6 The Skull, Brain and Associated Structures: Part I Applied Anatomy and Physiology

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6.1 The Scalp

The scalp is anatomically described as having five layers: **S**kin, Sub**C**utaneous tissue, **A**poneurosis, **L**oose areolar tissue and **P**ericranium (SCALP -a well known and useful acronym). Functionally however this can be considered as two layers—a deeper layer of pericranium and areolar tissue (which is attached to the skull), upon which sits a mobile superficial layer comprised of skin, subcutaneous tissue and the galea aponeurotica. It is between these two layers that the scalp moves and blood can collect—a scalp or subgaleal haematoma. Most scalp lacerations extend the full thickness of the upper layer.

The vessels and nerves of the scalp pass predominantly within the subcutaneous tissue. Anteriorly, the supraorbital and supratrochlear nerves and vessels exit their foramina in the supraorbital ridges, passing upwards towards the vertex of the scalp. Laterally, the superficial temporal artery and auriculotemporal nerve pass upwards superficial to the temporalis fascia. Segments of the superficial temporal artery can often be excised at this site and submitted for histology, to establish a diagnosis of temporal arteritis. Posteriorly, the posterior auricular and occipital arteries pass upwards within the scalp. These arrangements result in a scalp which has a very rich blood supply and can bleed profusely. In children particularly but also adults, blood loss from the scalp can be significant and sometimes result in hypovolaemic shock.

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6.2 The Skull

The skull consists of the calvarium (which contains the brain) and the facial skeleton. The calvarium consists principally of eight major bones

- Frontal (1)
- Sphenoid (2)
- Temporal (2)
- Parietal (2)
- Occipital (1)

A small part of the ethmoid bone completes the skull base anteriorly.

The calvarial bones are separated by five sutures: frontal, sagittal, lambdoid, coronal and squamous. The bones of the skull are derived almost entirely from neural crest cells, except for the basilar part of the occipital bone, which forms from mesoderm of the occipital sclerotomes. A useful mnemonic for the bones of the skull is "Old People From Texas Eat Spiders" (Occipital, Parietal, Frontal, Temporal, Ethmoid, Sphenoid).

Embryologically, the skull (cranium) develops within the mesenchyme surrounding the developing brain, and is divided into two parts (i) the neurocranium that encloses cranial cavity and protects the brain and (ii) the viscerocranium that forms the skeleton of the face.

1. The neurocranium is comprised of a cartilaginous portion that forms the base of the skull and a membranous portion that forms the cranial vault. The base of the skull is thus initially cartilaginous, formed by the fusion of several cartilages. These are formed by chondrification of the mesenchyme below the brain. They subsequently ossify to form the thick skull base. The vault of the skull develops from the mesenchyme on superior and lateral aspects of the developing brain (the membranous neurocranium). This tissue is derived from the neural crest cells and paraxial mesoderm. The vault is initially membranous, but later undergoes membranous ossification to form a number of flat membranous bones, which comprise the cranial vault. Primary centres of ossification later appear in each bone and grow towards its periphery, forming needle-like bony spicules.
2. Much of the viscerocranium is derived from the mesenchyme from the first pharyngeal arch (maxillary and mandibular processes) and the second pharyngeal arch. This mesenchyme undergoes membranous ossification to form the bones of the facial skeleton. This is described in more detail of the relevant chapters.

The bones of the skull are thus formed by (i) membranous ossification, (ii) cartilaginous ossification, or (iii) both. Accordingly, there are of three types of bones-membranous, cartilaginous and membranocartilaginous bones. The newborn skull presents two striking features:

1. A small viscerocranium (facial skeleton) compared to neurocranium.
2. Presence of fontanelles.

The relatively huge size of neurocranium is due to the relatively rapid development of the brain during foetal growth and early years of life. This reaches 25% of its adult size at birth and 75% by the age of 4 years. The face (viscerocranium) is small because of the small size of the facial bones, particularly the jaws and the virtual absence of paranasal air sinuses. These develop later throughout childhood and adolescence.

Fontanelles are the soft membranous areas in the vault of newborn skull. They are also sometimes referred to as "soft spots". These develop at sites where more than two bones meet and where the sutures widen to fill the gap. There are six fontanelles at birth—one at each angle of the parietal bone. Two in the sagittal midline (anterior and posterior fontanelles) and two on either side of the vault (sphenoid and mastoid). The most prominent of these is anterior fontanelle. This is diamond shaped and located where the two halves of the frontal bone and the two parietal bones meet. It can usually be seen to pulsate. All the fontanelles except the anterior, close within 3 or 4 months following birth. The anterior fontanelle usually closes completely between the second and third year of age. The presence of the fontanelles allows the bones of the skull to move slightly and overlap (moulding) during parturition, to facilitate birth. They also enable the skull to accommodate brain development postnatally. Palpation of anterior fontanelle can thus provide valuable information regarding whether ossification is proceeding normally and whether the intracranial pressure is normal or not. A sunken fontanelle can indicate dehydration, whilst a bulging, tense fontanelle is seen with raised intracranial pressure. In some babies, moulding can exert considerable tension at the "obstetrical hinge" (junction of the squamous and lateral parts of the occipital bone), such that the great cerebral vein (of Galen) can rupture during childbirth.

The calvarium is relatively elastic in consistency. This is why an impact can injure the underlying brain or tear vessels without fracturing bone. The skull is thickest over the vertex (between 0.5 and 1 cm). It is thinnest in the temporal region and where it forms the roof of the nose and orbit. Here the bones are so thin that light can pass through (you can verify this for yourself if you come across a real skull). All upper facial injuries should therefore be considered as potential anterior skull base fractures until radiologically excluded. Skull thickness varies from person to person. It is also affected by some diseases (notably Pagets disease and intraosseous meningioma). Where the surface bone is thin, it is protected by thick muscle (such as the temporalis). Radiologically, identification of sutures and vascular markings is important because these can be confused with fractures. Unlike the straight lines of a fracture, suture lines and vascular markings are tortuous.

6.3 Hyperostosis Frontalis Interna (HFI)

This is a benign normal variant of unknown cause in which there is increased thickness of the cancellous bone of the frontal bone. It is a progressive and symmetrical process with an incidence of 4–5% in the general population. Patients with HFI may suffer from headache and neuropsychiatric disorders such as epilepsy and dementia. HFI has also been linked to some endocrinopathies such as diabetes mellitus, toxic

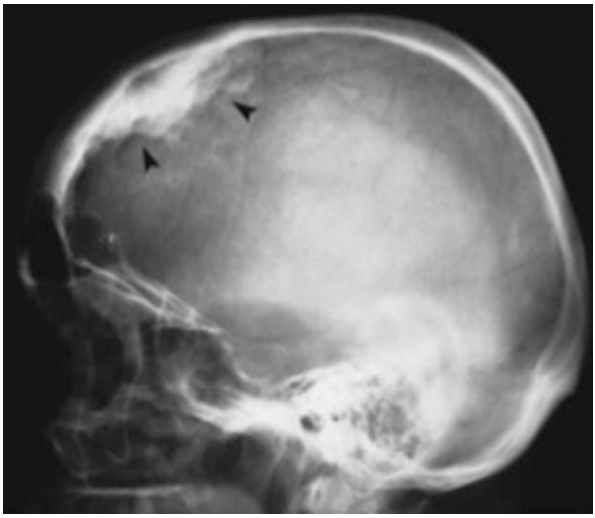

Fig. 6.1 (**a**) A lateral plain radiograph of the skull shows increased bone thickness in the inner surface of the frontal bone (*arrowheads*)

goitre, and acromegaly. Morgagni syndrome is characterised by obesity, hirsutism (abnormally excessive hair growth) and HFI (Fig. 6.1).

Internally, the skull is divided into the anterior, middle and posterior cranial fossae. The anterior cranial fossa (ACF) contains the anterior part of the frontal lobe of the brain. It extends back to the lesser wing of the sphenoid bone and lays above the orbits and nose. The bone here is very thin and often fractured following impacts to the upper face. Associated tears in the dura can result in CSF leaks. Tumours can also readily pass from the orbits and nose into the ACF and vice versa, resulting in unusual symptoms. The anterior fossa is perforated by the olfactory nerves only (CN I).

The middle cranial fossa is the largest of the three. It is continuous with the anterior cranial fossa above the lesser wing of the sphenoid and is separated from the posterior cranial fossa by the tentorium cerebelli. The middle fossa is occupied by the temporal lobes of the brain and above these is the remainder of the frontal, parietal and occipital lobes. The carotid arteries enter the skull and the CN II to VI leave the skull via the middle fossa floor. Because the bones are thick, fractures of the middle cranial fossa represent high energy impacts. If a fracture extends to the carotid foramen the vessel may sometimes be damaged. This can result in catastrophic brain haemorrhage or ischaemia. Tumours involving the skull base can present with cranial nerve symptoms and deficits. For this reason unexplained facial numbness or palsy requires further investigation (MRI or CT).

The posterior cranial fossa lies below the tentorium cerebelli. This contains the midbrain, pons, medulla and cerebellum. The major venous outflow of the brain passes through the posterior fossa, where the sigmoid sinus continues as the internal jugular vein. CN VII to XII also exit through the posterior fossa. The medulla continues as the spinal cord through the foramen magnum, which is also where the vertebral arteries and spinal root of the accessory nerve enter the skull. Injuries or tumours at this site can result in high spinal cord symptoms (sometimes fatally so).

6.4 Paget's Disease (Osteitis Deformans)

This condition is mentioned here, although the skull is not the only site involved. It is a slowly progressive disorder of unknown aetiology, which predominantly affects males over 50. One unproven theory is that Paget's disease may be a delayed or slow reaction to a myxovirus stimulus. Bone undergoes hyperactive turnover with alternating phases of resorption, vascularity and sclerosis. Most of the bones of the body can be involved. In the head and neck, the skull, maxilla and mandible may be affected. The classic presentation used to be a patient whose hat or dentures no longer fitted due to bone swelling. Today patients present more with bone deformity or pain. Headaches and symptoms secondary to vascular and nerve compression can also occur. Radiographically the skull and maxilla have a "cotton-wool" appearance, with hypercementosis around the roots of teeth, and loss of the periodontal space. This can make tooth extraction extremely difficult. Serum alkaline phosphatase is usually raised. Treatment is directed toward controlling the disease activity and managing its complications. In many patients no treatment may be necessary. Bone pain may require anti-inflammatory drugs (NSAIDs) or alternative analgesics. Surgery is occasionally required for damaged joints, fractures, severe deformity, or nerve entrapment. Bisphosphonates or injectable calcitonin (Miacalcin), may also be prescribed. Bisphosphonates are the mainstay of treatment for Paget's disease. A number of alternatives are available including alendronate (Fosamax), risedronate (Actonel) and etidronate (Didronel). Complications of Paget's disease include pain, fracture, deformity, nerve entrapment, arthritis and rarely, sarcoma. Bleeding can occur during surgery if affected bone is operated on. Paget's disease of the skull can result in progressive hearing loss. In most patients the prognosis is good.

6.5 The Meninges

The inner surface of the skull is lined by a tough fibrous membrane—the dura mater (sometimes referred to as the 'pachy' or thick meninges). This becomes more firmly attached to the skull with increasing age. The dura is reflected internally from the bones to form various membranes (falci), which separate and support the lobes of the brain. These include

1. the falx cerebrum, which separates the two cerebral hemispheres
2. the tentorium cerebelli, which separates the middle and posterior cranial fossae, and
3. the falx cerebelli, which separates the two cerebellar hemispheres.

The extradural space, between the dura and the skull, is a potential space only (Fig. 6.2). Normally it does not exist. Several large venous sinuses, providing the major venous outflow of the brain, lie within the dura. The superior sagittal sinus and inferior sagittal sinus lie along the upper and lower margins of the falx cerebrum respectively. The inferior sagittal sinus continues as the straight sinus, which passes along where the falx joins the tentorium. The straight and superior sagittal

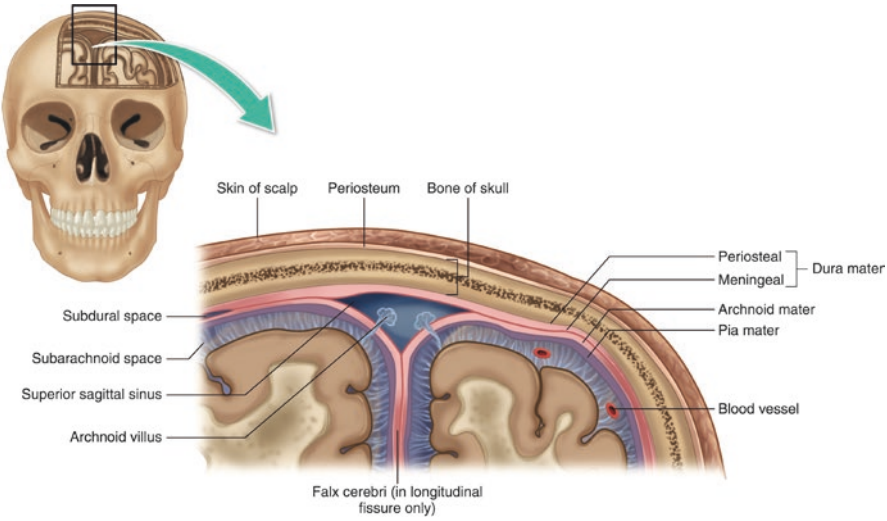

Fig. 6.2 Skull and dural anatomy

sinuses join at the confluence of the sinuses, or torcula, which lies internal to the external occipital protuberance. These sinuses continue laterally as the transverse sinuses, which lays within the lateral margin of the tentorium. This complicated anatomical arrangement of the sinuses usually results in most of the blood from the superior sagittal sinus passing into the right transverse sinus, and blood from the straight sinus passing into the left. The transverse sinuses continue as the sigmoid sinuses, which curve through the posterior fossa to the jugular foramen, where they become the internal jugular veins (Fig. 6.3).

Knowledge of the anatomy of the sinuses is important when assessing the impacts of depressed skull fractures. Large depressed fragments may impede venous drainage, resulting in widespread venous congestion and infarction within the brain. Furthermore attempts to elevate such fragments may result in catastrophic haemorrhage if the sinus lining is torn. When planning surgical access to the brain, bony cuts are placed to avoid the venous sinuses whenever possible.

