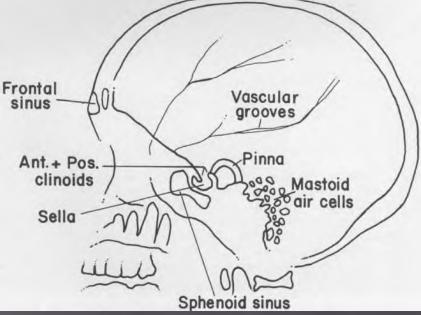
POLTAVA STATE MEDICAL UNIVERSITY DEPARTMENT OF ONCOLOGY AND RADIOLOGY WITH RADIATION MEDICINE ASSISTANT NESTULIA K.I. LECTURE: 8. CNS

- The newer imaging modalities have had a greater impact on the diagnosis of diseases of the skull, spine and central nervous system than on any other body system. **Computed** tomography (CT) and magnetic resonance imaging (MRI) have become the standard investigations for most disorders of the brain. Plain films are still the initial investigation for disorders of the bones of the skull - particularly fractures, but otherwise have limited uses. Radionuclide imaging has been almost entirely replaced by CT and MRI. Arteriography is now limited to demonstrating arterial stenoses, aneurysms and some arteriovenous malformations.
- In neonates and small infants it is possible to examine the brain and ventricles by *ultrasound* using the 'window' provided by the open fontanelle and many lesions can thus be diagnosed. Unfortunately the technique cannot be used in the same way for adults and older children because of the skull barrier. However, Doppler ultrasound is used in adults for screening carotid bifurcations in suspected atheromatous stenosis.

□ The usual set of skull films comprises a series made AP, **PA** (in several degrees of sagittal flexion of the neck), lateral (each side in turn close to the plate), as well as one of the basilar projections in which the ray is directed so that it superimposes the complex basilar structures upon the less complex calvarial cap. The lateral view of the skull shows the two halves of the coronal suture superimposed. The two parts of the lambdoidal suture are seen. Sutures usually remain visible throughout life, distinguishable from fracture lines by their serpiginous character and white margins, while a fracture will be more linear, not at all marginated, and usually more radiolucent. Study the normal skull films on the next slayds.



The Lateral View : 1, frontal sinus; 2, roof of right and left orbits superimposed; 3, anterior border of middle cranial fossa; 4, pituitary fossa; 5, sphenoid sinus; 6, maxillary antrum; 7, vascular groove; 8, pineal; 9, mastoid air cells.





Posteroanterior view: 1, frontal sinuses; 2, lesser wing of sphenoid; 3, greater wing of sphenoid; 4, superior orbital fissure; 5, wall of middle cranial fossa, 6, petrous bone; 7, mastoid air cells: 8, pineal; 9, superior orbital margin.. Anteroposterior views: 1 foramen magnum: 2. dorsum sellae of pituitary fossa: 3. petrous bone: 4. mastoid air cells; 5, pineal

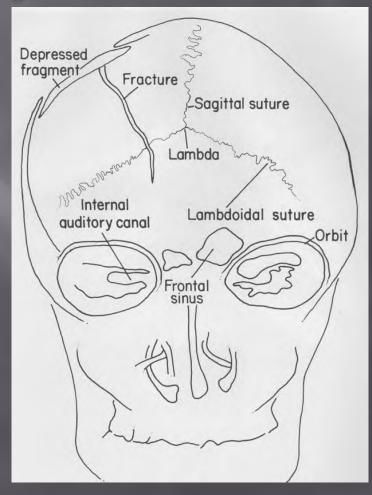


TRAUMA

Traumatic brain injuries include concussion, contusion, skull fracture, and hemorrhage, which may be epidural, subdural, subarachnoid, or intraparenchymal. Epidural hematoma results from rupture of a meningeal artery and follows a hyperacute course, whereas subdural hematoma results from rupture of bridging veins and follows an acute or a chronic course, depending on the severity of the injury. Trauma of the spinal cord produces a variety of neurologic deficits not only from direct neurologic trauma, but also from direct and delayed damage to the vasculature, with resultant paraplegia or quadriplegia, depending on the level of injury.

PA projection with fractures both linear and depressed. A plate of bone seen in tangent (between the arrows) is slightly depressed. This is not a simple linear fracture but a comminuted one, therefore. Note fillings in the teeth. Identify: odontoid seen through the nose, frontal sinuses, petrous tips with internal auditory canals seen through orbits.





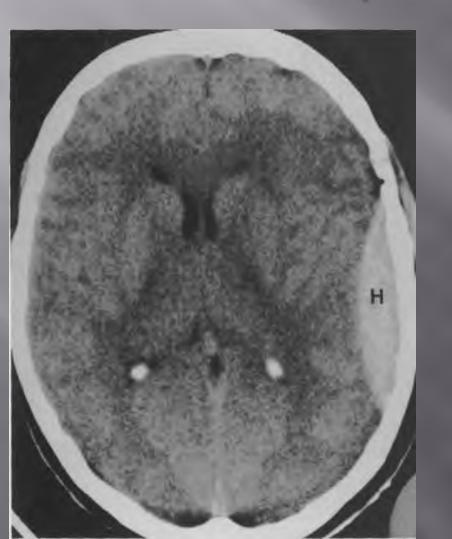
Epidural Hematoma

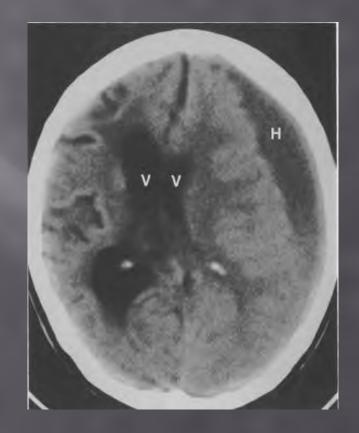
Traumatic brain injuries include concussion, contusion, skull fracture, and, in a small percentage of major head injuries, epidural hematomas. Usually, the bleeding is from arterial injury. Common localysations of epidural hematomas are the temporal fossa, the subfrontal region, and the occipital-suboccipital area. The temporal fossa epidural hematoma, which results from damage to the middle meningeal artery, is the most common epidural hematoma. The classic course is a period of unconsciousness due to a concussion, a period of lucidity as the dura mater initially slows the leakage of blood, and a rapid deterioration of consciousness. An aggressive diagnostic and surgical approach is required to save the patient.

Acute and Chronic Subdural Hematoma

A subdural hematoma usually results from an acute venous hemorrhage caused by rupture of cortical bridging veins. Acute subdural hematomas, which are often associated with skull fractures, usually develop within hours after injury. Associated massive cerebral or brainstem contusions or both contribute to a high mortality rate. Common signs are depressed consciousness, ipsilateral pupillary dilatation, and contralateral hemiparesis. Chronic subdural hematomas in infants can occur as a result of birth trauma. In adults, they are more common in the elderly, patients with chronic alcoholism, and patients receiving long-term anticoagulant therapy or who have a blood dyscrasia. The precipitating trauma is often trivial. Brain atrophy with an increase in the subdural space is a predisposing factor. A vascular membrane forms around the lesion within 2 weeks after the initial hemorrhage fills the available subdural space. The hematoma enlarges slowly until it produces symptoms. The clinical course can be subtle, with waxing and waning signs and symptoms. The differential diagnosis includes stroke, infection, or psychosis.

