The Skull, Brain and Associated Structures: Part III

8

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8.1 Head Injuries

8.1.1 Terminology in Head Injuries: Traumatic Brain Injury (TBI)

A "head injury" is one of the most common reason for attendance to the emergency department, particularly at weekends. Traumatic brain injury (TBI), a more precise term, represents a wide spectrum of severity. It has been estimated that 3.2 million people are living with long-term disability related to traumatic brain injury (TBI). This is the leading cause of morbidity and mortality between the ages of 1 and 45 years. Teenagers and the elderly are most at risk, although the causes vary demographically. Motor vehicle crashes are a main cause of head injuries in most ages, whilst falls are most common in people aged 65 or older. Whilst the majority of attendances are relatively minor, it is nevertheless important not to overlook the patient with a potentially serious intracranial injury. They can rapidly deteriorate. Other causes of an altered conscious level should also be considered in any agitated, confused or obtunded patient. Alcohol excess, drugs, hypoxia, hypotension, hypoglycaemia and other metabolic disturbances should always be considered. A systematic approach to the head injured patient should therefore always be followed, even in those with an apparent isolated head injury. This is discussed in the chapter on the injured patient.

Traumatic brain injury (TBI) can be either primary or secondary. "Primary" brain injury occurs at the time of the trauma. This is caused by impacts or inertial forces to the head that deform the intracranial tissues. Impacts usually produce focal injuries such as skull fractures, contusions and extradural/subdural haematomas.

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Inertial forces can occur without any impact to the head. These arise as a result of sudden acceleration or deceleration, translational and/or rotational forces, which can cause focal or diffuse brain injuries, such as subdural haematomas and contre coup contusions. Diffuse injuries commonly occur following rotational or angular acceleration. From a management perspective there is nothing that can be done about these instantaneous injuries. By the time medical assistance arrives the primary injury is completed and secondary brain injury is rapidly developing. Prevention is the only way to reduce their incidence and severity.

"Secondary" brain injury occurs after the initial event and arises as a result of complicating factors such as hypoxia, hypercarbia, hypotension, raised intracranial pressure (haematomas or cerebral oedema), cerebral herniation or, later on in the patient's recovery, infection. Secondary injury can develop hours or days after the initial trauma and may be a major factor in prognosis. Whatever the underlying problem, these all essentially result in the development of either hypoxia or inadequate cerebral perfusion. The focus of head injury management is therefore to prevent these complicating factors from causing secondary brain injury. By doing so it is hoped that recovery from the primary brain injury will occur through natural processes. Whether head injury management is successful or not is dependent on both the extent of primary brain injury and the ability to control and reverse confounding factors.

8.1.2 Primary Injuries to the Brain

Injury to the brain can be caused by external forces to the head that strain the tissues beyond their structural tolerance. These forces can be classified as contact or inertial. Contact or impact forces usually produce focal injuries (such as skull fractures, contusions and extradural or subdural haematomas). Inertial or acceleration/deceleration forces can occur without any impact. These can result in focal or diffuse brain injuries—without any external signs of head trauma. As a result of all these different forces, primary brain injury can take the form of:

8.1.2.1 Cortical Lacerations (Burst Lobe)

This is mechanical disruption to the substance of the brain, commonly caused by a penetrating injury or pushed-in bone fragment following a skull fracture. A laceration may also occasionally occur following severe shearing forces unassociated with fractures. In most cases, it usually results in significant bleeding and is thus commonly seen in association with an acute subdural and cerebral haematoma. Other brain injuries are frequently seen, including contusions and diffuse axonal injury. Lacerations are particularly common in the inferior frontal lobes and the poles of the temporal lobes. Soon after injury the affected brain usually swells significantly. Patients therefore usually present with a significantly reduced Glasgow coma scale. Craniotomy is therefore often urgently indicated in order to evacuate the subdural haematoma and/or debride the damaged brain. The prognosis is usually poor due to the extent of primary brain damage. A cerebral laceration associated

with large amounts of blood on CT imaging is generally regarded as an indicator of poor prognosis.

8.1.2.2 Cerebral Contusions/Haematoma

These occur when the brain strikes the inner table of the skull. Haemorrhage within the brain substance occurs after very severe TBI. This is usually associated with contusions of the surrounding tissue. "Coup" contusions arise from the skull bending or fracturing, which subjects the underlying cortical and pial vascular network to excessive strains. This can cause bleeding at or near the brain surface. Damage is likely to occur when the skull is "rebounding" from the impact and the vessels are experiencing tensile strain. "Contrecoup" contusions are thought to occur as a result of cavitation effects and inertial loading. As the brain moves towards the impact site it creates an area of negative pressure directly opposite the point of impact. This negative pressure may cause damage by exceeding the tensile strength of the tissues or by causing small gas bubbles to appear within the parenchyma. The return to normal pressure in the brain then causes the small bubbles to collapse—hence the term cavitation. Duret's haemorrhage (small lineal areas of bleeding in the midbrain and upper pons), is thought to result from disruption of the arteries at the time of downward displacement of the brainstem. This is usually seen in patients with severe herniation for 12–24 hours prior to death. Its presence thus often indicates a grave prognosis. Contusions are most common in the inferior frontal cortex and the anterior temporal lobes. These are composed of areas of punctate haemorrhage, oedema and necrosis. They can evolve over time and may therefore not be obvious on an initial CT scan. Depending on their size and location, they may cause significant mass effect, with midline shift or herniation.

8.1.2.3 Diffuse Axonal Injury

This refers to punctate contusions at the interface between the grey and white matter, with widespread disruption and shearing of axon sheaths. It usually occurs following high energy impacts, particularly when there has been a rotational or deceleration element. Unlike brain trauma that occurs as a result of a direct impact to the brain, DAI is the result of shearing forces that occur when the head is rapidly accelerated or decelerated. This may be seen following vehicle collisions, falls, and assaults. It can also occur as the result of child abuse such as in shaken baby syndrome. The effect is disruption of axons. CT imaging in diffuse axonal injuries can be normal, but often shows a tight, swollen brain, with or without petechial haemorrhages. The degree of brain swelling usually increases over the first 48 hours post injury. The prognosis for diffuse axonal injury is poor and surgical options are limited. It is seen in about half of patients with severe TBI and in a third of those who die. It is also a common cause of a persistent vegetative state.

8.1.2.4 Concussion

This is a transient impairment of consciousness following a minor or moderate head injury. It is probably a mild form of diffuse axonal injury.

8.2 Secondary Injuries to the Brain

This can occur as a result of varying processes, many of them ultimately resulting (directly or indirectly) in ischaemia to a part of, or all the brain. These include

(i) Hypotension (systolic blood pressure [SBP] < 90 mm Hg)
(ii) Hypoxemia (PaO_2 < 60 mm Hg; O_2 Saturation < 90%)
(iii) Hypocapnia ($PaCO_2$ < 35 mm Hg)
(iv) Hypercapnia ($PaCO_2$ > 45 mm Hg)
(v) Hypertension (SBP > 160 mm Hg, or mean arterial pressure [MAP] > 110 mm Hg)
(vi) Anaemia (Hemoglobin [Hb] < 100 g/L, or hematocrit [Ht] < 0.30)
(vii) Hyponatremia (serum sodium <135 mEq/L)
(viii) Hyperglycemia (blood sugar >10 mmol/L)
(ix) Hypoglycemia (blood sugar <4.6 mmol/L)
(x) Hypo-osmolality (plasma osmolality [P Osm] < 290 mOsm/Kg H_2O)
(xi) Acid-base disorders (acidemia: pH < 7.35; alkalemia: pH > 7.45)
(xii) Fever (temperature > 36.5 °C)
(xiii) Hypothermia (temperature < 35.5 °C)

8.2.1 Pathophysiology

The brain is the most sensitive structure in the body to hypogycaemia, hypoxia and ischaemia. Electrolyte disturbances can both aggravate and be caused by brain injury. Therefore one of the key elements of head injury management is to maintain an optimal environment which provides an adequate supply of well oxygenated and nutritious blood to the injured brain. If ischaemia occurs, it initiates a cascade of metabolic events that results in the production of oxygen free radicals, amino acids, cytokines, and other inflammatory products. Collectively, these result in disruption of ionic regulation and flow, notably ionised calcium, which plays a key role in neurodegeneration after injury. Free fatty acids, leukotrienes, and thromboxane B_2 are also released. These are also associated with neurodegeneration and poor outcomes. TBI also increases extracellular potassium, leading to disruption of the Na^+/K^+-ATPase cell membrane mechanisms, with subsequent cellular swelling and depolarisation of neurones. Hypoglycemia results in brain fuel deprivation, redistribution of cerebral flow and functional failure. This can usually be corrected by raising plasma glucose concentrations. However, brain death can also occur as a result of increased neuronal NADPH oxidase activation during glucose reperfusion. Thus when profound hypoglycaemia occurs, therapeutic hyperglycemia should be avoided. Precise glucose monitoring and control is therefore essential. Recently N-acetylaspartate (NAA) has been suggested as a possible 'marker' of neuronal health following brain injury. Decreased levels of NAA occurs in many neurological diseases that result in neuronal and axonal degeneration (including tumours,

epilepsy, dementia, stroke, hypoxia, multiple sclerosis and many leukoencephalopathies). Any major CNS disease resulting in neuronal and axonal damage, secondary ischaemia or toxicity will result in abnormalities of the metabolite. This can be detected with MR spectroscopy, potentially making it one of the most reliable molecular markers to assess brain recovery.

8.2.1.1 The Effects of Intracranial Swelling and Bleeding

The brain has a high metabolic demand and therefore requires adequate nutritional flow. To maintain stable cerebral blood flow (CBF), the brain's vasculature must respond to changes in arterial blood pressure (BP) or intracranial pressure. In the non-injured brain, this autoregulation maintains a constant supply of blood with a mean blood pressure (BP) between 50 and 160 mmHg. Cerebral blood flow must remain stable, so as to supply a steady amount of oxygen and glucose to the brain. This is possible by a variety of metabolic, myogenic and neurogenic mechanisms. However these mechanisms can become impaired following head injury. The cerebral perfusion pressure (CPP) is the difference between cerebral arterial and cerebral venous pressure. This is the driving force for cerebral blood flow. Practically this is measured as the ICP subtracted from the mean arterial pressure (MAP). CPP is normally over 70 mmHg and calculated by the equation CPP = MAP-ICP. Cerebral autregulation mechanisms appear to be locally mediated mainly by metabolic byproducts, or substrate deficits. Thee include (i) Carbon dioxide concentration in the brain parenchyma, (ii) pH of the blood (iii) Lactate (iv) Potassium and (v) Low oxygen. Autoregulation can be impaired by a variety of mechanisms

(i) Hypercapnea
(ii) Ischaemic stroke
(iii) Traumatic brain injury
(iv) Hypoxic brain injury
(v) Regional oedema, surrounding a space occupying lesion or a haematoma
(vi) Infection, e.g. meningitis or encephalitis
(vii) Malignant hypertension
(viii) Diabetic microangiopathy (after many years of uncontrolled diabetes)
(ix) Hepatic encephalopathy
(x) Septic encephalopathy

Applying some of the principles of physics to the intracranial contents, the Monro–Kellie doctrine, or hypothesis, is widely known and an important principle in intracranial pressure management. In essence the skull is a rigid container with 3 key constituents—brain tissue, cerebral blood and cerebrospinal fluid (CSF). Water, which makes up much of these components is incompressible. The Monro-Kellie doctrine states that the sum of intracranial volumes is constant and therefore an increase in any one of these compartments must be off set by an equivalent decrease in the other two. Compensatory mechanisms include movement of CSF into the spinal sac, increased reuptake of CSF and compression of venous sinuses. These

mechanisms reduce the circulating liquid component of the intracranial contents. Many of the MRI abnormalities seen in intracranial hypotension or CSF volume depletion can be explained by the Monro–Kellie hypothesis. These include meningeal enhancement, subdural fluid collections, engorgement of cerebral venous sinuses, prominence of the spinal epidural venous plexus, and enlargement of the pituitary gland.

Any growing intracranial mass lesion (haematoma, contusion, oedema, tumour, abscess etc) will initially be compensated for by displacement of venous blood and CSF, so the ICP will not rise. However when this compensatory mechanism is exhausted the ICP will rise and the CPP fall. Small increases in the volumes of intracranial constituents cause large increases in ICP. The Cushing reflex then comes into play, increasing the systemic BP in a physiological attempt to maintain cerebral flow. As a result, the pulse rate subsequently falls due to vagal stimulation. As ICP rises further, the CPP falls eventually to a point when there is no cerebral blood flow, no cerebral perfusion, cerebral infarction and brain death. Prior to this, brain structures begin to herniate (protrude through an opening). This results in pressure on the brainstem which can affect respiration patterns.

8.2.1.2 Neuro-endocrine Changes

The pituitary is particularly vulnerable to head trauma due to its anatomical location of the gland within the sella turcica and its fragile infundibular stalk with its tenuous vascular supply. Post-mortem studies have shown pituitary gland infarctions in up to one-third of patients who die shortly after sustaining a TBI. Proposed mechanisms include direct mechanical and shearing forces to the pituitary stalk with its long and vulnerable hypophyseal vessels. This can result in anterior lobe infarction. Raised intracranial pressure, systemic hypotension, hypoxia and local vasospasm may also contribute to pituitary ischaemia and dysfunction. The posterior pituitary is less susceptible to injury because of its more robust blood supply. Although pituitary insufficiency after TBI has been considered a rare event, recent reports have suggested permanent insufficiency may be more common than previously thought.

In the early stages of TBI, damage to both the hypothalamus and pituitary can occur with concomitant disturbances in hormone secretion. Moderate to severe TBI can result in central hypogonadism in 25–80%, thyroid hormone deficiency in 2–15%, hyperprolactinemia in more than 50%, GH deficiency in 20% and cortisol deficiency in 13%. One of the key hormones in the hypothalamus-pituitary-adrenal axis (HPA-axis) is cortisol. Deficiency of this is life-threatening condition and must be promptly diagnosed and treated. The roles of GH, IGF-1, oestrogen and testosterone upon brain function and neuronal plasticity are not fully understood, but it has been suggested these are important in both acute and long-term recovery. GH and IGF-1 receptors are abundant in the brain, GH is involved in vascular reactivity, vascular tone and CNS repair processes, while IGF-1 may be important in re-myelination and prevention of demyelination. Oestrogen and progesterone may be neuro-protective.

8.3 Assessing Traumatic Brain Injuries (Head Injuries)

Whenever possible this should include a witnessed account of the injury. Signs and symptoms of a more severe brain injury include

(i) Loss of consciousness at any time—With children, was a cry heard immediately?
(ii) GC Score <15 on initial assessment.
(iii) Focal neurological deficit.
(iv) Retrograde or anterograde amnesia.
(v) Persistent headache.
(vi) Vomiting or seizures post injury.

Initial assessment must therefore take these risk factors into consideration. Any history of previous neurosurgical intervention, use of anticoagulants, clotting disorders and alcohol abuse (acute or chronic) also increase the likelihood of brain injury, even after apparently minor trauma and should also be specifically enquired about. Patients aged 65 years and over, have a higher incidence of intracerebral haemorrhage following minor falls and should therefore be considered for CT, even if symptoms are minor and especially if they are on anticoagulant medication.

8.4 History

A history of trauma in most patients presenting with a head injury will usually be self-evident. However, consider the possibility of trauma in any patient with a coma of unknown cause. Intracranial bleeding may be either the cause or consequence on an accident. Therefore consider other possibilities in all patients who present following an unexplained fall (especially seizure or arrhythmia). In the acute setting, if the patient is comatose or confused and unable to give a history, any witnesses to the injury should be questioned regarding the precise circumstances of the injury and the condition of the patient immediately after.

Determine the mechanism of the injury, as this may give an indication of the amount of force applied to the head, the likelihood of specific intracranial (and other) injuries and possible prognosis. Patients sustaining a head injury from an assault or from being struck by a falling object are more likely to have more localised injuries (such as a depressed skull fracture, extradural haematoma etc) than patients sustaining acceleration/deceleration injuries, in which diffuse axonal injury is more likely. Suddenly stopping (a deceleration injury) will also transfer more energy to the brain than a stationary person struck by a moving object (an acceleration injury). Pedestrians and pedal cyclists fare worse than vehicle occupants in motor vehicle collision. However patients that have been ejected from car, or have fallen from a height are at higher risk of sustaining potentially serious head injuries. Penetrating head injuries not surprisingly, have a worse outcome than blunt trauma. Patients are more likely to present with a lower GCS and die early. Non-accidental

injury in children under 5 years old is also associated with a worse outcome, possibly because of cerebral infarction in this group.

Any loss of consciousness at the time of injury, will provide some indication of the severity of the primary brain injury sustained. If there was no loss of consciousness the likelihood of a serious primary brain injury is relatively low. Loss of consciousness and its duration should therefore be regarded as a marker of severe neurological injury. The clinical course following recovery may give an indication of any developing secondary brain injury. Any delayed loss of consciousness implies complications are developing. If the patient was fully recovered but is now deteriorating, or has an increasing headache, repeated nausea and vomiting, or seizures and is very drowsy, this would suggest and expanding intracranial mass or brain swelling. A history of of prior head injuries, particularly prior concussive episodes in amateur or professional sports, can indicate the potential for a worse prognosis and long-term outcomes. If the patient has received repeated head injuries over a short period of time (days or weeks) consider the possibility of second impact syndrome. Any history of drug or alcohol abuse, or prescription of anticoagulants, or a bleeding tendency can raise the risk of intracranial bleeding.

The age of the patient influences both the likelihood of developing TBI and its prognosis. TBI has a bimodal incidence distribution. Young adult males comprise the largest peak, as a result of alcohol related assaults, alls etc. and motor vehicle collisions. A second, smaller peak in incidence is also seen in the elderly, usually as a result of falls. With increasing age in the over 65 years of age group, prognosis generally falls. It has been reported that for any given severity of injury, women appear to fare less well and are more likely to develop brain swelling and intracranial hypertension than men. Genetic factors may also play a role—it has been suggested there is evidence that allele for apolipoprotein E (the same gene associated with Alzheimer's disease) predisposes to poor outcome after TBI.

The inability to lay down new memories after a head injury is generally referred to as posttraumatic amnesia (PTA). However, recent research has suggested that posttraumatic amnesia is in fact a misnomer. This is because the clinical state that occurs following head injury is actually one of severe inattention that prevents retention of new information. Thus "posttraumatic confusional state" may be a more accurate description of this phenomenon.

8.4.1 Providing Telephone Advice

Medical practitioners and nursing staff working in emergency departments and community practice are sometimes called by worried friends, relatives or bystanders for advice regarding any injuries. Guidelines for this are available online. In general, patients who have sustained a head injury should attend a hospital emergency department if they have any of the following risk factors:

(i) If the mechanism of injury suggests a high energy impact (call an ambulance)
(ii) Any loss of consciousness ('knocked out') as a result of the injury, even if the person has now recovered.

(iii) Amnesia for events before or after the injury
(iv) Persistent headache since the injury.
(v) Any seizures
(vi) Any vomiting episodes since the injury.
(vii) Any symptoms suggestive of focal neurological deficit since the injury.
(viii) Any previous brain surgery.
(ix) Any history of bleeding or clotting disorders.
(x) Current anticoagulant therapy such as warfarin.
(xi) Current drug or alcohol intoxication.
(xii) There are any safeguarding concerns (for example, possible non-accidental injury or if a vulnerable person is affected).
(xiii) Irritability or altered behaviour ('easily distracted', 'not themselves', 'no concentration', 'no interest in things around them'), particularly in infants and children aged under 5 years.

