Chapter 4

The head

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The head—basic anatomy and physiology

The scalp
Anatomists describe the SCALP as having five layers: Skin, Subcutaneous tissue, Aponeurosis, Loose areolar tissue and Pericranium. Functionally, it can be considered as two layers:
- a superficial layer from the skin to the galea apponeurotica; and
- a deep layer consisting of the areolar tissue and pericranium.

It is between these layers that the scalp moves. Most scalp lacerations extend the full thickness of the upper layer. The vessels and nerves lie in the subcutaneous tissue (anteriorly—supra-orbital and supratrochlear; laterally—superficial temporal artery and auriculotemporal nerve; posteriorly—posterior auricular artery and occipital artery). The scalp has a rich blood supply and can bleed profusely. In children blood loss can result in shock.

The skull
The skull consists of the calvarium, which contains the brain, and the facial skeleton. The calvarium consists principally of eight bones:
- frontal
- sphenoid (2)
- temporal (2)
- parietal (2)
- occipital bones.

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

The skull is thickest over the vertex. It is thinnest in the temporal region and where it forms the roof of the orbits and nose. (See for yourself. Get a real skull and hold it up to daylight. This graphically demonstrates how fragile some areas are.) Skull thickness and sinus volume can vary from person to person.

Internally the skull is divided into the anterior, middle, and posterior cranial fossae.

The anterior cranial fossa contains the anterior part of the frontal lobe of the brain. It extends back to the lesser wing of the sphenoid bone, and lies above the orbits and nose. The anterior fossa is perforated by the olfactory nerves only (cranial nerve I).

The middle cranial fossa is the largest. It is continuous with the anterior fossa above the lesser wing of the sphenoid. It is separated from the posterior fossa by the tentorium cerebelli. The middle fossa is filled by the temporal lobes of the brain and above them contains the remainder of the frontal lobes, the parietal and occipital lobes. The carotid arteries enter the skull and the cranial nerves II to VI leave the skull via the middle fossa floor.

The posterior cranial fossa lies below the tentorium cerebelli. It contains the midbrain, pons, medulla, and cerebellum. The major venous
The outflow of the brain is through the posterior fossa, where the sigmoid sinus continues as the internal jugular vein. Cranial nerves VII to XII also exit through the posterior fossa. The medulla continues with the spinal cord through the foramen magnum, which is also where the vertebral arteries and spinal root of the accessory nerve enter the skull.

**The meninges**

The inner surface of the skull is lined by the **dura mater**, which is a tough fibrous membrane (‘pachy’ or thick meninges). It becomes more firmly attached to the skull with age. The dura is reflected internally to form:

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, external to the dura, is a potential space only: normally it does not exist.

Large **venous sinuses**, which provide 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, respectively. The inferior sagittal sinus continues as the straight sinus, which lies where the falx joins the tentorium. The straight and superior sagittal sinuses join at the confluence of the sinuses, or torcula, which lies internal to the external occipital protuberence. The sinuses continue laterally as the transverse sinuses, which lie in the lateral margin of the tentorium. The anatomical arrangement of the confluence results in most of the blood from the superior sagittal sinus passing to the right transverse sinus and from the straight sinus to the left. The transverse sinuses continue as the sigmoid sinuses, which curve through the posterior fossa to the jugular foramen.

The cavernous sinuses lie alongside the sella turcica (pituitary fossa) and communicate with the sigmoid sinuses via the superior and inferior petrosal sinuses, which lie along the upper and lower borders of the petrous temporal bone. **The cavernous sinuses receive some venous drainage from the face and are thus a route whereby extra-cranial infection can gain access intra-cranially.** The importance of the venous sinuses is that they can bleed profusely if damaged. Particular care need to be taken if they have been potentially damaged, e.g. by depressed fractures.

**The arachnoid mater** lies deep to the dura. It is a flimsy membrane and consists of the parietal layer of the ‘lepto’ (or thin) meninges. The subdural space lies between the dura and arachnoid and is usually empty, although the two membranes are not adherent. 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 is very wide and forms the ‘basal cisterns’.

**The pia mater** is the visceral layer of the leptomeninges. It is firmly attached to the brain.
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 part of the 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 divides into the two posterior cerebral arteries. The latter supplies 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 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 is highly convoluted into gyri (the folds) and sulci (the clefts). This increases the area of the cortex, which is where the higher functions are organized.