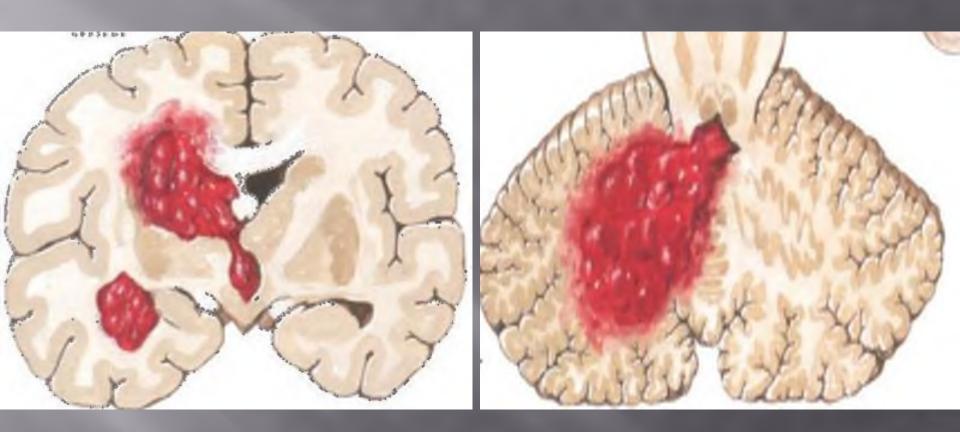
Extracerebral haematoma. (a) CT scan showing a high density lentiform area typical of an acute extradural haematoma (H). (b) CT scan in another patient taken a month after injury showing a subdural haematoma (H) as a low density area. Note the substantial ventricular displacement. V, ventricles.



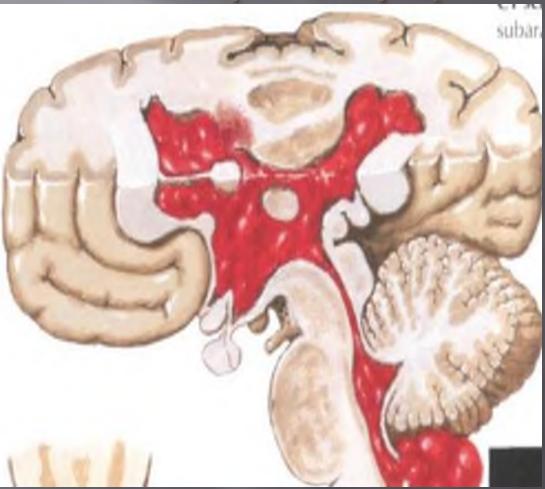


NEUROLOGIC DISORDERS OF INFANCY AND CHILDHOOD

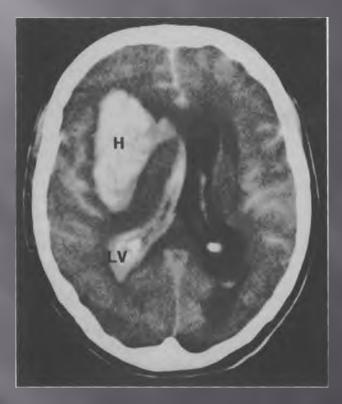
Many neurologic disorders of infancy and childhood result from birth trauma, prematurity predisposing to hemorrhage within the germinal matrix of the brain, and a wide spectrum of development defects involving abnormalities in the formation of the neural tube (anencephaly, encephalocele), neural proliferation and migration (microcephaly), and neural organization and myelination (porencephaly). The chronic motor dysfunction known as cerebral palsy often develops in surviving infants.

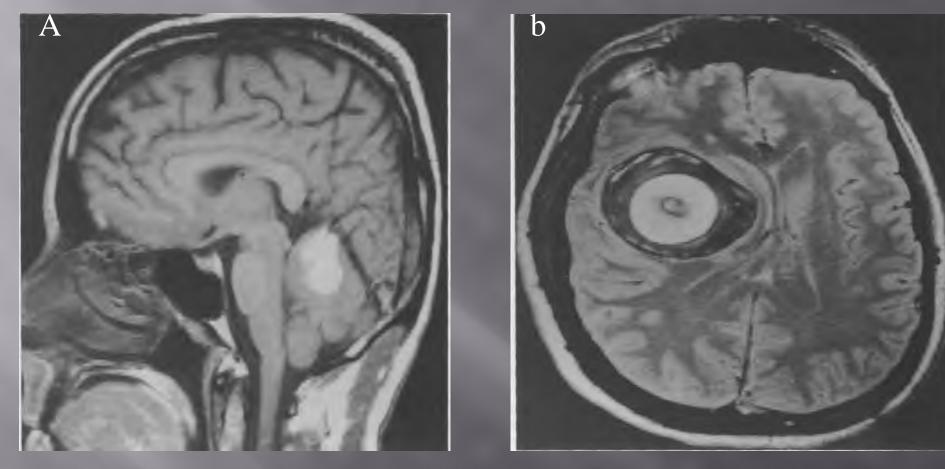


In the newborn, certain forms of intracranial hemorrhage are usually related to birth trauma, and these include subdural hemorrhage, subarachnoid hemorrhage, and posterior fossa hemorrhage. However, other factors, particularly prematurity and asphyxia, are involved in periventricular and intraventricular hemorrhage. Periventricular-intraventricular hemorrhage originates in the germinal matrix and occurs with increasing frequency in relation to the degree of prematurity of the infant. Such bleeding causes a high mortality rate. Surviving infants often develop cerebral palsy.



Intracerebral haemorrhage. CT scan showing the haematoma as a high density area (H). Blood is also seen in the displaced lateral ventricle (LV) and in the subarachnoid spaces over the cerebral hemispheres. The patient had suffered head trauma.



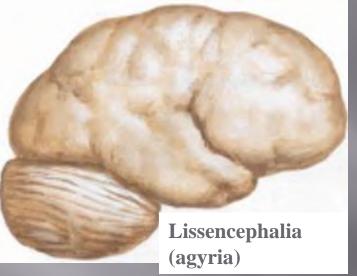


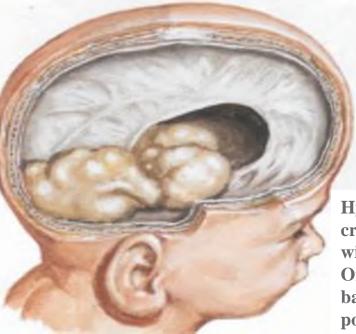
Cerebral haemorrhage on MRI. (a) A 7-day-old haemorrhage into the superior portion of the cerebellum is clearly shown as a high signal intensity collection on a TI-weighted image, (b) A chronic haemorrhage in the right cerebral hemisphere shows the complex mixture of high and low signals typical of old haemorrhage.