8.5 Examination of the Injured Head

As with all trauma, initial examination starts with a rapid primary survey (with particular consideration to the risk of cervical spine injury). This is discussed further in the chapter on the injured patient. With regards to the examination of the injured head, the Glasgow Coma Scale (GCS) is a quick and reliable method for rapid neurologic assessment. Both initial and post resuscitation GCS scores have been reported to correlate with 1-year outcomes following severe head injury. Examination for other injuries should be undertaken when the patient has been stabilised (notably neck/scalp/facial/ocular). Following ascertainment of the GCS score (whilst preparing for a CT scan if indicated), look for signs of external trauma. Bruising or bleeding around the head and scalp and blood in the auditory canal/behind the tympanic membrane may be clues to occult skull fractures and possible brain injury. However do not lift any skin flaps to visualise the underlying skull—if there are mobile fractures, these may be displaced resulting in catastrophic haemorrhage. Carefully assess the cranial nerves. Whilst the diagnosis is usually made following urgent CT scan, there are still useful clinical signs that may indicate specific injuries. These include

(i) Pupillary responses should be elicited. In the comatose patients, a unilaterally dilated pupil, may indicate impending herniation. This is usually on the side of a mass lesion (a true localising sign). However, unequal but reactive pupils may be seen in 20% of normal individuals. A dilated and unreactive pupil can also occur secondary to ocular injury (traumatic mydriasis), although this should never be assumed. Bilaterally fixed and dilated pupils and a GC score of 3 carries a very poor prognosis.
(ii) Isolated ophthalmoplegia secondary to traumatic brainstem injuries has been described and has a relatively benign prognosis.
(iii) Anosmia—this is a common symptom and has many possible causes. It can be difficult to elicit in the early stages of assessment of a head injury.

Nevertheless if present this may suggest shearing of the olfactory nerves at the cribriform plate. If anosmia is accompanied by rhinorrhea, a fracture of the anterior cranial fossa with CSF leak should be considered.

(iv) Abducent nerve (CN VI) palsies may indicate raised intracranial pressure.

(v) CN VII palsy, particularly in association with decreased hearing (CN VIII), may indicate a fracture of the petrous temporal bone.

(vi) Hearing loss (sensory neural) occurs in 20–30% of patients following head injuries. Consider the possibility of temporal bone fracture

(vii) In the drowsy or unconscious patient, focal neurological signs, particularly motor weakness, may suggest a localised contusion or early signs of herniation. Hemiparesis can result from either 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 (Kernohan's notch). Thus, hemiparesis does not help in determining the side of a mass lesion—it can be a 'false' localising sign. Flexor or extensor posturing implies extensive intracranial injury or raised intracranial pressure.

(viii) Tremors and dystonia may occasionally be seen but generally resolve overtime. These can sometimes indicate a severe head injury.

(ix) Aphasia implicates localised pathology.

(x) CSF rhinorrhoea, oculorrhea, or otorrhoea, or bleeding from the ear suggest an open skull base fracture involving the anterior and middle cranial fossa respectively. Fractures of the middle cranial fossa represent high-energy injuries

(xi) Battle's sign (bruising over the mastoid process): This indicates a likely fractured petrous bone.

(xii) Panda eyes or periorbital haematoma are highly suggestive of an anterior cranial fossa fracture.

Today, many specialists suggest that the term "concussion" should be avoided and replaced with the term "mild traumatic brain injury" (MTBI). All individuals with traumatic brain injury who are conscious and those who present with post-traumatic amnesia (PTA), should be assessed for

(i) Motor impairments—weakness, altered tone, balance and incoordination

(ii) Possible missed injuries

(iii) Pain

(iv) Bulbar problems affecting speech and swallowing

(v) Sensory dysfunctions that may impact on safety including hearing loss, numbness, visual problems (including reduced acuity, visual field loss, gaze palsies)

(vi) Reduced control over bowels and bladder

(vii) Cognitive dysfunctions such as impairments in attention, orientation and memory

(viii) Behavioural dysregulations including potential emotional/behavioural issues

8.6 Further Evaluation: The Role of Imaging

Today in most trauma centres, CT scanning is now the first choice of imaging in head trauma. The ability to scan patients rapidly also allows the cervical spine to be imaged at the same time. With high energy head trauma it is estimated that there is approximately a 5% risk of the cervical spine being significantly injured as well. Some centres now perform a 'pan-scan' in polytrauma, in which the head, neck, chest, abdomen and pelvis are all scanned during the same session. Several protocols have been developed to identify patients who will mostly benefit from head CT. These share many similarities, some of which are shown below. However it is always important to follow local protocols in your own hospital. In the United Kingdom, the National Institute for health and clinical excellence (NICE) has published a series of useful guidelines.

Current protocols available for head injury assessment
New Orleans Criteria—NOC (2000)
Canadian Assessment of Tomography for Childhood Head injury—CATCH rule (2010)
Canadian CT Head Rule—CCHR
CT in Head Injury Patients—CHIP rule
NICE.

8.6.1 Indications for Head CT: New Orleans Criteria (2000)

(i) Headache
(ii) Vomiting
(iii) Age more than 60
(iv) Drug or alcohol intoxication
(v) Persistent antegrade amnesia (short-term memory deficit)
(vi) Visible trauma above the clavicle
(vii) Seizure

High risk (for neurosurgical intervention)

(i) GCS score < 15 at 2 hours after injury
(ii) Suspected open or depressed skull fracture
(iii) Any sign of basal skull fracture (hemotympanum, 'raccoon' eyes, cerebrospinal fluid otorrhoea/rhinorrhoea, Battle's sign)
(iv) Vomiting (at least, two episodes)
(v) Age > 65 years

8.6.2 Indications for Head CT: Nexus II

(i) Evidence of significant skull fracture
(ii) Scalp hematoma
(iii) Neurologic deficit
(iv) Altered level of alertness
(v) Abnormal behaviour
(vi) Coagulopathy
(vii) Persistent vomiting
(viii) Age of 65 years or more

8.6.3 Indications for Head CT: CT in Head Injury Patients (CHIP)

Major criteria:

(i) Pedestrian or cyclist versus vehicle
(ii) Ejected from vehicle
(iii) Vomiting
(iv) Post-traumatic amnesia ≥4 h
(v) Clinical signs of skull fracture (such as palpable discontinuity of the skull, leakage of cerebrospinal fluid, raccoon eye bruising, and bleeding from the ear)
(vi) GCS score < 15
(vii) GCS deterioration ≥2 points (1 hour after presentation)
(viii) Use of anticoagulant therapy
(ix) Posttraumatic seizure
(x) Age ≥ 60 year

Minor criteria:

(i) Fall from any elevation
(ii) Persistent antegrade amnesia (any deficit of short-term memory)
(iii) Posttraumatic amnesia of 2 to 4 h
(iv) Contusion of the skull
(v) Neurologic deficit
(vi) Loss of consciousness
(vii) GCS deterioration of 1 point (1 hour after presentation)
(viii) Age of 40–60 years

8.6.4 CT Scanning in Children

The indications for carrying out a CT in children are different from those in adults. This is partly due to the structure of the paediatric skull, neurological development

and concerns regarding the use of radiation. The national institute for Health and Clinical Excellence (NICE) recommends CT if there are any of the following

(i) Loss of consciousness lasting more than 5 min (witnessed)
(ii) amnesia (antegrade or retrograde) lasting more than 5 min
(iii) abnormal drowsiness
(iv) three or more discrete episodes of vomiting
(v) clinical suspicion of non-accidental injury
(vi) post-traumatic seizure with no history of epilepsy
(vii) GC Score less than 14
(viii) Clinical suspicion of an open or depressed skull injury, tense fontanelle or any basal skull fracture (haemotympanum, 'panda' eyes, cerebrospinal fluid leakage from the ear or nose, Battle's sign)
(ix) focal neurological deficit,
(x) if the child is under 1 year, the presence of bruise, swelling or laceration of more than 5 cm on the head
(xi) A dangerous mechanism of injury (high-speed road traffic accident either as pedestrian, cyclist or vehicle occupant, fall from a height of greater than 3 m, high-speed injury from a projectile or an object).

An alternative guideline is the Children's head injury algorithm for the prediction of important clinical events—CHALICE (2006). This is based on the history, physical examination findings and the mechanism of trauma. The criteria are:

8.6.4.1 History

(i) Witnessed LOC of >5 min duration
(ii) History of amnesia (either antegrade or retrograde) of >5 min duration
(iii) Abnormal drowsiness (defined as drowsiness in excess of that expected by the examining doctor)
(iv) ≥3 vomits after head injury (a vomit is defined as a single discrete episode of vomiting)
(v) Suspicion of non accidental injury (defined as any suspicion of non accidental injury by the examining doctor)
(vi) Seizure after head injury in a patient who has no history of epilepsy

8.6.4.2 Examination

(i) GC Score less than 14, or GC Score less than 15 if the patient is younger than one year old
(ii) Suspicion of penetrating or depressed skull injury or tense fontanelle
(iii) Sign of basal skull fracture (defined as evidence of blood or cerebrospinal fluid leakage from the ear or nose and panda eyes)
(iv) Battle's sign, haemotympanum, facial crepitus or serious facial injury)

(v) Positive focal neurological sign (defined as any focal neurological sign, including motor, sensory, coordination or reflex abnormality)
(vi) Presence of bruise, swelling or laceration >5 cm if the patient is younger than one year old

8.6.4.3 Mechanism

(i) High-speed road traffic accident either as a pedestrian, cyclist or occupant (defined as accident with speed >40 miles/h or 64 km/h)
(ii) Fall of more than 3 meters in height
(iii) High-speed injury from a projectile or an object

If any of the above criteria are present, a CT scan is indicated.

8.7 Classification and Common Types of Head (Brain) Injuries

Head injuries are usually classified for management, epidemiological, and research purposes into minor, moderate and severe, based upon the Glasgow coma score.

8.7.1 Head Injury Severity Score

- Minimal—GC Score 15, No LOC or amnesia
- Mild—GC Score 14, or GCS 15 plus brief LOC or impaired alertness /memory
- Moderate—GC Score 9–13 or LOC >5 min or focal neurological deficit
- Severe—GC Score 5–8
- Critical—GC Score 3–4

Head injuries are also classified by their specific injury. Fractures themselves often do not require specific management, but their presence is an indication of severe injury. Contusions and haematoma carry risk by virtue of their 'mass effect'—the larger this is, the greater the chance of developing raised ICP.

8.8 Concussion (Mild Traumatic Brain Injury: MTBI)

Concussion accounts for a considerable number of the "head injuries" that occur worldwide and which present to the emergency department. In many cases diagnosis is usually a clinical one and is often used synonymously with the term "mild traumatic brain injury" (mTBI). However, some authorities believe that concussion represents a subset of mTBI and should be regarded separately and managed differently. This has major implications for patients at risk of repeated injuries, notably

professional sportsmen or women. Multiple definitions of concussion and mTBI currently exist. However they all contain similar characteristics.

In general, concussion is defined as a traumatically induced transient disturbance of neurological function. This is caused by a number of complex pathophysiological processes. It typically manifests as rapid, but transient neurological dysfunction which quickly resolves spontaneously. In some cases symptoms may evolve over several minutes to hours following injury. This type of trauma is thought to represent a very mild form of diffuse axonal injury, with no permanent structural or histological changes to the brain. At the cellular level, it is thought that axonal stretching at the time of injury results in disruption in the neural membrane, allowing leakage of K+ ions and an influx of Ca^{2+}. This results in an 'excitotoxic' cascade and subsequent dysfunction. The patient often appears confused and disorientated. There then follows a cascade of neurometabolic changes, which in conjunction with changes in cerebral perfusion can result in confusion and possibly loss of consciousness, particularly if this involves the reticular activating system. Conventional neuroimaging studies will often show normal findings and over a short period of time resolution of symptoms typically occurs. Although signs and symptoms can be subtle and transient, some can become persistent (such as impaired concentration and balance). This can place affected patients at risk of further injury.

Concussed individuals are therefore more likely to sustain a further concussion. Furthermore, the force required to cause this may be less then in an individual who has never been concussed. All previously concussed patients are now recognised to be at risk of developing "second-impact syndrome." This occurs when further injury is sustained to an incompletely recovered brain. However, this additional injury can result in severe disruption to the autoregulatory mechanisms, leading to vascular engorgement, increased cerebral blood volume and the risk of cerebral oedema.

Common signs and symptoms of a concussion include

(i) Headache
(ii) Sleep disturbances
(iii) Dizziness
(iv) Amnesia
(v) Nausea/vomiting
(vi) Emotional changes/irritability Photophobia
(vii) Difficulty concentrating/confusion
(viii) Photophobia
(ix) Loss of balance
(x) Vision changes

Typically a patient is "knocked out" for several minutes. The patient subsequently wakes up and usually 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. They should be instructed to avoid a second concussion/head injury (if due to sports). Documented advice should be provided, including

risks of complications and when they might need to return. Return to play protocols are now widely used in most contact sports.

Despite its designation as a "mild" head injury, concussions are far from benign. Concussed patients often suffer from short-term symptoms including headache, fatigue, irritability, photosensitivity, confusion and difficulty with memory or sleep. Many of these symptoms are non-specific and cannot be attributed to any structural cause. In some patients symptoms can persist—referred to as 'post-concussion syndrome'. It has also been suggested that there is an increased risk of developing depression and dementia in such patients.

In recent years a lot of research has been published into concussion and mTBI. A wide range of neuroimaging modalities has been reportedly used to investigate the clinical and pathophysiological effects of mild brain injury. These include CT, PET, SPECT, and various MRI techniques (diffusion tensor imaging, MR spectroscopy and functional MRI). Since recovery of neural network connectivity and metabolic imbalances tends to lag behind apparent clinical recovery, these investigations have been used to help determine complete recovery. Clinical testing of visual function may also be a sensitive means to assess minor head injury and screen for sports-related concussion. These include the King Devick (K-D) test, which involves rapid number naming. This has the advantage of being able to be undertaken by non-physicians. Fortunately most individuals recover from concussion within days to weeks, with complete resolution of symptoms and cognitive impairment. General management therefore focuses on mental and physical rest until the individual is symptom-free. Following this, a graduated increase in physical activity is allowed before the individual can return to full activities (including sports).

8.8.1 Second-Impact Syndrome

Second-impact syndrome (SIS) is a rare condition in which rapid brain swelling develops, sometimes catastrophically, following a second concussion before the symptoms of an earlier one have completely resolved. This is distinct to repetitive head injury—where the patient receives a series of minor head injuries over a prolonged period of time and later develops a slow decline in cognitive abilities. This is called chronic traumatic encephalopathy. With SIS the second blow may occur minutes, days or weeks after the initial injury. Neither impact needs to be necessarily severe for this to occur. Second-impact syndrome can be fatal and is often disabling. Its cause is unknown, but it is thought to be as a result of the development of cerebral oedema following loss of cerebral autoregulation. In severe cases neurological deterioration can occur over a short period of time. This can quickly result in loss of consciousness and ultimately respiratory failure.

Most cases of SIS occur in young people, who may have a predisposition. This has important implications in sports enthusiasts and professional athletes—current guidelines recommend not to return to play before symptoms from any head injury have fully resolved. Patients who participate in contact sports (such as boxing, soccer, American football, rugby, ice hockey and professional wrestling) are not

surprisingly at increased risk. When SIS is diagnosed, management focuses on the control of cerebral oedema and any raised intracranial pressure. Depending on the severity of symptoms this may require full neuro intensive care.

8.9 Skull Fractures

Skull fractures are generally classified in three ways (i) by pattern (linear, comminuted, depressed), (ii) by anatomical location (vault convexity, base) and (iii) by skin integrity (open, closed). Very rarely do they require surgical repair. With open and depressed fractures, surgical debridement and elevation may be required. Otherwise, all fractures need a period of observation in order to identify the early development of intracranial complications.

8.10 Linear Fractures

Linear fractures are the most common type of skull fracture and usually occur over the lateral convexities of the skull. When compared to vascular markings and sutures, fractures are usually relatively straight with well-defined margins and are usually several centimetres long. The edges of long fractures might be separated by several millimetres. Skull fractures heal slowly and therefore it might not be possible to determine how old a fracture is from its appearance. This has important forensics implications. However, new fractures 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 (such as bleeding from the ear, battle's sign, CSF rhinorrhoea or panda eyes).

The main significance of linear skull fractures is that they signify an increased risk of developing an intracranial haematoma. Linear skull fractures that involve the squamous portion of the temporal bone can be accompanied by a tear of the middle meningeal artery, resulting in an extradural haematoma. They can also cause facial nerve injury. The fractures themselves do not require stabilisation or treatment when the scalp is closed. They are managed just as a minor head injury without a fracture—observation and basic care. Patients should be CT scanned and can be discharged after 12–24 hours if asymptomatic (Fig. 8.1).

8.10.1 Growing Skull Fracture

This is a rare complication of a linear skull fracture in young children (usually under 2 or 3 years). Here, the dura is torn under the fracture, allowing an expanding pouch of arachnoid to pass through the dural gap and skull fracture. This acts as a one-way valve, trapping cerebrospinal fluid and causing progressive erosion and expansion of the fracture edges. As a result the fracture enlarges. In addition, the pulsating,

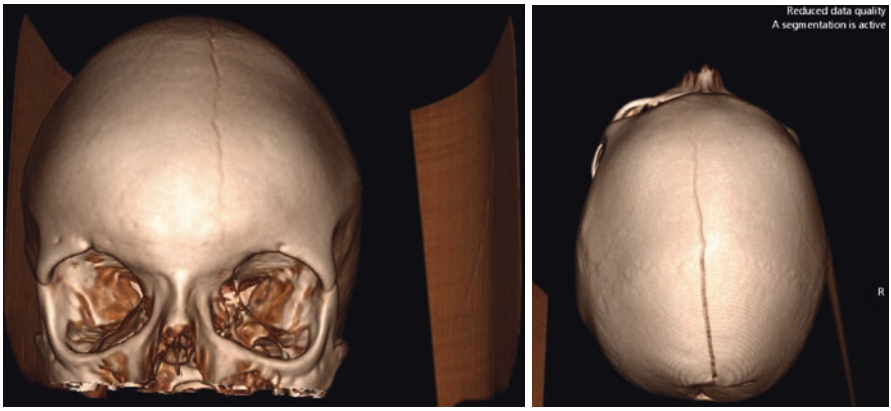


Fig. 8.1 Linear fractures

growing brain places tensile forces across the edges of any unrepaired dural laceration. Occasionally part of the brain may herniate through the skull defect, resulting in neurological deficit. Therefore these fractures must be carefully followed up and if necessary surgically repaired with closure of the dura and replacement or repair of the bony defect. Growing skull fractures are sometimes referred to as Leptomeningeal cysts. However this is a misnomer, as it is not a cyst, but an extension of an encephalomalacia.

8.10.2 Depressed Fractures

These are usually round, with linear fracture lines radiating out from the centre, resulting in multiple fragments. They usually result from a blunt force from an object with a relatively small surface area, such as a hammer. In some cases, impacted fragments may become "wedged" into position by the surrounding bone edges. All fractures should be urgently evaluated further by CT-scanning. Depressed fractures are often associated with contusions of the underlying brain and an overlying scalp laceration. If the dura is torn there is a significant risk of intracranial contamination. Although often open in adults, depressed fractures can be closed in young children ("ping-pong ball" fracture). Fractures should be considered for elevation if they are depressed by an amount greater than the skull thickness. The aims of treatment are to i) relieve pressure on the brain, ii) minimise the risk of epilepsy, iii) correct any cosmetic deformity and iv) preventing infection (if open). Treatment involves wound toilet and removal of any foreign bodies, ideally within 24 hours. Dural closure is essential to prevent CSF leaks from the wound and (in children) brain herniation into the fracture area. The risk of long-term epilepsy is believed to be increased if there is a dural tear, intracranial haematoma, over 24 hours of

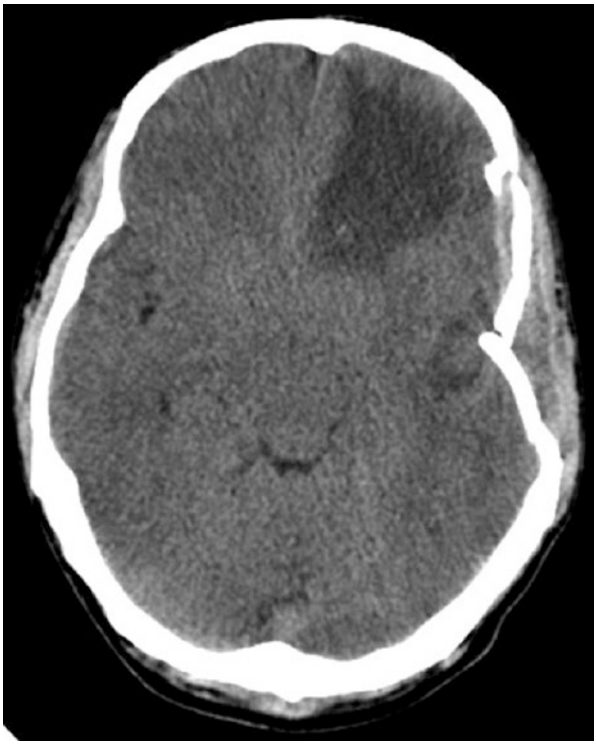


Fig. 8.2 Depressed fracture

post-traumatic amnesia, or early seizures. Closed minimally depressed fractures can otherwise be managed conservatively. The greatest cosmetic deformity occurs in the forehead. Exploration is more urgent for a large, depressed fracture when imaging suggests dural laceration, brain penetration, simultaneous frontal sinus fracture, mass effect, or underlying haematoma (Figs. 8.2 and 8.3).