Cortical functions are crossed, with one hemisphere dealing with the function of the other side of the body. The left hemisphere is dominant for speech in 99% of right-handed individuals (as 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.

Localization of cortical functions:
- **Primary motor area.** Pre-central gyrus of frontal lobe (body image inverted with leg area on the medial hemisphere surface). The basal ganglia are also highly important.
- **Primary sensory area.** Post-central gyrus of parietal lobe (body image inverted with 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 lesion causes minor deficit only).
- **Emotions.** Inferior frontal lobe, tip of temporal lobe and cingulated gyrus (on medial surface above corpus callosum). Other deep parts of the limbic system are also involved (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**: (dominant) numeration, calculation; (non-dominant) body image and awareness of external environment.

* Dominant hemisphere only.

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

The hypothalamus is concerned with autonomic function and endocrine control through the pituitary gland.

**Cerebellum**

The cerebellum is concerned with **balance and coordination.** It consists of two hemispheres and the midline vermis. It 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.

**Brainstem**

The midbrain, pons, and medulla contain nuclei for the third to twelfth cranial nerves, together with descending and ascending fibre tracts. The midbrain contains a gaze-control centre. The brainstem **reticular formation** contains centres for the vital functions (wakefulness, pulse and blood pressure control, breathing).

**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 median foramen of Magendie and the lateral foramina of Luschka.

Each of the ventricles contains the frond-like choroid plexus, which produces **CSF.** The total volume of CSF in a normal adult is 150 ml, but only 22 ml are in the ventricles, the rest being in the sub-arachnoid space. CSF production is **450 ml per day** so CSF is replaced three times per day. CSF is absorbed through the arachnoid villi over the cortical surface by a passive, pressure-dependent process. Blood in the CSF can block this process resulting in **raised intra-cranial pressure (ICP).**
The head—neurological examination

Neurological examination cannot be considered in isolation from the rest of the body. For instance, poor respiratory or cardiac function can impair neurological function by causing cerebral ischaemia (hence you cannot accurately assess ‘D’ until ‘A, B & C’ have been optimized.) Remember focal neurological dysfunction might be related to lesions elsewhere; e.g. metastases, brain abscesses.

Neurological examination should be carried out as appropriate. The following should be considered:

- **Glasgow coma scale**: orientation, eye opening and verbal response;
- **cranial nerve examination**;
- **limb function**:
  - appearance (deformity, wasting, abnormal movement);
  - muscle tone;
  - power in each muscle group;
  - limb reflexes;
  - sensation (touch, pain, vibration, temperature) in each dermatome;
- **co-ordination**:
  - Rhomberg’s test for equilibrium;
  - gait;
- **Higher cerebral functions**:
  - language ability: expressive, receptive and nominal dysphasia;
  - reading ability: dyslexia;
  - writing ability: dysgraphia;
  - calculation ability: dyscalculia;
  - object recognition: agnosia;
  - ability to perform specific tasks: dressing, geographical (follow route) and constructional (copy drawing) apraxia;
  - memory test: immediate, short-term, long-term, verbal, and visual memory (cannot be tested if confused or dysphasic);
- **reasoning and problem solving ability**;
- **mental state**: degree of anxiety, mood, emotional behaviour, inhibition, speed of thought and response.

Examination of the unconscious patient

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

- resuscitation status;
- Glasgow coma scale;
- pupil responses;
- eye movements and fundoscopy;
- signs of injury;
- abnormal skin colour (cyanosis, jaundice, rubor in carbon monoxide poisoning);
- needle-stick marks (drug overdose);
- smell of breath (alcohol, ketosis, uraemia, cyanide);
- brainstem reflexes;
- limb tone;
- limb movements (spontaneous, localizing, flexion, extension, or absent);
- limb reflexes and plantar response.
Glasgow coma scale (GCS)

The importance of the GCS, like many investigations, is that it is a 'snapshot' of the patient's condition at the time it was taken. To be of use it must be repeated again and again at suitable intervals. Only this way will it be possible to quickly pick up any improvements or deterioration in the patient's condition.

The GCS was devised as a means of consistently describing the depth of unconsciousness and monitoring any change. It involves assessing three responses:

- eye opening
- motor
- verbal.

Patients should be described according to the three responses, as this gives a clearer indication of their status (e.g. eye opening to speech, disorientated, and localizing pain and not just 'GCS 12').