Brain Malformations

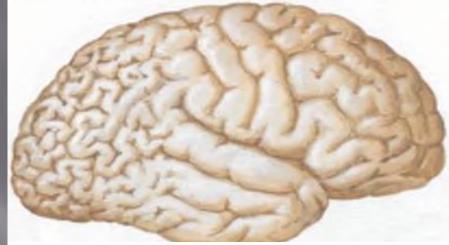
The time of onset of prenatal injury predicts the type of maldevel-opment and resultant prenatal encephalopathy characterized by defects in the formation of the neural tube (first trimester), neural proliferation and migration (second trimester), and neural organization and myelination (third trimester). Defects in neural tube formation in the first trimester result in anencephaly, encephalocele, or holoprosencephaly (arrhinencephalia), the latter characterized by a single ventricle with defective olfactory and optic systems, and impairment of caudal closure results in meningomyelocele. **During the phase of neuronal proliferation, a decrease in number of neurons** leads to microcephaly, whereas an increase results in megalencephaly. With defective neuronal migration, gyral formation does not occur, resulting in lissencephalia (smooth brain) or other lesions, such as agenesis of the corpus callosum. Abnormalities in intrauterine cerebral blood flow, if severe, can result in the rare disorder of hydranencephaly and, if less severe, porencephaly characterized by cystic spaces in the brain parenchyma.

Brain Malformations

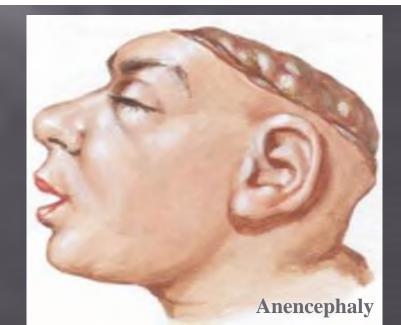




Hydranencephaly cranial cavity filled with cystic sac. Only remnants of basal ganglia and posterior lobe.



Microgyria. Of occipital and posterior temporal lobes



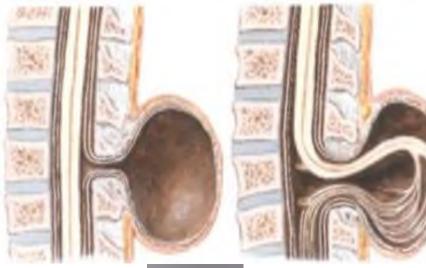
Arteriovenous malformation. Enhanced CT scan showing the enlarged abnormal vessels (arrows). Arteriovenous malformation. MRI scan (Tlweighted) showing signal void from fastflowing blood in the vascular malformation (arrows).



Spinal Dysraphism

Spinal dysraphism includes several conditions characterized by congenital failure of fusion of the midline structures of the spinal column. The resultant clinical spectrum ranges from an asymptomatic bony abnormality (spina bifida occulta) to severe and disabling malformation of the spinal column and spinal cord (meningomyelocele). Lesions in the lumbosacral region and higher may produce paraplegia and loss of bowel and bladder control; hydrocephalus develops in approximately 90% of cases. The hydrocephalus is related to a congenital deformity of the hindbrain, known as the Arnold-Chiari malformation, in which the posterior fossa structures are downwardly displaced into the spinal canal and interfere with the circulation and absorption of CSF

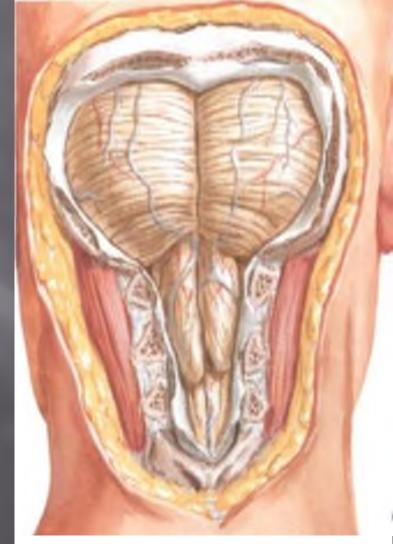
Spinal Dysraphism



Meningocele

Meningomyelocele





Arnold-Chiari malformation

Spina bifida. With central cicatrix

Hydrocephalus

Hydrocephalus, characterized by enlargement of the ventricles of the brain, results from increased formation or decreased absorption of CSF (communicating hydrocephalus) or from blockage of one of the normal outflow paths of the ventricular system (obstructive hydrocephalus). Obstructive hydrocephalus often results from a congenital stenosis of the cerebral aqueduct of Sylvius, but a brainstem tumor or a posterior fossa tumor encroaching on the fourth ventricle that obstructs one of the medial or lateral apertures can produce the same effect. In adults, brain tumors are the usual cause of obstructive hydrocephalus. Communicating hydrocephalus may occur in premature infants after intraventricular hemorrhage. In children and adults, communicating hydrocephalus with increased intracranial pressure may follow an intracranial hemorrhage or infection. Adults also may have normal-pressure hydrocephalus, which must be differentiated from ventricular dilatation secondary to brain atrophy (hydrocephalus ex vacuo).

Hydrocephalus



Potential lesion sites in obstructive hydrocephalus. 1. Interventricular foramina (of Monro); 2.Cerebral aqueduct (of Sylvius); 3. Lateral apertures (of Luschka); 4. Median aperture (of Magendie)

Section through brain. Showing marked dilation of lateral and 3rd ventricles

Lateral ventricle 3rd ventric

4th ventric

Brain Tumors in Children

Brain tumors in children are found most commonly in the posterior fossa. The more common astrocytomas and medulloblastomas develop from the parenchyma of the cerebellum. Symptoms include evidence of cerebellar dysfunction (ataxia of the trunk and extremities) and obstruction of CSF flow, leading to headache, nausea, and vomiting. Other tumors include ependymomas, which originate from the ependymal cells lining the ventricular system, and brainstem gliomas. Treatment of posterior fossa tumors involving a combination of surgery, radiation therapy, and chemotherapy, can yield a favorable prognosis, whereas the prognosis for brainstem gliomas is generally poor.

CEREBROVASCULAR DISEASE

Cerebrovascular disease presents as a transient ischemic at-tack or the more severe and persistent neurologic deficit of stroke. It stems from underlying pathology of the extracranial or intracranial cerebral vasculature. The major categories are ischemic strokes due to thrombosis, embolism or hypoxia, and hemorrhagic strokes due to rupture of a cerebral vessel. Global cerebral ischemia is caused by hypotension, hypoperfusion, and low flow states and results in multifocal infarcts in the border zones (watershed areas) at the interface between the perfusion zones of 2 major arteries or more diffuse encephalopathy.