8.10.3 Depressed Fractures Over Dural Sinuses

Depressed skull fractures over a venous sinus require careful consideration. Elevation of these fractures can result in massive blood loss if a depressed fragment has been plugging a tear in the sinus wall. There are thus two options in management—(i) the fracture can either be carefully elevated, whilst attempting to gain control of the venous sinus and preparing for significant blood loss, or (ii) if the fracture site is not grossly contaminated and there is no intracranial hypertension it can be managed by local débridement and irrigation. This is followed by serial scans to assess for brain abscess (Figs. 8.4, 8.5, 8.6).

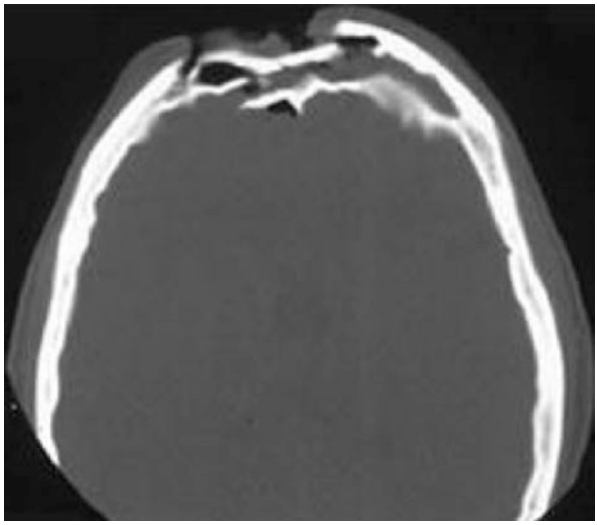


Fig. 8.3 Depressed fracture involving frontal sinus

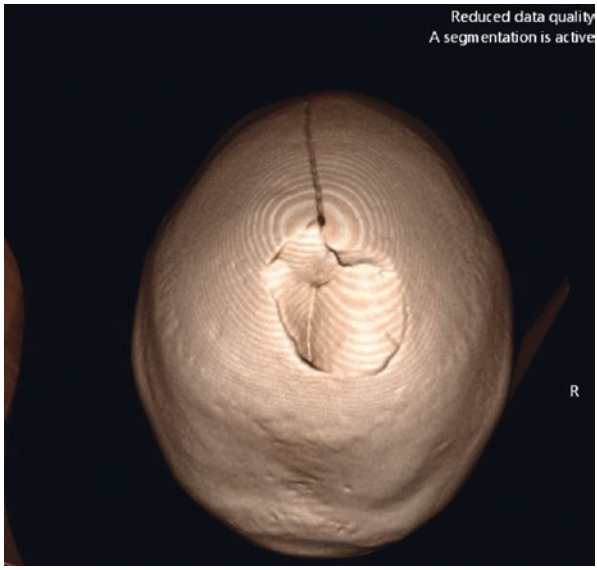


Fig. 8.4 Depressed fractures

8.10.4 Basal Skull Fractures

These can occur following severe blunt trauma to the forehead or the occiput. They are most common in the anterior skull base, often involving the cribriform plate (disrupting the olfactory nerves) and resulting in CSF rhinorrhoea. More posterior basal skull fractures (involving the middle and posterior cranial fossae) may extend through the petrous bone and internal auditory canal, thereby damaging the acoustic and the facial nerves. A greater degree of force is required to produce fractures of the middle or posterior cranial fossa bases than for a vault fracture. The anterior cranial fossa however is relatively fragile. Basal skull fractures are difficult to see on

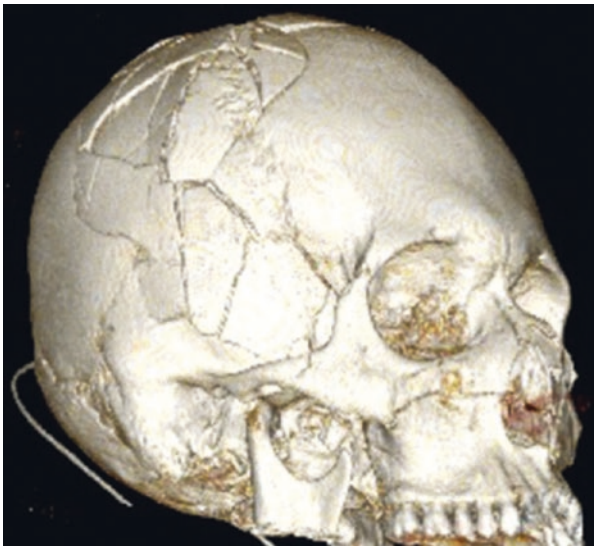


Fig. 8.5 Extensive skull fractures

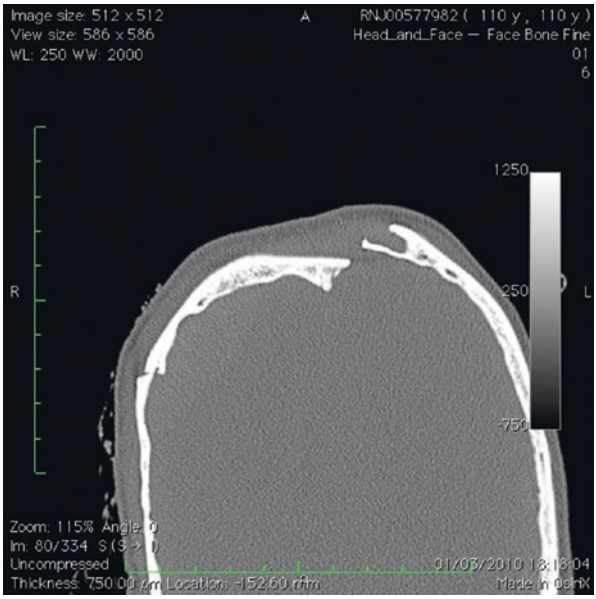


Fig. 8.6 Extensive depressed frontal fracture involving frontal air sinus

plain skull x-ray and are usually diagnosed initially on clinical grounds. Pneumocephalus should always raise suspicion for a fracture. A lateral cervical spine view may occasionally show fluid in the sphenoid sinus. CT scan is still required to confirm the presence of a fracture and assess for any intracranial injury.

With basal skull fractures the underlying dura is often torn, possibly resulting in leakage of CSF from the nose or ear. These communications provide a route for bacteria to enter the intracranial space. Basal fractures without a CSF leak are managed in a similar way to vault fractures. CSF otorrhoea usually settles

conservatively. CSF rhinorrhoea also frequently settles, but the fistula might reopen later with a risk of delayed meningitis, sometimes years later. Surgical repair should therefore be considered and discussed with the patient, especially if a large defect is seen on a coronal CT scan. Prophylactic antibiotics are not usually indicated as they have not been shown to prevent meningitis (antibiotics penetrate poorly into CSF in the absence of infection). They can also lead to colonisation by resistant organisms. The pneumococcal vaccine is indicated in such cases.

With petrous bone fractures, the fracture can run either longitudinal to the bone or transverse across it. This is discussed further in a chapter on the ear. Longitudinal fractures are more common and often involve the tympanic membrane or external ear canal, resulting in otorrhea. Transverse fractures result from higher-energy impacts and can damage the middle ear ossicles or the facial and vestibulocochlear nerves. This can result in facial palsy and deafness. Fractures that pass close to, or involve the carotid canal can also be associated with injury to the carotid artery (dissection, occlusion, pseudoaneurysm formation). These can be asymptomatic or result in life-threatening bleeding or stroke. Traumatic internal carotid artery dissection (TICAD) is a form of blunt cerebrovascular injury (BCVI), commonly caused by motor vehicle accident. In young adults this is a common cause of cerebral ischaemic stroke, accounting for approximately 20% of cases. Involvement of the petrous segment of the carotid canal should therefore be carefully evaluated in all petrous bone fractures. Further imaging with CT angiography and venography (CTA, CTV) may be required.

Complications associated with basilar skull fractures include

(i) Cerebrospinal fluid leak/fistula
(ii) Meningitis
(iii) Pneumocephalus
(iv) Cavernous sinus thrombosis
(v) Carotid dissection, pseudoaneurysm or thrombosis
(vi) Carotid-cavernous fistula
(vii) Injury to cranial nerves III, IV, VI, VII and VIII

8.10.5 Tension Pneumocephalus

Tension pneumocephalus (TP) is mentioned here as it is almost exclusively related to fractures of the skull base. This is a very rare condition in which there is progressive build-up of air within the cranial cavity, due to a one way valve-effect at the site of the fracture. Pneumocephalus can also arise following surgery, tumours and infections. Positive pressure ventilation via bag and mask, repeating nose blowing by the patient and the historical use of nitrous oxide (N_2O) anaesthesia are all possible causes. Although in most cases, pneumocephalus is generally asymptomatic, when the gas is of sufficient volume it will produce a mass effect resulting in symptoms of raised intracranial pressure. Depending upon its site clinical features may mimic that of an intracranial bleed. Diagnosis can only be made on CT scan. The radiological sign that is most widely reported as specific to tension pneumocephalus

is the 'Mount Fuji' sign,—the presence of subdural free air results in compression and separation of the frontal lobes. It is said that the displaced frontal lobes and the widening of the interhemispheric fissure simulates the silhouette of Mount Fuji! Management of tension pneumocephalus requires the urgent relief of pressure (craniotomy, burr holes and rarely needle aspiration) (Figs. 8.7 and 8.8).

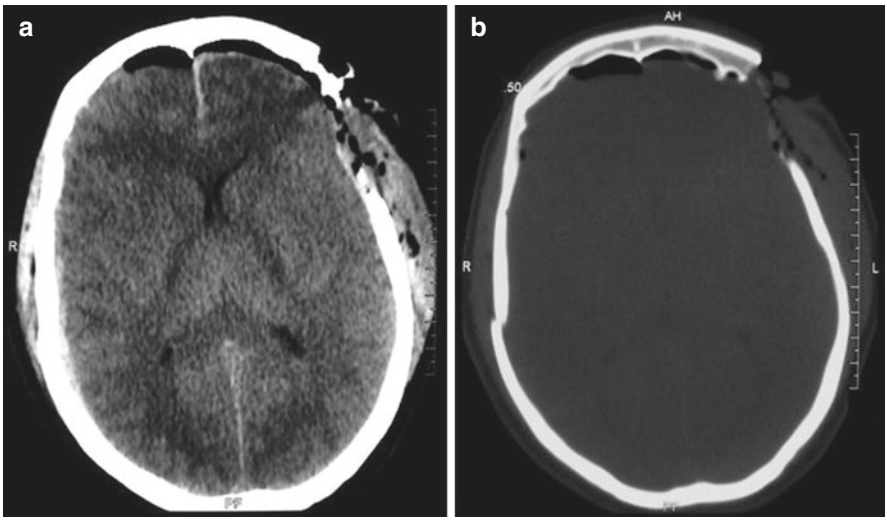


Fig. 8.7 Axial cranial CT scan on parenchymatous (**a**) and bone (**b**) windows revealing an open skull fracture in the left frontal area. There is a posttraumatic brain edema with a pneumocephalus in the frontal convexity

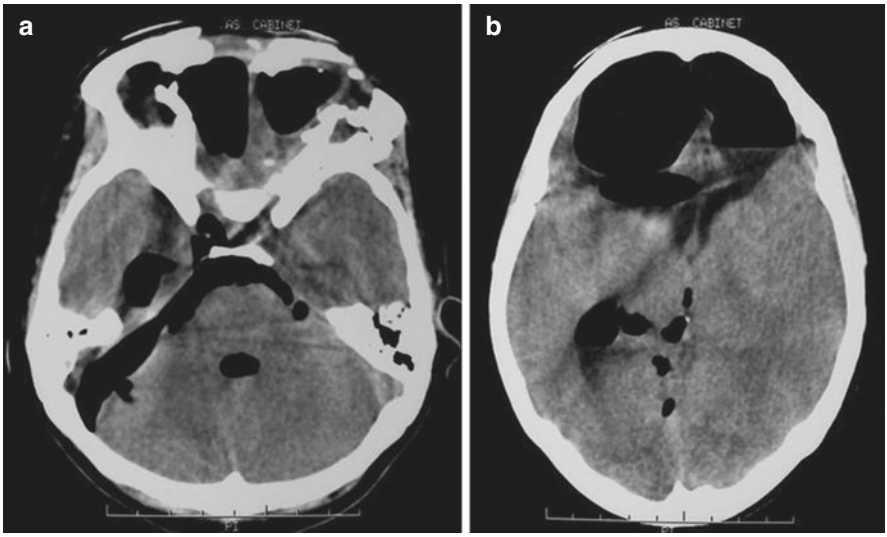


Fig. 8.8 (**a**, **b**), Axial CT scans resealing multiple cranio-orbital fractures and extensive pneumocephalus. This patient developed a post-traumatic bacterial meningitis

8.10.6 Orbital Roof Fractures

These fractures often require multidisciplinary assessment. Orbital roof fractures can occur in isolation, or associated with more widespread fractures involving the frontal bone and the frontal sinus. Fragments of the orbital roof may become displaced into the orbit and associated with swelling or injury to the orbital contents. They can also be displaced intracranially. Both can be associated with dural tears. Excessive production of tears from the eye, or an associated laceration should therefore raise concerns for oculorrhea. Distinction should be made between isolated and non-isolated orbital roof fractures. In non-isolated cases, early repair may be indicated, particularly if the fracture extends into the frontal sinus. Management of these fractures is controversial, but severe injuries put the patient at risk of meningitis, encephalocele, and brain abscess. With isolated orbital roof fractures, management depends on the degree of displacement of the bony fragment and any adverse effect it has on the function of the globe. In the absence of a functional deficit these injuries can often be left, but the patient should be reviewed regularly for ocular and neurological complications (such as exophthalmos, diplopia, or painful pulsation). Distinction should also be made between children and adults. In general, it is advised to be even more conservative in children to avoid disturbing growth potential.

8.10.7 Frontal Sinus Fractures

Frontal sinus fractures can result in significant morbidity if not managed correctly. However management is still controversial, particularly with minimally displaced and otherwise symptom-free fractures of the posterior sinus wall (posterior 'table'). Fractures here can be accompanied with dural tears, increasing the risks of intracranial infection, but this is not always the case. Those fractures which prevent free drainage of the sinus (often seen in association with nasoethmoidal fractures) can result in both mucocele formation and infection. Management often involves multiple specialties, such as neurosurgery, maxillofacial surgery and ENT. Treatment options include observation, obliteration of the sinus and cranialisation (ie remove all of the sinus-forming mucosa, incorporating the sinus space into the intracranial space). Despite this, in the first instance, these should be regarded as skull fractures and assessed accordingly (Figs. 8.9, 8.10, 8.11, 8.12, 8.13, 8.14).

8.11 Intracranial Haematomas

It is generally accepted that the risk of a patient developing an intracranial haematoma, is related to the (i) patient's level of consciousness and (ii) the presence or absence of a skull fracture. Rapid deterioration in the Glasgow coma scale in the

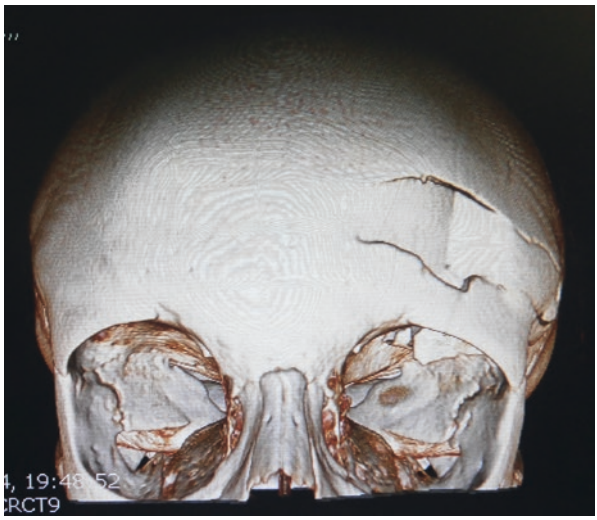


Fig. 8.9 Depressed fratcure which may extend towards frontal sinus

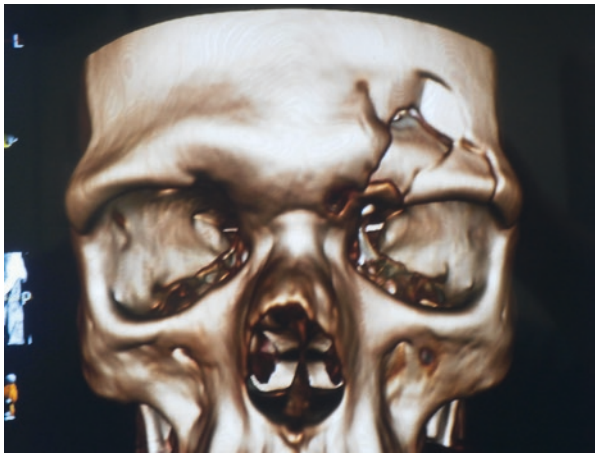


Fig. 8.10 Depressed frontal sinus fracture

presence of the skull fracture would be highly suggestive of an intracranial haematoma. Several types of haematoma are known to develop, these can occur in isolation or in combination.

8.12 Cerebral Contusions

Contusions (bruising) often occur at the poles of the brain due to a contra-coup type injury (that is, the contusion is located on the opposite site of impact). As the head is thrown in one direction and then recoils back, the untethered brain (floating in CSF) continues its motion, thereby striking the adjacent bone. These injuries can be

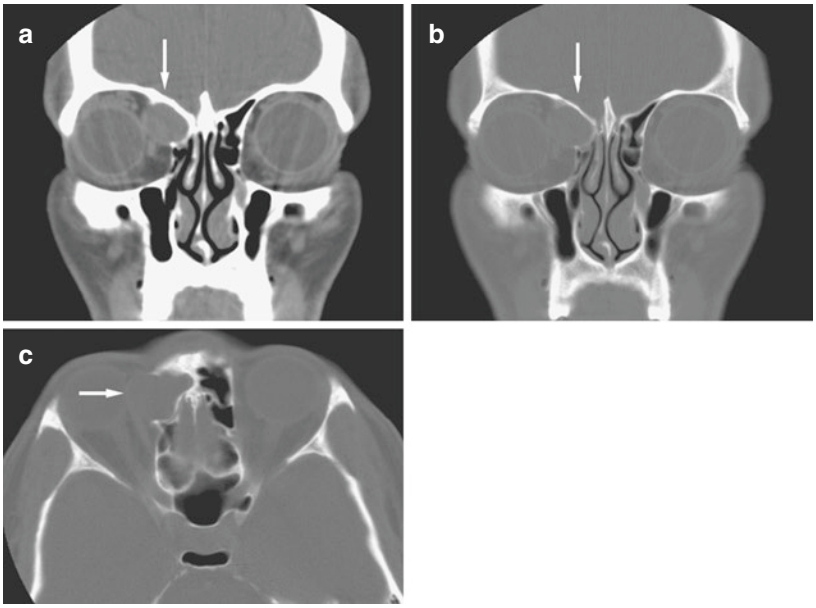


Fig. 8.11 Frontal sinus mucocele; 50-year-old male with diplopia and right eye down and laterally deviated. (**a**) Coronal CT image, soft-tissue window, shows ovoid well-defined soft tissue mass in right orbit (*arrow*) with displacement of globe laterally down. (**b**) Coronal CT image, bone window, shows bone expansion and absent frontal sinus wall against orbit (*arrow*), but otherwise normal bone structures. (**c**) Axial CT image shows well-defined delineation of bone expansion (*arrow*)

associated with marked oedema and raised intracranial pressure. Small contusions are usually treated conservatively, with close observation of the patients GCS. However lobectomy or evacuation of an intracerebral haematoma may be necessary with larger contusions if the ICP cannot be controlled medically.