Progression down the scale indicates a worsening condition and a worsening prognosis. Following trauma, a person who has no eye opening, no motor, and no verbal response (GCS 3) is unlikely to survive (see Table 4.1).

Glasgow coma scale in young children

In young children the modifications shown in Table 4.2 can be used.

**Table 4.1** Glasgow coma scale

<table>
<thead>
<tr>
<th>Eye-opening response</th>
<th>Motor response</th>
<th>Verbal response</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Obey commands</td>
<td></td>
<td>6</td>
</tr>
<tr>
<td>spont.</td>
<td>Localizes pain*</td>
<td>Orientated‡</td>
<td>5</td>
</tr>
<tr>
<td>To speech</td>
<td>Abnormal flexion</td>
<td>Words only</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>Extension</td>
<td>Sounds only</td>
<td>2</td>
</tr>
<tr>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>1</td>
</tr>
</tbody>
</table>

* Must bring hand higher than chin to supra-orbital pain.
‡ Orientated to time, place and person.

**Table 4.2** Glasgow coma scale in young children

<table>
<thead>
<tr>
<th>Eye-opening Response</th>
<th>Motor response</th>
<th>Verbal response</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous movement</td>
<td>Localizes pain</td>
<td>Usual vocalization</td>
<td>5</td>
</tr>
<tr>
<td>Spontaneous</td>
<td>Normal flexion</td>
<td>Reduced vocalization</td>
<td>4</td>
</tr>
<tr>
<td>To speech</td>
<td>Abnormal flexion</td>
<td>Cries only</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>Extension</td>
<td>Moans only</td>
<td>2</td>
</tr>
<tr>
<td>Nil</td>
<td>Nil</td>
<td>Nil</td>
<td>1</td>
</tr>
</tbody>
</table>
When discussing the GCS make it clear which score you are using. The original GCS had a maximum score of 14 not 15. Some units may still use this score, so be clear, especially if you are transferring or receiving a patient elsewhere.

The cranial nerves
See Table 4.3. Note the following:
- CNIII—gaze deviation and ptosis are only seen in a conscious patient.
- Pupillary inequality can occur in 20% of normal people; both pupils will react to light in this case.
- CNVII—taste is via the nervus intermedius, which joins the trigeminal (CNV3) for distribution to the tongue.
- Weber’s test: place tuning fork in centre of forehead. If sound is heard best in deaf ear, deafness is conductive.
- Rinne’s test: hold tuning fork by ear until sound inaudible, then move fork to mastoid process. If sound is heard deafness is conductive.

Cranial nerve (brainstem) reflexes

Pupillary reflexes
Afferent II, efferent III parasympathetic: 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), but the affected pupil will constrict to light in the opposite eye. If there is a III palsy, that pupil will not react to light in either eye, but the opposite pupil reacts to light in both eyes.

Corneal reflex
Afferent V, efferent VII: stroke the cornea with cotton wool and watch for blinking.

Grimace reflex
Afferent V, efferent VII: press on the supra-orbital nerve at the orbital margin and watch for facial grimacing. Any limb or autonomic (pulse rate and blood pressure elevation) responses should also be recorded.

Gag reflex
Afferent IX, efferent X: stimulate the posterior pharynx and watch for gagging.

Oculocephalic and oculovestibular reflexes
Afferent VIII, efferent III, IV, and VI: these are the same reflex pathway stimulated by different methods.

In the oculocephalic (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.
### Table 4.3 The cranial nerves