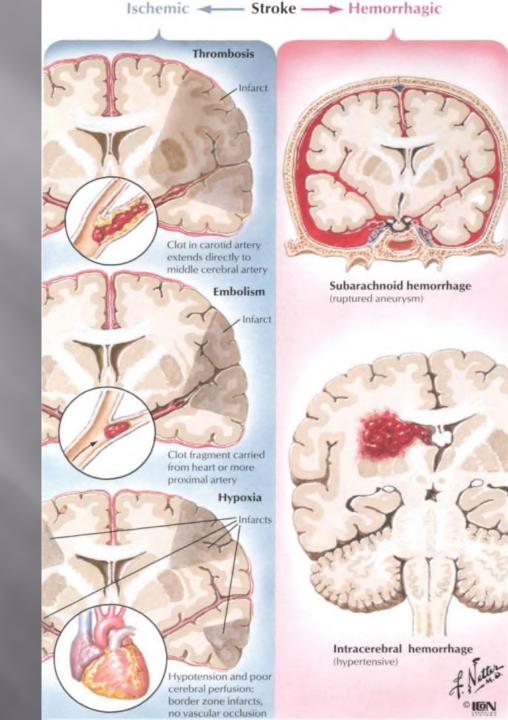
CEREBROVASCULAR DISEASE

Significant obstruction of a component of the carotid or vertebrobasilar arterial tranks leads to focal cerebral ischemia or I infarction. *In situ* thrombosis of a cerebral artery is usually secondary to atherosclerosis or, less commonly, arteritis associjated with infections or collagen-vascular diseases. Other leases of cerebral infarction are due to emboli to the cerebral vasculature from thrombi formed in a diseased heart, the aorta, or a major extracranial cerebral artery. The effects of arterial occlusion can be mitigated to a variable extent by the collateral circulation, particularly through the circle of Willis at the base of the brain. Pale, nonhemorrhagic infarcts are produced by in situ thrombosis, whereas hemorrhagic infarcts due to influx of blood from collateral vessels are produced with cerebral emboli. The distinction between infarction due to *in situ* thrombosis versus embolization is important for optimal clinical treatment, which does not call for the use of anticoagulants in cases of hemorrhagic infarcts due to cerebral emboli.

Hypertension is the most common and important cause of primary intracerebral (intraparenchymal) hemorrhage. Other causes include vascular malformations and hematologic disorders. Hypertension produces cerebral arteriolosclerosis and Charcot-Bouchard microaneurysms. Rupture of the microaneurysm leads to hemorrhage into the brain parenchyma, with frequent extension into the ventricles and subarachnoid space. Hypertensive hemorrhages originate in the basal ganglia in approximately 75% of cases and other sites in the remainder. The most common cause of a major primary subarachnoid hemorrhage is the rupture of a saccular (or berry) aneurysm, located at bifurcation sites of the arteries of the circle of Willis.

Diagnosis of Stroke

Stroke refers to a constellation of disorders in which brain injury is caused by a vascular disorder. The 2 major categories of stroke are ischemic, in which inadequate blood flow due to thrombosis, embolism, or generalized hypoxia causes one or more localized areas of cerebral infarction, and hemorrhagic, in which bleeding in the brain parenchyma or subarachnoid space causes damage and displacement of brain structures. The clinical spectrum of focal cerebral ischemic events includes transient ischemic attacks, residual ischemic neurologic deficit, and completed infarction.



Atherosclerosis, Thrombosis, and Embolism

Atherosclerosis is characterized by the development of foci of intimal thickening composed of variable combinations of fibrous and fatty material and known as fibrous (atheromatous) plaques. Such lesions tend to form adjacent to branch points in arteries. The fibrous plaques may remain static, regress, progress, become calcfied, or develop into complicated atheromatous lesions called dangerous or vulnerable plaques because they are responsible for clinical disease. Complications include loss of endothelial integrity, overt surface ulceration, aggregation of platelets and fibrin on the eroded plaque surface, hemorrhage in the plaque, formation of mural thrombi, embolization of plaque contents or thrombotic material or both, and total arterial occlusion by thrombus. The consequences of thrombotic occlusion are variable and unpredictable depending on the extent of disease and the amount of preexisting collateral blood flow. Thrombotic occlusion often results in tissue infarction

Lacunar Infarction

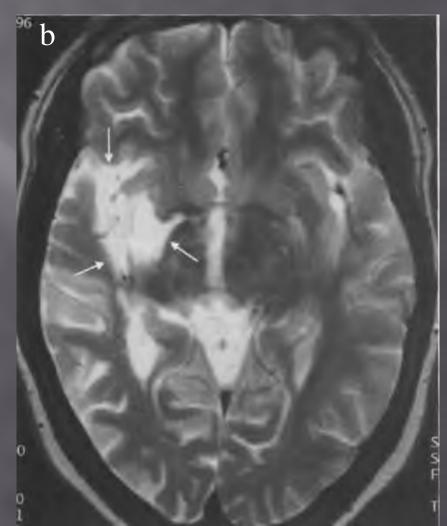
Atherosclerosis involves large- and medium-sized cerebral arteries, whereas hypertension produces disease of small penetrating arteries of the brain. Progressive arteriolosclerosis develops in the small vessels. Hyaline and fibrinoid material thickens the wall and obliterates the lumen. The lacunae (holes), the small, round lesions deep in the brain parenchyma, are commonly found in the brain at autopsy. Some lesions are clinically significant. A small infarct in the base of the pons or internal capsule can produce a pure motor hemiplegia with contralateral weakness of the face, the arm, and the leg but no sensory, visual, or intellectual defects. Other lesions can produce pure sensory strokes. Lacunar lesions in the pons can produce several syndromes, including hemiparesis coupled with ataxia.

Lacunar Infarction



Head of caudatenucleus Putamen Glolous pallidum Thalamus **Cere**bral infarction, (a) Unenhanced CT scan showing a low density region of the left cerebral hemisphere conforming to the distribution of the middle cerebral artery (arrows), (b) MRI scan of another patient with a right middle cerebral artery tentory infarct. The infarcted area (arrows) shows patchy high signal intensity on this T2-weighted image. The arrows point to the anterior and posterior extent of the infarcted brain tissue.