The cavernous sinuses are paired structures that are composed of multiple venous sinusoids. They lie alongside the sella turcica (pituitary fossa) and communicate with the sigmoid sinuses via the superior and inferior petrosal sinuses, which pass along the upper and lower borders of the petrous temporal bone. Each cavernous sinus receives blood from superior and inferior ophthalmic veins, pterygoid plexus, sphenoparietal sinuses and superficial cerebral veins. They also drain freely between each other (Fig. 6.4). Extracranial infection can therefore potentially pass intracranially via the ophthalmic veins, resulting in devastating septic thrombosis of the cavernous sinus. The internal carotid artery and cranial nerves III, IV, V_1, V_2 and VI also pass through the sinus. Cranial nerve VI passes through

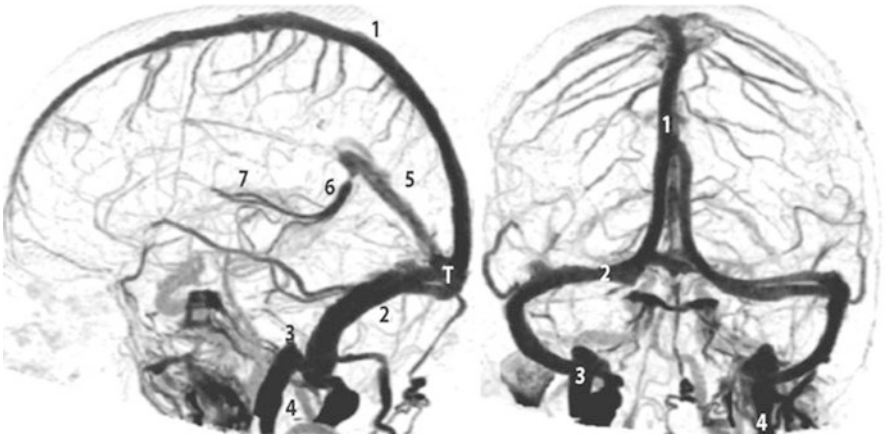

Fig. 6.3 Digital subtraction angiography (DSA) of the cerebral venous circle with MIP lateral and frontal view: superior sagittal sinus (1), transverse sinus (2), sigmoid sinus (3), internal jugular vein (4), torcular Herophili or sinus confluence (T), straight sinus (5), Galen vein (6), internal cerebral veins (7)

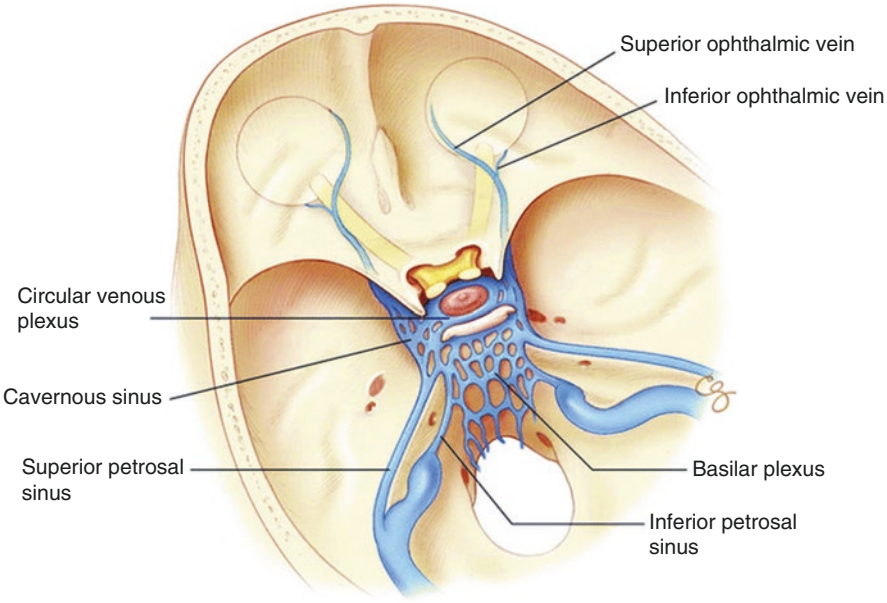

Fig. 6.4 Anatomy of the cavernous sinus drainage system. From BCSC Neuro-ophthalmology. Reproduced with permission from American Academy of Ophthalmology

the middle of the sinus alongside the internal carotid artery, the remaining nerves pass through the lateral wall of the sinus. This complex anatomical arrangement has important implications in the diagnosis and management of caroticocavernous fistula.

The arachnoid mater lies deep to the dura. This is a relatively flimsy membrane comprising the parietal layer of the 'lepto' (or thin) meninges. The subdural space between the dura and arachnoid is usually empty. However the two membranes are not adherent and blood can easily collect within this space. The subarachnoid space lies deep to the arachnoid and contains the cerebrospinal fluid (CSF). This helps to support and cushion the brain. At various places, mostly around the base of the brain, the subarachnoid space becomes very wide and forms the "basal cisterns".

The pia mater is the visceral layer of the leptomeninges. This is firmly attached to the brain.

6.5.1 Arachnoid Cysts

These are congenital lesions lined by arachnoid membrane and filled with CSF. They have been suggested to increase the risk of subdural haemorrhage or hygroma following head trauma, possibly as a result of rupture of the cyst or its vessels.

6.5.2 Cavernous Sinus Syndrome (CSS)

This is a rare condition which highlights the complex anatomical arrangement of the cavernous sinus and associated structures. It is characterised by ophthalmoplegia, proptosis, conjunctival congestion, trigeminal sensory loss and Horner's syndrome. These signs and symptoms result from the involvement of the cranial nerves passing through the cavernous sinus. CS pathology accounts for 5% of all cases of ophthalmoplegia. The most common cause of CSS is a neoplastic lesion (primary, secondary or from local spread). Other causes include (i) thrombophlebitis, (ii) aspergillosis, (iii) Tolosa-Hunt syndrome, (iv) inflammatory pseudotumour, (v) aneurysm of the internal carotid artery, (vi) carotid–cavernous fistula and (vii) dural arteriovenous shunt. Conditions that produce painful ophthalmoplegia can also mimic CSS (vasculitis, basal meningitis, diabetes mellitus and ophthalmoplegic migraine), making this a difficult diagnostic condition. Contrast-enhanced MRI is usually required.

6.6 Cerebral Blood Supply

The internal carotid and vertebral arteries supply the brain. The internal carotid arteries divide into the anterior and middle cerebral arteries. The anterior cerebral artery supplies the inferior surface of the frontal lobe and the anterior-medial surface of the hemisphere, extending a short distance onto the lateral surface. This includes the leg area of the motor cortex. The middle cerebral artery supplies most

of the lateral surface of the hemisphere. This includes the trunk, arm and face areas of the motor cortex, speech area (on the dominant side) and auditory cortex.

The two vertebral arteries unite to form the basilar artery, which then divides into the two posterior cerebral arteries. These supply the inferior surface of the temporal and occipital lobes and the posterior part of the medial surface of the hemisphere, also extending a short distance onto the lateral surface. This includes the visual cortex.

The vertebro-basilar system also supplies the cerebellum and brainstem. The combination of these vessels results in the circle of Willis—an arterial 'ring' supplied by the internal carotid and vertebral systems. These anastomose around the optic chiasm and infundibulum of the pituitary stalk. This arrangement allows equalisation of blood-flow between the two sides of the brain and provides an anastomotic circulation if part of the ring becomes occluded. A complete circle of Willis (in which no component is absent) is only seen in 20–25% of individuals. Posterior anomalies are common and are seen in nearly 50% of anatomical specimens. Anterior anomalies are less common. Congenital absence of one or both internal carotid arteries (ICA) may also occur but is rare. If one ICA is absent there is a high incidence of associated flow related aneurysms.

6.7 The Blood-Brain Barrier (BBB)

This is composed of a complex network of cells that make up the cerebral capillaries and post-capillary venules. Under normal conditions it provides a selectively permeable barrier between the vasculature of the brain and the brain itself, regulating the passage of substances into the brain. The BBB therefore functions to provide a constant supply of nutrients and maintain ion homeostasis, whilst at the same time protecting against wide variations in blood composition and harmful chemicals. This includes restricting access of some drugs. Following trauma, infection and other local pathological processes, this homeostatic function can be severely disrupted. Early increase in BBB permeability has been reported following diffuse injuries, whilst a biphasic disruption has been reported in more focal injuries. This can result in vasogenic cerebral oedema, which can significantly alter intracranial pressure. The loss of barrier integrity following acute brain injury also allows peripheral immune cells to enter the brain and exacerbate inflammatory processes.

6.8 Arteriovenous Malformations (AVMs)

Cerebral arteriovenous malformations are believed to be congenital but more recent evidence suggests that they develop in childhood. They have a prevalence of around 0.01%. Of these approximately 15% of patients are asymptomatic, 20% present with seizures and approximately 65% present with intracranial haemorrhage (discussed later). As with AVMs elsewhere in the body, there is an abnormal shunting of flow between arterial inflow and venous outflow, without a normal capillary network. Radiographically, two major types of shunts are described (i) plexiform nidus (a collection of small vessels with shunting, often seen in the brain parenchymal) and (ii) a fistula (a single-lumen communication between an artery and vein),

although these are often found together in a single lesion. Anatomically cerebral AVMs may be divided into dural, pial, or mixed. One particular type of malformation is a fistula of the vein of Galen malformation. This is rare and results from the persistence of the embryologic prosencephalic vein of Markowski, which drains the arteriovenous shunt but not normal brain tissue. The true vein of Galen fails to develop. Other anomalies of cerebral venous drainage occur. Diagnosis of AVMs relies on imaging (CTA, MRA) and treatment usually involves endovascular, surgical or radiotherapeutic procedures.

6.9 The Brain

Each of the cerebral hemispheres is divided into four lobes. The central sulcus separates the frontal lobe from the parietal lobe, the parieto-occipital fissure separates the parietal and occipital lobes and the Sylvian fissure separates the temporal lobe from the frontal and parietal lobes. The cortical surface of the brain is further convoluted into multiple gyri (folds) and sulci (clefts). These increase the area of the cortex, the site where the higher functions are organised.

Cortical functions are crossed, that is, one hemisphere deals with the function of the other side of the body. The left hemisphere is dominant for speech in 99% of right-handed individuals (since the right hemisphere is dominant in 1%, dysphasia can occasionally be caused by a right cerebral lesion). There is a 50:50 likelihood of either hemisphere being dominant for speech in left-handed individuals, but right hemisphere dominance is more likely if there is a strong family history of left-handedness. Functional MRI can help distinguish dominance and is often important in surgical planning.

6.9.1 Localisation of Cortical Functions

- Primary motor area—Precentral gyrus of the frontal lobe (here the body image is inverted with the leg area on the medial hemisphere surface)
- Primary sensory area—Postcentral gyrus of parietal lobe (again the body image is inverted with the leg area on the medial hemisphere surface)
- Speech motor area*—(Broca's area) Infero-lateral frontal lobe (just above tip of temporal lobe)
- Speech interpretation area*—(Wernicke's area) Inferior parietal lobe and upper temporal lobe (behind primary sensory area)
- Visual cortex—Tip of occipital lobe, especially medial surface
- Auditory cortex—Superior temporal gyrus
- Higher intellectual functions—Tip of frontal lobe (unilateral lesions cause minor deficit only)
- Emotions—Inferior frontal lobe, tip of temporal lobe and cingulate gyrus (on the medial surface, above corpus callosum). Other deep parts of the limbic system are also involved (these are a series of structures that surround the lateral ventricle, including the hippocampus, amygdala and fornix)

- Olfactory function—Infero-medial temporal lobe
- Other parietal lobe functions include
  - (dominant) numeration, calculation
  - (non-dominant) body image & awareness of external environment

**Dominant hemisphere only* (Fig. 6.5)

The two hemispheres are connected by the commissures, the largest and most important of which is the corpus callosum. Descending white matter tracts from each cortex converge to form the internal capsule en route to the brainstem. Here the motor fibres are condensed into the posterior limb. A small lesion here can therefore produce a major deficit. Ascending sensory fibres (except olfaction) relay in the thalamus. This is lateral to the internal capsule. The remaining basal ganglia are

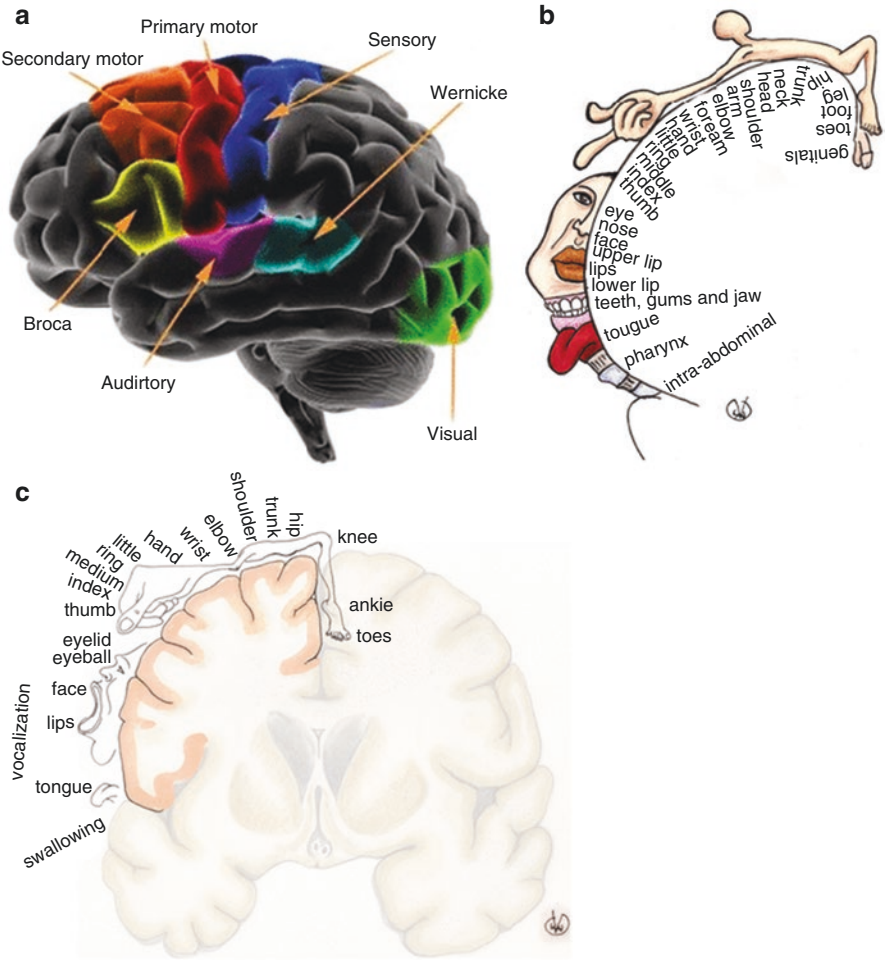

Fig. 6.5 Schematic representation of the main cortical areas: motor, sensory and special sensory areas of the left hemisphere

concerned with motor function, and have complex interconnections. The hypothalamus is concerned with autonomic function and endocrine control through the pituitary gland.

6.9.2 The Cerebellum

The cerebellum (Latin for "little brain") is concerned with fine motor control. Although it accounts for approximately 10% of the brain's volume, it contains over 50% of the total number of neurons in the brain. It does not initiate movement, but contributes to fine coordination and precision once under way. The cerebellum consists of two hemispheres and the midline vermis. These receive input from the spinal cord, vestibular system and other parts of the brain, which are integrated to facilitate fine motor activity. The cerebellum is divided into three lobes (anterior, posterior and flocculonodular lobe), but these divisions are not usually obvious on external inspection. Damage to the vermis causes ataxia and unsteadiness on sitting (truncal ataxia). Damage to the cerebellar hemispheres causes incoordination on the same side of the lesion. In general, patients lose coordinated voluntary movements and develop problems maintaining balance and posture. These include

1. Decomposition of movement. Most movements involve the coordinated activity of many muscle groups and different joints to produce a smooth movement. Patients with cerebellar dysfunction are unable to produce this. Instead, they often break the movements down into their component parts. For example, when touching one's finger to one's nose, the patient first performs the shoulder movement, then the elbow and finally the wrist movement in sequence, rather than in a uniform motion.
2. Intention tremor. When making a movement to a target, cerebellar patients produce an involuntary tremor that increases as they approach the target (for example, if reaching for a cup).
3. Dysdiadochokinesia. Difficulty performing rapidly alternating movements.
4. Deficits in motor learning.