<table>
<thead>
<tr>
<th>No</th>
<th>Nerve</th>
<th>Function</th>
<th>Test</th>
<th>Palsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Olfactory</td>
<td>Smell</td>
<td>Various smell bottles (test each nostril separately)</td>
<td>Loss of smell (anosmia)</td>
</tr>
<tr>
<td>II</td>
<td>Optic</td>
<td>Vision</td>
<td>Visual activity, visual fields, pupillary responses, fundoscopy</td>
<td>Blind eye, visual field defect or loss of acuity, papilloedema</td>
</tr>
<tr>
<td>III</td>
<td>Occulomotor</td>
<td>Eye movements</td>
<td>Eye movement in all directions, pupillary responses</td>
<td>Ptosis, eye deviated down and outwards, unreactive dilated pupil</td>
</tr>
<tr>
<td>IV</td>
<td>Trochlear</td>
<td>Eye movements</td>
<td>Eye movement down when looking medially</td>
<td>Inability to look down when looking medially</td>
</tr>
<tr>
<td>V</td>
<td>Trigeminal</td>
<td>Facial sensation, muscles of mastication</td>
<td>Sensation in three trigeminal divisions, corneal reflex, jaw movement</td>
<td>Loss of facial sensation, loss of corneal reflex, jaw weak and deviates to side of lesion of opening, wasting of mastication muscles (chronic)</td>
</tr>
<tr>
<td>VI</td>
<td>Abducent</td>
<td>Eye movements</td>
<td>Eye movement laterally</td>
<td>Inability to look laterally</td>
</tr>
<tr>
<td>VII</td>
<td>Facial</td>
<td>Facial movements, Taste to anterior tongue</td>
<td>Facial movements Sweet, bitter, salt taste</td>
<td>Loss of facial movement UMN: forehead spared substances, LMN: forehead affected, Loss of taste</td>
</tr>
<tr>
<td>VIII</td>
<td>Vestibulocochlear</td>
<td>Hearing, Equilibrium</td>
<td>Hearing, Weber’s and Rinne’s tests balance and equilibrium</td>
<td>Deafness nystagmus, loss of equilibrium</td>
</tr>
<tr>
<td>IX</td>
<td>Glossopharyngeal</td>
<td>Pharyngeal posterior tongue sensation and taste motor to upper pharynx</td>
<td>Pharyngeal sensation, gag reflex</td>
<td>Loss of gag reflex and pharyngeal sensation</td>
</tr>
<tr>
<td>X</td>
<td>Vagus</td>
<td>Visceral parasympathetic supply (extensive) Larynx and pharynx motor function</td>
<td>Pharyngeal movement, gag reflex, Laryngoscopy</td>
<td>Loss of gag reflex and pharyngeal movement, Hoarse voice, vocal cord paralysis</td>
</tr>
<tr>
<td>XI</td>
<td>Accessory</td>
<td>Trapezius and sternomastoid motor function</td>
<td>Trapezius and sternomastoid power</td>
<td>Weakness of trapezius and sternomastoid</td>
</tr>
<tr>
<td>XII</td>
<td>Hypoglossal</td>
<td>Tongue movements</td>
<td>Tongue movements</td>
<td>Tongue deviates to side of lesion</td>
</tr>
</tbody>
</table>

UMN = upper motorneurone, LMN = lower motorneurone.
Head injuries—pathophysiology

The brain is the most sensitive organ in the body to hypoxia and ischaemia. Therefore it is essential to maintain an adequate supply of well oxygenated blood to the injured brain.

**Autoregulation** maintains a constant supply of blood to the brain between a mean blood pressure (BP) of 50 and 160 mmHg. However, this mechanism is usually **impaired following head injury**. The cerebral perfusion pressure (CPP) is the force driving blood through the brain and is normally over 70 mmHg. It is related to the BP and intra-cranial pressure (ICP) by:

$$\text{CPP} = \text{BP} - \text{ICP}.$$  

A developing **intra-cranial mass lesion** will initially be compensated for by displacement of venous blood and CSF, so the ICP will not rise. When this compensatory mechanism has been exhausted the ICP will rise and the CPP fall. The **Cushing reflex** then comes into play, increasing the BP to maintain cerebral blood flow. The pulse rate also falls due to a vagal reflex. When this compensatory reflex fails progressive cerebral ischaemia will occur leading to cerebral infarction and brain death. A vicious circle becomes established with hypoxia, hypotension and cell breakdown products, which worsen cerebral oedema, contributing to the deterioration.

**Brain herniation**

Three types of herniation can occur when a mass lesion develops intra-cranially.

- **Sub-falcine herniation.** One hemisphere is displaced beneath the falx, which is seen as midline shift on a CT scan. This can obstruct the foramen of Monro anteriorly, causing unilateral ventricular dilatation and compress the posterior cerebral artery against the falx posteriorly, causing a posterior cerebral infarct.

- **Trans-tentorial herniation.** The uncus of the medial temporal lobe herniates through the tentorial notch. This compresses the oculomotor nerve (dilatated pupil), and the midbrain.