8.13 Extradural Haematomas (EDH)

Extradural (or epidural) haematomas (EDH) occurs about 4% of TBI patients. They are usually associated with a skull fracture or separation across a suture (diastasis). These represents one of the most urgent neurosurgical lesions, as severe brain compression can develop rapidly from high pressure arterial bleeding, necessitating rapid evacuation.

The majority of EDHs are located in the temporal or parietal regions, following a tear of the middle meningeal artery. They can also occur in the frontal and occipital regions and (rarely) in the posterior fossa (where obstruction of the dural venous sinuses can occasionally result in venous infarction of the brain). Generally speaking, EDH are rare in young children. This is because their skulls are relatively soft and pliable and fracture edges are not usually sharp enough to lacerate the artery. At

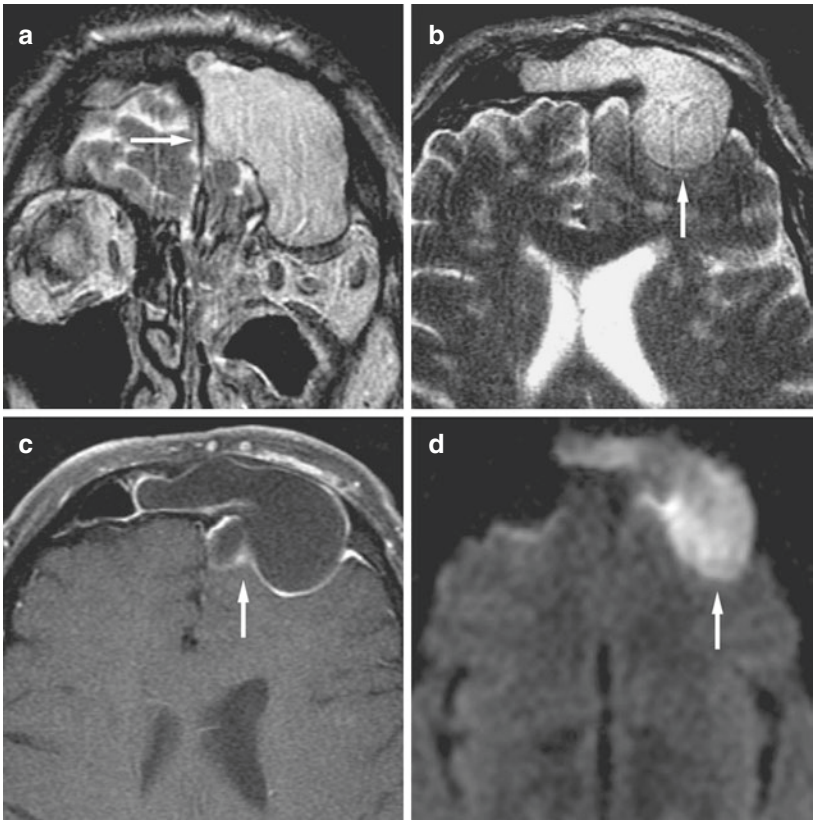
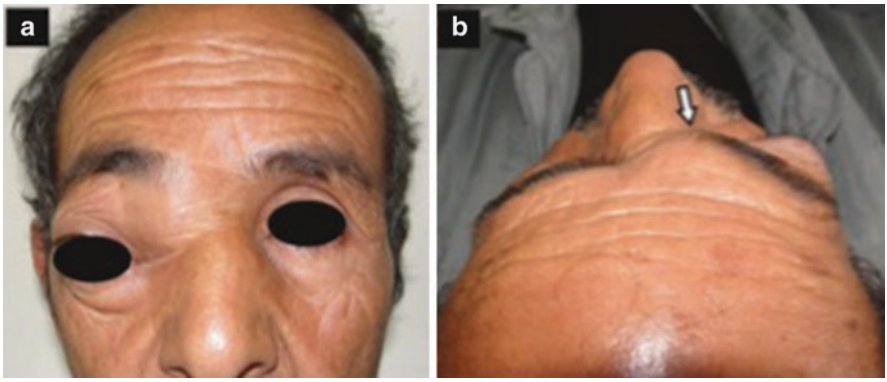


Fig. 8.12 Frontal sinus mucocele; 60-year-old male presented with headache and diplopia. (**a**) Coronal T2-weighted MRI shows intermediate signal large expansive mucocele (*arrow*). (**b**) Axial T2-weighted MRI shows expansion into cranial fossa with displacement of left frontal lobe (*arrow*). (**c**) Axial T1-weighted post-Gd MRI shows no enhancement except in thin peripheral rim (*arrow*). (**d**) Axial diffusion-weighted MRI shows high signal (*arrow*)

the other end of the age spectrum, EDH is also rare in the elderly, because the dura is more adherent to the skull. Any bleeding is thus more likely to be contained whilst the artery thromboses. The typical history of EDH is one in which the patient presents with delayed deterioration. Following the initial injury the patient may be concussed but soon makes a good recovery. However as the dura is slowly stripped away from the skull initial pressure effects are compensated for as described in the Monroe-Kellie doctrine. Although this interim period of recovery, referred to as the "lucid interval" is well known, patients rarely make a complete recovery and often complain of symptoms. It is for this reason that ongoing symptoms following a head injury need further evaluation.

Spread of an extradural haematoma is limited by the suture lines of the skull, where the dura is very adherent. Because an extradural space does not normally exist, the clot must strip the dura from the inner table of the skull as it enlarges. As



Figs. 8.13 and 8.14 This 55-year old man had a chronic right proptosis with diplopia and recurrent episodes of fever. Clinical pictures show frontal (**a**) and superior (**b**) views. Note facial deformity and frontal swelling (*arrow*)

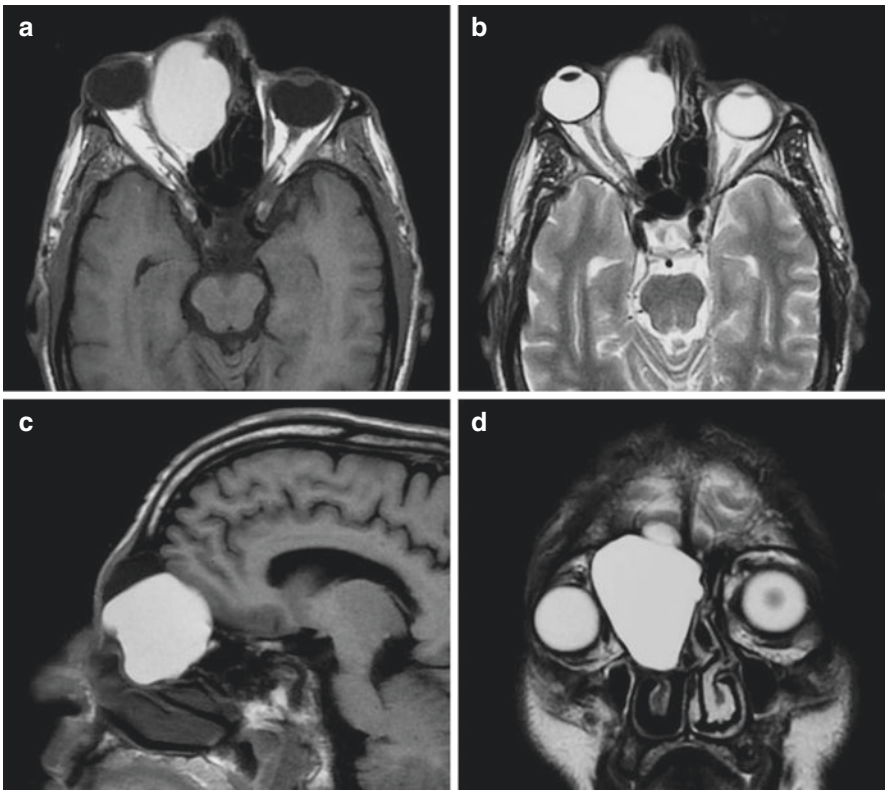


Fig. 8.14 Axial craniofacial T1-weighted (**a**) and T2-weighted (**b**) MR images. Sagittal T1-weighted (**c**) and coronal T2-weighted (**d**) MR images show the right frontoethmodial cystic lesion with intraorbital extension

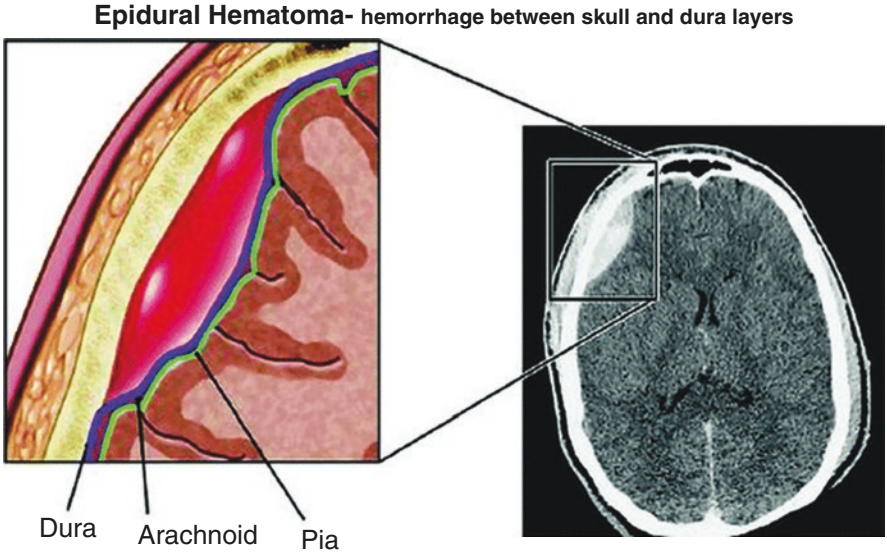


Fig. 8.15 Epidural hematoma - anatomical and CT scan correlates. From Freeman and Aguilar [191]. Reproduced with permission from Elsevier

a result, extradural haematomas appear as biconvex (lens) shaped opacities on CT and are mostly high density. Lower density areas within them are due to unclotted blood (Figs. 8.15, 8.16, 8.17, 8.18).

Generally speaking, the prognosis is very good if they are treated early enough. Very small extradural haematomas with minimal symptoms can often be left (although they should be discussed with the local neurosurgical unit). Larger haematomas and those with evidence of neurological deficit require urgent surgical evacuation. The higher the GCS at the time of surgery, the better the prognosis.

8.14 Subdural Haematomas

Subdural haematomas (SDH) are seen in about 25% of all comatose patients following TBI. Compared to isolated EDH, the degree of underlying brain damage is more severe. The SDH develops between the surface of the brain and the inner surface of the dura (hence their name) as a result of tearing of the bridging veins over the cortical surface of the brain, or disruption of the dural venous sinuses or their tributaries. Typically the haematoma spreads over most of the cerebral convexity of one hemisphere, initially as a relatively thin-layer. Cerebral oedema and contusions are also commonly associated (Fig. 8.19).

Subdural haematomas are classified as acute, subacute, or chronic, each having a characteristic appearance on computed tomography (CT). An acute subdural

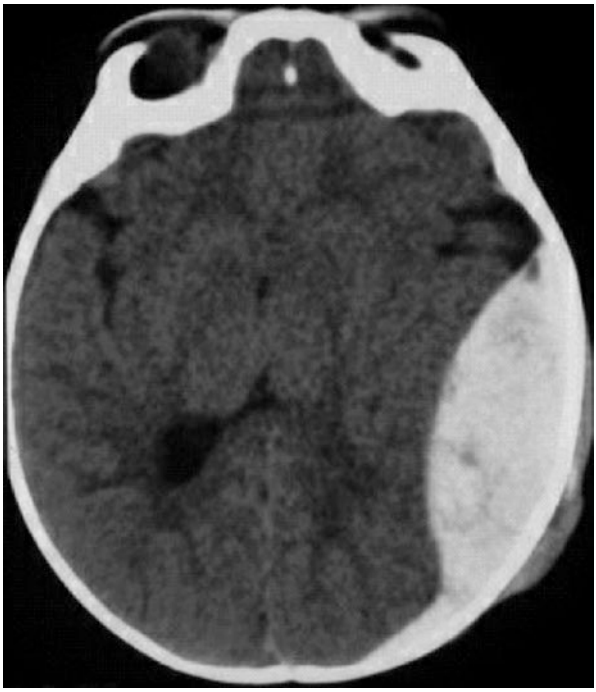


Fig. 8.16 Left extradural haematoma

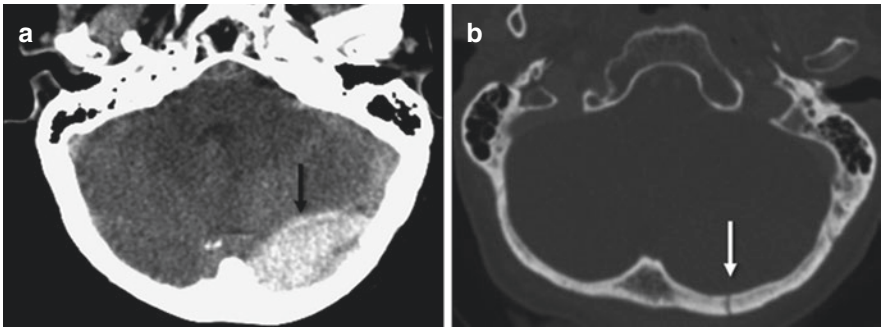


Fig. 8.17 (**a**) CT - Axial soft tissue window through the posterior fossa demonstrating a large epidural hematoma (*black arrow*), causing mass effect on the fourth ventricle. (**b**) Bone window CT demonstrates underlying occipital bone fracture (*arrow*). From Baugnon and Hudgins [133]. Reproduced with permission from Elsevier

haematoma, identified within 72 hours of injury, generally appears on CT as a high-density, homogeneous crescent-shaped mass paralleling the skull. However, up to 10% of acute subdural haematomas may be isodense with brain because of the low haemoglobin content. Chronic haematomas are hypodense relative to the brain. Interestingly there have been rare reports of intracranial subdural haematoma (SDH) that have migrated into the spine, resulting in radiculopathy.

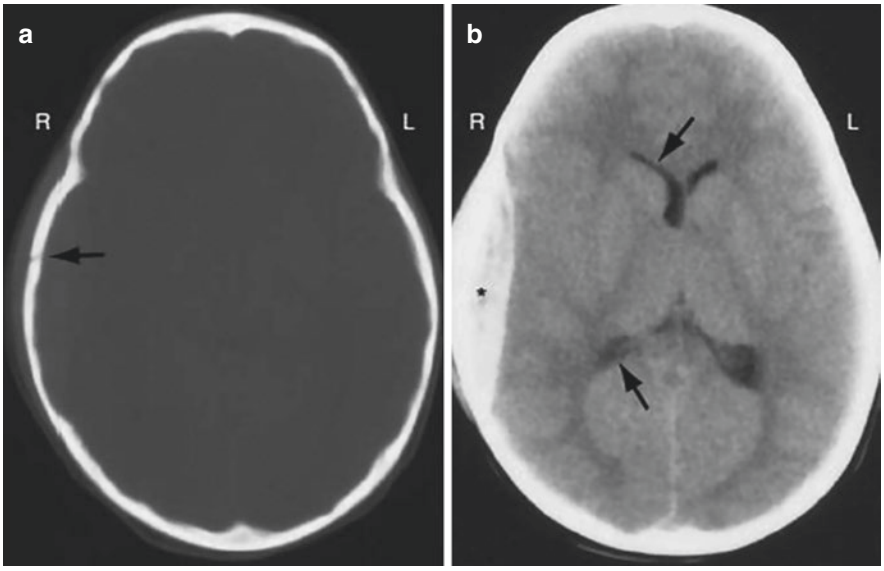


Fig. 8.18 Epidural hematoma in a 3-year-old girl following head injury in MVA. (**a**) CT set to show bony details reveals a fracture of the right temporal bone (*arrow*), (**b**) CT set to reveal soft tissue details reveals a lens-shaped hyperdensity (*) and compression of the right lateral ventricle (*arrows*). From Nolte [168]. sixth Ed. Reproduced with permission from Elsevier

8.14.1 Acute Subdural Haematoma

This may occur as a result of tearing of the bridging veins between the brain and skull. If identified early and treated promptly before significant 'mass effect' occurs, the prognosis can be good. However in some cases a laceration of the brain surface (burst lobe) may also occur. This has a considerably worse prognosis, due to the associated primary injury to the brain itself. Any expansion of the subdural haematoma will increase the likelihood of secondary brain injury. Bleeding can extend over a wide area of the lateral cortical surface. Unlike EDH, skull fractures are not aways present. Acute subdural haematoma is more common than EDH, occurring in about 30% of severely head injured patients. They appear crescent shaped on CT scan as the blood is not contained by the dura and therefore spreads across the surface of the brain. Mortality rate in patients with subdural haematomas is high, especially if associated with midline shift, the presence of underlying brain swelling or contusions, obliteration of the basal cisterns and the presence of traumatic SAH (Fig. 8.20).

Symptoms usually appear soon after the head injury, approximately 24–48 hours later. There may be a history of loss of consciousness. Patients often feel drowsy, and nauseous, or have a very bad headache. Confusion, slowing of the speech and limb weakness make quickly develop. Thin acute subdural haematomas can be treated conservatively, but large ones need prompt evacuation via a craniotomy. In

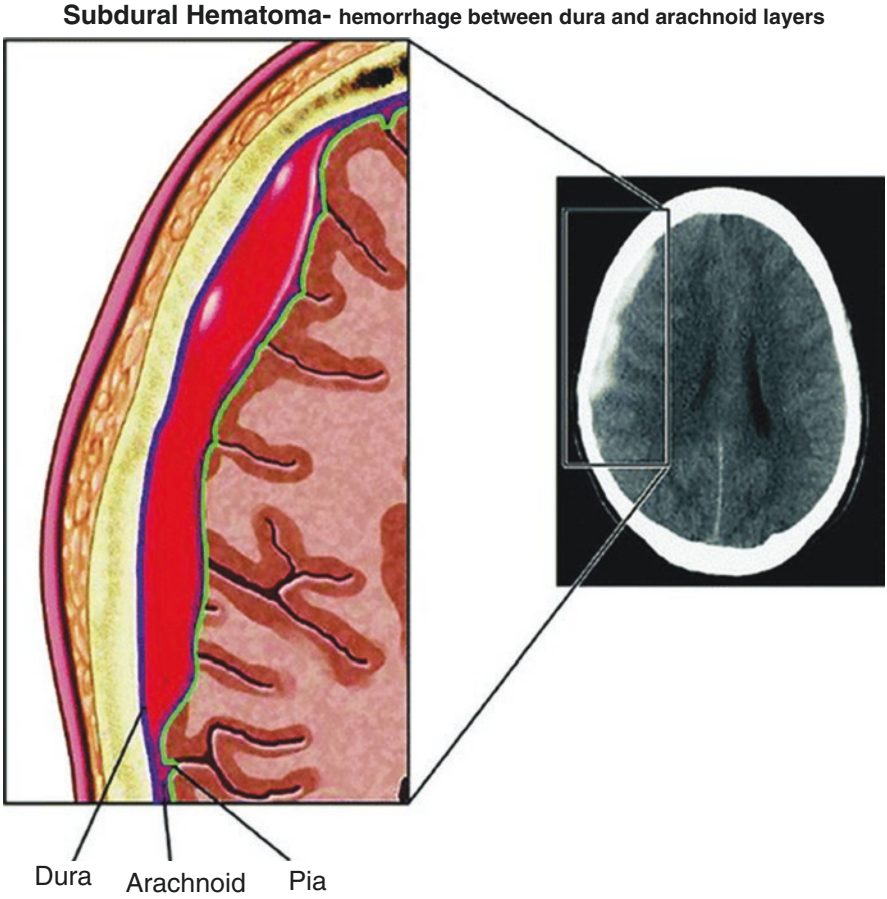


Fig. 8.19 Subdural hematoma - anatomical and CT scan correlates. From Freeman and Aguilar [191]. Reproduced with permission from Elsevier

many cases by the time the patient has reached the operating theatre the blood has clotted and become jelly-like in consistency. This is now too viscous to drain via a relatively small burr hole. In the subacute form, symptoms will be similar to the acute form but will only become apparent after 3–7 days.

