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 petaechial haemorrhages. The degree of brain swelling usually increases over the 48 h 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.

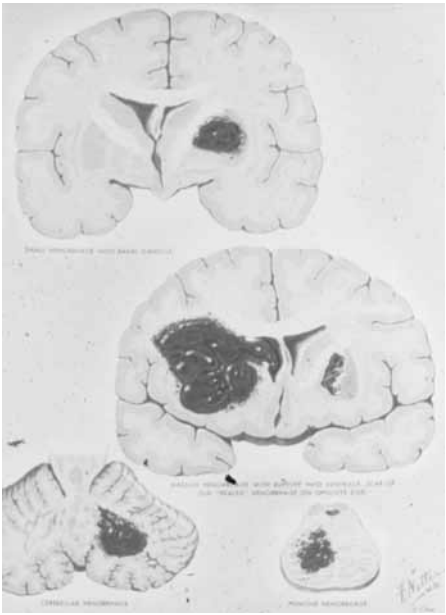

Fig. 4.2 Intra-cerebral haematoma—a 'secondary' brain injury.

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:

- 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**.

- Minor: GCS 13–15.
- 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 symptomatic.

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.

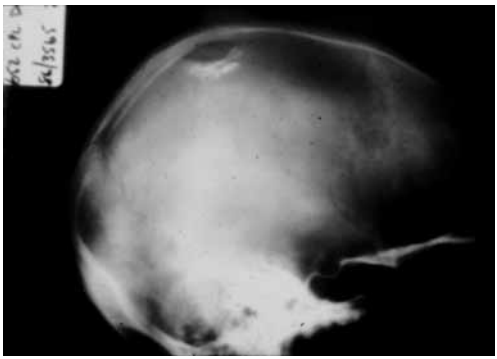

Fig. 4.3 Depressed skull fracture.

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 subdurals should de 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.

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

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, C_2H_5OH);
- amnesia (retrograde vs anterograde);
- penetrating injury;
- ?FB;
- battles sign;
- CSF oto/rhinorrhea;
- 'panda' or 'racoon' eyes.

Table 4.4

	Orientated	Confused or worse
No skull fracture	1:6000	1:120
Skull fracture	1:32	1:4

One of several different types of Haematoma might develop.

AQ: Please provide Table caption.

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 (JVO_2) 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 JVO_2 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 pO_2 and normal pCO_2;
- removal of mass lesions;
- diuretics;
- mannitol;
- frusemide;

- inotropes- maintain CPP by BP elevation;
- hyperventilation—reduce pCO_2 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!**

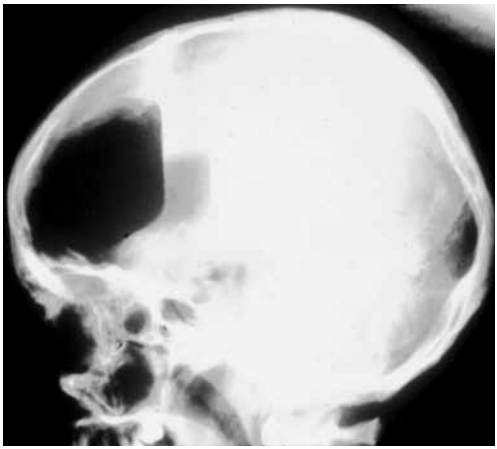

Fig. 4.4 A 'tension' pneumocephalocele.

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**.