- **Tonsilar herniation.** The cerebellar tonsils herniate through the foramen magnum causing brainstem compression (coning). This is the ultimate cause of brain death.
Fig. 4.1 Complex interplay of factors regulating ICP.
Head injuries—general points

‘Primary’ brain injury occurs at the time of the trauma. As clinicians there is nothing we can do about this. Prevention is the only way to reduce this. However, ‘secondary’ brain injury occurs after the initial event and is due to complications such as hypoxia, hypercarbia, hypotension, raised intra-cranial pressure (haematomas or cerebral oedema), cerebral herniation of infection. One way or another these all result in either hypoxia or inadequate cerebral perfusion.

The aim of head-injury management is to prevent secondary injury by regular observation and rapid correction if any deterioration occurs. This helps promote a physiological milieu that encourages natural recovery of the primary injury.

Primary brain injury

Primary brain injury can take the form of:

- **Cortical lacerations (burst lobe)** usually results also in an acute subdural haematoma together with a cerebral haematoma and surrounding contusions. The affected brain usually swells markedly. A craniotomy is necessary for evacuation of the subdural and debridement of the damaged brain. The prognosis is usually poor due to the extent of the primary brain damage.

- **Cerebral contusions.** This is discussed under Intra-cranial haematomas

- **Diffuse axonal injury** consists of widespread disruption of axon sheaths due to a high-energy impact. It is particularly associated with a rotational element to the force. **Concussion**, a transient impairment of consciousness following a minor or moderate head injury is probably a mild diffuse axonal injury. The CT scan in diffuse axonal injuries can be normal, but more often shows a tight swollen brain with or without petechial haemorrhages. The degree of brain swelling usually increases over the 48h post-injury. The prognosis for diffuse axonal injury is poor.

The commonest causes of head injuries are:

- motor vehicle collisions;
- assaults;
- falls from a height;
- sporting accidents.

Alcohol is involved in about 30% of head injuries.
Fig. 4.2 Intra-cerebral haematoma—a ‘secondary’ brain injury.
Head injuries—assessment

History
The following are extremely important and should be determined in all cases:

- **Time of injury and any changes in condition.**
- **Mechanism of injury:** suddenly stopping (a deceleration injury) will transfer more energy to the brain than a stationary person struck by a moving object (an acceleration injury):
  \[ E = \frac{1}{2}mv^2. \]
- **Conscious state immediately after the injury:** for baseline observation.
- **Any delayed loss of consciousness:** this implies complications are developing.
- **Any suggestion of compound or penetrating injury:** bleeding from the ears, CSF loss from the nose or ears. Penetrating injuries through the orbit can be easily overlooked.
- **Period of post-traumatic amnesia:** for prognostic reasons.
- **Any ongoing symptoms.**

Examination

- **Resuscitation status:** any deficiency needs immediate correction—before taking a history if necessary.
- **Conscious state:** Glasgow coma scale.
- **Focal neurology:** cranial nerve and limb neurology. Unequal but reactive pupils occur in 20% of normal individuals. A dilated unreactive pupil is usually on the side of a mass lesion (a true localizing sign). The usual sequence is initial pupillary constriction as the III nerve is irritated followed by dilatation as a palsy occurs. A hemiparesis can be caused by a mass lesion pressing on the opposite motor cortex, or a mass on the same side compressing the opposite cerebral peduncle against the edge of the tentorium. Thus, a hemiparesis does not help in determining the side of a mass lesion and is considered a false localizing sign.
- **Local signs of injury.**
- **CSF rhinorrhoea or otorrhoea, bleeding from the ear:** a compound skull base fracture.
- **Battle’s sign (bruising over the mastoid process):** a fractured petrous bone.
- **Panda eyes (well circumscribed peri-orbital bruising):** an anterior fossa skull base fracture.
- **Scalp lacerations, abrasions, swelling; etc.** Consider whether a laceration overlies a fracture.
- **Examination for other injuries:** this should be repeated when the patient has been stabilized.

Investigations

Plain skull X-rays need not be performed if the patient is to have a CT scan.

- **Skull X-ray indications:** At the time of writing these are undergoing major review in the UK by the National Institute of Clinical Excellence (NICE).
• any loss of consciousness or amnesia;
• suspected penetrating injury;
• CSF or blood loss from nose or ear;
• significant scalp laceration, bruise, or swelling;
• violent mechanism of injury, including >60 cm fall in a young child;
• persisting headache and/or vomiting.