6.9.3 The Brainstem

The brainstem connects the cerebrum with the spinal cord. It consists of the midbrain, medulla oblongata and the pons. Motor and sensory neurons travel through the brainstem, facilitating signals between the brain and the spinal cord, thereby coordinating motor signals to the body. In addition, the brainstem also connects the cerebrum with the cerebellum. The brainstem reticular formation controls life supporting autonomic functions of the body including:

- Alertness
- Arousal

- Breathing
- Blood Pressure Control
- Heart Rate

Most of the cranial nerves originate in the brainstem. The midbrain, pons and medulla contain the nuclei for the third to twelfth cranial nerves, together with the descending and ascending fibre tracts (Fig. 6.6). The midbrain also contains a gaze-control centre. Strokes within the brain stem can therefore have devastating consequences. These may be localised by the pattern of deficits. Brain stem strokes are often caused by obstruction of blood vessels, and have occured with a very forceful neck movement, such as after a roller coaster ride. Vertigo (spinning) is a common early symptom of brain stem strokes.

Locked-in Syndrome This is described later. In essence, a large stroke in the upper brain stem can sever the connections between the brain and the body. In such cases, the person may retain consciousness and thought, but becomes paralysed except for eye movements. Patients can still receive sensory stimuli to the thalamus.

6.9.4 The Ventricular System

The two lateral ventricles are C-shaped cavities within the cerebral hemispheres. They connect via the Foramen of Monro with the midline, slit-like third ventricle. This in turn, connects via the cerebral aqueduct with the pyramidal fourth ventricle, between the brainstem and cerebellum. The exit from the fourth ventricle is via the medial foramen of Magendie and the lateral foramina of Luschka. Each of the

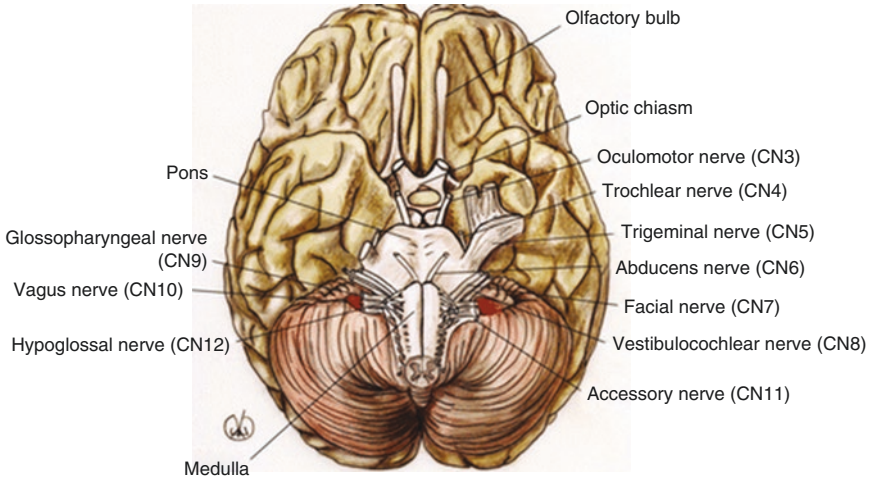

Fig. 6.6 Anatomy of the brainstem, with cranial nerves (cn)

ventricles contains the frond-like choroid plexus, which produces CSF, but only 22mls of CSF is within the ventricles, the rest being in the subarachnoid space. Blood in the CSF can block this absorption resulting in raised intracranial pressure (ICP) (Figs. 6.7 and 6.8).

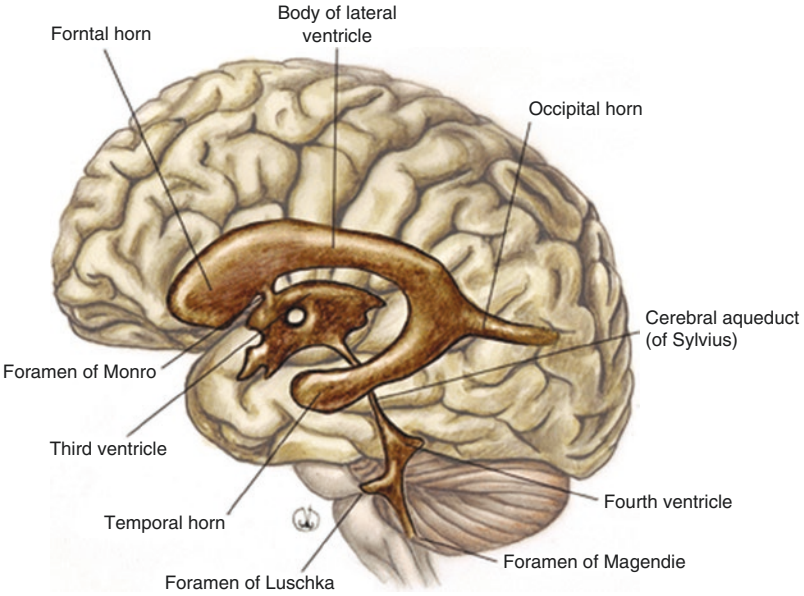

Fig. 6.7 Anatomy of the ventricular system

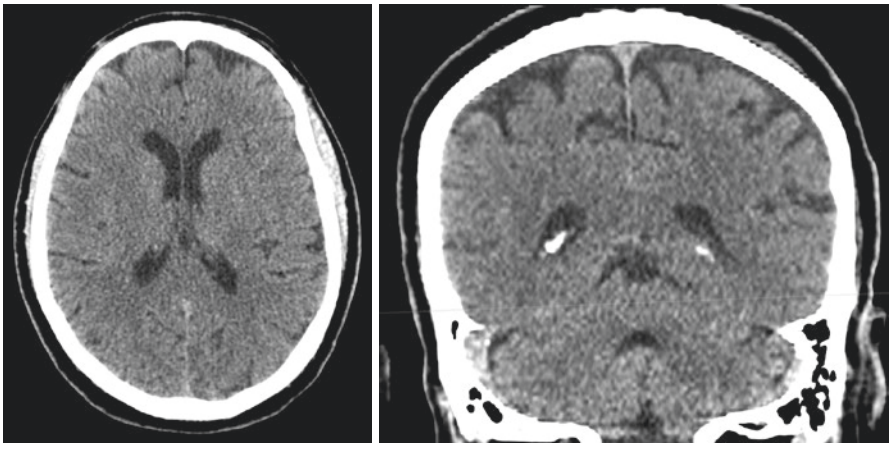

Fig. 6.8 Normal ventricles on CT

6.10 CSF and Cerebral Perfusion Pressure

CSF protects and supports the brain. It is produced by both active and passive filtration of plasma by the choroid plexus. Fluid is secreted at an approximate rate of 20 mL/h in adults (400–600 mL daily), irrespective of the intracranial pressure (ICP). However the rate of CSF production does show diurnal variation, with peak production in the late evening and early morning. The normal volume of CSF is about 150 mL in adults, with a turnover 3–4 times daily. Age, body mass and notably diseases can all affect the rate of CSF production. Other diseases and some injuries can affect resorption.

The majority of CSF production occurs in the lateral, third and fourth ventricles. After circulation through the subarachnoid space, the CSF is then resorbed into the venous system by the arachnoid granulations within the dural surface of the superior sagittal sinus (Fig. 6.9). Unlike CSF secretion, resorption is a passive process, driven by the hydrostatic pressure differential between the subarachnoid space and the venous circulation. Resorption requires the intracranial pressure (ICP) to be 1.5–7.0 cm H2O greater than venous pressure. Most diseases that cause hydrocephalus do so by interfering with this process (a rare exception is the choroid plexus papilloma—a tumour of the choroid plexus that produces excessive CSF). As a result of impaired resorption a higher pressure gradient is required to filtrate CSF back into the venous circulation. Therefore an abnormally high ICP develops.

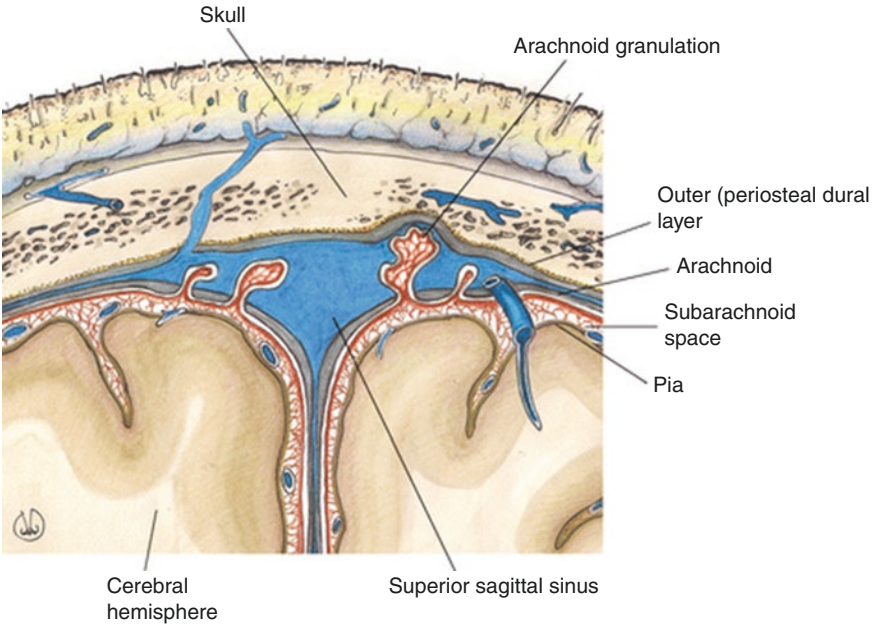

Fig. 6.9 Anatomy of the meningeal structures of the cerebral regions, coronal section

The cerebral perfusion pressure (CPP) is the pressure gradient which enables cerebral blood flow (i.e. cerebral perfusion). Without adequate perfusion, the brain will quickly die. The aim of management in all trauma patients is thus to ensure that the brain is adequately perfused with oxygenated blood and nutrients. In health, cerebral perfusion is normally maintained within a relatively narrow limit. The perfusion pressure is derived from the pressure gradient between the cerebral arterial pressure (often calculated using the mean arterial pressure—MAP) and the intracranial pressure. This is a complex relationship, which can be affected by both local changes in blood flow, as well as changes in the patients systemic BP. Raised intracranial pressure (ICP) can also adversely affect perfusion. In the healthy brain, wide variations in BP and intracranial pressure can be compensated for by auto regulation. However in the injured or diseased brain this compensatory mechanism can be severely disrupted, placing the brain at risk of inadequate perfusion. Normally the MAP is between 60 to 160 mmHg and the ICP about 5–15 mmHg, resulting in a CPP of about 50–150 mmHg.

6.11 Intracranial Pressure (ICP)

Intracranial pressure (ICP) is controlled by the volumes of CSF, blood and brain tissue within the cranium. These volumes can vary slightly, but is accommodated for by changes in the CSF and blood (blood volume can fluctuate by up to 40% and CSF resorption can increase to reduce the size of the ventricles by up to 90%). Brain tissue itself is also compressible, but only to a small amount. As a result of these adaptive mechanisms the intracranial volume can cope with around 100–150 mL of additional 'pathology' (haematoma, tumour, swelling), without there being any increase in the ICP. However, when these mechanisms are exhausted, just a small increase in additional volume will result in a rapid rise in ICP. Intracranial hypertension is the most common cause of death after severe TBI (Figs. 6.10, 6.11, 6.12 and 6.13).

The upper limit of normal ICP in adults and older children is usually about 15 mm Hg, (range of 5–15 mm Hg). Transient physiologic changes can occur as a result of coughing or sneezing. These can often produce pressures exceeding 30–50 mm Hg, but the ICP quickly returns to its normal pressure. The ICP waveform is normally pulsatile, secondary to cardiac and respiratory activity. These can also affect changes in the cerebral blood volume. Normal inspiration improves cerebral venous return, with a concomitant drop in cerebral blood volume. Mechanical ventilation and intrathoracic disease, which increase intrathoracic pressure may therefore adversely affect the ICP.

6.12 Brain Swelling

Swelling of all or part of the brain may occur secondary to (i) increased water within the brain tissue (cerebral oedema), (ii) increased intravascular blood volume (congestive brain swelling), or (iii) a combination of these. Cerebral oedema has many

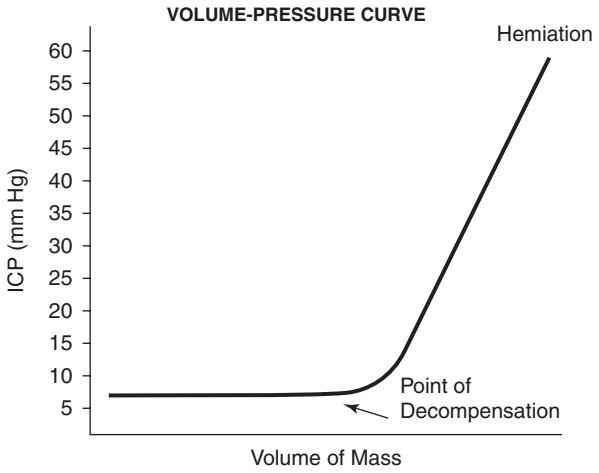

Fig. 6.10 Volume pressure curve

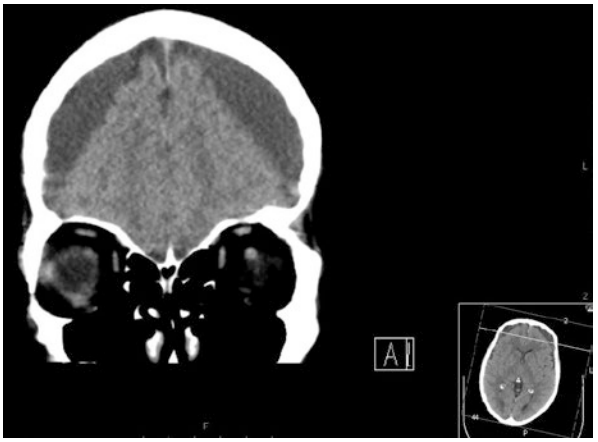

Fig. 6.11 Significant mass effect from bilateral chronic subdural haematoma

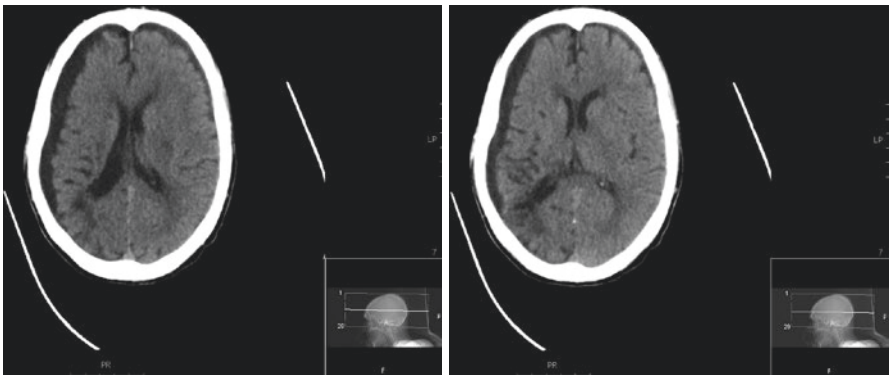

Fig. 6.12 Mass effect. Note distortion of ventricles

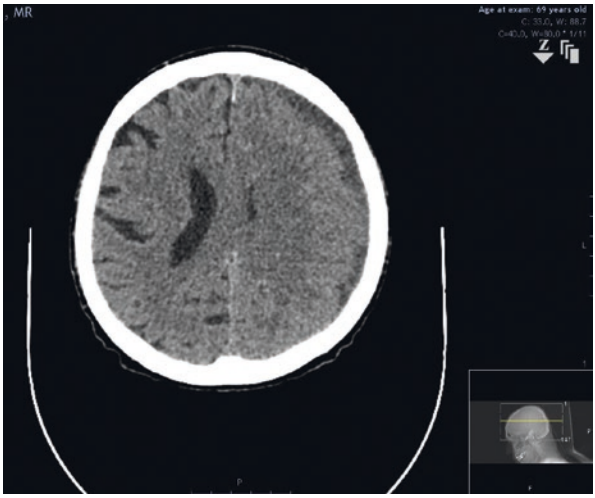

Fig. 6.13 Mass effect. Note distortion of ventricles

causes and may be classified into cytotoxic, vasogenic, osmotic, hydrocephalic-interstitial and hydrostatic.