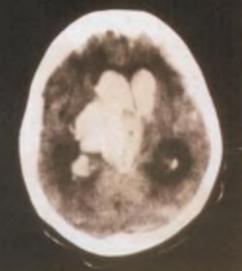


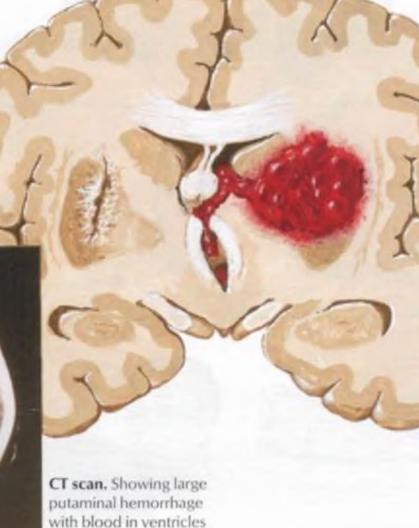
Intracerebral Hemorrhage

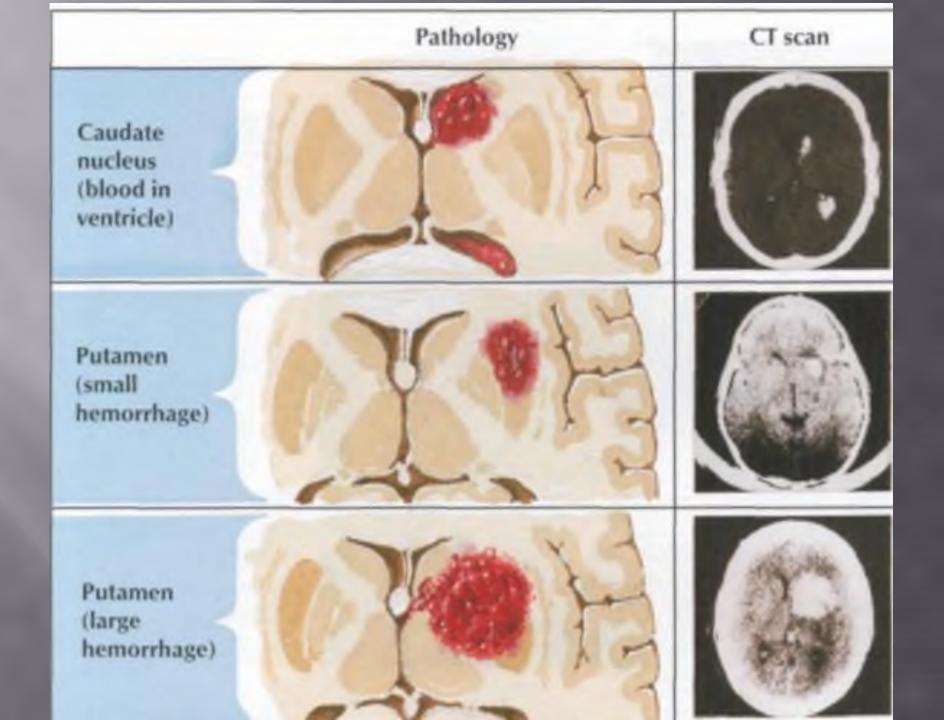
Hypertension is the most common and important etiologic factor in intracerebral hemorrhage. Over time, degenerative changes of the small arteries lead to the formation of microaneurysms. The penetrating lenticulostriate branches of the middle cerebral artery are most commonly involved, but similar changes can occur in small vessels in other parts of the brain. Hemorrhages tend to dissect through white matter pathways, thereby disrupting the cerebral cortex. The enlarging hematoma may extend onto the cerebral surface, producing subarachnoid hemorrhage or rupture into the ventricles. Hypertensive hemorrhage typically occurs in regions where small lacunar lesions develop and involve, in descending order of frequency, the putamen, the cerebral white matter, the thalamus, pons, the cerebellum, and the caudate nucleus. Hemorrhages usually begin while the patient is awake and engaged in daily activity. As the hematoma expands, the focal neurologic deficit gradually increases during a period of minutes or a few hours.

Intracerebral Hemorrhage

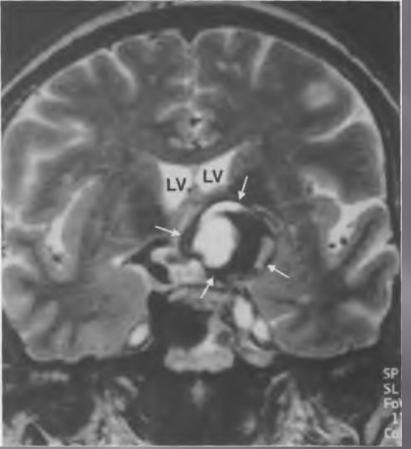
Moderate-sized intracerebral hemorrhage. Involving left putamen, with rupture into lateral ventricle. Brain distorted to opposite side. Scar of healed hemorrhage on right side.



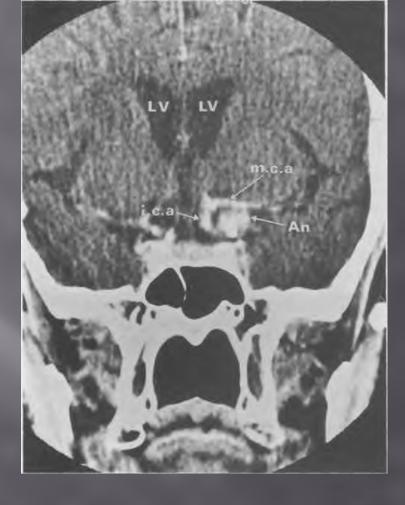




	Pathology	CT scan	Pupils	Eye movement	Motor and sensory deficits	Other
Caudate nucleus (blood in ventricle)	A	\bigcirc	Sometimes ipsilaterally constricted	Conjugate deviation to side of lesion. Slight ptosis.	Contralateral hemiparesis, often transient	Headache, confusion
Putamen (small hemorrhage)	T.H		Normal	Conjugate deviation to side of lesion	Contralateral hemiparesis and hemisensory loss	Aphasia (if lesion on left side)
Putamen (large hemorrhage)	(YOH	0	In presence of herniation, pupil dilated on side of lesion	Conjugate deviation to side of lesion	Contralateral hemiparesis and hemisensory loss	Decreased conscious- ness
Thalamus	No H		Constricted, poorly reactive to light bilaterally	Both lids retracted. Eyes positioned downward and medially. Cannot look upward.	Slight contralateral hemiparesis, but greater hemisensory loss	Aphasia (if lesion on left side)
Occipital Iobar white matter	T		Normal	Normal	Mild, transient hemīparesis	Contralateral hemianopsia
Pons			Constricted, reactive to light	No horizontal movements. Vertical movements preserved.	Quadriplegia	Coma
Cerebellum	And Marine		Slight constriction on side of lesion	Slight deviation to opposite side Movements toward side of lesion impaired, or 6th cranial nerve palsy.	Ipsilateral limb ataxia. No hemiparesis.	Gait ataxia, vomiting



MRI scan (T2-weighted)
showing haemorrhage
surrounding a ruptured middle
cerebral artery aneurysm. The
haemorrhage (arrows) shows
the typical mixture of very high
and very low signal intensity.
LV, lateral ventricles.



Aneurysm (An) of the left internal carotid artery. LV, lateral ventricle; i.e.a., internal carotid artery; m.e.a., middle cerebral artery.