**CT scan indications:**
• reduced GCS or neurological signs persisting after resuscitation;
• neurological deterioration in resuscitated patient;
• skull fracture or suture diastasis;
• epileptic fits;
• diagnosis uncertain;
• tense fontanelle in a child.

**Head injury classification**

Head injuries are classified for management, epidemiological, and research purposes as minor, moderate, and severe, based upon the total GCS score. When discussing the GCS, make it clear which score you are using. The original GCS had a maximum score of 14 not 15. Some units may still use this score, so be clear, especially if you are transferring or receiving a patient elsewhere.

- Moderate: GCS 9–12 (or 7–8 with eye opening).
- Severe: GCS 8 or less.
Skull fractures

The main worry in 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 (they are due to a cylindrical vessel indenting the bone and so the thickness of the skull overlying them varies across their diameter).

- **Sutures** lie in well-defined locations, but sometimes additional sutural bones might be present. Their margins are highly tortuous.

**Sometimes following a head injury, a suture might become separated (diastasis of a suture) and this should be managed as a fracture.**

**Linear fractures**

These are usually relatively straight with well-defined margins and are usually several centimetres long. The margins of long fractures might be separated by several millimetres. Skull fractures heal slowly and it might not be possible, from its appearance, to determine how old a fracture is, particularly if an individual is prone to multiple head injuries. However, a new fracture will be painful and tender and there will often be a degree of scalp swelling. Fractures of the skull base are difficult to see on plain skull X-rays and should be suspected on the basis of clinical features.

**The main significance of linear skull fractures is that they signify an increased risk of developing an intra-cranial haematoma.** They are managed the same as a minor head injury without a fracture—observation and basic care. They should be scanned and can be discharged when asymptomatic.

**Basal skull fractures**

A greater degree of force is required to produce a basal fracture than a vault fracture. They are difficult to see on plain skull X-ray and are usually diagnosed on clinical grounds. They are usually visible on a fine cut CT scan with bone windows.

Basal fractures without a CSF leak are managed similar to vault fractures. CSF otorrhoea usually settles conservatively. CSF rhinorrhoea also frequently settles, but the fistula might re-open with a risk of late meningitis, sometimes years later. Surgical repair should therefore be discussed with the patient, especially if a large defect is seen on a coronal CT scan. Prophylactic antibiotics are not indicated as they have not been shown to prevent meningitis (antibiotics penetrate poorly into CSF in the absence of infection) and they lead to colonisation by resistant organisms.

**Depressed fractures**

These are usually round with linear fracture lines radiating from the centre, resulting in several fragments. All fractures should be evaluated further by CT scanning. They are usually compound in adults, but can be closed in young children (‘ping-pong ball’ fracture). Compound fractures should be elevated if they are depressed by an amount greater than the skull thickness. The principal aim of surgery is wound toilet and removal of any foreign bodies to reduce the risk of infection, and so surgery should be
performed within 24 h. There is a risk of long-term epilepsy, which increases if there was a dural tear, an intra-cranial haematoma, over 24 h of post-traumatic amnesia and whether there were any early fits. Closed depressed fractures can be left alone unless they are in a cosmetic area or a significant depression is associated with a neurological deficit, although abnormal neurology is very rare in depressed fractures.

Fig. 4.3 Depressed skull fracture.
Intra-cranial haematomas

The risk of developing an intra-cranial haematoma had been determined with relationship to the orientation of the patient and the presence of a skull fracture (see Table 4.4). One of several different types of haematoma might develop.

Concussion

This is a temporary disturbance in brain function following relatively minor head injuries. Structurally the brain remains undamaged. Typically the patient is ‘knocked out’ for several minutes. Prolonged episodes of unconsciousness are rare. In any event the patient rapidly wakes up and makes a full recovery. So long as there are no other complicating medical or social factors such patients can go home providing they can be carefully observed. If the patient does not fully recover do not treat this as concussion.

Extradural haematomas

Extradural haematomas are usually associated with a skull fracture or suture diastasis. The commonest site is temporal, due to a tear of the middle meningeal artery, but they can also occur in the frontal and occipital regions. They are rare in young children, as their skull fractures are not sharp enough to damage the artery, and in the elderly, as their dura is usually firmly adherent to the skull. They classically present with delayed deterioration due to the dura being only slowly stripped from the skull. However, only a minority of patients are completely asymptomatic during this 'lucid interval'.