6.12.1 Cytotoxic Oedema

Cytotoxic (intracellular) oedema occurs as a result of an influx of sodium ions and water following failure of the intracellular ion pump. This may occur secondary to ischaemia of the high energy-dependent pumps, or following direct injury to the mitochondria. All cells are affected, although astrocytes are reported to be affected more. Because the blood brain barrier (BBB) is usually intact, there is no enhancement on CT or MRI scans after intravenous contrast enhancement.

6.12.2 Vasogenic Oedema

Impairment of the BBB leads to vasogenic oedema and leakage of water from the intravascular into the extravascular compartments. This distinction can often be made with diffusion-weighted imaging (DWI). Cytotoxic and vasogenic oedema often occur together or sequentially.

6.12.3 Osmotic Oedema

This can occur following a significant reduction in serum osmolality (for example, following excessive use of intravenous dextrose solutions, or inappropriate

secretion of antidiuretic hormone syndrome). This results in increased intracellular water.

6.12.4 Hydrocephalic-Interstitial Brain Oedema

Periventricular extravasation of water can occur as a result of high-pressure hydrocephalus secondary to obstruction. This is an uncommon type of oedema that may be seen following head injury.

6.12.5 Hydrostatic Oedema

This occurs as a result of unfavourable hydrostatic pressure gradients between blood vessels and brain. Arterial hypertension combined with decompressive craniectomy can produce extensive brain oedema in the decompressed zone. The increased hydrostatic pressure gradient also opens the blood-brain barrier (BBB) resulting in additional vasogenic oedema.

6.13 Brain Swelling Following Head Injury

Some degree of brain swelling is usually present in most cases of traumatic brain injury and this can contribute towards a raised ICP. Localised swelling may occur around cerebral contusions or intracerebral haemorrhage. Diffuse swelling of one of the cerebral hemispheres can also occur following acute subdural haematoma. Diffuse swelling of the entire brain can also occur and not surprisingly, is usually related to a poor prognosis.

6.14 Trigeminocardiac Reflex (TCR)

This is an odd but common brain stem reflex, which can often be elicited during clinical examination or treatment. Stimulation of any of the branches of the trigeminal nerve sends an afferent signal via the Gasserian ganglion to the trigeminal sensory nucleus. This connects via the reticular formation to the motor nucleus of the vagal nerve, from which efferent pathways depress the myocardium. Sudden development of bradycardia, cardiac arrest, arterial hypotension, apnoea and gastric hypermobility can all occur. The oculocardiac reflex (OCR), a variant of the TCR is commonly seen during orbital or ocular surgery. TCR has also been reported during transsphenoidal surgery, microvascular decompression of the trigeminal nerve, percutaneous radiofrequency thermocoagulation and other endovascular procedures in neurosurgery.

Clinically, there is usually sudden onset of bradycardia. This can follow stimulation of the trigeminal nerve anywhere throughout its course. Subtypes of TCR have

been described, based on trigger points. Central (proximal) TCR is triggered following stimulation of the intracranial course of the trigeminal nerve (from the Gasserian ganglion to the brainstem). Peripheral (distal) TCR is elicited following stimulation of the trigeminal nerve anywhere outside the cranium beyond the Gasserian ganglion. Peripheral TCR is further subdivided based on the branch of the trigeminal nerve, into an ophthalmocardiac reflex (OCR) and maxillomandibulocardiac reflex (MCR).

Pressing on the eyes, or placing ice cubes in the mouth are two manoeuvres that have been previously taught to help slow down the heart in patients with supraventricular tachycardia. These exploit the TCR as a temporary measure to treat symptomatic SVT. The Diving Reflex (DR) Is also believed to be a variant of this reflex. This is characterised by the involuntary breath-holding, slowing of the HR and reduction of limb blood flow that occurs when suddenly immersed or soaked in cold water (commonly when taking a very cold shower, or the infamous "Ice Bucket Challenge").

6.15 Bulbar Palsy and Pseudobulbar Palsy

These are rare types of motor neurone disease that affect the lower cranial nerves. Bulbar palsy is a lower motor neurone palsy that affects cranial nerves IX, X, XI, and XII. Pseudobulbar palsy is an upper motor neuron palsy that affects the corticobulbar tracts of the Vth, VIIth, IXth, Xth, XIth, and XIIth cranial nerves. Any condition which disrupts or damages the nerve nuclei or corticobulbar tracts can result in bulbar or pseudobulbar palsy. Common causes include (i) stroke, (ii) multiple sclerosis, (iii) infections, (iv) motor neurone disease, (v) syringobulbia, (vi) Guillain-Barre syndrome, (vii) neurosyphilis and (viii) brain stem tumours. Both conditions are seen mostly in elderly men and present with progressive dysarthria and dysphagia. Patients with pseudobulbar palsy may also have a lack of facial expression and difficulty chewing. Lower motor neurone signs (atrophy and fasciculations of the tongue, absent gag reflex) differentiate bulbar palsy from pseudobulbar palsy, which presents with upper motor neurone signs (spastic tongue, exaggerated gag and jaw jerk reflexes). Diagnosis is mainly clinical although CSF analysis and MRI are often required. Treatment is mainly supportive (anticholinergics for drooling and baclofen for spasticity). Prognosis is generally poor.

6.16 Important Considerations When Taking a History

Because of the embryological, anatomical, physiological and functional overlaps between the head, face, eyes and cervical spine, its perhaps not too surprising that pathology arising in one site can present with symptoms in another, especially the head. Simple 'eye strain' and TMJ dysfunction for example can both present with headache. As such, the questions posed when assessing for possible intracranial disease may sometimes involve these adjacent sites. These aspects are discussed in the relevant chapters as well as here. From a purely 'brain' viewpoint, the following points should be considered.

6.17 Sudden Loss of Consciousness

This is often referred to as a "blackout" and the causes can range from simple vasovagal attacks ('faints'), to seizures. When taking a history it is important to ask about the onset, duration and frequency of the episodes. Often a witnesses account is useful. Exacerbating factors, preceding and associated symptoms such as an aura, nausea or vomiting are also important. A detailed past medical history is important to rule out non-neurological causes, such as low blood glucose in diabetes and cardiac causes.

6.17.1 Lethargy

This is a sense of tiredness, exhaustion, or lack of energy. Whilst it can be a symptom of intracranial pathology (such as raised intracranial pressure or encephalitis) the differential diagnosis is far reaching. Diseases and disorders affecting most, if not all of the body's systems can result in fatigue. Lethargy can also be the result of mental health conditions, notably depression. Other causes are of unknown origin, such as chronic fatigue syndrome. As such, it is important to take a careful and detailed history, noting any patterns to the lethargy, associated symptoms, work/social life patterns, stress-related factors, sleep patterns, medications and any other psychological/psychiatric symptoms. Clarifying the circumstances under which fatigue becomes a problem is very important in identifying the cause. Lethargy by itself is less likely to be arising from intracranial pathology, but if other neurological symptoms are present further investigations may be indicated.

6.17.2 Headache

Headaches are one of the more common neurological problems presenting to GPs and emergency departments. They are painful and debilitating often resulting in absence from work or school. Whilst most causes are benign, headache may be the first sign of a serious condition (commonly giant cell arteritis, raised intracranial pressure and infection). The most common primary headaches are tension-type headache, migraine and cluster headache. A careful history is thus vital in diagnosing the cause of headache. Key features include (i) worsening headache with fever, (ii) sudden-onset headache reaching maximum intensity within 5 min, (iii) neurological deficit or cognitive dysfunction, (iv) impaired level of consciousness, (v) triggers, such as coughing (valsalva), and ocular symptoms. These are discussed later in this chapter. There are many causes of headache, ranging from simple tension headache to a devastating aneurysmal subarachnoid haemorrhage (SAH). The focus of the history should be on the timing or onset of the headache. The location and character as well as duration and frequency are also important. Exacerbating and relieving factors will often give a clue, as will the presence of any associated symptoms. The past medical and social history should include dietary habits, such as excessive caffeine ingestion as well as prescribed and recreational drug use.

6.18 Head Injuries

This is discussed later in this chapter, under 'Traumatic brain injuries'. It is important to ask about the nature of the injury and any symptoms which have resolved or persist (notably loss of consciousness, visual symptoms, loss of smell and taste and limb deficits). Preceding alcohol or illicit drug use should be noted. Details about the incident may alert to the severity of the injury. For example, in an road traffic collision, were other passengers in the car killed, or was there a prolonged extraction time? In such cases there should be a low threshold to investigate thoroughly for serious impact injury. This is discussed further in the chapter on the injured patient.

6.19 Examining the Head and Associated Structures

This is tailored according to the suspected pathology (trauma, infections, tumours etc.). There are various elements which make up a comprehensive examination, and this can be time consuming and difficult to do well. When assessing the sensory system, we have to rely on the patient's subjective assessment of any stimulus applied. Patients must be encouraged to report any changes in sensation, or if anything feels different from normal. Usually there will not be a complete absence of sensation, but rather a dulling or diminution. Comparison to the opposite side is therefore helpful, although still imprecise. Comments such as, 'I can feel it but it's not as sharp', should be noted. Many of the muscles supplied by the cranial nerves are not as powerful as those in the limbs and quantitative assessment of power may be difficult. Neurological examination should not be considered in total isolation from the rest of the body. Poor respiratory or cardiac function, for example, can impair neurological function as a result of cerebral ischaemia. Remember to check the patient's blood pressure and glucose levels. Note the following.

6.19.1 Conscious Level: The Glasgow Coma Scale

The Glasgow coma scale (GCS) is probably the single most important component of any neurological examination, particularly following injuries. It often indicates the degree of urgency when assessing the patient. The scale (or score) was devised and published in 1974 as a reproducible and reliable measure of patient's level of consciousness. This was designed to replace ill-defined terms such as 'stuporous' and 'obtunded' which are vague descriptors of consciousness. Using the GCS provides a more objective description. It has three components—eye opening, best motor response and verbal response. A fully alert and orientated person has a GC Score of 15. A dead body has a GC Score of 3 (not zero). Whilst this is a significant improvement, the Glasgow coma score alone, in which only the numbers are stated (with no description) is limited in its usefulness. This is because

1. One or more components of the GCS may not be measurable—such as patients who are unable to open their eyes because of swelling, or patients who are intubated or have a tracheostomy.

2. Use of the score alone results in the loss of useful clinical information. For example, a patient with a GC Score of 9 can be either (1) Obeying commands, eyes open to pain and no speech or (2) Localising to pain, no eye opening and inappropriate words. Although the scores are the same, the second scenario is more concerning.

Practically, it is easier to simply state each individual response, rather than calculate the GC Score. This avoids mistakes when estimating the patient's conscious level. Confusion can also arise with variations in the use of the score (whether spastic flexion is included), giving a maximum of 14 or 15 points. Withdrawing to pain is often inappropriately used in place of flexion, which is incorrect.

6.19.2 Components of the Glasgow Coma Scale (Corresponding Score in Brackets)

Eye opening
EO spontaneously (4)
EO to speech (3)
EO to pain* (2)
EO none (1)
*do not test with supraorbital pressure as patient will instinctively close their eyes.
Verbal response
Orientated (5)
Confused (4)
Inappropriate words (3)
Incomprehensible sounds (2)
None (1)
Best motor response
Obeys commands (6)
Localises pain (5)
Flexion to pain (4)
Abnormal (spastic) flexion* (3)
Extension to pain (2)
None (1)
*1976 amendment included spastic flexion between flexion and extension.

6.19.3 Paediatric Variation of the Glasgow Coma Scale

Motor and eye opening same as adult
Verbal depends on age/ability:
5 point scale:
Babbles/coos/words as per usual (5)
Less than usual ability or spontaneous irritable cry (4)
Inappropriate crying (3)
Occasionally whimpers/moans (2)
No response (1)

6.19.4 Assessing Higher Mental Function

In addition to the Glasgow coma scale a mental status examination may be required. This usually includes the abbreviated mental test score (AMTS) or mini mental state examination (MMSE). Specific test for higher functions may also be applied. Fundoscopy is useful for assessing for raised intracranial pressure.

The Mini–Mental State Examination (MMSE) (Folstein test) is a 30-point questionnaire that is used to measure cognitive impairment and screen for dementia. It can be used to measure progression of cognitive impairment over time. The main elements of the test include

1. Orientation in time (from broadest to most narrow—year, month, day, time)
2. Orientation in place (from broadest to most narrow—country, region, address, ward).
3. Registration—repeating named prompts "what is this?"
4. Attention and calculation—"starting at 100 subtract 7 and keep going" (serial sevens), or spelling a simple word backwards.
5. Recall—"who am I?"
6. Language—Naming a pencil and a watch.
7. Repetition—Speaking back a phrase.
8. Complex multi step commands—this varies, but may involve drawing a figure.

The Abbreviated mental test score is a simpler 10 point version and includes

1. Age
2. Time to nearest hour
3. An address to be repeated at the end of the test
4. Where are you now? (name of hospital etc.)
5. Year
6. Recognition of 2 people e.g. doctor, nurse
7. Date of birth
8. Year second world war began
9. Name of present monarch
10. Count backwards from 20 to 1

Other higher function tests include

1. Cerebellar (see below)
2. Language ability: expressive, receptive
3. Nominal dysphasia—name the strap of a watch, or nib of a pen
4. Reading ability: dyslexia
5. Writing ability: dysgraphia
6. Calculation ability: dyscalculia Number skills
7. Gnosis "knowing things": agnosia—Geography, objects, people
8. Praxis "doing things": dyspraxia—dressing, Dress, draw, write
9. Memory test: immediate, short-term, long-term, verbal and visual memory (cannot be tested if confused or dysphasic)

10. Reasoning and problem solving ability
11. Mental state: degree of anxiety, mood, emotional behaviour, inhibition, speed of thought and response.