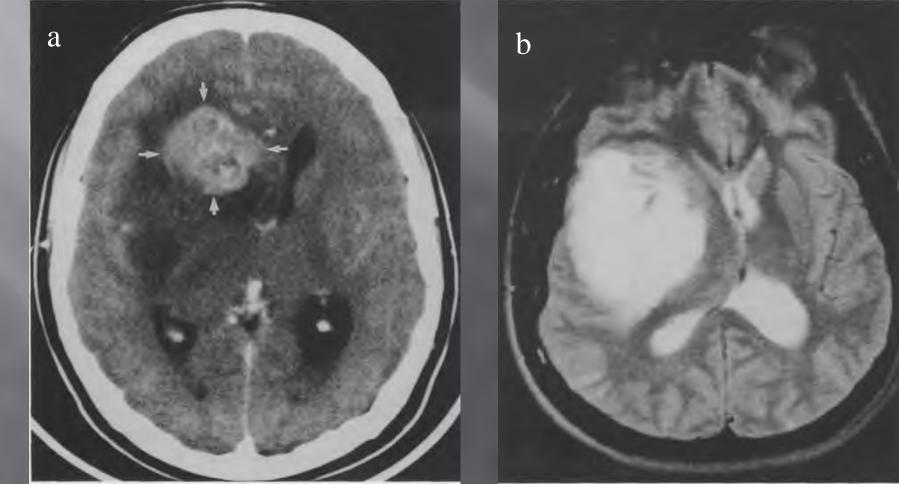
BRAIN TUMORS

Tumors of the central nervous system are either primary or metastatic. The more common metastatic brain tumors may take origin from virtually any primary neoplasm, but the most frequent are lung, breast, melanoma, kidney, and colon. The primary tumors of the central nervous system are classified as gliomas and nonglial neoplasms, including neuronal tumors and meningiomas. The gliomas are the most common primary tumors of the brain and include astrocytomas, oligodendrogliomas, and ependymomas. In children, most braintumors arise in the posterior fossa and include astrocytomas and medulloblastomas of the cerebellum and gliomas of the brainstem, whereas in adults, most brain tumors arise in the cerebral hemispheres. The distinction between benign and malignant lesions is blurred because of the infiltrative growth pattern, frequent involvement of vital structures, and the tendency for lower-grade lesions to transform over time to higher-grade lesions, including the glioblastoma multiforme. Meningiomas are typically benign tumors of adults that arise from the meningoepithelial cells of the arachnoid, become attached to the dura, and produce symptoms by compression of adjacent structures. Most tumors of peripheral nerves are derived from Schwann cells. Acoustic neuroma is a single lesion that produces a mass effect in the cerebellopontine angle. Neurofibromatosis, or von Recklinghausen disease, is the prototype of a group of inherited disorders known as phacomatoses, in which defects of the neural crest lead to multifocal lesions of the nervous system and the skin.

Gliomas

Patients with brain tumors present with symptoms resulting from either increased intracranial pressure or focal brain dysfunction. Gliomas, the most common tumors of the brain, arise from the glial supporting tissue rather than the neurons. The tumors show differentiation toward any of the normal glial components (astrocytoma, oligodendroglioma, ependymoma, and ganglioneuroma). The tumors of each cell type range from moderately well-differentiated, slow-growing neoplasms to pleomorphic, rapidly growing tumors, the most common of which is the glioblastoma multiforme. The glioblastomas are characterized by vascular proliferation and necrosis and cellular pleomorphism. The prognosis, which varies with the location and type of tumor, is difficult to determine because glioblastomas may show a mixed pattern with high-grade areas adjacent to low-grade areas, and low-grade tumors tend to progress over time to highgrade lesions.

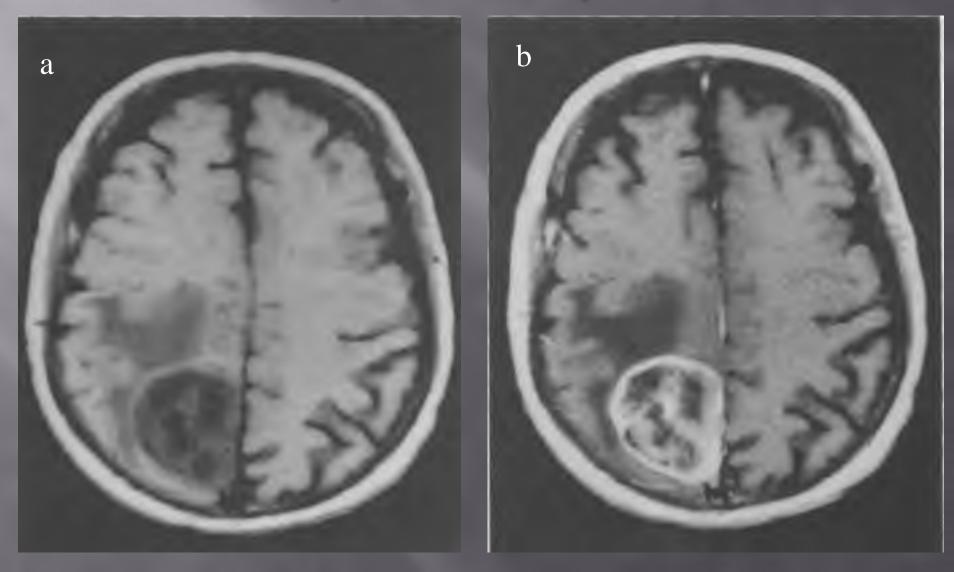
Glioma, (a) CT scan, post i.v. contrast, showing round mass (arrows) with contrast enhancement and surrounding oedema. Note the compression and displacement of the adjacent lateral ventricles, (b) MRI scan (T2-weighted) in another patient, showing a large, high-intensity rounded lesion with displacement of the adjacent ventricular system.



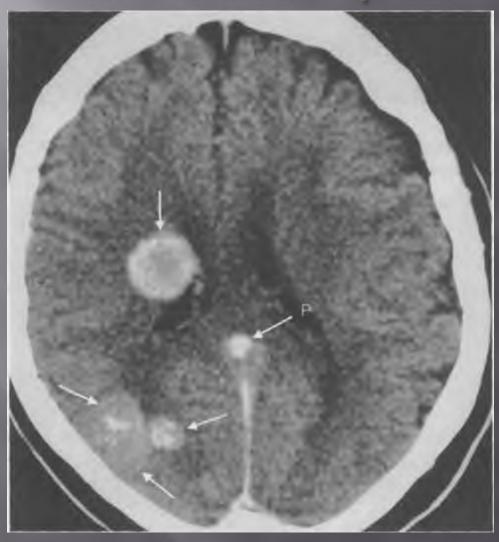
Tumors Metastatic to the Brain

Certain common neoplasms, particularly carcinomas of the lung and the breast, as well as less common neoplasms, including carcinoma of the kidney and melanoma, have a propensity to metastasize to the brain or spinal cord. Metastatic brain tumors are more common than primary brain tumors. Brain metastases may be the first manifestation of an aggressive tumor such as lung cancer. Most metastatic tumors reach the brain through the bloodstream (hematogenous metastases) and become localized at the border between white and gray matter, although occasionally a tumor may spread directly to the brain by local extension from a head and neck cancer or via Batson venous plexus. Metastatic tumors are usually well demarcated and solid, but they may be cystic. Some tumors may be hemorrhagic at the time of presentation, confusing the real diagnosis. The lesions are frequently multiple. CSF examination may yield evidence of meningeal carcinomatosis.

(a) Pre-and (b) postcontrast enhancement (with intravenous gadolinium) shows the obvious partial enhancement of the tumour. Note the adjacent low intensity white matter oedema.



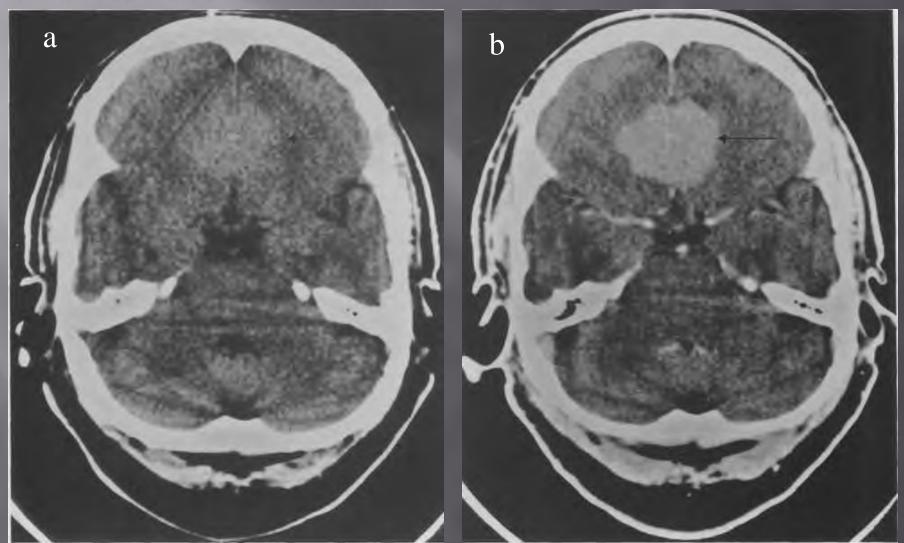
Metastases. Enhanced CT scan showing several rounded areas of increased density (arrows). The round density in the midline is due to the pineal.