Extradural haematomas are lentiform (lens) shaped on CT scans and are mostly high density. Low-density areas within them are said to be due to unclotted blood.

Very small extradurals with minimal symptoms can often be left alone (although they should all be transferred to neurosurgical units for observation), but most need a craniotomy for evacuation. The prognosis is very good if they are treated early enough.

Acute subdural haematomas

Acute subdural haematomas are due to:

- a tear of a bridging vein between the brain and skull, in which case the prognosis is good with prompt treatment; or
- a laceration of the brain surface (burst lobe), which has a worse prognosis.

There need not be a skull fracture with subdural haematomas. They are commoner than extradurals and can extend over a wide area of the lateral cortical surface. They are crescent-shaped on CT scans.

All patients with acute subdurs should be transferred to neurosurgical units for management. Thin subdurals can be treated conservatively with close observation, but significant ones need a craniotomy as the clotted blood is too thick to drain via burr holes.

Cerebral contusions and haematomas

In cerebral contusions blood is interspersed between the neurones and glia, whereas with cerebral haematomas the bleeding forms a cavity within
the brain. However, cerebral contusions can enlarge and result in a haematoma. Contusions often occur at the poles of the brain due to a contra-coup injury; i.e. the brain striking the inner surface of the skull after it has come to an abrupt stop.

They can be associated with marked oedema and a greatly raised intracranial pressure (ICP). They are usually treated conservatively, but a lobectomy (or evacuation of an intra-cerebral haematoma) can be performed if the ICP cannot be controlled and only one lobe is involved.

The affected brain usually resorbs to form a porencephalic cyst. The prognosis is usually poor, with survivors often having some degree of cognitive, personality or memory change.

1 Chronic subdural haematomas

Chronic subdural haematomas are thought to be due to repeated minor bleeding following a minor head injury several weeks previously. The head injury can be so trivial that it cannot be remembered in 50% of cases. They usually occur in the elderly, but can also occur in babies due to non-accidental injuries. They are often associated with coagulopathies and alcoholism.

They can cause a wide variety of symptoms, including headaches, reduced consciousness, and focal neurology. Therefore, consider this in all elderly patients with intermittent confusion. Chronic subdural haematomas are usually treated by burr hole drainage and have a good prognosis but might recur, especially with a persistent coagulation disorder.

Criteria for SXR

At the time of writing these are undergoing major review in the UK by the National Institute of Clinical Excellence (NICE). May vary with different units—check local policy:

- mechanism of injury;
- LOC;
- vomiting;
- severe headache;
- visual disturbance;
- fits, faints, neurological deficit;
- GCS <15;
- difficulty in assessment (child, C₂H₅OH);
- amnesia (retrograde vs anterograde);
- penetrating injury;
- ?FB;
- battles sign;
- CSF oto/rhinorrhea;
- ‘panda’ or ‘racoon’ eyes.

Table 4.4 Risks of intracranial haematoma following head trauma

<table>
<thead>
<tr>
<th></th>
<th>Orientated</th>
<th>Confused or worse</th>
</tr>
</thead>
<tbody>
<tr>
<td>No skull fracture</td>
<td>1:6000</td>
<td>1:120</td>
</tr>
<tr>
<td>Skull fracture</td>
<td>1:32</td>
<td>1:4</td>
</tr>
</tbody>
</table>

One of several different types of Haematoma might develop.
Criteria for admission
May vary with different units—check local policy:
- skull fracture (proven or suspected);
- GCS less than 15;
- FND;
- epilepsy;
- unable to assess;
- elderly;
- infants;
- ?NAI;
- ethanol;
- mechanism of injury;
- social;
- risk factors, e.g. warfarin.

Criteria for neurosurgical consultation/CT scan
May vary with different units—check local policy:
- skull fracture + GCS < 15;
- penetrating injury;
- depressed fracture;
- deterioration;
- pupillary asymmetry;
- FND;
- Cushings;
- compound fracture;
- FB;
- coma;
- GCS > 15 after 8 h;
- anaesthetised + any head injury.
- coma after resuscitation.