8.14.2 Chronic Subdural Haematomas

Chronic subdural haematomas are thought to occur as a result of repeated episodes of minor bleeding that develop following a minor head injury. The injury itself may have occurred several weeks or even months previously and often is often trivial that it cannot be remembered in about half of the patients. This particular haematoma is commonly seen in the elderly who may already have a

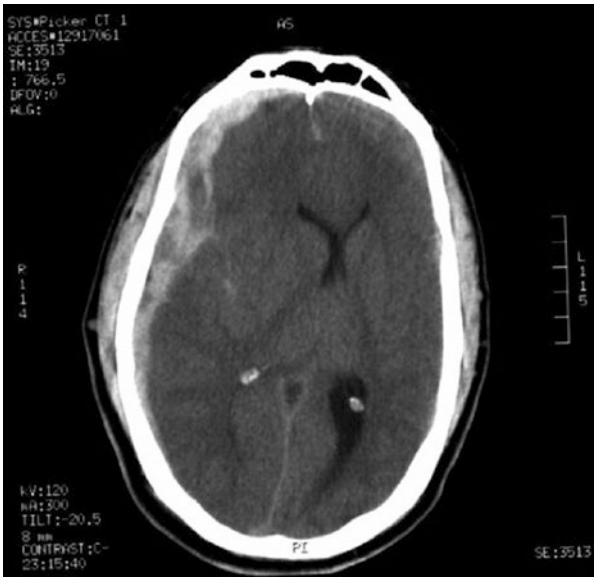


Fig. 8.20 Right acute subdural haematoma

degree of brain atrophy. Pre-existing confusion or dementia can therefore complicate the clinical picture. Patients are prone to falls, many of which may not even be recognised. Although common in the elderly, subdural haematoma can occur at any age. Other risk factors include anticoagulants/anti-platelet drugs, bleeding diatheses, alcohol abuse, epilepsy and patients on haemodialysis. In people over the age of 60 the blood vessels around the brain become more friable. In addition, the brain shrinks a little, stretching the vessels. This makes them more susceptible to tears and more likely to continue to bleed. Alcohol misuse can affect the clotting of the blood. It can also result in shrinking of the brain. Alcoholics are also more likely to fall and sustain a head injury. In babies a subdural haematoma can be caused by tearing of veins in the subdural space. This may be caused by both accidental and non-accidental injury. Chronic subdural haematoma can also occur in babies, most often following non-accidental injuries (Fig. 8.21).

Patients can present with a wide variety of symptoms, including headaches, reduced consciousness and focal neurological deficit. Therefore consider this diagnosis in all elderly patients who presents with intermittent confusion or symptoms suggestive of TIA. A history of trauma is helpful, but may not always be remembered. A high index of suspicion is therefore required. Symptoms may not usually appear until about 2–3 weeks after the initial head injury. In some patients it may be months later. An inflammatory process occurs and membranes can develop over time and there is some evidence that steroids can be used to treat these which may be helpful in those unfit for surgery. Symptoms tend to progress gradually. There is often loss of appetite, nausea and/or vomiting. Headache may subsequently develop, which becomes progressively more severe. The patient may also develop increasing

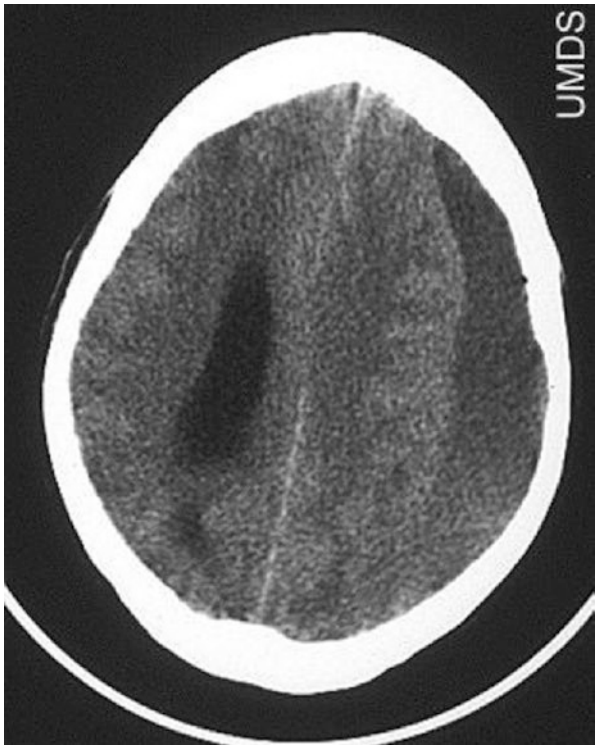


Fig. 8.21 Chronic subdural haematoma

weakness of the limbs on one side of the body, speech difficulties, increasing drowsiness, confusion or personality changes. Sometimes a seizure can occur.

Chronic subdural haematomas can be treated by burr hole drainage as the blood is usually liquefied. They generally have a good prognosis but they can recur, especially in patients with a coagulation disorder.

8.14.3 Traumatic Subarachnoid Haemorrhage

This often results from tearing of the corticomeningeal vessels. Though common after severe TBI, subarachnoid haemorrhage does not produce a haematoma or mass effect. However, it may be associated with an increased risk for posttraumatic vasospasm, which can adversely affect cerebral perfusion and prognosis (Fig. 8.22).

8.14.4 Subdural Hygroma

This is a collection of blood-free fluid in the subdural space. Most are believed to be derived from chronic subdural haematomas. They are commonly seen in elderly patients but can occur in children after an infection. Another cause of subdural

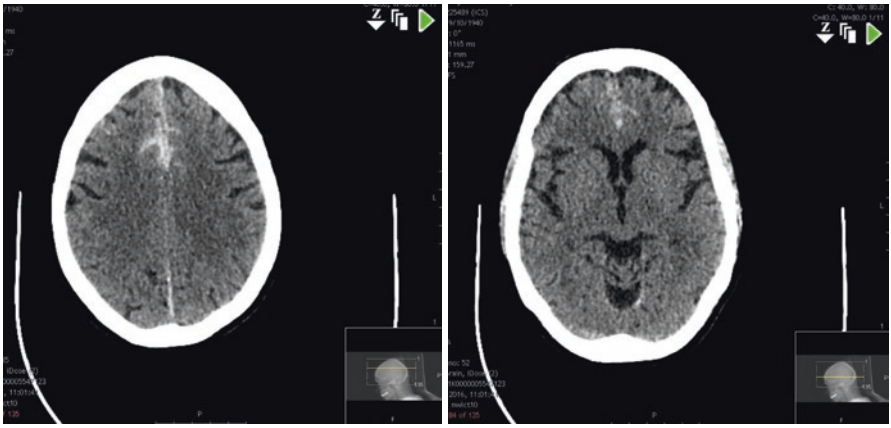


Fig. 8.22 Traumatic subarachnoid haemorrhage

hygroma is a sudden decrease in CSF pressure following placement of a ventricular shunt. This can lead to leakage of CSF into the subdural space.

8.15 Brain Herniation

Brain herniation is a potentially deadly consequence of very high pressure within the skull. Part of the brain is squeezed across the internal rigid structures. In severe cases the brain can herniate across the falx cerebri, tentorium cerebelli, and even through the foramen magnum. Herniation can be caused by a number of conditions that result in a "mass effect" and significant increase in the intracranial pressure. Whilst this is most commonly seen following trauma, other causes include spontaneous intracranial haemorrhage (stroke) and an intracranial tumour. Patients are usually very drowsy or unconscious and quickly develop abnormal limb posturing. One or both pupils may become dilated and fail to respond to light. Vomiting can also occur due to compression of the vomiting centre in the medulla oblongata. Depending on the precise site of herniation, patients may develop limb weakness on the same side as the lesion causing the shift, or on the side opposite. Damage to the midbrain (containing the reticular activating network) quickly results in coma. Eventually compression on the cardio-respiratory centres in the medulla oblongata results in respiratory and cardiac arrest.

Herniation can also occur in the absence of a significantly raised ICP, when mass lesions occur at the borders of brain compartments. In these circumstances local pressure is increased at the site of the herniation, but this pressure is not transmitted throughout the rest of the brain. Therefore the ICP may remain within normal limits and the patient can otherwise be symptom-free. However in these cases the herniated tissue is still under pressure preventing perfusion. This can result in localised infarction which can be fatal unless rapidly diagnosed and treated.

Brain herniation is usually described as either supratentorial or infratentorial. This is based on the site of herniation relative to the tentorium cerebella—the dural fold which separates the cerebral hemispheres from the cerebellum. Supratentorial herniations occur above the tentorial notch. These include.

(i) Uncal (transtentorial). Here the innermost part of the temporal lobe (uncus), is displaced towards the tentorium, putting pressure on the midbrain. This can result in a CNIII palsy. Occasionally there may be compression of the contralateral cerebral crus, which contains descending corticospinal tract fibers. This leads to ipsilateral hemiparesis, a so called "false" localising sign. Unless decompression is quickly achieved, increasing pressure on the brainstem will result in a decorticate posture, respiratory depression, bradycardia and finally death.
(ii) Central. In central herniation, parts of the temporal lobes on both sides of the cerebral hemispheres are squeezed through a notch in the tentorium cerebelli. This is often rapidly fatal.
(iii) Cingulate (subfalcine/transfalcine). The innermost part of the frontal lobe is displaced under part of the falx cerebri. This can occur when one hemisphere swells. This does not put as much pressure on the brainstem but may interfere with circulation of the frontal lobes, or it may progress to central herniation.
(iv) Transcalvarial. Here, the brain is displaced through a fracture or the gap created following a craniectomy.
(v) Tectal (posterior)

Infratentorial herniations include

(i) Upward herniations. Increased pressure in the posterior fossa can cause the cerebellum to pass upwards through the tentorial notch (cerebellar herniation). The midbrain is also pushed through the notch. This is also known as a transtentorial herniation.
(ii) Downward herniations (cerebellar/tonsillar). In tonsillar herniation, also referred to as "coning", the cerebellar tonsils are displaced downward through the foramen magnum resulting in compression on the lower brainstem and upper cervical spinal cord. This can result in major dysfunction to the respiratory and cardiac centres. Following trauma, patients are usually unconscious at this stage. However with other pathologies they may still be awake and may complain of intractable headache and neck stiffness.

Tonsillar herniation of the cerebellum can also occur in a non-traumatic context. This is known as a Chiari malformation (formerly an Arnold-Chiari malformation). Four types have been described as a result of very different disease processes and with different symptoms and prognosis. These can be found in asymptomatic patients as an incidental finding, or can rarely present as a life-threatening emergency. Suspected causes of tonsillar herniation include (i) malformation of the posterior fossa with limited space for the cerebellum, (ii)

hydrocephalus which displaces the tonsils down, (iii) dural tension, pulling the brain caudally. Connective tissue disorders, such as Ehlers Danlos Syndrome, have also been associated with tonsillar descent. Chiari malformation is now being diagnosed more frequently by radiologists, as a result of the increased use of MRI, especially upright MRI which is more sensitive in detecting this condition. Cerebellar tonsillar ectopia (CTE) is a term used by radiologists to describe cerebellar tonsils that are "low lying" but that do not meet the radiographic criteria for a Chiari malformation (the cerebellar tonsils lie at least 5 mm below the level of the foramen magnum).

8.16 Penetrating Head Injuries

A penetrating head injury is serious an open injury in which the dura mater has been breached. As such, this places the patient at high risk of intracranial infection. Common causes mostly include high-velocity projectiles, but these can also occur from objects such as knives, hammers, humane animal slaughter devices ('captive bolt') or tearing of the dura by depressed skull fractures. Gunshot wounds to the head are often fatal as shockwaves from the projectile causes damage to the brain beyond its path (Fig. 8.23). In many respects these injuries are similar to closed head injuries in that the patients will experience primary brain injury followed by secondary brain injury, however with these injuries patients also have a significant increased incidence of infection. Initial management of penetrating head injuries is the same as closed injuries although antibiotics should also be given. Haemorrhage is common and may be harder to control, especially if there is bleeding from torn dural sinuses, or cerebral vessels which cannot be immediately accessed. It is

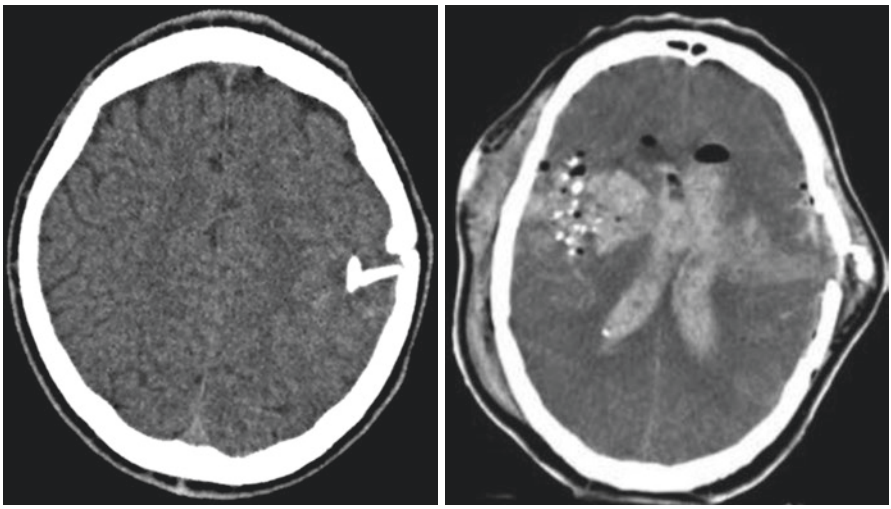


Fig. 8.23 Penetrating brain injury

important to be especially careful when examining a suspected penetrating, or open head wound, especially those sited in the midline. Disruption of the tissues can result in tearing of the venous sinus and exsanguinating haemorrhage. Exploring such wounds in the emergency department should therefore not be undertaken.

8.17 High Energy Penetrating Injuries

Craniocerebral gunshot wounds (GSWs) and penetrating injuries from blast injuries are frequently encountered by military surgeons, less so by civilian neurosurgeons. These are, not surprisingly, some of the most difficult injuries seen in the emergency department. The ballistics of the wound should always be considered, notably the type of weapon used, its proximity and the bullet's caliber. Depending on the amount of kinetic energy transferred, cavitation along the path of the projectile can result in a variable amount of damage. The main effects of cerebral GSWs are usually brain swelling, intracranial haemorrhage and cortical lacerations from metallic, bony and other foreign bodies. Very often, the prognosis is poor, resulting in death or the patient recovers with significant neurological deficits. Aggressive management is usually required for patients with a Glasgow Coma Score 6–12, although this is controversial. Following initial resuscitation, the revised GCS (which may indicate an improvement in the patient's condition), together with the amount of brain damage seen on CT, should be used to determine the likelihood of recovery and help in the decision-making process. Excessive crystalloid, hypotension, hypoxia and hypercapnia should all be avoided. Coagulopathy may also develop, which complicates management and is generally a poor prognostic factor. Clinical factors associated with poor outcome include.

(i) GCS < 5 (following resuscitation)
(ii) Dilated, unreactive pupil(s)
(iii) Occipital entry wound
(iv) Brainstem injury
(v) Bi-hemispheric (bilateral) injuries
(vi) Injury to the 'eloquent' part of the brain (sensory processing or linguistic ability)
(vii) High-velocity missile injury
(viii) Hypotension
(ix) Major intracranial vascular injury
(x) High ICP
(xi) Onset of diabetes insidious
(xii) Suicide attempt (because of close range)
(xiii) Increased retrieval time
(xiv) Coagulopathy or disseminated intravascular coagulation (DIC)
(xv) Advanced age

Bilateral frontal lobe injuries (often seen following suicide attempts) have a surprisingly better chance of survival than other bilateral injuries. However cognitive deficits and personality change are usually significant. Minor penetrating

injuries to the brain with small entry wounds may only require local debridement, wound closure and antibiotics. More severe, but localised injuries with ongoing haemorrhage and mobile or depressed fragments usually require exploration via a limited craniotomy. However more severe penetrating injuries will require extensive surgery and neuro intensive care—if survival is anticipated and a decision is made to operate. Surgery may include decompressive craniectomy, debridement, evacuation of haematomas, dural repair and insertion of an ICP monitor. With these more devastating injuries, patients may survive but not surprisingly will have severe disabilities. Complications of these injuries are varied and severe and include (i) pseudoaneurysm, (ii) cerebral vasospasm (secondary to subarachnoid haemorrhage), (iii) cerebral abscess, (iv) meningitis, (v) ventriculitis, (vi) epilepsy and (vii) hydrocephalus. Today, lead toxicity is uncommon. Nevertheless lead levels should be monitored if there are a significant number of retained metallic fragments. Plastic surgery may be required if there is significant scalp tissue loss.

8.18 Low Energy Penetrating Injuries

Penetrating injuries due to knives, nail guns and other sharp objects are uncommon. Machete injuries to the head are common in some parts of the world. These may result in depressed, open fractures, with or without neurological deficit. The principles of management of these injuries are the same as for gunshot wounds. Any visible foreign body should not be removed until the patient is anaesthetised and the surgeon has control of the major vessels involved.

8.18.1 Penetrating Orbital Roof Injuries

Transorbital intracranial penetrating injuries are relatively rare but can occur when a foreign body enters via the orbital roof or less commonly, through the superior orbital fissure. Entry via the optic canal is extremely rare. Most penetrating cranial injuries in children are due to sharp objects penetrating the orbit. These include pencils and tree branches following falls. The possibility of intracranial penetration should therefore always be considered in any penetrating orbital injury (in both adults and children). These may appear trivial at first glance and can be easily underestimated if the penetrating object has been removed. Children may thus present later, with a high fever, drowsiness, confusion, meningism, or ocular symptoms as a result of delayed infection (orbital or frontal lobe abscess).

The direction of the penetration should be ascertained if possible. Penetration through the thin orbital roof often leads to frontal lobe laceration or contusion. CSF leakage is usually evident, through the overlying skin wound. Penetration through the superior orbital fissure is potentially more serious. This is because the foreign body enters the middle cranial fossa and can pass close to the brain stem or cavernous sinus. If a foreign body passes through the optic canal, injury to the internal carotid artery should be suspected.

When penetrating orbital or cranial injury is suspected, urgent CT is the investigation of choice. Whilst CT can easily detect metallic objects, wooden and other organic foreign bodies are much harder to identify. Intracranial wood may show varying degrees of attenuation. Bubbles of gas should be regarded with suspicion and not simply attributed as air from the sinus or an artifact. Over time, a wooden foreign body absorbs water and approaches tissue density, making it even harder to identify. The history of the injury is therefore extremely important. Angiography may also be required to identify injury to the intracranial vessels. If there is no metallic foreign body, MR imaging, including MR angiography should be considered. Organic material may still be difficult to visualise, although MR imaging may delineate some foreign bodies.

Management is extremely difficult. Ideally the foreign body needs to be removed and the tissues debrided, but to do so requires sufficient access to deal with any major haemorrhage that occurs. If the patient is awake, the foreign body should be left in situ and an urgent referral made to the on-call neurosurgical team. If the patient is unconscious or agitated they will probably require airway protection and resuscitation. In all cases antibiotic should be given, following the advice of the neurosurgical team or a microbiologist. Agricultural injuries are usually at high risk of infection.

8.19 Blast Injuries to the Brain

Blast-induced traumatic brain injury (bTBI) is a potentially devastating injury. The pathophysiology of the blast is more complex than a GSW. Explosion can cause injury to the brain by three main mechanisms

(i) a transient but high pressure shock wave is transmitted through the skull (and possibly funnelled through its openings—orbits, nasal cavity etc). This contains a huge amount of energy and travels over the speed of sound. It is followed by a "low pressure" phase, with both occurring within a few milliseconds. Multiple shock waves may therefore arise from a single explosion.
(ii) metal fragments and other foreign bodies may penetrate the skull and enter the brain
(iii) hot gases generated by the blast result in skin and respiratory burns. This can result in hypovolaemia and hyperaemia, thereby aggravating any secondary brain injury.