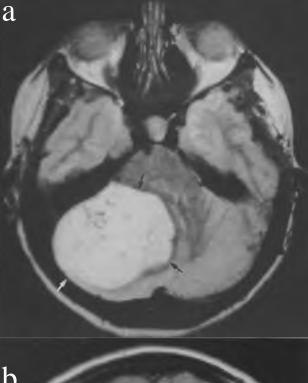


Meningiomas

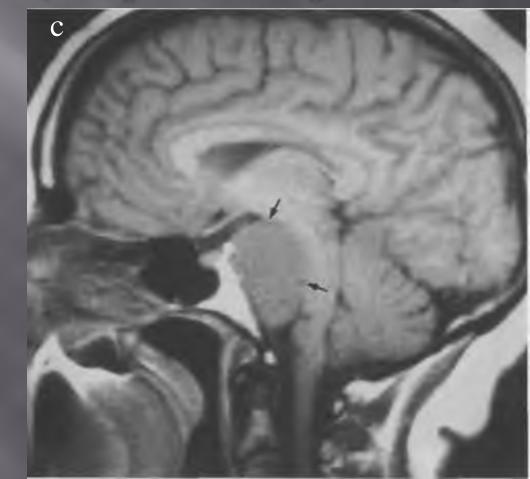
Meningiomas are the most common of the benign brain tumors. Their incidence increases with age, with a moderate female preponderance. Meningiomas, which arise from arachnoid cells in the meninges, are nearly always benign, but rare malignant variants occur. Most meningiomas are composed of groups of cells arranged in a whorled pattern without identifiable cell membranes (syncytial type), sometimes containing large numbers of calcified psammoma bodies (psammomatous type). **Fibroblastic and transitional variants also occur. The symptoms** depend on location of the tumor, the growth rate, and adherence to adjacent structures rather than on histologic type. Meningiomas may extend into venous structures, such as the superior sagittal sinus, or erode into the bone of the skull.

Meningioma, (a) Precontrast image showing that the density of the meningioma (arrow) is slightly greater than the brain substance owing to fine calcification in the tumour, (b) Enhanced CT scan showing a large midline tumour (arrow) beneath the frontal lobes. Note the marked contrast enhancement.





Multiplanar imaging capability of MRI in a patient with a large meningioma in the posterior fossa (arrows), (a) T2-weighted axial section, (b) TIweighted coronal section, (c) Meningioma (arrows) of the clivus, in a different patient, pressing on the pons (TI-weighted midline sagittal section).



Pituitary Tumors

Pituitary tumors of the adenohypophysis are classified on both a functional and an anatomical basis. Using standard histology, the tumors are classified as eosinophilic adenoma, basophilic adenoma, and chromophobe adenoma. The eosinophilic adenoma is associated with acromegaly, and the basophilic adenoma is associated with Cushing syndrome. The chromophobe adenoma, the most common type of tumor, may be nonfunctioning. A more accurate classification can be obtained by immunocytochemical staining for specific hormones. Clinically, important features include the degree of sella turcica enlargement and erosion and the type of suprasellar extension. Precise delineation of tumor extent can be obtained with a combination of CT and MRI scans and angiography.

Normal nituitary fossa: 1. anterior clinoid process: 2. nosterior clinoid process; 3/ dorsum sellae; 4. floor. The white line forming the floor and

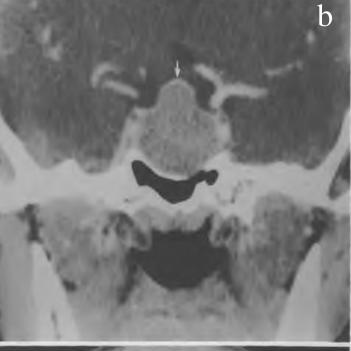
the dorsum sellae.

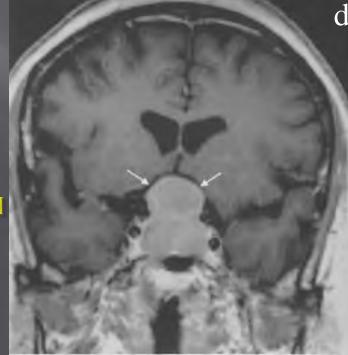
Pituitary tumour causing enlargement of the pituitary fossa with a sloping floor. The floor appears as a double line on the lateral view (arrows).



Pituitary tumour, (a) Computed tomography sean after contrast shows a mass in the pituitary fossa which enhances vividly (arrows), (b) Direct coronal postcontrast CT scan in another patient, showing a large tumour expanding the pituitary fossa and projecting superiorly (arrow), (c) Sagittal MRI scan of a pituitary tumour (arrows) in another patient, (d) Coronal MRI scan, postcontrast, in a similar patient (the arrows point to the tumour).

a

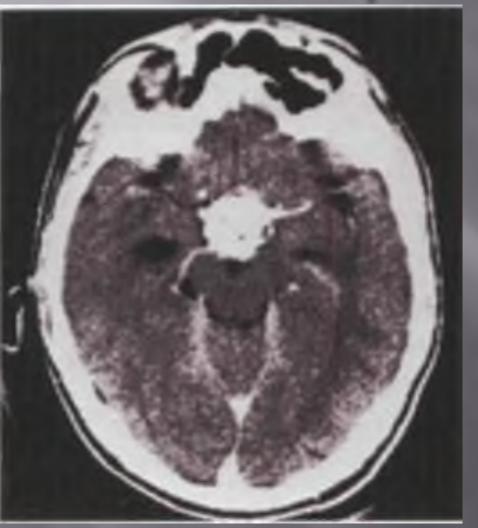


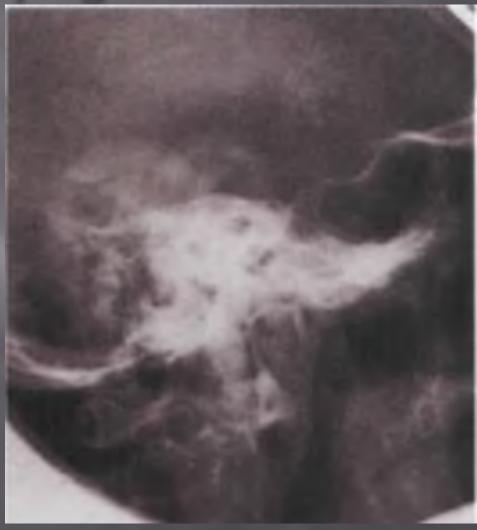


Craniopharyngiomas

Craniopharyngiomas are the most common parasellar tumors in children, but they also occur in adults. Craniopharyngiomas arise from remnants of the Rathke pouch derived from the embryonic pharynx. The lesion is composed of clusters of columnar and cuboidal epithelial cells. The tumor may be solid or cystic because of formation of degenerative areas containing oily fluid, calcium, and keratin. The tumor routinely extends to the optic chiasm. A craniopharyngioma produces visual symptoms secondary to compression of the optic tract. Approximately 50% of patients have endocrine dysfunction, with diabetes insipidus, panhypopituitarism, and gonadal deficiency in adults and growth retardation and obesity in children. Hydrocephalus, often with papilledema, also can develop in children with this tumor.