Cerebellar testing

- Dysmetria
- Finger-to-nose test
- Ankle-over-tibia test
- Dysdiadochokinesis
- Rapid pronation-supination
- Ataxia
- Assessment of gait
- Nystagmus
- Intention tremor
- Staccato speech

6.19.5 Cranial Nerve Examination

Examination of the cranial nerves should be undertaken routinely. With practice, a quick 'cranial nerve survey' can be undertaken in just a few minutes. Detailed examination may be required when assessing stroke patients, or where demyelinating conditions or tumours are suspected. The nerves are often examined roughly in numerical order but this is not essential. Some are more easily assessed when grouped together (notably those supplying the extra ocular muscles).

6.19.5.1 Olfactory Nerve

Formal testing of the olfactory nerve is often omitted, although a note should be made of any reported loss in the patient's sense of smell. Ensure that there is free flow of air by occluding each nostril in turn and asking the patient to sniff in. The olfactory nerves can be tested using standard bottles containing substances such as peppermint, coffee or lavender. If there is doubt about a claim of anosmia, try testing with ammonia solution. This will result in a reaction even in the presence of anosmia.

6.19.5.2 Optic Nerve

This is discussed in detail in the chapter on the eye. Visual acuity can be easily tested with a Snellen's chart, or chart for small print. Deterioration of at least two lines is significant. Colour vision can be tested with Ishihara plates or a bright red object. The visual fields are assessed in comparison to your own (assuming yours is normal). Use an ophthalmoscope to assess pupil size and reactivity. This assesses both the optic nerve and the oculomotor nerve (the efferent pathway). Then use the ophthalmoscope to examine each eye in turn. Check that the optic disc is clear—intracranial disease can affect the appearances of the disk, notably papilloedema or

optic atrophy. Cupping of the disc may be seen in patients with glaucoma—a possible cause of headache. Note also the vessels of the retina. When assessing the pupils it is worth noting that pupillary inequality can occur in 20% of the normal population (Anisocoria). Check previous records and ask relatives. Don't forget glass eyes.

6.19.5.3 Oculomotor, Abducent and Trochlear Nerves

These three nerves are usually examined together as they supply the extraocular muscles. This is also discussed in the chapter on the eye. The oculomotor nerve supplies most of the muscles to the eye and carries the parasympathetic fibres which constrict the pupil. This is balanced by the sympathetic fibres. Failure of sympathetic tone is a feature of Horner's syndrome. The patient should be asked to follow an object with their eyes, noting any irregularity in movement, obvious restriction, ptosis or lid lag. Ask the patient if they see one or two images. Is there is complete failure of the eye to move laterally, this is most likely due to paralysis of the lateral rectus muscle, supplied by VI nerve.

6.19.5.4 Trigeminal Nerve

The trigeminal nerve is largely a sensory nerve although it does have a motor component (in the mandibular division). Lightly touch each side of the face with a finger or piece of cotton wool and ask the patient if it feels normal and symmetrical. Those areas supplied by the ophthalmic, maxillary and mandibular branches should be tested. Remember that cutaneous branches from C2 and C3 (Great auricular nerve) also supply the face just in front of the ear. The motor components of the trigeminal nerve can be assessed by asking the patient to clench their teeth. Both masseters should feel firm. The contracting temporalis may also be felt. The corneal reflex can also be elicited but this is often omitted.

6.19.5.5 Facial Nerve

This is assessed by asking the patient to forcefully close their eyes, raise their eyebrows, show a 'toothy grin' and purse their lips tightly. Paralysis of the facial nerve causes the affected side of the face to appear to droop. This is more marked with a lower motor neurone (LMN) lesion. With an upper motor neurone (UMN) lesion the forehead is generally spared. This is because the muscles to the forehead have bilateral innervation by the upper motor neurones. This is an important distinction which helps differentiate between a UMN lesion (such as a pseudobulbar palsy) and a LMN lesion (commonly Bell's palsy).

6.19.5.6 Vestibulocochlear Nerve

Testing of the vestibular component of the nerve requires specialist expertise and involves such tests as Hallpike's manoeuvre. Formal testing of the cochlear component requires audiometry. A simple but crude test involves whispering, rubbing the patient's hair close to the ear, or use of a high-frequency tuning fork. If there is significant loss in one ear, Weber's test should be performed. This is discussed in greater detail in the chapter on the ear.

6.19.5.7 Glossopharyngeal and Vagus Nerves

Assessment of these nerves is difficult. Ask the patient if they have any problems swallowing. Then ask them to open their mouth wide and say 'Ahh'. This sound should be clear and the palate and uvula should lift symmetrically. The gag reflex can also be tested by touching the pharynx with a tongue depressor, however this is best omitted if there are risks from vomiting (particularly in a supine patient). Isolated lesions of the Glossopharyngeal nerve are very rare. Taste to the posterior third of the tongue is supplied from this nerve, but this can be difficult to assess.

6.19.5.8 Accessory Nerve

The accessory nerve supplies the trapezius and sternocleidomastoid muscles. This can be assessed by asking the patient to shrug their shoulders against resistance. They should also push their head forwards against your hand (although this should not be undertaken if there are any concerns regarding a neck injury).

6.19.5.9 Hypoglossal Nerve

Ask the patient to protrude their tongue and note any asymmetry, fasciculation or deviation. If the tongue deviates to one side, this suggests a hypoglossal nerve lesion. With a LMN lesion, the tongue will deviate towards the side of the lesion. With a UMN lesion, it will deviate away from the side of the lesion.

No	Nerve	Function	Test	Palsy
I	Olfactory	Smell	Various smell bottles e.g. coffee, lemon (test each nostril separately)	Loss of small (anosmia)
II	Optic	Vision	Visual acuity, visual fields, pupillary responses, fundoscopy	Blind eye, visual field defect or loss of acuity
III	Occulomotor	Eye movements	Eye movement in all directions, pupillary responses	Ptosis, eye deviated down and outwards, unreactive dilated pupil
IV	Trochlear	Eye movements	Eye movement down when looking medially	Inability to look down when looking medially
V	Trigeminal	Facial sensation, muscles of mastication	Sensation in 3 trigeminal divisions, corneal reflex, jaw movement	Loss of facial sensation, loss of corneal reflex, Jaw weak & deviates to side of lesion on opening, wasting of mastication muscles (chronic)
VI	Abducent	Eye movements	Eye movement laterally	Inability to look laterally

No	Nerve	Function	Test	Palsy
VII	Facial	Facial movements Taste to anterior tongue	Facial movements, Sweet, bitter, salt taste substances	Loss of facial movement UMN: Forehead spared LMN: Forehead affected Loss of taste
VIII	Vestibulocochlear	Hearing equilibrium	Hearing, Weber's & Rinne's tests balance & equilibrium	Deafness nystagmus, loss of equilibrium
IX	Glossopharyngeal	Pharyngeal & posterior tongue sensation & tasteMotor to upper pharynx	Pharyngeal sensation, gag reflex	Loss of gag reflex & pharyngeal sensation Deviation of the uvula
X	Vagus	Visceral parasympathetic supply (extensive) Larynx & pharynx motor function	Pharyngeal movement, gag reflex Laryngoscopy	Loss of gag reflex & pharyngeal movement Hoarse voice, vocal cord paralysis
XI	Accessory	Trapezius & sternocleidomastoid motor function	Trapezius & sternocleidomastoid power	Weakness of trapezius & sternocleidomastoid
XII	Hypoglossal	Tongue movements	Tongue movements	Tongue deviates to side of lesion

Clinically, the glossopharyngeal, vagus, spinal accessory and hypoglossal nerves and the cervical sympathetic plexus should be considered as a functional unit as well as individually. Although lesions affecting one of these nerves may dominate a patient's clinical features, often one or more of the other nerves will be involved on careful testing. Isolated glossopharyngeal nerve dysfunction is unusual and pathologic processes often involve the entire IX, X, XI complex. When this occurs careful localisation with imaging is required. This may need to include the entire head and neck region and sometimes the upper chest. For example, the vagus and cervical sympathetic nerves have a long course between the brain and the chest, whilst the glossopharyngeal and vagus also contain important pathways involved in referred pain.

Sometimes, lesions beyond the expected course of a nerve, or pathology involving an associated organ will be the cause of symptoms. For example, malignancy of the tongue base or throat can result in otalgia (referred via the glossopharyngeal nerve). Involvement of the vagal nerve can also result in ear pain. Thus it is important to understand the anatomical course of these important nerves and appreciate the possible symptoms and neurologic deficits that can occur from more 'remote' pathology. For example, glossopharyngeal nerve pathology can result in otalgia, dysphagia, odynophagia, throat pain, loss of the cough or gag reflex, loss of taste of the posterior one third of the tongue, dry mouth hypotension, tachycardia, or bradycardia. Important presentations include

1. Lesions within the skull such as the at the cerebellopontine angle. These may present as lower CN deficits.

2. Jugular Fossa Syndromes (Vernet syndrome (IX–XI), Collet-Sicard syndrome (IX–XII), Villaret syndrome (IX–XII and the sympathetic plexus)). Generally speaking if three of the five nerves are involved, the lesion will be at or near the skull base. If just two nerves are involved more comprehensive imaging is necessary.
3. Referred otalgia is an important symptom that may occur in upper aerodigestive tract disease. Other causes of otalgia need to be quickly excluded (ear, temporomandibular joint, parotid diseases) followed by careful assessment of the nasopharynx, oropharynx and hypopharynx undertaken. Otalgia (as a result of middle ear disease) can be caused by eustachian tube dysfunction as a result of nasopharyngeal carcinoma. This is discussed further in the chapters on the ear and throat.
4. Vocal cord paresis may be the only sign of a wide range of conditions including posterior fossa meningioma, thyroid cancer or mediastinal tumours or even an aortic dissection.

Glossopharyngeal neuralgia should therefore be a diagnosis of exclusion and requires careful assessment particularly is symptoms are worsening.

6.19.6 Peripheral Neurological Examination

Symptoms and signs in the upper and lower limbs have many causes, notably neurological and vascular. If symptoms and signs are confined to a single limb, consider local pathology as well as peripheral nerve disorders. However with symmetric findings or if all four limbs are involved spinal-cord and central lesions should be considered. Thus a peripheral neurological examination should always be part of the assessment of the central nervous system. This part of the examination can be quite time-consuming, but if done well and interpreted correctly it will prove to be very helpful in making the diagnosis.

Examination starts with general observations. Ask the patient to walk forward a few steps, turn around, and then walk back. As they do so assess their gait. Ask them to walk heel-to-toe, assessing for any unsteadiness. Note also any signs of foot-drop and the absence of any normal arm swing. Next, perform Romberg's test, assessing for impaired proprioception or vestibular dysfunction—ask the patient to stand with their feet together and eyes closed, this is positive if the patient becomes unsteady when the eyes are closed. Finally look at both the upper and lower limbs for signs of (i) muscle wasting/fasciculation, (ii) asymmetry, (iii) abnormal resting posture and abnormal movements. Clawing of the hands and foot (pes cavas) should be noted. If there is apparent wasting, feel for muscle bulk and determine if it is proximal or distal, or involves a specific muscle group.

The upper and lower limbs are then examined. Each is assessed for

1. Tone—ask the patient to relax and check the tone, assessing for resistance during flexion-extension and pronation-supination and rolling the legs. Compare both sides

'Clasp-knife' rigidity (High resistance that suddenly releases) or clonus may be seen in pyramidal lesions, whist 'cog-wheel' rigidity (jerky resistance) or 'lead pipe' rigidity (Increased resistance throughout movement) occurs in extrapyramidal lesions notably Parkinson's disease. If the limbs appear floppy with reduced tone, consider lower motor neurone lesions. Gegenhalten is a term used to describe resistance to passive change, where the strength of antagonist muscles increases with increasing examining force. This is common in dementia.

2. Power—Examine each of the main joints in turn, comparing both sides, assessing the power in each muscle group. Start proximally and moving distally. Conventionally the MRC scale of muscle power is usually used. This is noted in the chapter on the back of the neck.
3. Co-ordination—This assesses the patient's ability to perform smooth and precise movements. They include (i) Finger-nose test: Placing your finger a short distance from the patient and ask them to rapidly touch their own nose and then touch your finger, repeating back and forth. At the same time slowly change the location of your own finger. (ii) Dysdiadochokinesia test: Ask patient to rapidly alternate pronation-supination on one hand against the back of the other. This test can indicate cerebellar disease (iii) Heel-shin test: Ask the patient to place one heel on the other knee and slide the heel down and up the shin and (iv) Tapping test: Rapidly tap their foot against your hand. Like the hand test impairment in rapidly alternating movements may indicate cerebellar disease.
4. Reflexes—These include the bicep, brachioradialis, triceps, patella and calcaneal (ankle jerk). Test also the plantar reflexes—scratch the sole of the foot with a slightly sharp object (a key), starting at the heel, along the lateral side of the foot, curving towards the big toe. Look for a plantar response in the big toe. A positive Babinski sign occurs when the big toe extending and abducts. This indicates an UMN lesion. If there are difficulties in eliciting reflexes, ask patient to clench their teeth or interlock fingers and pull apart (Jendrassik manoeuvre).
   Reflexes in the newborn and infant are different. Infant reflexes (or primitive reflexes) are usually evaluated. Each of these reflexes disappears at a certain age as the infant grows. They include
   - Blinking. An infant will close their eyes in response to bright lights.
   - Babinski reflex. As the infant's foot is stroked, the toes will extend upward.
   - Crawling. If the infant is placed on his or her stomach, he or she will make crawling motions.
   - Moro's reflex (or startle reflex). A quick change in the infant's position will cause the infant to throw the arms outward, open the hands, and throw back the head.
   - Palmar and plantar grasp. The infant's fingers or toes will curl around a finger placed in the area.
5. Sensation—This is assessed by pin prick, light touch, proprioception, and vibration. Proprioception is performed by instructing the patient to close their eyes, moving the distal interphalangeal joint of the index finger and big toe, and asking them to indicate the direction of movement. If proprioception cannot be detected, move to

next proximal joint. At the same time Graphesthesia may be tested for. This is the ability to recognise writing or shapes drawn on the skin purely by the sensation of touch. Use a small blunt object, not a pen! This tests cortical higher functions.
Pronator Drift—is a test undertaken in the upper limb. Ask patient to close their eyes with their hands in a supine position. Look for any involuntary pronation of the hands—this occurs in UMN lesions. With suspected spinal cord injury a rectal examination should be undertaken, testing for anal tone and sensation.

6.19.7 Brainstem Reflexes

These are important in patients who are in a coma. If their brainstem reflexes are absent they are unlikely to survive

- Pupillary reflexes (afferent CNII, efferent CNIII). Shine a torch into each eye in turn and watch for pupillary constriction. If one eye is blind there will be no response in that pupil (direct reflex) or the opposite pupil (consensual reflex). However the affected eye's pupil will constrict to light shone in the opposite eye. If there is a CNIII palsy that pupil will not react to light in either eye, but the opposite pupil reacts to light in both eyes.
- Corneal reflex (afferent CNV, efferent CNVII). Stroke the cornea with cotton wool and watch for blinking.
- Grimace reflex (afferent V, efferent VII). Press on the supraorbital nerve at the orbital margin and watch for facial grimacing. Any limb or autonomic (pulse rate & blood pressure elevation) responses should also be recorded.
- Gag reflex (afferent CNIX, efferent CNX). Stimulate the posterior pharynx and watch for gagging.
- Oculocephalic & oculovestibular reflexes (afferent CNVIII, efferent CNIII, IV & VI). These are the same reflex pathways, stimulated by different methods. In the occulocephalic (doll's eyes) reflex the head is turned briskly to one side. If the reflex is preserved the eyes will turn to the opposite side as if maintaining gaze on the same point. This cannot be tested on conscious patients as voluntary control over gaze predominates. In the oculovestibular reflex ice cold water is irrigated into the external auditory canal, after ensuring it is not blocked by wax or that the ear drum is perforated. If the reflex is preserved nystagmus will develop due to stimulation of the semicircular canals by convection currents. This should not be tested on conscious patients as severe vertigo and vomiting will result.