External wounds can be deceptive and may not reveal the true extent of any internal damage. The brain is very vulnerable to blast injury and blast waves frequently cause severe cerebral oedema. Each wave transfers kinetic energy throughout the brain, damaging neurones and blood vessels. Depending on the amount of energy transferred this results in a spectrum of injury, ranging from mild to fatal injuries. Oedema, contusion, diffuse axonal injury (DAI), haematomas, and haemorrhage can all occur. Persistent focal cerebral vasospasm has also been reported.

Management principles following resuscitation include rapid cranial decompression, early repair of skull base injury and CSF diversion. Brain swelling usually occurs within a few hours of the injury, but mortality may be decreased if treated with early decompressive craniectomy. Early diagnosis and management of any vascular injuries and delayed facial or cranial reconstruction may also be required. SAH, vasospasm and pseudoaneurysms can also occur following blast injury.

Milder blast injury can produce neurological complications ("shell shock" or "blast concussion"). These include behavioural, psychological, and cognitive symptoms, such as retrograde amnesia, headache, confusion, difficulty concentrating, mood swings, difficulty sleeping and anxiety. Such symptoms can take months or even years to develop. A potential biomarker for these injuries being investigated is the altered expression of p11 protein, a member of the large family of S100 proteins. Advances in neuroimaging (diffusion tensor imaging—DTI) may also help diagnosis due to its ability to detect changes in the white matter.

8.20 Pituitary Necrosis

The pituitary is particularly vulnerable to head trauma due its anatomical location (within the sella turcica), vascular supply and fragile stalk. Infarction of the gland has been reported in up to one-third of patients who have died from traumatic brain injury. Mechanical /shearing injuries to the delicate vasculature in the pituitary stalk results in anterior lobe infarction. Secondary injuries may occur from raised intracranial pressure, profound or prolonged hypotension, hypoxia and vasospasm. The posterior pituitary appears to be less susceptible to injury due to its more robust vascular supply.

Pituitary insufficiency following traumatic brain injury may be more common than previously thought. In the acute phase hypothalamic and pituitary dysfunction may result in disturbances in hormone secretion. This may include the hypothalamic-pituitary-adrenal axis which results in inadequate cortisol secretion. This is a life-threatening condition. Other hormones may be involved, including growth hormone, oestrogen and testosterone, but the importance of these on the patient's immediate survival are less. Nevertheless, central hypogonadism, thyroid hormone deficiency, hyperprolactinemia, GH deficiency and cortisol deficiency have all been reported inpatients following moderate and severe traumatic brain injury. Clinically, these are similar to Sheehan's syndrome (also known as postpartum pituitary gland necrosis).

8.21 Post traumatic Intracranial Aneurysm and Caroticocavernous Sinus Fistula (CCF)

Post traumatic intracranial aneurysms usually occur following penetrating injuries. They can also occur following blunt trauma (particularly with middle cranial fossa fractures) and rarely following surgical procedures. They comprise less than 1% of

all intracranial aneurysms and are usually pseudoaneurysms of the internal carotid artery. These are usually diagnosed following CT angiography. Untreated, an expanding aneurysm can result in an increase in intraocular pressure (with the risk of blindness), cavernous sinus herniation into the sphenoid sinus (with the high risk of uncontrollable epistaxis) and the development of hemiplegia, as a result of a "steal" phenomenon.

Post-traumatic carotid-cavernous fistula (CCF) is an abnormal communication that develops between the intra-cavernous portion of the internal carotid artery and the surrounding cavernous sinus (Fig. 8.24). This occurs in approximately 0.3% of patients following severe head trauma. A high flow arteriovenous shunt develops, resulting in a significantly increased pressure within the cavernous sinus and its tributaries. This elevated pressure results in impaired venous drainage of those structures that normally drain into the sinus. With large shunts the flow may even be reversed. Those structures that traverse the sinus can also be affected. Symptoms of CCF include headache, diplopia, ptosis, chemosis, pulsating exophthalmos, and ophthalmoplegia (as a result of paresis of cranial nerves III, IV, and VI). Rarely the

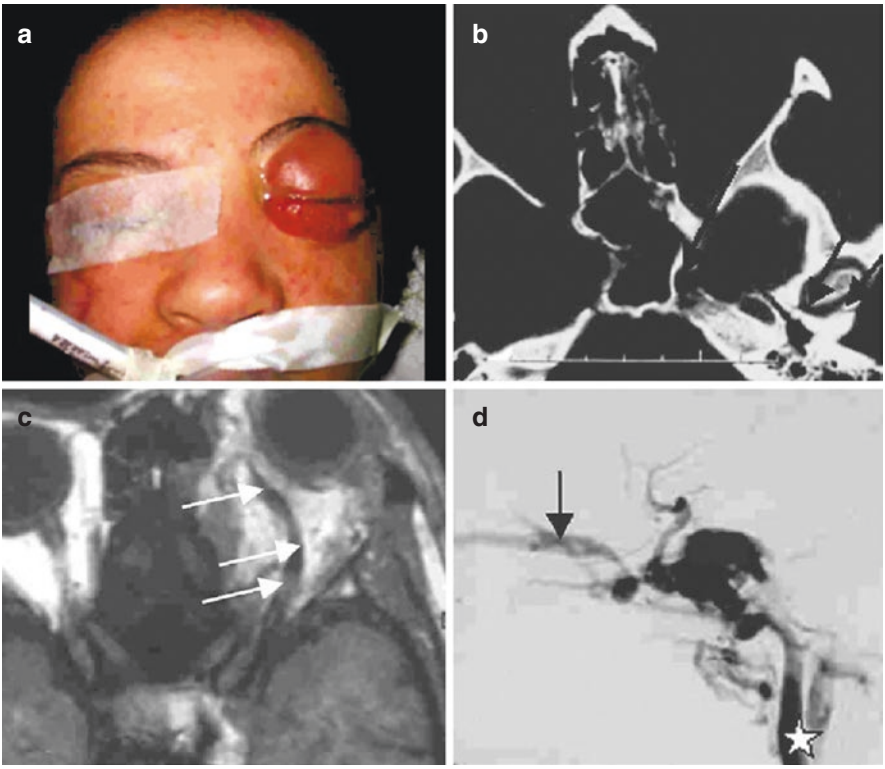


Fig. 8.24 a-d Left-sided carotid cavernous fistula (**a**) Chemosis and pulsatile exophthalmus of the left eye. (**b**) The fracture line crosses the glenoid fossa and goes towards the cavernous sinus (*arrows*), (**c**) T1-weighted MRI showing exophthalmus and huge dilatation of the ophthalmic vein (*arrows*), (**d**) Internal carotid artery (*star*) angiography showing the arterial-venous fistula, feeding the cavernous sinus (*CS*) and the ophthalmic vein (*arrow*).

patient can present with massive epistaxis. On examination there may be a 'machinery-type murmur' heard over the forehead or eye. Ophthalmoplegia is also accompanied by loss of sensation over the ipsilateral forehead (supraorbital nerve) due to the involvement of the ophthalmic division of the trigeminal nerve. Exophthalmos (proptosis) is usually painful and there may be visual impairment. If the patient is not treated, blindness can result. The differential diagnosis includes superior orbital fissure syndrome, orbital apex syndrome, orbital compartment syndrome and cavernous sinus syndrome. In the absence of a clear history of trauma, suspect an inflammatory, neoplastic, or vascular pathology. CT or MR imaging typically shows a dilated superior ophthalamic vein, CT angiography will confirm the diagnosis. Current management is typically endovascular and involves the interventional radiology team. Some specialists recommend that dural CCF patients without aggressive symptoms, aged more than 70 years, or with slow flow and mild inflow into the cavernous sinus can be managed conservatively. Spontaneous resolution has been reported. CCF patients with aggressive symptoms and/or cortical venous reflux on angiography require embolisation. Haemorrhage and/or congestive infarction may occur. Onyx is a commonly used liquid embolic agent.

8.22 Shaken Baby Syndrome (Abusive Head Trauma/Non Accidental Head Injury)

This is a form of child abuse which occurs as a result of violent shaking a young child (< 5 years of age) or infant. It often presents as a triad of symptoms—subdural haematoma, retinal haemorrhages and brain swelling, often with no external signs of injury and is often fatal or results in severe brain damage. Fractures of the long bones may also be associated with this.

There is a wide spectrum of severity of abusive head injury. Injuries range from mild to severe and can result in death. Rotational injury is especially damaging and likely to occur in shaking trauma. Victims of Non accidental head injury (NAHI) may showed signs of irritability, failure to thrive, lethargy, vomiting and seizures. Bulging of the fontanelles and increased head circumference may suggest brain swelling.

Infants with mild injuries who survive are at risk for learning and behavioural problems and seizures. Those with severe injuries who survive often have significant problems, including:

- intellectual disability
- learning and behavioural problems
- seizures
- blindness
- motor problems
- persistent coma (persistent vegetative state)

The differential diagnosis includes hydrocephalus, sudden infant death syndrome (SIDS), seizure disorders, vitamin C deficiency and some infectious, congenital and

metabolic disorders. CT scanning and magnetic resonance imaging are often used to diagnose the condition.

Injuries are not always confined to the head. Infants may be strangled, gagged, and smothered as well as sustaining injuries to the neck, chest, and abdomen. Some injury patterns are closely associated with abuse and rarely seen with other conditions. These include rib fractures to the side and back of the chest and pattern injuries (which match the aetiology, such as a ligature or handprint). Emergency staff should always be vigilant to the possibility of child abuse. Clues include when a severely injured infant or child presents with a caregiver who gives a history that is inconsistent with the clinical findings. If any member of the treating team is concerned, they must report these concerns as dictated by local policy. The exact procedure varies from country to country.

8.23 Management Principles of Head Injuries

8.23.1 Prehospital Care of Head Injuries

The acutely injured brain is particularly vulnerable to secondary brain injury as a result of systemic hypotension, reduced perfusion, hypercarbia, hypoxemia, hypoglycaemia and an elevated ICP. Prevention of these is thus crucial to management. Care of the head injured patient therefore always begins with securing a patent airway, restoring normal breathing and ensuring adequate circulation. Early endotracheal intubation (usually orotracheal) is usually required, especially in comatose patients. This not only protects the airway from aspiration, but also enables oxygenation and ventilation. Patients are often sedated and pharmacologically paralysed before intubation, because irritation of the oropharynx can cause transient hypertension, tachycardia, agitation and an increased ICP.

Supplemental oxygen is also provided before and immediately after intubation. Ventilatory rates are around 10 to 12 breaths per minute for adults, 20 breaths per minute for children and 25 breaths per minute for infants. Therapeutic hyperventilation is now generally no longer undertaken, unless neurological deterioration occurs during transfer. Excessive hyperventilation can result in cerebral vasoconstriction, reducing an already low cerebral blood flow and exacerbating cerebral ischaemia. Rapid fluid resuscitation and restoration of a normal BP are also important—hypotension is associated with a doubling of the mortality rate in severe head injury. Active bleeding should be stopped. In all patients with a Glasgow Coma score of 8 or less, spinal immobilisation should also be carried out. Once the airway and haemodynamic stability is achieved, immediate transport to a designated trauma centre should occur without delay.

8.23.2 Emergency Department Care

Much of this is discussed in the chapter on the injured patient. Important handover information from the paramedics includes the mechanism of injury, initial vital signs, GCS, procedures undertaken, medications given and haemodynamic stability

during transport. Most trauma centres now follow the Advanced Trauma Life Support guidelines. The airway or tracheal intubation are reassessed. Adequate oxygenation is confirmed following pulse oximetry and arterial blood gas analysis and thereafter monitored using an oxygen saturation monitor or arterial line. Intravenous access is confirmed or established and fluids are infused as required. Any life-threatening injuries (notably haemorrhage, tension pneumothorax and cardiac tamponade) should be treated immediately upon discovery.

Oxygen saturation is monitored continually along with the blood pressure. Other interventions/investigation include a Foley catheter, orogastric tube, blood specimens for glucose, electrolytes, complete blood count, platelets, clotting profile, type and crossmatch, toxicology and when indicated, pregnancy test. Neurologic examination is then quickly performed, including a GCS assessment, pupillary size and reaction to light. The head should be gently inspected for any lacerations or penetrating wounds. Large lacerations are dressed or temporarily sutured to prevent further haemorrhage. Haemotympanum, periorbital/mastoid ecchymosis, and CSF rhinorrhea, oculorrhea, or otorrhea should also be noted.

All this occurs against a background of obtaining imaging as soon as possible. Depending on the mechanism of injury and suspected injuries sustained, initial x-ray evaluation may include chest, pelvis, and lateral cervical spine films. However many trauma centres now proceed directly to the CT suite for a 'pan scan'. This involves CT of the head, neck, torso and pelvis—in essence from the top of the head to the knees. CT scan of the head is usually performed at intervals of 10 mm or less. This will identify haematomas, contusions, brain swelling, midline shift and the patency of the basal cisterns.

Surgical mass-effect lesions should be evacuated immediately. Diagnostic burr holes are rarely required today, but may occasionally be undertaken if the patient has lateralising neurologic deficits and a unilateral fixed and dilated pupil, but is so haemodynamically unstable that they require an immediate laparotomy or thoracotomy, before a head CT scan can be obtained.

8.23.3 Scalp Lacerations

Treatment of lacerations can be a traumatic experience for patients, especially children. Clinical evaluation should identify associated serious head injury, laceration of the galea, or fracture. Haemorrhage can be significant from these. This can be easily overlooked since in everyday practice only a minority of scalp lacerations require hospital admission for haematoma. Nevertheless, this can be an issue if interhospital transfer is required. Large head wounds tend to cause a greater than expected blood loss and can contribute to patient destabilisation relatively quickly. This can result in haemodynamic instability if unrecognised (especially in children and occipital lacerations in supine patients). After haemostasis is achieved the laceration should be thoroughly cleansed and quickly closed ideally in two layers if possible. Never blindly explore or probe a scalp laceration to see if there is a fracture—particularly midline lacerations. Sutures are preferred over staples for large, gaping wounds, but take more time to apply. If urgent, place an over and over continuous suture to achieve mass closure. If time allows interrupted two layer sutured

closure is preferable. The deep suture is passed through the aponeurotic layer, where the blood vessels pass. The use of tissue glue is acceptable for small lacerations, but wounds can fall apart and gape if it is not used properly. A hair apposition technique has also been reported in which the wound is closed by bringing together the hair on either side, making a single twist, which is then secured with tissue glue. This has the advantage of being time efficient and can be undertaken by all levels of staff.

Degloving scalp injuries require urgent treatment. Not only can they resulting in catastrophic blood loss, but they can also be highly contaminated. These are serious and potentially debilitating injuries. They are a form of avulsive soft tissue injury which, commonly occurs when long hair is trapped industrial high-speed rotary machines. The plane of separation is through the loose connective tissue (between the galea aponeurotica and the periosteum). Consequently the underlying skull is often exposed. Depending on the mechanism of injury the tissues may be highly contaminated and need to be carefully cleaned prior to closure. Depending on the size and extent of the injury prognosis varies, but generally these injuries do surprisingly well if managed correctly.

In paediatric patients, large scalp lacerations are probably better repaired in the operating room, rather than the emergency department. Sedation or general anaesthesia can be provided enabling thorough wound examination and irrigation prior to repair. Finally, in all patients, remember to investigate and if necessary give antitetanus prophylaxis. Scalp sutures can usually be removed after 7–10 days, or resorbable sutures can be used.

8.23.4 Potentially Significant Head Injuries

Ongoing observation of patients is still an important part of their management, notably in patients with abnormal CT finding but who do not require surgery and in patients with persisting neurological symptoms and signs. Observation should occur during the entire hospital episode, whether in the emergency department or following admission. These are initially undertaken on a half-hourly basis until the GCS is 15. The minimum documented observations are: i) GCS; ii) pupil size and reactivity; iii) limb movements; iv) respiratory rate; v) heart rate; vi) blood pressure; vii) temperature and viii) blood oxygen saturation. A typical frequency of observations is half-hourly for 2 hours, then 1-hourly for 4 hours, then 2-hourly thereafter. Should the patient deteriorate at any time after the initial 2-hour period, observations should revert to half-hourly and follow the original frequency schedule. It is important to make sure support staff know when to call for an urgent review. If any of the following occur, urgent reassessment and possibly CT is required

(i) Development of agitation or abnormal behaviour.
(ii) A sustained (at least 30 min) drop of 1 point in GC score (this is more worrying if this drop is in the motor response score).
(iii) Any drop of 3 or more points in the eye-opening or verbal response scores of the GCS, or 2 or more points in the motor response score.

(iv) Development of severe or increasing headache or persisting vomiting.
(v) New or evolving neurological symptoms or signs such as pupil inequality or asymmetry of limb or facial movement.

Observation in infants and young children (under 5 years) is difficult and therefore should only be performed by experienced staff. Most patients can be discharged the following day if they are asymptomatic. Patients who do not require admission should be given written guidelines of when to return to hospital and should only be discharged if there is a responsible adult who can take them home and call for assistance if required.

8.23.5 Transferring Head Injured Patients

Transfers are an inevitable process in most head injured patients. This includes transferred to the radiology department for CT scanning. Patients may also need to be urgently transferred to another hospital where there are neurosurgeons. Very rarely do neurosurgeons travel to see the patient. The decision to transfer a patient must be made by senior medical staff at the referring and receiving hospital. It is important to ensure that the patient is quickly resuscitated and in the best possible condition before they are transferred. Thorough resuscitation and stabilisation of the patient before transfer is the key to avoiding complications during the journey. Key considerations include includes.

(i) The fundamental requirement for transfer is to ensure satisfactory oxygen delivery. In adults, a mean blood pressure greater than 80 mmHg, PaO_2 greater than 13 kPa and $PaCO_2$ between 4.5–5.0 kPa should be achieved.
(ii) There should be a designated consultant in the referring and receiving hospitals with overall responsibility for the transfer of patients. Local guidelines should be available between the two hospitals.
(iii) Thorough resuscitation and stabilisation of the patient must be completed before transfer to avoid complications during the journey—this may include the need to undertake thoracotomy, laparotomy or pelvic fixation to control haemorrhage. If the patient remains hypotensive despite resuscitation, they should not be transported until this is addressed. Correction of major haemorrhage takes precedence over transfer. Persistent hypotension will adversely affect neurological outcome.
(iv) Intubate and ventilate all patients with a Glasgow Coma Score less than or equal to 8. This requires adequate sedation and muscle relaxation to avoid an increase in (ICP), and measures to prevent aspiration of gastric contents. Intubation is usually achieved by rapid sequence induction with in-line stabilisation of the cervical spine. “At risk” patients may also require intubation. This may include patients with mandibular fractures, or significant facial swelling, which may place the airway at risk during transfer or if the GCS has fallen by 2 or more points. The decision to intubate these patients

should be made by the designated consultant led team. However, if patients are being transferred for observation only, it is best to avoid intubation and sedation if safe to do so. This should be discussed with the on-call neurosurgical team.

(v) Intravenous volume loading should be undertaken to maintain or restore peripheral perfusion, blood pressure and urine output. Hypovolaemic patients tolerate transfer poorly. The amount and choice of fluid should be discussed with the receiving hospital. A central venous catheter may be required.

(vi) If the patient has had a seizure, loading with anticonvulsant agents (usually phenytoin) should be considered prior to transfer.

(vii) Unstable or compound long bone fractures should ideally have emergent preliminary toilet and be splinted, with minimal delay.

(viii) In some cases intravenous mannitol may be given (under the direction of a neurosurgeon) to buy time by reducing intracranial pressure. The maximum dose is 1 g/kg—in a 70 kg adult, this is 350mls of 20% mannitol. Hypertonic saline is increasingly used to reduce ICP, but should always be discussed with the receiving team before prescribing.

(ix) Patients with brain injuries should be accompanied by a doctor with appropriate training and experience in the transfer of patients with acute brain injury.

8.23.6 Monitoring Should Include

- Pupillary size and reaction to light.
- ECG.
- Pulse oximetry.
- Invasive blood pressure.
- Urine output by urinary catheter.
- Capnography.
- Central venous pressure monitoring where indicated.
- Temperature (preferably core and peripheral).

