperation. One died after several unsuccessful reoperations from meningitis. It was a posttraumatic leak through the clivus. All others were frontal or ethmoidal fistulas.

Two patients of the Paris material were not reopened. One died from infection, the other one was finally cured by an internal ventricular shunt. Three patients were successfully reoperated. Two patients needed multiple operations, one of them with success and one with failure (a sphenoidal case, resulting in chronic rhinorrhea).

In all cases of failure or recurrence a reoperation is necessary after careful re-evaluation of the exact location of the fistula or the fistulas. Some few cases are reported with recurrent or persistent fistulas in spite of several attempts at operative closure. In the series of Laun (1982) were 28 recurrences. 19 of them had to be reoperated 3 times and 5 of them more than 3 times. In such cases of repeated recurrence or failure we strongly advise to add CSF drainage to the open surgery. This can be a temporary lumbar or ventricular drainage, but it is probably safer to perform permanent internal drainage: ventriculo-atrial, ventriculo-peritoneal or lumbo-peritoneal, as proposed by Greenblatt *et al.* (1973). In our opinion this should not be the primary mode of treatment but only a last resource in leaks which persist in spite of careful repair of the dura. Some series contain cases in which recurrences closed themselves spontaneously. But no reports exist with follow-up studies long enough to evaluate the risk of a late meningitis.

Senior Author's Address

We must draw the attention of our readers to the fact that not all problems in the treatment of traumatic CSF fistulas have been solved, especially concerning operative indications and preservation of olfactory function.

On the one hand it is true that surgical repair of a dural tear is not a difficult operation and even younger neurosurgeons still in training, can perform it beautifully. On the other hand we must admit that the indication for operative treatment in some special cases may remain debatable and

need thorough discussion to make the best choice. This is especially true for those traumatic cases in which rhinorrhea stops spontaneously during the first postoperative week, because there remains, without operative treatment, a meningitis risk as high as about 10%. In such cases we advise operative treatment—without regard to the spontaneous cessation of the rhinorrhea—in all cases with complete anosmia, in all cases with compound frontal or ethmoidal fractures and in cases with larger linear fractures whether located frontally, ethmoidally or sphenoidally.

The legal aspect has also to be considered, especially if no operation is advised. A patient may be tempted to sue the neurosurgeon in case of late meningitis, arguing that his future had not been adequately assured. The pros and cons of operative treatment have thus to be discussed thoroughly with the patient and his family and this has to be documented in written form.

We must underline that the diagnosis of a CSF leak can be difficult in more severely injured patients, and also that its precise location—including the possibility of multiple posttraumatic fistulas—may pose problems. For traumatic cases in the past also the timing of an operative intervention has been a problem. In our opinion this problem is now solved in favour of delayed operation.

The figures of postoperative anosmia, recurrencies and failures clearly show that further diagnostic and operative/technical efforts are needed to solve these remaining problems.

Summary

CSF fistulas are a major complication of head injury but also occur spontaneously or symptomatically in connection with tumours of the skull base, empty sella syndrome, ethmoidal encephalomyelocele, intracranial hypertension or postoperatively in connection with operations on skull base tumours or ENT operations. Their main risk is the possibility of meningitis.

The main clinical symptom is CSF leakage from the nose, but meningitis may be the first manifestation. Isotope cisternography and metrizamide CT cisternography are the most important methods for precise localization, sometimes also for verification of a suspected fistula.

Most traumatic CSF fistulas of the frontal and ethmoidal region have to be treated operatively. The method of choice is the transfrontal approach and the closure of the fistula opening using a pedicled pericranial flap or fascia lata graft.

Most sphenoidal fistulas have to be treated by packing the sphenoidal sinus with muscle.

The treatment methods of the rare spontaneous and symptomatic CSF fistulas are also described.

The results of operative treatment are satisfactory. About 6% recur-

rences, which as a rule can be cured by reoperation, and a mortality rate of about 1–3% seem to be an acceptable price for prevention of an otherwise unavoidable and oftenly deadly meningitis. Future efforts are necessary to improve the operative technique in order to reduce the incidence of anosmia.

Our descriptions and advice are based not only on literature reports but also on our own experiences with a combined material of 237 cases operated on for rhinorrhea.

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