6.19.8 External Examination

It is important to be gentle and systematic. Following trauma, open injuries tend to bleed profusely and can be quite distracting. Congealed blood can also hide important and useful clinical signs. Examination of the scalp should be done carefully, avoiding probing of wounds. Examination will usually be guided by the suspected problem (trauma,

sinusitis, meningitis, headache, subarachnoid haemorrhage). A comprehensive head examination will include examination of the eyes, nose, ears, oropharynx and the neck. These elements in examination are described in the relevant chapters in this book.

In infants and young toddlers, changes in the shape and size of the cranium may reflect changes in the intracranial contents. In young children, before closure of the sutures, increased intracranial pressure may result in widening of that suture lines, which may be palpable and visible on radiographs. If the intracranial pressure has been raised for a prolonged period of time, a mottled decalcification or 'copper-beaten' appearance of the skull and demineralisation of the dorsum sellae may also be seen on x-rays. Bulging of the anterior fontanelle is a useful sign in raised intracranial pressure in infants. Hydrocephalus can also result in enlargement of the skull and may be detected by measuring head circumference during check-ups. Standardised charts with normal values are available for this. Subdural fluid which may occur in meningitis and subdural or extradural haematomas, can also cause enlargement of the skull in infants. Conversely, a small head circumference may indicate failure of normal brain development. Premature closure of sutures causes characteristic distortions of the head and may restrict brain expansion. Some of these are described in the chapter on embryology. Untreated, some of these can result in problems related to chronically raised intracranial pressure.

In adults, changes in the shape and size of the skull are more unusual. However, meningioma can result in bone proliferation and produce bony asymmetry of the skull. This should be suspected if a bony prominence is noted on the opposite side to a neurological deficit, or in a newly presenting seizure. Some metabolic conditions can also result in characteristic skull abnormalities, for example prominent supraorbital ridges (frontal bossing) is often seen in patients with acromegaly.

Following trauma to the head, examination should be undertaken very carefully. Once the patient has undergone a primary survey (see the chapter on the injured patient) carry out a quick general inspection, noting any obvious asymmetry, lumps, signs of trauma and obvious neurological deficit. Ecchymoses at the base of the occiput (Battle sign) or around the eyes but contained by the orbital rim ("raccoon eyes") suggest basal skull fractures. If the patient is well enough, clean off any excess blood or secretions so that a more thorough inspection can be undertaken. In addition to the Glasgow coma scale it is also important to assess the eyes, noting particularly eye movements, pupil responses and the patients vision. These can be affected by diseases and injuries both within and out with the skull. Depending on the presenting problem, palpation of the skull may be required. This should be undertaking carefully, particularly if there is a history of trauma. Start with the scalp, looking carefully in any hair bearing skin. Injuries and scalp disorders can be easily hidden by thick hair. If there is an obvious exposed skull fracture do not manipulate it. Cover it with a sterile dressing and arrange urgent imaging. Percussion over the frontal sinus may elicit tenderness if there is sinusitis. Again, that should be avoided in the context of trauma. Numbness of the forehead my indicate fractures involving the orbit, or supraorbital ridges. The supraorbital ridges, nasal bridge and occipital region should also be carefully palpated. Assessment of the neck may also be required as part of the examination. This is necessary if the history suggests (i)

vascular insufficiency (vertebrobasilar) (ii) Meningitis (also look for photophobia and Kernig's sign), (iii) injury (image first)—see the chapter on the back of the neck, (iv) malignancy or infection.

In the non-trauma scenario, distortion or inflammation of various sensitive intracranial structures (blood vessels, meningies and cranial nerves) can result in pain. This may be detected by gently rotating the patients head and asking them to localise any pain. Movements of the brain within the cerebrospinal fluid stretches these sensitive structures, which would normally not result in discomfort. Migraine for example can result in sensitisation of the cranial blood vessels. Other more serious conditions resulting in inflammation or anatomical distortion will also bring on these symptoms. Malignancies as well as infections of the head and neck can affect these structures resulting in extraocular movement disorders and other cranial nerve palsies.

Auscultation over the skull can occasionally be useful with vascularised lumps (e.g. haemangioma, A-V malformation) or following trauma (surgical emphysema/carotid bruit). It should also be done in anyone presenting with unilateral headaches focal seizures, or subjective tinnitus (discussed in the chapter on the ear). A caroticocavernous fistula can also result in a bruit, best heard over the closed eyelids. Using the bell of a stethoscope, vascular bruits should be listened for over the mastoid region, temporal region, forehead, closed eyes, and in bald patients, over the scalp. Bruits may be caused by arteriovenous malformations on the surface of the brain, or lining the skull (Fig. 6.14). Loud bruits, detectable over the entire head, may occur in young children with congenital arteriovenous malformations in the area of the internal cerebral veins and the vein of Galen. They may also have an enlarging head, as a result of hydrocephalus. Diffuse bruits in infants may also result from meningitis or severe anaemia.

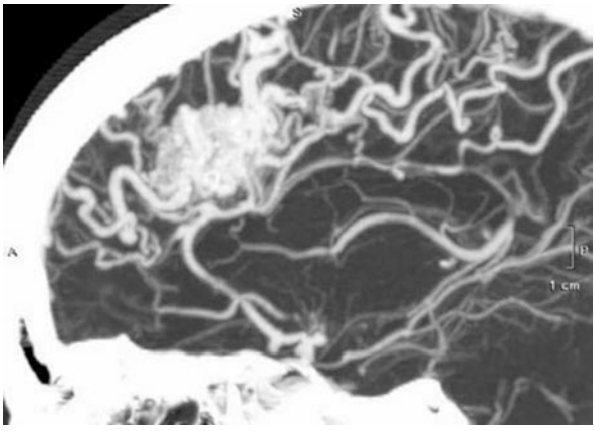

Fig. 6.14 Arteriovenous malformation (AVM). Sagittal maximum intensity projection (MIP) image demonstrates a 30 mm AVM in the frontal lobe. Note the complex superficial venous drainage pattern. Aneurysms are also present in the distal internal carotid artery (ICA) posteriorly and the anterior communicating artery

6.19.9 Examination in the Unconscious Patient

Neurological examination is limited in unconscious patients, but the following should be assessed

- Pattern of breathing (Cheyne-Stokes/Kussmaul)
- Glasgow coma scale
- Pupil responses/Papilloedema
- Spontaneous eye movements/deviation
- Signs of injury
- Neck stiffness (?meningism)
- Abnormal skin colour (cyanosis, jaundice, rubor in carbon monoxide poisoning)
- Needle-stick marks (drug overdose)
- Smell on their breath (alcohol, ketosis, uraemia, cyanide)
- Brainstem reflexes
- Limb tone
- Limb movements (spontaneous)
- Limb reflexes and plantar response

Note also the resuscitation status of the patient. The entire neurological assessment can be adversely affected if the patient is not fully resuscitated.

6.19.10 Some Useful Clinical Signs

6.19.10.1 Facial Nerve Palsy

Facial nerve paralysis is characterised by unilateral facial weakness. Following a head injury this can indicate a fractured base of skull. Depending on the cause, other symptoms include associated hearing loss, hyperacusis and loss of taste. There may also be reduced salivation and tear secretion, although this is rarely detected clinically. Other signs may be related to the cause of the palsy, such as vesicles in the ear (seen in shingles). Acute facial pain radiating from the ear may precede the onset of symptoms.

6.19.10.2 Intercanthal Distance

If this is increased the patient may have detached or displaced canthi secondary to an underlying nasoethmoidal fracture. Although distances are often assigned for the intercanthal distance, this is better assessed by looking at any recent photographs of the patient. If the intercanthal distance is increased remember to check for CSF leakage.

6.19.10.3 Anosmia

Injury to the head is thought to be one of the most common causes of loss of smell, the extent of which is determined by the site and severity of the injury. The

olfactory nerves run across the skull base anteriorly making them vulnerable to injury. Anosmia can also occur due to infiltration of the olfactory nerves (anterior cranial fossa tumour). Inflammation of the nasal passage (seen in chronic rhinosinusitis), especially when associated with nasal polyps can result in long-term deficit. Very occasionally people are born with no working sense of smell (congenital anosmia). Sometimes this may be part of a condition called Kallmann syndrome, which includes a hypothalamic defect with lack of hormone production and fertility problems. A range of other medical causes can also lead to problems with the sense of smell, notably liver failure, underactive thyroid gland and diabetes. These can be screened for with blood tests. Epilepsy, Parkinson's, Alzheimer's Diseases and Idiopathic anosmia make up the remainder of most causes.

6.19.10.4 Racoon (Panda) Eyes

Any well-defined 'blackeye' suggests a fracture of the associated orbit. Bleeding from the fracture tracks forwards, but is contained by the attachment of the orbital septum. This results in a relatively sharp margin to the haematoma. If both eyes are involved the patient is said to have panda or raccoon eyes. These often indicate a fractured base of skull (anterior cranial fossa), high LeFort or nasoethmoid fractures.

6.19.10.5 Third Nerve Palsy

Paralysis of the third cranial nerve affects the medial, superior, and inferior recti, and the inferior oblique muscles. As a result the eye is unable to move upwards, downwards or inwards. At rest it looks laterally and downwards owing to the unopposed lateral rectus and superior oblique muscles respectively. Reduced activity in the levator palpebrae superioris results in ptosis—drooping of the upper eyelid. Causes of oculomotor nerve palsy are very diverse. Palsy with pupillary sparing is often termed a medical third palsy and often has an ischaemic or diabetic aetiology. Full assessment of oculomotor nerve function involves testing of movement, reaction to light, and accommodation. In severe head injuries a third nerve palsy represents third nerve compression from an expanding intracranial lesion. In this scenario the patient will have a significantly reduced GCS as well.

6.19.10.6 Superior Orbital Fissure Syndrome (SOFS)

The superior orbital fissure (SOF) is bound laterally by the greater wing of the sphenoid, medially by the lesser wing of sphenoid, and superiorly by the frontal bone. It lies at the apex of the orbit at the junction between the roof and the lateral orbital wall. The fissure acts as a pathway between the orbit and middle cranial fossa. Traumatic superior orbital fissure syndrome is an uncommon complication of craniofacial trauma seen in less than 1%. There is ophthalmoplegia, ptosis, proptosis of eye (from oedema), a fixed dilated pupil and anaesthesia of the upper eyelid and forehead. This occurs if fractures extend into the

SOF, or it can occasionally indicate a carotid aneurysm—usually part of a significant injury. Non traumatic causes include tumours of the orbit (lymphoma or rhabdomyosarcoma) or adjacent structures, infection, inflammatory disorders and vasculitic and ischaemic diseases. Orbital inflammation may be isolated, or associated with systemic inflammation such as Wegener's granulomatosis, polyarteritis nodosa, sarcoidosis, or rarely, temporal arteritis. Fungal infection of the orbit is a rare cause of both SOFS and orbital apex syndrome and is usually secondary to sinus infection. The cavernous sinus extends from the superior orbital fissure to the dorsum sella. Tolosa-Hunt syndrome (THS) is caused by a granulomatous inflammatory process that involves the anterior cavernous sinus/superior orbital fissure region. This is diagnosed by excluding traumatic, infective, vascular, neoplastic, metabolic, and inflammatory causes.

6.19.10.7 Orbital Apex Syndrome

This presents with the same clinical findings as SOF syndrome, but in addition the patient also has reduced visual acuity. This represents a severe insult to the orbital apex. Following trauma this represents a high energy injury. It can also occur as a result of other non-traumatic causes.

6.19.10.8 Haemotympanum

Blood that can be visualised behind the ear drum may be indicative of a fracture of the middle cranial fossa. It can also occur following trauma and some disorders of the middle ear. In traumatic haemotympanum the tympanic membrane appears dark blue, purplish or sometimes almost black. This has traditionally been said to be pathognomonic of a temporal bone fracture. However, haemotympanum can also occur as a result of retrograde haemorrhage after severe epistaxis. In this scenario the haemotympanum appears bright red, presumably due to the presence of oxygenated blood (from the carotid arterial system, which supplies the nose) rather than deoxygenated blood (from venous bleeding associated with temporal bone fractures).

6.19.10.9 Battles Sign (Mastoid Ecchymosis)

This is a crescent-shaped bruise that appears behind one or both ears, around the mastoid region. There may also be a palpable bogginess of the area. This is usually seen several days following injury. It is a sign of a fractured base of skull. Bleeding from the ear and haemotympanum may also occur, although these may also be seen with fractures of the mandibular condyle.

6.19.10.10 CSF Rhinorrhoea/Otorrhoea

"Tramlining"—Fractured base of skull. This is discussed later in this chapter (Fig. 6.15).

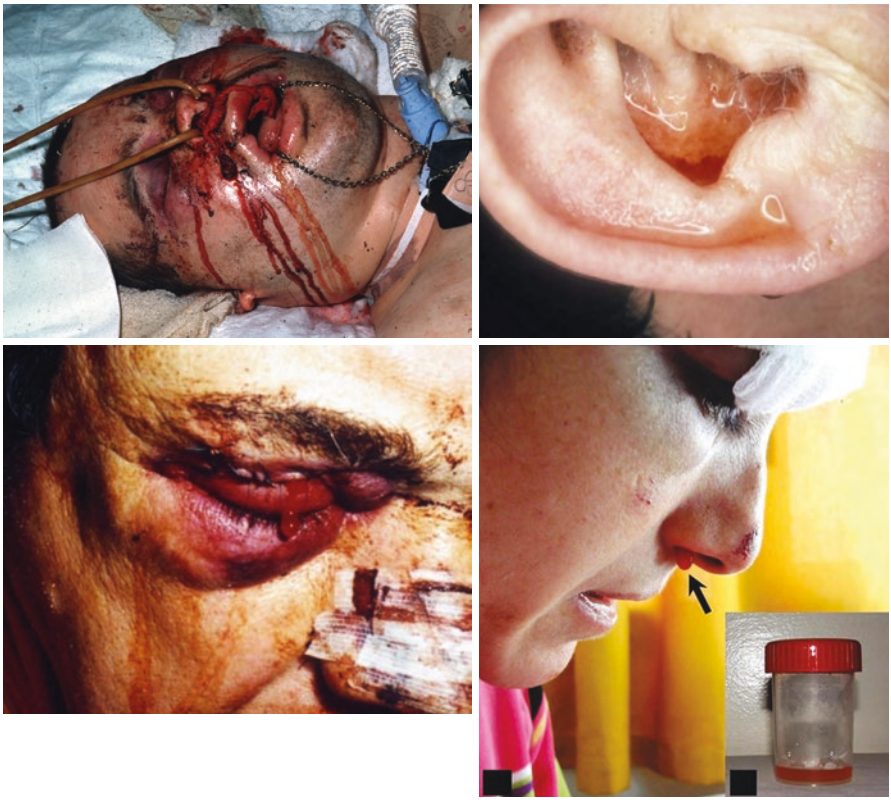

Fig. 6.15 CSF leakage can be hidden or obvious. The site of exit may give an indication of where the dural tear is sited. Patients should be advised not to blow their nose as intracranial air can collect with mass effect

6.20 Investigating Symptoms and Signs

6.20.1 Laboratory Tests

Full blood count, Urea and Electrolytes, coagulation screen, group and save, and alcohol levels are commonly required in patients with severe head related symptoms. Pituitary function can also become deranged and pituitary related hormones may need assays at a later stage. A WCC should be taken in all suspected infections and patients presenting with severe headaches (for underlying infections). CRP or ESR are usually very high in temporal arteritis and should prompt administration of steroids and urgent referral. Lumbar puncture (LP) may be required for CSF

analysis, although this would normally be undertaken on the ward. Tau protein and neuro filaments in CSF have been reported as potential biomarkers for axonal injury. These molecules are also found in some neurodegenerative diseases. Beta2 transferrin is a specific test for CSF in suspected leaks. Blood cultures should be taken in suspected meningitis/encephalitis.