8.23.7 Investigations Undertaken Prior to Transfer Should Include

- Arterial blood gas.
- Appropriate X-rays (chest, cervical spine, pelvis etc).
- FBC and coagulation screen.
- Blood sugar.
- Blood should be cross matched if appropriate and sent in the transferring ambulance.

8.23.8 Head Injuries in Children

Traumatic brain injury (TBI) in children is a leading cause of death and disability. Causes are often age dependent. Infants are usually dropped accidentally or fall from a table, chair, bed etc. Toddlers often fall down stairs as they learn to walk or when they explore their environment and also fall off play equipment. Older children often fall off bikes and scooters or from climbing equipment. Adolescents sustain head injuries during contact sports or activities involving vehicles. The types of injuries that result include both injuries to the skull and brain that are pathophysiologically similar to adults and those that are not. Like adults, head injuries in children can result in concussion (mild head injury with alteration in mental status). Skull fractures can involve any bone of the skull but most commonly involves the parietal bones. Brain injuries can be parenchymal contusions, bleeding (parenchymal, epidural, subarachnoid, subdural or ventricular), diffuse axonal injury or diffuse brain swelling.

Differences that occur compared with adults, do so because of age-related changes within the developing brain, differing mechanism of injuries and children's differing ability to withstand trauma. Children have relatively larger heads compare to adults and are therefore at greater risk of Head injury. Biomechanically, the paediatric skull and brain are more plastic and can therefore deform and absorb impact energy in a different way to adults. Cerebral white-matter has relatively little myelin compared to adults and the neonatal brain is therefore more 'watery' in consistency, making it more susceptible to injury. Consequently, different pathological mechanisms are reported to occur during acceleration and impact forces. The infant skull is also less rigid, and has open sutures, which can result in differential movement between the bones following injury. This phenomenon is well known in obstetrics, in which intracranial haemorrhage can occur following normal vaginal delivery. Neonates are also prone to conditions such as cephalic and subgaleal haematoma. Acute extradural haematoma is relatively rare in young infants as the dura is firmly adhered to the inner surface of the skull, especially around the suture lines.

Shaking a child usually produces slight deformation of the skull, but the high plasticity of the skull results in shearing forces between the skull and adjacent cortical vessels and brain. These can result in stretching and shearing injuries to the vessels and brain parenchyma—shaken baby syndrome. As with adults the paediatric scalp is highly vascularised and is a potential cause of lethal blood loss. Compared to adults even a relatively small loss of blood volume can lead to haemorrhagic shock in a newborn, infant, and toddler.

Traumatic head injury in children is also predisposes to the development of coagulopathy. This has been suggested to be a manifestation of disseminated intravascular coagulation (DIC) and has been associated with intravascular thrombosis of small vessels and neuronal death. Children with a low GC Score (less than 9) are at an increased risk of being coagulopathic. This has been shown to worsen prognosis.

Hyperglycemia after traumatic brain injury in children has also been associated with poorer outcome. Serum glucose levels greater than or equal to 300 mg/dL on admission is a significant risk factor for mortality. Inability to normalisation blood glucose within 48 hours has also been associated with worse prognosis.

Various markers have been proposed for the diagnosis and determination of prognosis of brain injury in children. Some of these have previously been discussed. S100 calcium-binding protein B, or S100B, is a biomarker of acute neurological disorder. S100B has been reported to be elevated in children after significant traumatic brain injury, occurring within 6 hours after impact. Whilst promising, currently S-100B cannot replace clinical examination and CT. Neuron-specific enolase (NSE), a glycolytic isoenzyme in neurons and neuroendocrine cells, is also related to brain injury and has been reported to be predictive of the development of intracranial pathology.

Assessment of head injuries in children can be difficult. Many of the symptoms and signs which would result in concern in adults are often present in children, even following minor injuries. These include vomiting, drowsiness and headaches. Initial assessment of the child should be made by a suitably trained member of staff and carefully documented.

Full cervical spine immobilisation may be required if

(i) GC Score < 15 on initial assessment
(ii) There is neck pain or tenderness
(iii) Focal neurological deficit
(iv) Paraesthesia in the extremities
(v) Any other suspicion of cervical spine injury (including mechanism)

Neck immobilisation should be maintained until appropriate investigations had been performed and a serious injury ruled out.

As with adults, the gold standard in diagnosis of acute intracranial injury is CT scan of the head. Whilst this is generally widely available, can be completed rapidly and usually provides a definitive diagnosis, concerns have been reported regarding the radiation dose required and risks of later malignancy. Generally speaking, the younger the child, the greater the risk of carcinogenesis as a result of exposure to ionising radiation. Children also usually require sedation or anaesthesia, each with their own attendant risks. Indications for CT in children include (i) new seizures, (ii) decreased GCS, (iii) suspected skull fracture, (iv) focal neurological defect, (v) CSF leak. CT should be performed if a child has more than one of the intermediate risk factors (witnessed loss of consciousness >5mins, drowsiness, 3 or more discrete vomiting episodes, amnesia or worrying mechanism of injury). If only one intermediate risk factor is present the child should be observed for a minimum of 4 hours post head injury (with regular neurological observations every 30 mins, then hourly, then 2 hourly). If there is any deterioration, or persistent drowsiness or vomiting, CT is usually indicated.

Unfortunately vomiting in children is very common and non-specific, but it is a sign of raised intracranial pressure (ICP). Therefore the vomiting child should be

monitored closely. At the same time it is important to look for other features of raised ICP including decreased GCS, Cushing's triad, eye movement anomalies, unequal or dilated pupils. In children under 5 years of age, a change in behaviour or reduced wakefulness may indicate an abnormal conscious level and should be monitored.

One useful tool in assessment of Head injuries in children is the The CHALICE clinical decision rule. This has been designed to be applied to any child less than 16 years of age, with the aim of determining which children require a CT scan of the head. It is based on a number of variables which rely on the clinician's judgement and the accuracy of the information provided by the witness. Despite these potential shortcomings this test has been shown to be very reliable in identifying severe head injuries. Inclusion criteria (ALL must be satisfied for the CHALICE rule to be applied). The Paediatric Glasgow Coma Scale (PGCS) is also commonly used to assess consciousness in children and define the severity of head injuries.

Treatment depends on the history, the mechanism of the injury, clinical findings and results of the CT scan, if undertaken. As with adults this may range from discharge home with head injury instructions, observation in the emergency department, admission to a ward, or occasionally intracranial pressure monitoring, evacuation of an intracranial haematoma (e.g. subdural or epidural haematomas) or elevation of a depressed skull fracture. Patients may be discharged home only if clinically well, no risk factors identified and the patient has achieved GCS 15 or normal level of consciousness. If the child returns with similar symptoms CT is probably indicated. Non-accidental head injury (NAHI) should be considered in children under one with unexplained bruising or injury. Therefore all infants under 1 year with a suspected head injury should be admitted and referred to the Paediatric team.

8.23.9 Growing Skull Fracture

In developing infants or young children, a 'leptomeningeal cyst', or herniated brain and dura mater can result in enlargement of the fracture line. This occurs as a result of localised pulsations of the growing brain and pressure within the fracture.

8.24 Other Issues

8.24.1 Post-Concussion Headache

This can occur in isolation, or be part of a post concussion syndrome. Patients may develop symptoms similar to a tension-type headache. This is often associated with dizziness and loss of concentration, which can affect the ability to work, drive etc. Headaches which are progressive, or those which change in character warrant further investigations.

8.24.2 Post-Traumatic Amnesia (PTA)

This is a common problem that can occur immediately following TBI. The patient is unable to remember events that occur after the injury and may be confused and unable to state his or her name, where he or she is, and what time it is. About a third of patients with mild head injury can recall only some events. Two types of amnesia are described (i) retrograde amnesia (loss of memories that were formed shortly before the injury) and (ii) anterograde amnesia (problems with creating new memories after injury). Retrograde amnesia sufferers may regain memory later, but memories are not regained with anterograde amnesia because they were not processed properly initially. Longer periods of amnesia following injury may indicate a longer recovery time and risk of post-traumatic epilepsy. PTA may also be linked to the likelihood of psychiatric and behavioural problems later.

8.24.3 Driving

Patients should be advised to contact their driving regulation authority and their insurance company. If they have received a significant head injury, this may result in a restriction from driving for fixed period (typically around 6 months).

8.24.4 Chronic Traumatic Encephalopathy (CTE)

Studies have also found that TBI may be associated with progression of some diseases, including epilepsy, Alzheimer's disease, Parkinson's disease, ischaemic stroke and psychiatric diseases. Chronic traumatic encephalopathy (the so-called "punch-drunk syndrome", or dementia pugilistica), is a term which was once used to describe a distinctive and irreversible neuropsychiatric condition that affected boxers. However many of today's contact sports can result in repeated mild traumatic brain injuries (mTBIs), and similar symptoms. Sports commonly associated with this risk include American football, Boxing, ice hockey, soccer, rugby, martial arts and cycling. Other causes include physical abuse, head banging and poorly controlled epilepsy.

Repeated exposure to mild brain injuries can result in long-term neurological deterioration and a range of disturbances. Linear and rotational acceleration can cause diffuse axonal injury within the cortical and subcortical areas. Pathologically, this results in increased membrane permeability, ionic changes and cell death. Together with chronic inflammation and an auto immune response, these processes can result in deposition of a "tau protein", which results in neurodegeneration.

Symptoms include confusion, slowed muscular responses, hesitant speech, tremors, and parkinsonism. Currently, CTE is a diagnosis of exclusion because only post-mortem examination can confirm the diagnosis. Cerebral atrophy,

neuronal loss, gliosis, degeneration of the substantia nigra and argyrophilic neurofibrillary tangles are features found during post-mortem. In severe cases there may be atrophy of the temporal lobe, thalamus, hypothalamus, and mammillary bodies. Research is currently focused on early diagnosis using neuroimaging techniques and biomarkers (magnetic resonance imaging and functional MR).

8.24.5 Heterotopic Ossification (Neurogenic Myositis Ossificans)

This is an odd phenomenon which has been well described. It is defined as the formation of bone within the soft tissues, usually muscle. This should not be confused with the metastatic calcification that can occur in hypercalcaemia, or dystrophic calcification related to tumours. Ectopic bone can develop spontaneously following TBI and especially if local trauma to the soft tissues has also occurred. Prolonged immobility, multiple fractures, burn injuries and coma may all contribute to the risk of heterotopic ossification. Several hypotheses have been proposed including vasomotor and metabolic disorders and the role of oestrogenic precursor cells. However the precise cellular mechanisms remain unclear. Whatever the pathophysiology, there appears to be a relationship between the nervous system and bone metabolism and several neuropeptides and neurotransmitters have been found in the bone tissue, including glutamate, substance P, leptin, vasoactive intestinal peptide (VIP) and catecholamines. These have all been shown to modify osteoblastic and osteoclastic activity. The hip is the most commonly involved site. Early detection and prevention with physical therapy, NSAIDs or bisphosphonates have shown good results.

8.25 Head Injury Instructions

All patients (and their carers) should be provided with "head injury instructions" when discharged from hospital. Occasionally symptoms suggestive of brain injury or intracranial bleeding may take several hours, or even days to develop following injury. In rare cases (notably in the elderly), symptoms from a slow bleed (chronic subdural haemorrhage) can develop weeks or even months later. 'Head injury instructions' should provide the necessary information (including symptoms to look out for), regarding when the patient should seek help or return to the emergency department. These typically include

(i) Drowsiness when the patient would normally be fully awake.
(ii) Worsening headache—which does not settle with simple analgesia
(iii) Confusion, strange behaviour and any problems with understanding or speaking.
(iv) Inability to remember events before or after the head injury.
(v) Vomiting

(vi) Loss of use of part of the body—for example, weakness in an arm or a leg.
(vii) Dizziness, loss of balance or walking strangely.
(viii) Fitting or collapse followed by feeling strange afterwards.
(ix) Any visual problems, such as blurring of vision or double vision.
(x) Blood or clear fluid leaking from the nose or ear.
(xi) New deafness in one or both ears.
(xii) Unusual breathing patterns.

The Patient should also be advised

(i) Do not take any alcohol or drugs for the following few days.
(ii) Do not take sleeping tablets or sedatives unless prescribed by a doctor.
(iii) Discuss with your doctor about playing contact sports such as rugby or football. It is often advised not to play contact sports for at least three weeks following a minor head injury.
(iv) Do not drive, ride a motorbike or bicycle, or operate machinery until completely recovered.
(v) Show a relative or friend this advice leaflet so they too know what symptoms to look out for. Stay within easy reach of a telephone and medical help for the following few days.

It is quite common for children to want to sleep for short periods. However, this will appear to be a normal 'peaceful' sleep, after which they will wake up fully. Children should be allowed to sleep if they wish, but they should be woken up after an hour or so. They may be grumpy about being woken up, but that is reassuring. The child can then go back to sleep again, but should be checked a few times during the night. When asleep, check to see that the child appears to be breathing normally and is sleeping in a normal position.

8.26 Screening Tests for Concussion

Concussion can prevent patients from returning to work and in some cases (such as working in a hazardous environments) can necessitate a change of career. It can also prevent professional athletes from returning back to their sport for a prolonged period of time. Therefore any screening test that provides accurate prognostic information would be highly desirable. Unfortunately, at present, no test exists that is 100% sensitive and specific in screening for concussion. A number of scoring systems have been devised such as the Balance Error Scoring System (BESS), the Timed Tandem Gait test (TGT) and more objective sensorimotor assessments, but none have been shown to be completely reliable. The Standardised Assessment of Concussion (SAC) is a cognitive test that evaluates orientation, concentration, and memory. The Sport Concussion Assessment Tool, third Edition (SCAT-3) consists of a series of tests, including a focused physical exam, a 22 point checklist, GCS, cognitive and sensorimotor assessments. The King-Devick test (KDT) is a visual-based test involving rapid number-naming in which the patient reads aloud cards of

single-digit numbers as quickly as possible. Oculomotor assessment (eye-tracking) has also been used in the diagnosis of concussion. Perhaps the most simple way to screen for concussion is to ask the patient about relevant symptoms (headache, dizziness, difficulty concentrating, confusion and visual disturbances). Other symptoms include nausea, drowsiness, amnesia, irritability and feeling dazed. However, none of these symptoms are specific for concussion.

8.27 Advanced Head Injury Management

Most patients with significant brain injury and those who have undergone neurosurgical intervention usually require management on a neuro-intensive care unit (NICU). Once the patient has been transferred, management consists of various strategies aimed at maintaining haemostasis. The goal of critical care management of patients with severe traumatic brain injury is to optimise cerebral perfusion and avoid measures that may cause cerebral ischaemia. This generally involves:

- Stabilisation of the patient.
- Prevention of intracranial hypertension.
- Maintenance of an adequate and stable cerebral perfusion pressure (CPP).
- Avoidance of systemic, secondary brain insults.
- Optimisation of cerebral blood flow and oxygenation.

In the initial post-operative recovery period, cerebral perfusion needs to be optimised and all efforts are made to keep the intracranial pressure at a normal level. This is a highly specialist area of care. Many patients will therefore have the following undertaken or placed

- Endotracheal intubation and controlled ventilation
- ICP monitor
- Arterial line for invasive BP monitoring
- CVP line
- Urinary catheter
- Nasogastric tube

Other investigations that have been reported but which are not in widespread use include:

(i) Jugular venous oximeter (JVO2)—This is an indicator of both cerebral oxygenation and cerebral metabolism, reflecting the ratio between cerebral blood flow (CBF) and cerebral metabolic uptake of oxygen. A catheter is passed up to the jugular bulb at the base of the skull and measures the amount of oxygen being extracted by the brain. If the patient is being vigorously hyperventilated (to reduce the ICP), cerebral vasoconstriction can occur, worsening cerebral ischaemia. This can be detected by the JVO2 (comparing the amount of oxygen being extracted by the brain to the amount of hyperventilation being applied).

(ii) Brain tissue oxygen tension (PbtO2)—This measures focal cerebral oxygenation using an invasive probe. Since this provides a highly focal measurement, it is mainly used to monitor oxygenation in critically perfused areas of brain tissue
(iii) Cerebral microdialysis—This is an invasive bedside monitor that analyses brain tissue biochemistry. A catheter is inserted into damaged brain tissue to measure biochemical changes (glucose, lactate, pyruvate, glycerol, and glutamate). However its use is very limited.
(iv) Transcranial Doppler Ultrasonography is a non-invasive method to measure cerebral blood flow. It is increasingly used in the neurocritical setting for many conditions, including TBI.
(v) Electrophysiological monitoring—Electroencephalogram (EEG) is a useful tool for monitoring the depth of coma, detecting sub-clinical seizures, or seizure activity in pharmacologically paralysed patients.
(vi) Near infrared spectroscopy (NIRS). This is a continuous, direct, and non-invasive monitor that measures cerebral oxygenation and cerebral blood volume (CBV). NIRS is based on the differential absorption properties between haemoglobin (Hb) and cytochrome oxidase. From this the degree of tissue deoxygenation can be evaluated.
(vii) Brain temperature—After head trauma, a temperature gradient between the brain and body temperature of up to 3 °C (higher in the brain) may occur. This elevated temperature can act as a secondary insult to the injured brain.

8.28 Critical Care Management

Guidelines for the management of severe TBI are widely available. These may vary slightly depending on local policies and protocols, but generally include.

8.28.1 Analgesia, Sedation and Paralysis

Endotracheal intubation, mechanical ventilation, surgical interventions, nursing care and ICU procedures are all potential causes of pain. Narcotics, such as morphine, fentanyl and remifentanil, should be considered as first line therapy to provide analgesia, mild sedation and depression of airway reflexes. These are administered either as a continuous infusion, or as intermittent boluses. Adequate sedation potentiates analgesics, limits agitation, and facilitates nursing care and mechanical ventilation. Propofol is often the hypnotic of choice in many patients with an acute neurologic problem, as it is easily titratable and rapidly reversible. Benzodiazepines may also be used. Routine use of neuromuscular blocking agents (NMBAs) to paralyse patients with TBI is not usually recommended. These should be considered as second line therapy in patients with refractory intracranial hypertension.

8.28.2 Mechanical Ventilation

Patients with severe TBI are usually intubated and mechanically ventilated. Hypoxia (O_2 saturation < 90%, or PaO_2 < 60 mm Hg), should be avoided. Prophylactic hyperventilation to a PaCO2 < 25 mm Hg is not generally recommended as it can further compromise an already critically reduced cerebral perfusion. This is because excessive hyperventilation results in cerebral vasoconstriction and secondary ischaemia. Thus, hyperventilation is recommended only as a temporary measure to reduce an elevated ICP.

8.28.3 Haemodynamic Support

Haemodynamic instability is common in patients with severe TBI. Hypotension, (SBP < 90 mm Hg or MAP <65 mm Hg), is a common problem and is very detrimental to that brain. A drop in the patient's BP is associated with a significant increase in mortality following TBI. Common causes of hypotension include haemorrhage from associated injuries, polyuria secondary to diabetes insipidus, myocardial contusion, spinal shock and the use of certain drugs which can result in adrenal insufficiency. Anaemia is also common and can also result in secondary brain injury. This should also be avoided, with a target haemoglobin of approximately ≥100 g/L or a haematocrit ≥0.30. Coagulation abnormalities should also be quickly corrected.

8.28.4 Hyperosmolar Therapy

Mannitol is an effective method of reducing the ICP. However prophylactic administration is no longer recommended. The effective dose is 0.25–1 g/kg, intravenously over 15 to 20 minutes. Hypertonic Saline solutions are now suggested as alternatives to mannitol. These have a number of beneficial effects in head-injured patients, including expansion of the intravascular volume, extraction of water from the intracellular space, decreasing the ICP and an increase in cardiac contractility. Prolonged administration has been reported to be associated with a lowered ICP and controlled cerebral oedema. There are also less adverse effects such as renal failure, pulmonary oedema, or pontine demyelination.

8.28.5 Temperature Modulation

Evidence for the benefits of hypothermia in TBI is not entirely clear. Moderate systemic hypothermia (32 °C to 34 °C), does appear to reduce cerebral metabolism and CBV, decrease ICP and increase the CPP and has also been shown to reduce the extent of axonal damage. Clinical studies have also shown that patients experiencing periods of hyperthermia in the intensive care unit may

demonstrate worse outcomes after TBI. Therefore the temperature should be carefully monitored and controlled and any fever should be investigated and aggressively treated.