Possible outcomes
- Death.
- PVS/FND/post-concussion syndrome.
- Epilepsy.
- Irritability/personality change.
- Pyrexia.
- Diabetes insipidus.
- Hydrocephalus.
- Meningitis.
- ARDS/stress ulcer.
**Head injuries—definitive care**

**Scalp lacerations**
Scalp lacerations should be thoroughly cleansed and closed, in two layers if possible. The use of tissue glue is acceptable for small lacerations. The possibility of foreign bodies or an underlying fracture should be considered if the patient has not been X-rayed. Also remember anti-tetanus prophylaxis. Scalp sutures can usually be removed after 7 days.

Observations should be performed hourly, and half-hourly in higher risk patients. Most patients can be discharged the following day if asymptomatic. Stable patients who need longer admission can have their observation frequency reduced to 2-hourly. Patients not admitted should receive written guidelines of when to return and should only be discharged with a responsible adult who can call for assistance when required.

**Transfer arrangements**
- **Fully resuscitate ABCs in all patients before transfer**—this may include a laparotomy or pelvic fixation to stop bleeding.
- Intubate and ventilate comatose patients.
- If patients are being transferred for observation only, avoid intubation and sedation (discuss with neurosurgeons), if safe to do so.
- Intravenous mannitol can be given to gain time by reducing intra-cranial pressure.
- Transfer with experienced anaesthetist.
- Transfer promptly!

**Advanced head injury management**
On intensive care (ICU) patients will have at least the following inserted:
- endotracheal tube;
- ICP monitor;
- arterial catheter—BP monitoring;
- CVP line;
- urinary catheter;
- Naso-gastric catheter.

If the ICP is difficult to control, a jugular venous oximeter (JVO₂) may also be inserted. This is passed up to the jugular bulb at the skull base and measures the amount of blood being extracted from the brain. If the patient is being vigorously hyperventilated to reduce the ICP, cerebral vasoconstriction can occur, worsening cerebral ischaemia. This can be detected on JVO₂ by an increase in the amount of oxygen being extracted by the brain and the amount of hyperventilation reduced.

**ICP management**
The following measures can be used to lower ICP in severe head injuries. The first four are commonly used; the other measures are increasingly less successful and less frequently used:
- ventilation- to maintain a normal pO₂ and normal pCO₂;
- removal of mass lesions;
- diuretics;
- mannitol;
- frusemide;
CHAPTER 4 The head

- inotropes—maintain CPP by BP elevation;
- hyperventilation—reduce pCO2 to 3.5 kPa;
- barbiturates—lowers cerebral metabolism;
- hypothermia—lowers cerebral metabolism;
- CSF drainage (the ventricles are usually small and difficult to cannulate so this is not often used);
- decompressive craniotomy—allows additional space for the brain to expand into.

CSF leaks

Facial fractures that extend into the base of the skull (e.g. Le Fort II, Le Fort III, naso-ethmoidal and occasionally fractures involving the mandibular condyle) can tear the dural lining and allow cerebral spinal fluid (CSF) to leak from the nose (rhinorrhoea) or from the ear (otorrhoea). Clear CSF tends to mix with blood and presents as a heavily blood-stained, watery discharge. This trickles down the side of the face, where peripherally the blood tends to clot while the non-clotted blood in the centre is washed away by CSF. This creates two parallel lines referred to as ‘tramlining’. One test for CSF is the ‘ring test’ (allow drops to fall on blotting paper, blood clots centrally, the CSF diffusing outwards to form a target sign). Other tests include examining for eosinophils and sugar. This is helpful in distinguishing between CSF and mucous. More sensitive indicators include B2 transferrin and tau protein, although practically it is easier to simply assume that a leak is present. **Tell the patient not to blow their nose for three weeks. If they do the increased pressure can force air intra-cranially through the tear, which then cannot escape. This is the neurosurgical equivalent of a tension pneumothorax!**

![Fig. 4.4 A ‘tension’ pneumocephalocele.](image)
Head injuries in children
These can be difficult to assess. Many of the features, which would lead to concern in adults are often present even with minor injuries (vomiting, drowsy, headaches, etc.). Carefully consider the mechanism of injury, other injuries present and whether the parents are capable of taking the child home for close observations. Interpretation of skull X-rays can be difficult as large fractures may be confused with wide sutures or vascular markings. CT scans are difficult to get and nearly always require general anaesthesia in an uncooperative child. If in doubt either refer or admit.

Remember NAI.