6.20.1.1 Emerging Tests

1. Research is currently looking at the role of Protein S100B, glial fibrillary protein and neuron-specific enolase. These are currently being investigated as potential biomarkers in head injuries. There are at least 21 different types of S100 proteins. The protein is located in the cytoplasm and is a regulator of a number of cellular processes, such as cell cycle and differentiation. Due to its reported high sensitivity, S100-B has been incorporated into some clinical guidelines. Glial fibrillary acidic protein (GFAP) is a neuro filament protein, exclusively expressed by astrocytes. This has been suggested to be a more specific biomarker for brain injury than S100-B. G-enolase (neuron-specific enolase), a glycolytic enzyme, is another potential biomarker of traumatic brain injury (TBI)
2. MicroRNAs (miRNAs) have also been reported as possible biomarkers for TBI. These play an important role in the expression of messenger RNA. Apolipooprotein-E has been reported as another biomarker for various neurological and systemic diseases. This is an important molecule in lipid metabolism, and has been suggested to be an important risk factor for dementia and other neurodegenerative diseases.
3. Matrix metalloproteinases (MMP) are a family of endopeptidases involved in a number of neurological diseases, in which neuroinflammation plays a significant role. MMP-9 degrades components of the basal lamina, leading to disruption of the blood–brain barrier and vasogenic brain oedema. Elevated MMP-9 concentration may be found in plasma and CSF during the acute stages of TBI.
4. Fibronectins are glycoproteins that promote cell–cell and cell–matrix interactions. Two forms exist i) plasma fibronectin and ii) cellular fibronectin (c-Fn). High plasma levels of c-Fn in TBI patients might be indicative of the loss of blood brain barrier integrity. Blood levels of both MMP-9 and c-Fn have been reported to have good predictive values after thrombolytic therapy in acute ischemic stroke. However, the relationship between TBI outcome and blood levels of MMP-9 and c-Fn is unknown.
5. Circulating cell-free DNA (CFD) in the blood has been reported as a possible biomarker for a variety of conditions such as infection, inflammation, trauma, in critically ill patients. This has been extended to traumatic brain injury (TBI). Elevated CFD have been reported to correlate with mortality. An early fall in CFD (within 24 h) may predict better outcomes.
6. The neuropeptide substance P (SP) may have an important role in vascular permeability and brain water content following TBI. This may represent a pharmacological target for the treatment of oedema and increased ICP.

6.20.2 The Role of Imaging

6.20.2.1 Plain Films

Although CT scanning has now made plain films virtually obsolete, this may not be the case in all countries. 'Skull X-Rays' are therefore described here for completeness. Skull radiography is useful when looking for calvarial fractures, penetrating injuries and radiopaque foreign bodies. Plain views (or sometimes incidentally, when the skull base is visualised on a lateral cervical spine view), may occasionally show fractures, enlargement of the sella turcica, or a fluid level in the sinuses—all useful clues. In the context of trauma, the main difficulty with interpreting skull x-rays lies in distinguishing fractures from vascular marks and sutures.

- Vascular marks usually run upwards and posteriorly from the skull base and their margins are usually less well defined. This is because the depth of vessel indentation in the bone varies across its width.
- Sutures typically lay in well-defined locations, although sometimes additional sutural bones might be present. Suture margins are highly tortuous.
- Sometimes following a head injury, a suture might become separated (diastasis of a suture). This should be managed as a fracture.

With regards to other pathologies, a 'Copper-beaten' appearance is seen in patients with chronic raised intracranial pressure, where the internal table of the bone becomes deformed. 'Pepper-pot' appearances suggest bony deposits, commonly seen in multiple myeloma. Paget's disease also affects the skull. Any 'moth eaten' appearance suggests bony pathology and therefore indicates further imaging. Fibrous dysplasia can also appear quite dramatic, but in many cases can be diagnosed on imaging alone without the need for a biopsy.

6.20.2.2 CT/MRI Scanning

Computed tomography (CT) is now the imaging modality of choice in most trauma centres, as it provides rapid and accurate diagnosis of many injuries, including those to the head (skull fractures, intracranial haematoma, haemorrhage, brain contusions and signs of raised intracranial pressure) (Figs. 6.16, 6.17, 6.18, 6.19 and 6.20).

Suspected space occupying lesions, infections, strokes and many other intracranial diseases are also readily identified. Calcification is common in meningiomas which may be diffuse, speckled or peripheral. Adjacent bony sclerosis and thickening is common but when seen, should prompt a careful search for an intraosseous component. Purely intraosseous meningiomas are less common and can mimic metastatic disease, primary bone malignancies, osteomyelitis, leukaemia and other fibro-osseous lesions. CT may also be required to determine the safety of a lumbar puncture (LP), which is contraindicated if there is raised ICP. Fresh blood can be readily seen on CT and this can often rapidly diagnose subarachnoid haemorrhage in patients with acute headache. CT angiography may be considered in carotid

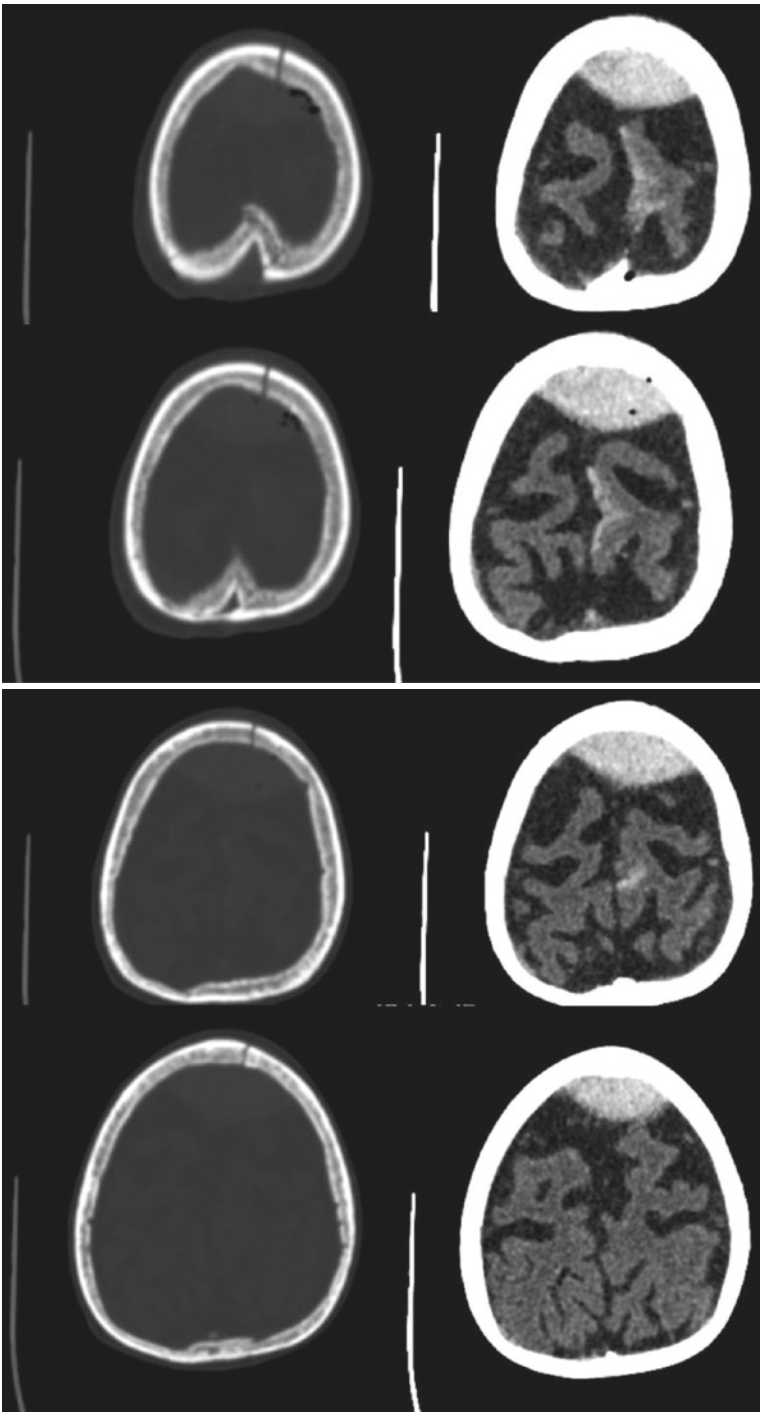

Fig. 6.16 CT must be viewed on both bone and 'soft tissue' settings, depending on the suspected pathology. Bone settings alone may miss obvious problems

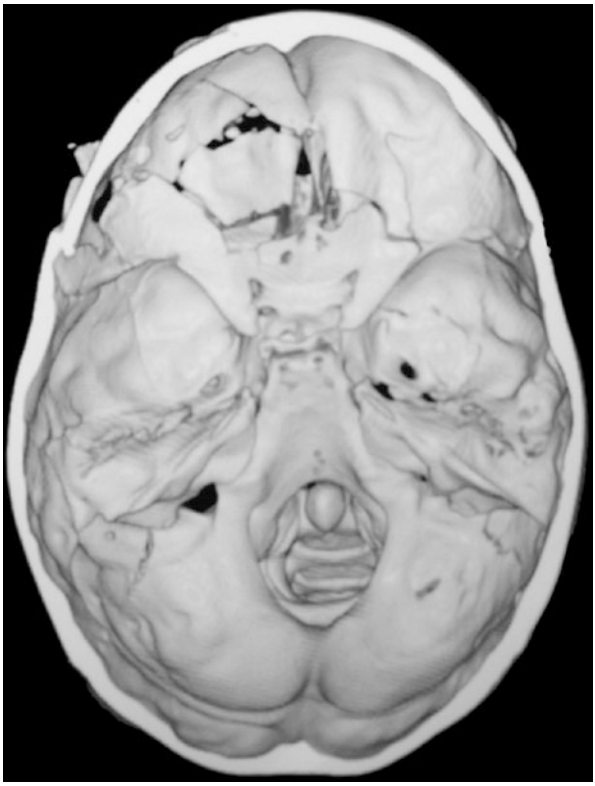

Fig. 6.17 3D reconstructions are invaluable in appreciating overall fracture patterns

dissection or penetrating injuries and CT venography maybe indicated in suspected cerebral venous thrombosis. Tumours and infective foci can also be defined and staged using contrast imaging.

MRI of the brain can also detect many of these conditions, but is not as readily available in the emergency situation. It provides excellent visualisation of haematomas and tumours (Figs. 6.21 and 6.22). Magnetic resonance angiography is also helpful in evaluating arterial injuries, including dissection and occlusion. The ability to rapidly and accurately screen the cerebrovascular system non-invasively has led to improved stroke care and treatment. Both the arterial and venous systems can be rapidly imaged with and without contrast. However it is contraindicated if there is a suspected foreign body. MRI of the anaesthetised patient can be very problematic to carry out and takes much longer than CT. MRI may be performed later in some cases of traumatic brain injury for prognostic reasons (patients with diffuse axonal injury).

Imaging also plays a critical role in the management of CSF leaks by precisely identifying the site of the leak. In addition to obvious sites of fracture, the two most common sites for CSF rhinorrhoea are the olfactory fossa (along the vertical

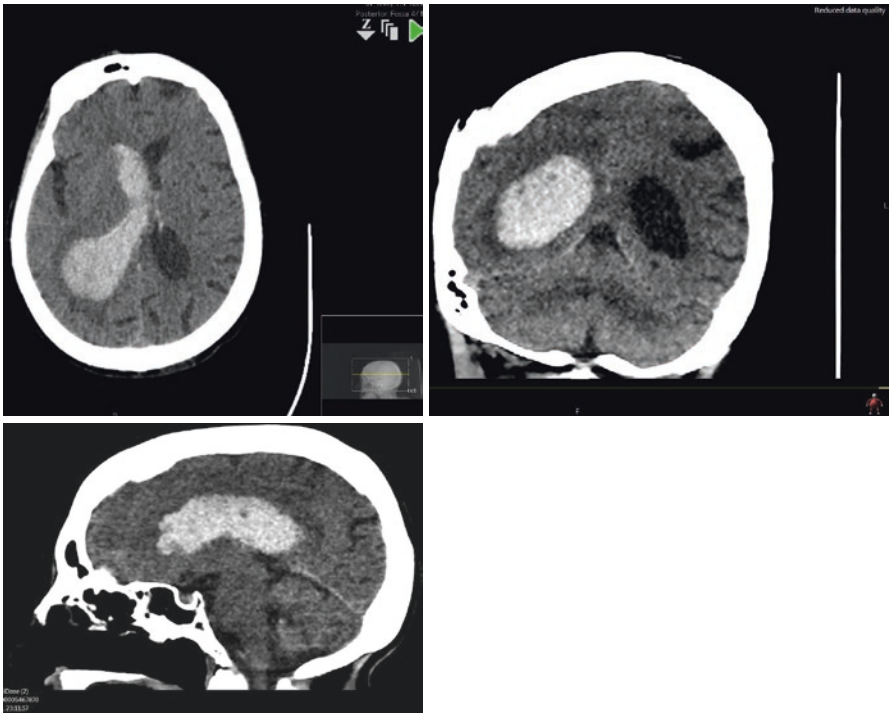

Fig. 6.18 Intraventricular haemorrhage

attachment of the middle turbinate) and the superior and lateral walls of the sphenoid sinus. Defects can usually be detected by multiplanar CT with or without MRI. Intrathecal contrast CT cisternography may occasionally be required in complex cases, such as those with multiple potential sites of leakage or when the surgical approach may be complicated. MRI cisternography may be useful but lacks bony detail.

Today, neuroimaging plays a key role in both diagnosis and in the understanding of neurodegenerative diseases and traumatic encephalopathies. Neuroimaging also plays an important role in the understanding of brain function, neuroplasticity and recovery. Functional MRI (fMRI) may be used to detect abnormal white matter blood oxygen level–dependent (BOLD) signals in patients with chronic TBI. This can assess cerebral function and functional deficits or disturbances. It may therefore be of value in the assessment of epilepsy, particularly if surgery is planned. Diffusion-weighted MR techniques are also used to assess early signs of cerebral ischaemia, tumours and infection. Diffusion tensor imaging (DTI) is an advanced MRI technique that can identify structural changes in axonal integrity. Although not used acutely, this is of value in identifying cortical dysplasias and in surgical planning in epilepsy.