Craniopharyngiomas





Craniopharyngioma. CT scan.

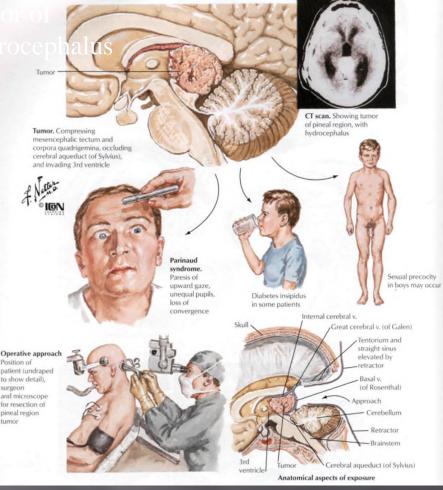
Tomogram. Flocculent calcification in craniopharyngioma

Tumors of the Pineal Region

The pineal gland has a strategic central location in the brain surrounded by vital structures, including the posterior third ventricle. Symptoms result from compression or involvement of these vital structures by the pineal tumor. Pineal tumors can be classified into tumors of germ cell origin, tumors of the pineal parenchyma, and a miscellaneous group. Tumors of germ cell origin are germinomas and teratomas. Germinomas, which comprise approximately half of all pineal tumors, are most common in adolescents and have a marked predilection for males. Teratomas have a similar male predilection. These tumors usually present with endocrine abnormalities. The germinoma usually spreads via the CSF but is radiosensitive, whereas teratomas are not invasive. Pinealcytoma is well circumscribed and noninvasive. It occurs at any age and has no sex predilection. The malignant pineal blastoma is composed of primitive cells resembling medulloblastoma and spreads within the CSF. Other pineal tumors include benign meningiomas and cysts.

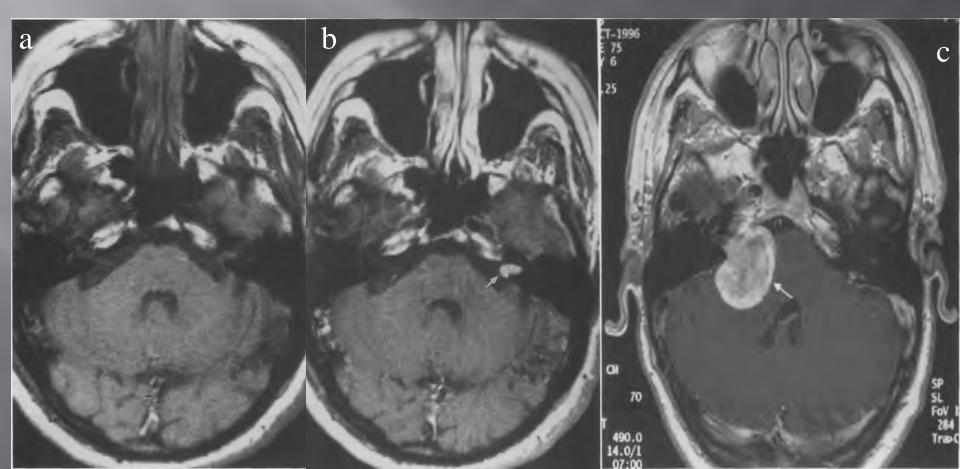
Tumors of the Pineal Region

CT scan. Showing tumor pineal region, with hydrod



Acoustic neuroma

Neurofibromas of the acoustic nerve arise in the internal auditory canal or immediately adjacent to the internal auditory meatus in the cerebellopontine angle. When large, they can be recognized at CT or MRI. When small, they may only be identifiable with MRI. Contrast enhancement improves their visibility with either technique. Acoustic neuroma, (a) Precontrast MRI scan; the acoustic neuroma is virtually invisible, (b) Post gadolinium enhancement; the small acoustic neuroma (arrow) in the left internal auditory canal is clearly demonstrated, (c) A different patient with a larger right-sided acoustic neuroma (arrow) in the cerebellopontine angle (enhanced TI-weighted scan).



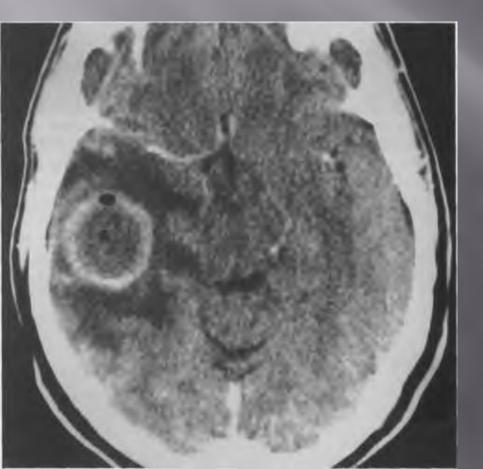
INFECTIOUS DISEASES

 Infections of the central nervous system may develop as a result of seeding of microorganisms via the hematogenous route, direct implantation from trauma or medical intervention, local spread from a contiguous site such as the paranasal sinuses, or retrograde spread along a peripheral nerve, as is the case with certain viral infections such as herpes simplex and rabies.

Infectious meningitis

Infectious meningitis of the leptomeninges and the cerebrospinal fluid (CSF) presents with fever, somnolence, and stiff neck. Examination of the CSF is important to differentiate acute pyogenic bacterial meningitis (numerous white blood cells with neutrophil predominance, high protein, low glucose) from aseptic (viral) meningitis (lymphocytic pleocy-tosis, moderate protein increase, normal glucose) and chronic forms of meningitis, including tuberculous meningitis (pleo-cytosis with mononuclear cells or mixed mononucle, and neutrophils, markedly increased protein level, and moderately reduced or normal glucose level). Parameningeal'm fections consist of brain abscess, subdural empyema, and spinal epidural abscess. Neurosyphilis occurs late in the course of approximately 10% of untreated patients jm may be manifest as meningeal-meningovasc ular disease, dementia paralytica (general paresis), or tabes dorsalis. A number of viruses can produce encephalitis or encephalomyelitis, characterized by meningeal and parenchymal, particularly perivascular, inflammation.

Cerebral abscess in temporal lobe. Postcontrast CT scan showing a spherical mass with central low density and marked ring enhancement from the edge of the abscess (A). A small bubble of gas is seen at the top of the abscess.



Small cerebral abscess in a patient with AIDS. MRI scan (Tlweighted, postcontrast) shows ring enhancing lesion in the upper brain stem (arow).