Quantitative magnetic resonance angiography (QMRA) is a non-invasive technique which can be used to quantify blood flow in the vasculature. It can visualise

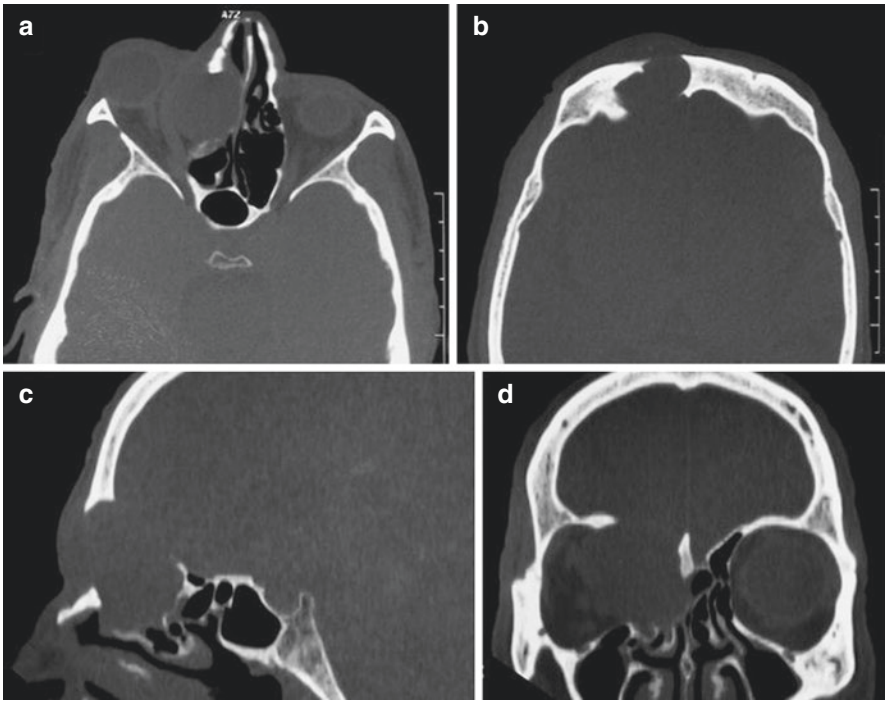

Fig. 6.19 Case 13.2. Axial (**a, b**), sagittal (**c**), and coronal (**d**) craniofacial CT scans on bone windows. This cystic lesion causes thinning and loss of the surrounding bony structures, with intraorbital and intracranial extension

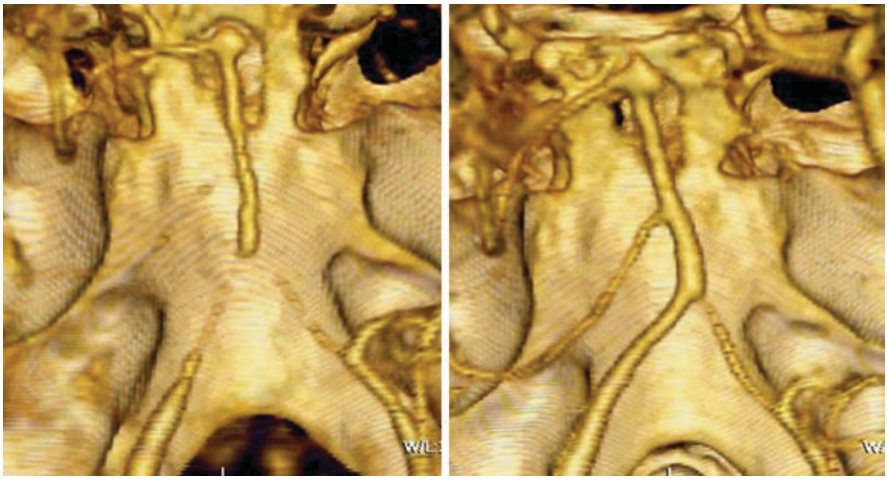

Fig. 6.20 (**a, b**) Vasculopathy. (**a**) Patient with sarcoid causing intracranial vasculopathy. Note narrowing of the basilar artery, distal vertebral arteries, and anterior inferior cerebellar artery. (**b**) Same patient after therapy demonstrates marked improvement in vessel calibre

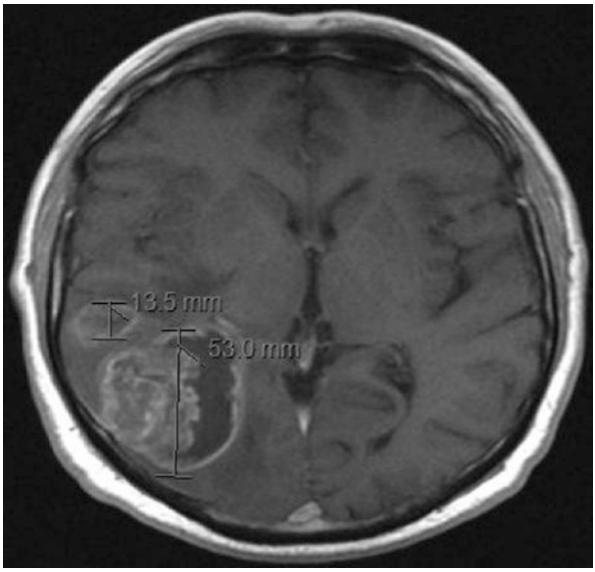

Fig. 6.21 MRI with gadolinium of right occipital glioblastoma

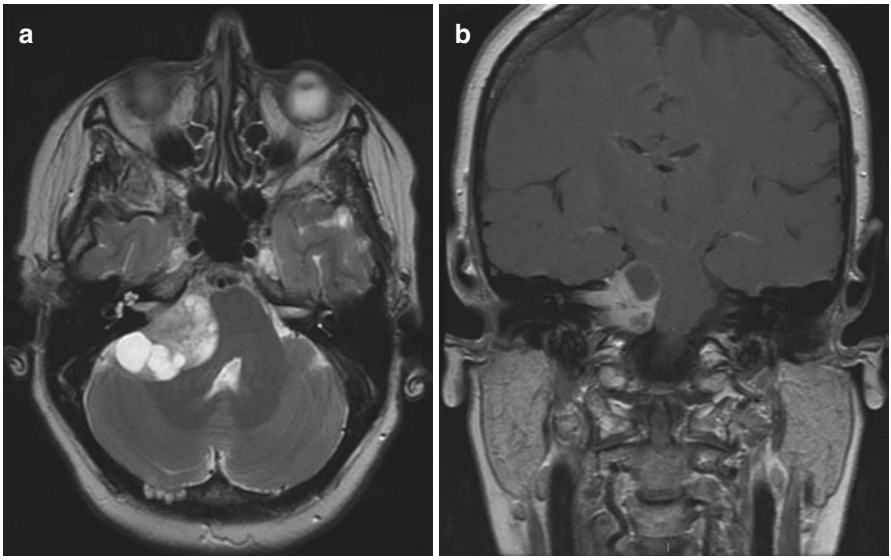

Fig. 6.22 T2 axial (**a**) MRI and T1 with gadolinium coronal section (**b**) showing right acoustic neuroma

extracranial and intracranial vascular anatomy as well as measure blood flow in the cerebrovascular system using computer software, known as non-invasive optimal vessel analysis (NOVA). NOVA has been used in vertebrobasilar ischaemia, to document vascular bypass patency, to evaluate intracranial stent stenosis after placement, to determine whether patients will tolerate carotid occlusion and to quantify carotid blood flow changes after carotid endarterectomy and carotid angioplasty and stenting for atherosclerotic disease.

6.20.3 Cerebral Angiography

Although cerebral angiography has been the gold standard for assessing the cerebral vasculature, in many hospitals CT angiography has largely replaced this in the initial evaluation of many injuries and diseases. Intracranial vascular occlusion and dissection can occur in up to 10% of blunt trauma victims and should always be considered in patients with focal neurological deficits which cannot be explained by the injuries on initial CT. Traumatic intracranial aneurysms may become symptomatic days to weeks after the trauma. Hence, angiography is not routinely indicated during initial assessment except in patients with brisk epistaxis or a mechanism of injury that implies a vascular injury. However conventional angiography can allow the use of endovascular techniques in managing such vascular injuries so still has a role in some cases. Recent developments in interventional neuroradiology have now made possible the ability to re-vascularise some regions of the central nervous system (CNS). These procedures may help salvage CNS tissue and function as a result of ischaemia. Over the last few decades intra-arterial thrombolysis in acute stroke and pharmacological dilation or balloon angioplasty of intracranial vasospasm has become widespread.

Digital subtraction angiography (DSA) is also the current gold standard for the diagnosis of intracranial atherosclerosis. However, this is invasive and carries the risk of stroke. Along with transcranial Doppler (TCD) and computed tomographic angiography (CTA), MR angiography is becoming more popular. This is less invasive but still able to visualise intracranial vessels. Recent advances in MRI techniques now enables visualisation of small perforating vessels originated from large intracranial arteries. MRA for imaging intracranial atherosclerotic plaques and the cerebral collateral circulation in the circle of Willis is a potentially promising tool (Figs. 6.23 and 6.24).

6.20.4 PET/SPECT

Positron emission tomography scanning (PET) and single photon emission computed tomography (SPECT) are functional neuroimaging techniques that provide complementary information to the other anatomical techniques. A radioisotope is

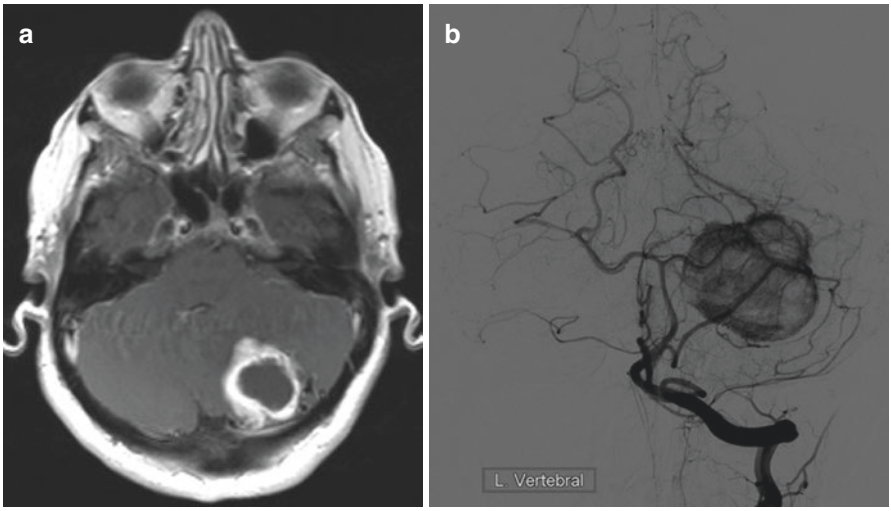

Fig. 6.23 t1 axial MRI with gadolinium (**a**) and catheter angiogram (**b**) showing cerebellar haemangioblastoma

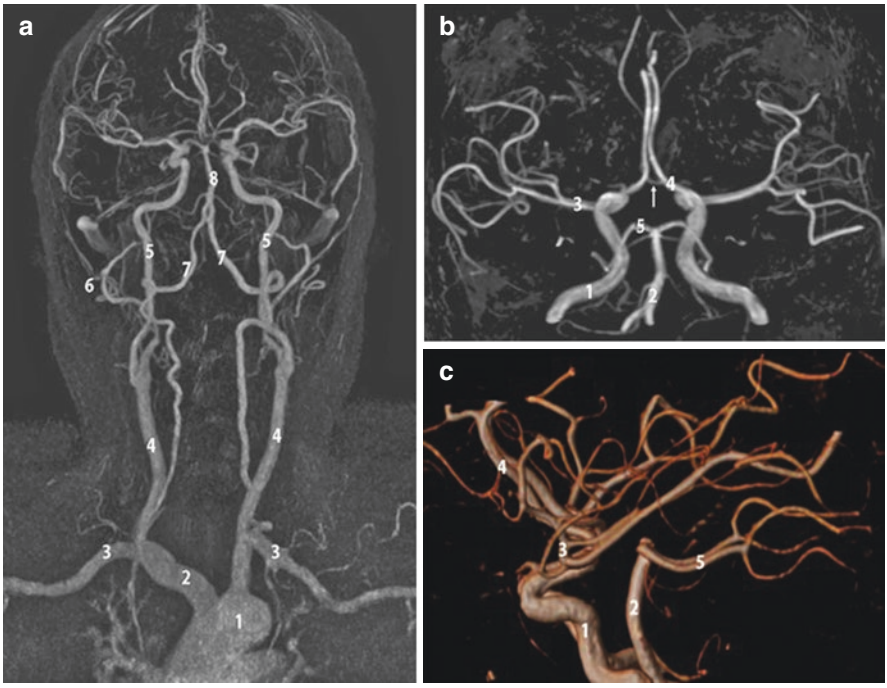

Fig. 6.24 (**a**) MRA of epiaortic arteries: aortic arch (1), brachiocephalic trunk (2), subclavian (3), common carotid (4), internal carotid (5), external carotid (6), vertebral (7), basilar (8), (**b–c**) 3D TOF-MRA reconstruction, oblique MIP (**b**) and lateral VRT (**c**): internal carotid (1), basilar artery (2), middle cerebral (3), anterior cerebral (4) and posterior cerebral (5) arteries

administered and transported into neurons, depending on their consumption of glucose or oxygen. PET therefore produces an image of cerebral energy metabolism. Using these techniques energy metabolism, glucose utilisation, regional cerebral blood flow and neurotransmitter synthesis can all be estimated. Single photon emission computed tomography uses radiotracers labeled with single-photon-emitting isotopes to produce images of cerebral function that show regional cerebral blood flow, cerebral blood volume, and blood-brain barrier permeability.

6.20.5 Ultrasound

Recent advances have made ultrasound an increasingly accurate and useful means of detecting intracranial pathology in infants, with similar sensitivities to MRI. Ultrasound offers clear advantages over other imaging modalities such as MRI and CT. It is less expensive, avoids radiation, usually does not require sedation and is portable. Doppler and linear sonography also play a key role in the evaluation of the paediatric brain.

6.21 Intracranial Pressure Monitoring: External Ventricular Drain

ICP monitoring has become an integral part of neurocritical care. It is used in the management of traumatic brain injury and hydrocephalus. Basic requirements of ICP monitoring include: minimal trauma during placement, negligible risk for infection, no CSF leakage, easy to handle, reliable and able to function during other diagnostic and therapeutic procedures. An external ventricular drain (EVD), or ventriculostomy drain, connected to an external gauge is currently the "gold standard" for measuring ICP. This can be placed at the bedside. The drain is inserted into the lateral ventricle with its tip in the foramen of Monro. The catheter is then tunnelled subcutaneously to minimise CSF leakage and risks of infection. Ventricular pressure (which represents ICP) is thus transmitted to an external transducer. Most transducers can be connected to ICU monitoring systems to allow real-time ICP measurements to be displayed.

EVDs can also serve as a therapeutic device to remove CSF and lower the ICP. This may be necessary following subarachnoid or intraventricular haemorrhage, in which the ICP becomes raised secondary to hydrocephalus. Complications include malposition, occlusion, haemorrhage, and infection, which necessitate removal of the drain. Accurate placement of an EVD can be difficult and in some patients due to the small size of the ventricles, or following ventricular shift (from a lesion or oedema).