8.28.6 Seizure Prophylaxis

Prophylactic therapy is generally not recommended for preventing late post-traumatic seizures. However it is recommended to prevent early post-traumatic seizure in TBI patients who are high risk of developing seizures (GC Score < 10, cortical contusion, depressed skull fractures, subdural haematoma, extradural haematoma, intracerebral haematoma, penetrating TBI, and seizures that have occurred within 24 hours of injury).

8.28.7 Deep Vein Thrombosis Prophylaxis

Severe TBI patients are at significant risk of developing deep vein thrombosis (DVT) and pulmonary embolism. Graduated compression stockings and compression devices are recommended unless their use is prevented by lower extremity injuries. In the absence of any contraindications, low molecular weight heparin (LMWH) or low dose unfractionated heparin should also be used along with with mechanical measures. However in the presence of an acute traumatic intracerebral haemorrhage, LMWH maybe contraindicated.

8.28.8 Stress Ulcer Prophylaxis

Severe TBI is a well-known risk factor for stress ulcers (Cushing's ulcer). Prophylaxis includes early enteral feeding, and pharmacological measures such as H_2-blockers, proton-pump inhibitors and sucralfate.

8.28.9 Nutritional Support

Severe TBI patients are usually in a hypermetabolic, hypercatabolic and hyperglycemic state. They also have altered G.I. function. Malnutrition increases mortality and therefore early enteral feeding is recommended whenever possible.

8.28.10 Glycemic Control

Stress hyperglycemia is a common secondary brain insult. Maintaining blood glucose levels within normal limits is therefore important because both hyperglycaemia and hypoglycemia can result in further brain injury.

8.28.11 Fluids and Electrolytes

The aim of fluid management is to maintain euvolemia or moderate hypervolemia (CVP = 8–10 mm Hg; PCWP = 12–15 mm Hg). Negative fluid balance is associated with an adverse outcome. Isotonic crystalloids should be used for fluid management and normal saline (NS) is currently the recommended solution. However aggressive fluid resuscitation with NS may result in hyperchloremic acidosis. Dextrose 5% solutions should be avoided.

8.28.12 General Intensive Care

Raising the head of the bed to 30–45° will help reduce ICP and improves CPP. It also lowers the risk of ventilator-associated pneumonia (VAP). Keeping the head and neck of the patient in a neutral position also improves cerebral venous drainage and reduces ICP. It is important to avoid compression of the internal and external jugular veins from a tight cervical collar or tape. This can impede cerebral venous drainage and result in an increase in the ICP. Other measures include turning the patient regularly, providing eye care, mouth and skin hygiene, administrating a bowel regimen (to avoid constipation and increase of intra-abdominal pressure and ICP) and physiotherapy.

8.28.13 Cerebral Vasospasm

Symptomatic vasospasm after TBI is treated similar to vasospasm after SAH. Nimodipine appears to be effective in reducing the effects of vasospasm.

8.29 ICP Management

This is a critical aspect of neuro-intensive care. A raised intracranial pressure will reduce the cerebral perfusion and if this is prolonged the patient will quickly succumb. Close monitoring is therefore essential. This may include repeated CT of the head. If there is a large intracerebral haematoma which can be removed surgically, this is often the first measure to reduce the ICP. Recommendations in ICP management can be found in the Brain Trauma Foundation Guidelines. Following a traumatic brain injury, ICP management is commenced in the emergency department, with simple measures (keep the patient head up, avoid neck constriction and ensure that the cervical collar is not too tight). This is followed by ‘medical’ measures in severe head injuries, after any intracranial haematoma has been removed, or where there is no surgically treatable cause. These measures include

- Elevation of the head of the bed to 30 degrees
- Controlled ventilation to maintain a normal pO2 and low-normal pCO2.

- Sedation and muscle relaxants
- Mannitol or hypertonic saline
- Inotropes—maintain CPP by MAP elevation
- Barbiturates-lowers cerebral metabolism
- Hypothermia-lowers cerebral metabolism
- Control of seizures
- CSF drainage
- Decompressive craniotomy. This provides additional space for the brain to expand into. This is controversial and although it reduces ICP, it may result in an increased number of disabled or vegetative survivors. However so far, clinical and experimental data suggest this to be an effective treatment in reducing mortality in patients with refractory intracranial hypertension. Massive cerebral ischaemic infarction and traumatic brain injury are the most frequent indications for DC.

8.29.1 Intracranial Pressure Monitoring

ICP monitoring is now an integral part of the management severe TBI in most trauma centres. Patients with contusions or haematomas visible on head computed tomography scans, and Glasgow Coma Scale scores of 8 or less, may benefit from intracranial pressure monitoring. Many guidelines recommend that this should be performed in all salvageable patients with a severe TBI and an abnormal computed tomography (CT) scan. ICP monitoring allows earlier detection of an expanding intracranial lesion, provides guidance during therapy, enables drainage of cerebrospinal fluid (thereby reducing the ICP) and thus helps improve CPP. Currently available methods for ICP monitoring include various devices which can be sited in the extradural, subdural, subarachnoid, parenchymal and ventricular spaces. Ventricular catheters are preferred by many specialists as these are reported to be the most accurate, low-cost, and reliable method of monitoring ICP. They allow continuous monitoring and CSF drainage if required. Treatment for intracranial hypertension is generally started when the ICP exceeds 20–25 mm Hg (following CT to exclude any intracranial masses which may need surgical removal). Potential complications of ICP monitoring include infection, haemorrhage and malposition. If an external ventricular drain is used this is at risk of blockage and ventriculitis.

8.30 Decompressive Craniectomy and Hemicraniectomy

Decompressive craniectomy (DC) is a surgical procedure in which a large segment of the skull overlying the swollen brain is removed, thereby creating space to accommodate the increased cerebral volume (swollen brain). This results in a reduction of the ICP and an increase in the cerebral blood flow. The CPP and cerebral microcirculation, is thus improved which improves tissue oxygenation.

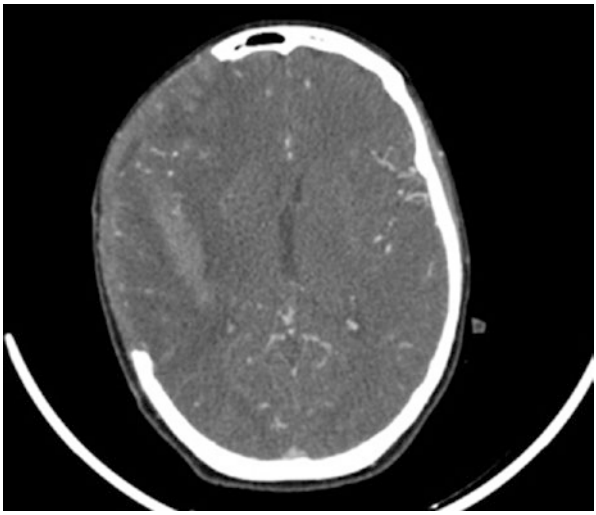


Fig. 8.25 Decompressive craniectomy

Decompressive craniectomy (DC) can be either bifrontotemporoparietal or a hemicraniectomy. It may be indicated in the management of intractable intracranial hypertension following failure of medical measures to control the ICP. It is performed as a life-saving procedure and must be undertaken within a few hours of the ICP becoming unacceptably high. However this is a major surgical procedure with significant morbidity and therefore many patients may not be suitable candidates. Nevertheless it is currently an often undertaken procedure in the management of elevated ICP. DC has also been used in the non-trauma setting, in the management of cerebral oedema after cerebral infarction, or following subarachnoid haemorrhage. DC has been shown to improve CPP, patient survival, as well as reduce the extent of infarction after extensive ischaemic stroke.

However decompressive craniectomy remains an area of controversy. Increased survival has also been associated with an increase in the number of survivors with moderate to severe disability (Fig. 8.25).

8.31 CSF Leaks

Leakage of CSF occurs in about 2% of all head injuries and up to 1/3 of all skull base fractures. CSF can leak from the nose (rhinorrhoea) or from the ear (otorrhoea). It can also occasionally pass into the orbit or through the scalp and into the eyelid, in which case there is no outward leakage. In very rare cases, CSF can pass through the orbit and exit via the eye, mimicking tear formation. This is called CSF oculorrhea and can be easily overlooked. Aside from trauma, other causes of CSF leaks includes intracranial or orbital tumours. Any erosive pathology can, in principle, eventually breach the bone and dura resulting in a leak. However patients usually present with other symptoms before the breach occurs. These less common

pathologies should be considered whenever there is an absence of trauma in the history.

Following trauma, CSF leaks are more commonly associated with fractures of the anterior cranial fossa. Here, the dura is tightly adherent to the underlying thin bones and is easily torn by displaced fractures (usually of the posterior wall of the frontal sinus). Orbital roof fractures can also result in CSF egress into the orbit. CSF fistulas are also commonly associated with the ethmoid and sphenoid sinus. Leaks originating from the ethmoid sinus may be caused by trauma (accidental or following surgery), or they can be spontaneous. A highly pneumatised sphenoid sinus may also predispose to leaks.

Most leaks develop either immediately or soon after trauma (within 48 hours). Many resolve spontaneously, the majority within the first 24 to 48 hours. It is also likely that many leaks may go unrecognised, especially in ICU patients. However delayed presentation can also occur. Nearly 95% of delayed leaks will manifest within 3 months of the injury. CSF rhinorrhea persisting for more than 7 days carries a significant risk of developing meningitis. Therefore patients who fail to respond to conservative measures often require surgery to prevent infective complications. Late onset CSF rhinorrhoea (after three months) following a closed head injury is rare, but it has been reported to occur several years later. Delay occurs because the dural defect is initially 'plugged' with herniated brain tissue, granulation tissue, or sinus mucosa. These tissues temporarily form a seal, although they do not provide a barrier against ascending infection. The herniated tissue itself also prevents the dura from healing naturally. Any subsequent displacement from this site (following a minor injury, coughing, sneezing etc) breaks in the seal resulting in rhinorrhea.

8.31.1 Other Causes of CSF Leaks

Iatrogenic causes account for about 16% of CSF rhinorrhea. These include endoscopic sinus surgery, skull base surgery, trans-sphenoidal pituitary surgery and craniofacial procedures. Other causes include erosion of the skull base by tumours, infection, mucocele and following irradiation. Any condition that increases the ventricular pressure may be important in the pathogenesis of CSF leaks. Congenital causes include developmental meningoencephaloceles. However spontaneous leaks are rare and may or may not be associated with a raised ICP. Sustained raised ICP is thought to result in remodelling with thinning of the skull base, ultimately resulting in bone erosion and the creation of an 'osteodural' defect in a pneumatised part of the skull base. Here, the bone is thin and can perforate allowing CSF to pass out through the nose. Spontaneous CSF leaks may therefore be a sign of idiopathic intracranial hypertension. Rare congenital causes of a CSF leaks include a failure of closure of the anterior neuropore (see chapter on embryology). This leads to herniation of the meninges and brain through the defect (encephaloceles). The most common causes of CSF otorrhea are fractures of, or following surgery to the temporal bone. Rarely spontaneous CSF otorrhea can occur from arachnoid granulations,

anatomic variations or dysplasias. Some cases are caused by middle ear and mastoid defects or inner ear malformations that act as a conduit for fluid. These can present in childhood or early adult life.

8.31.2 Diagnosing and Treating CSF Leaks

Clinical diagnosis is not completely reliable. However, when the patient presents with a watery discharge from the nose or ear following an injury to the head or upper face, it is often best to assume that this is CSF in the first instance. All patients attending the emergency department with a unilateral clear watery, salty tasting nasal discharge associated with nonspecific headaches should raise suspicions for CSF rhinorrhoea.

Confirmation of a fistula can sometimes be done by leaning the patient forward and observing for several minutes. In the acute phase, CSF tends to mix with blood and typically presents as a heavily blood-stained, watery discharge. In supine patients, this trickles down the side of the face. Peripherally along the track the blood tends to clot, while the non-clotted central blood is washed away by CSF stream. This creates two parallel lines referred to as "tramlining". Patients with a CSF leak and benign intracranial hypertension may also have bilateral papilloedema.

A simple bedside test for CSF is the "ring test" (allow a few drops to fall onto blotting paper—the blood clots centrally, whilst the CSF diffuses outwards to form a 'target sign'). Beta-2 transferrin assay is currently the gold standard for diagnosing the presence of CSF. This is a serum protein containing four negatively charged sialic acid groups. The molecule is found almost exclusively in CSF and therefore this is a highly specific test. Although CSF should also test positive for glucose, this is not a very specific or sensitive test and is generally not recommended. Interpretation of any positive results can be complicated by contamination with glucose-containing fluid (such as tears, nasal mucus, and blood). Furthermore, meningitis or other intracranial infections lower the concentration of glucose in CSF resulting in a false-negative result.

Once confirmed, the site of the leak must be identified. If there has been recent trauma, the site is often self evident on CT. With delayed presentation or non traumatic causes, high-resolution (HR) CT and MRI scans are generally the primary investigations of choice. Thin-cut CT can usually visualise the skull base in detail and may show small dehiscence and fractures. Fractures, air-fluid levels in nearby sinuses, and pneumocephalus are all highly suggestive of a dural tear, but these can be misleading as some bony defects may not coincide with the actual site of the leak. MRI is particularly useful in non-traumatic CSF leaks, in distinguishing tumours and infections. In patients with multiple skull base defects, MRI is also used to assess for the presence of meningoencephalocele.

Localisation may also be possible with nasal endoscopy, although this technique by itself is often insufficient, particularly in patients who have not undergone sinonasal surgery. Cisternography with intrathecal radioactive isotope is also a

well-established method of confirming and localising CSF fistulas. This uses water-soluble iodine contrast material. However, cisternography studies have variable sensitivities, radiation exposure, and poor anatomic localisation. Intrathecally injected fluorescein performed with or without blue-light endoscopy is another method for localisation, but this carries potential side effects including cardiac arrhythmias, seizures, headaches, and cranial nerve defects.

Initial conservative measures involve acetazolamide, laxatives, bed rest with the head elevated and avoidance of straining, such as sneezing etc. In some patients a lumbar drain may be useful. Conservative treatment maybe tried for 2 to 4 weeks. However if the leak does not settle by then, surgery is usually required. Repair of dural defects depends upon the site of the defect and its size. Both intracranial and/or extracranial approaches are possible, including endoscopic techniques. The dural defect can be closed using pericranium, fascia lata, or temporalis fascia with fibrin glue as a sealant. In selected cases endoscopic repair can provide excellent visualisation, precise graft placement, minimal damage to the surrounding tissues, preservation of olfactory function, shortened operating time and faster recovery time.

8.31.3 Orbital CSF Fistula (CSF Oculorrhea)

This is rare, but can occur as a result of trauma, intracranial or orbital tumours eroding into the subarachnoid space, or following surgery to the orbital roof. CSF can also leak into the orbit but remain contained within it or the eyelids as an orbitocele. This is also rare. Cranio-orbital fistulas should be considered in any patient presenting with an apparent fluid collection in the upper eyelid, or abnormal fluid leakage from a periorbital wound which is not lacrimation. There are several ways to differentiate tears from CSF. Posture is perhaps the simplest method. Sitting that patient forwards will raise the intracranial pressure and encourage CSF to leak from any wounds. CSF normally has approximately two thirds the serum glucose concentration, whereas tear glucose levels are insignificant. The fluid can also be tested for β2-transferrin as previously noted. An urgent CT will usually confirm the presence of a fluid collection and often the likely cause. Intracranial air is highly suggestive of a CSF leak. Depending on the cause and size of the defect, surgery may be necessary to repair damage to the orbit and/or cranium.

8.32 Spontaneous Cerebrospinal Fluid Leaks

This has been reported to occur in 5 out of every 100,000 people. Some patients chronically leak CSF despite repeated attempts at dural repair and patching. Spontaneous CSF leaks are sometimes referred to as high-pressure leaks when and increased ICP contributes to the development of the CSF leak. Idiopathic

intracranial hypertension (IIH) has been increasingly recognised as a cause of spontaneous CSF leakage. Patients who develop spontaneous leaks may develop sudden onset of acute headache, increasing throughout the day and often worse when the patient is upright. In addition patients may experience neck pain or stiffness, dizziness, nausea, and vomiting. On examination, leaking CSF can sometimes be seen through the nose or ear. The cause of spontaneous leaks is unknown, but may be because of an underlying connective tissue disorder or problems with the spinal venous drainage system. Early identification of these patients is important to prevent failure of any CSF leak repair and to prevent visual loss from papilloedema. Additionally, patients with IIH and chronically raised ICP need close follow up to watch for the development of a CSF leak.

8.33 Syndrome of the Trephined

Also called the "sinking skin flap syndrome" this is an unusual syndrome in which neurological deterioration occurs following craniotomy and removal of a large skull bone flap (Fig. 8.26). Symptoms can be related to posture. In severe cases there can be deterioration in sensorium with a sunken scalp flap and worsening midline shift. Management involves supporting the scalp by replacing the original bone (if kept), or cranioplasty (reconstruction using synthetic materials such as titanium or polymers).

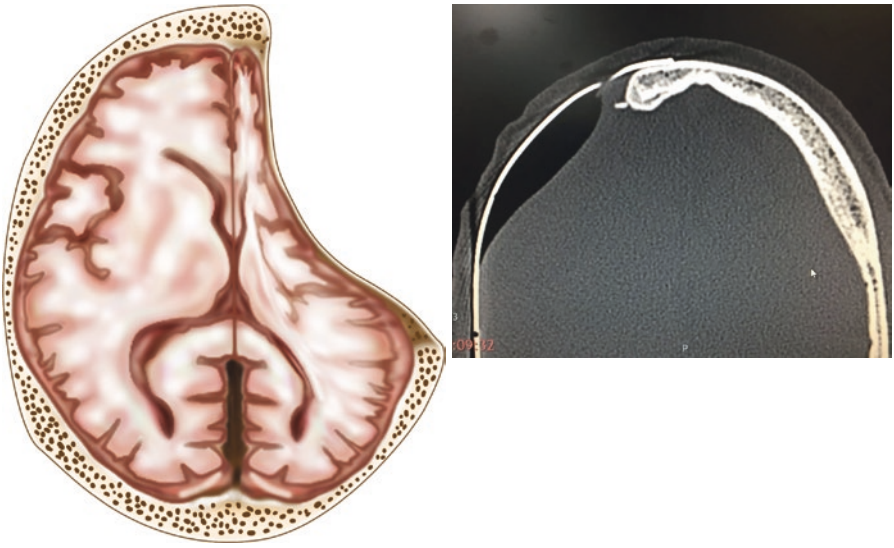


Fig. 8.26 Syndrome of the trephined

8.34 Brain Death

This is defined as the irreversible loss of all functions of the brain, including the brainstem. The patient has now permanently lost the potential for consciousness and the capacity to breathe. To establish the diagnosis there must be (i) coma, (ii) absence of brainstem reflexes and (iii) apnoea. Any patient who is diagnosed as brain dead is also legally and clinically dead. Diagnosis of brain death is primarily clinical. No other tests are required if full clinical examination, which includes two additional assessments of brain stem reflexes and an apnoea test are conclusive. Today, protocols exist to help effectively and quickly determine if a patient is 'brain dead'. Whilst these may vary slightly between centres, the main elements involve

(i) Determining the presence of a clear cause of and the irreversibility of coma (eg severe head injury, intracerebral haemorrhage, ischaemic brain insults, fulminant hepatic failure). Clinical and neuroimaging evidence of an irreversible CNS catastrophe should be confirmed.
(ii) Exclusion of any condition that might confound examination of cortical/brain stem function (e.g. Shock/hypotension, Hypothermia, Drugs known to alter neurologic, neuromuscular and electroencephalographic testing, severe underactivity of the thyroid gland, encephalitis, Guillain-Barre syndrome, some encephalopathies, and severe hypophosphataemia.
(iii) Complete neurological examination to confirm absence of spontaneous movement, decerebrate or decorticate posturing, seizures, shivering, response to verbal stimuli and response to noxious stimuli administered through a cranial nerve path way. Absent pupillary, corneal, oculovestibular (ice water), oculocephalic, cough and gag reflexes.
(iv) Failure of the heart rate to increase by more than 5 beats per minute after 1–2 mg of atropine intravenously. This indicates absent function of the vagus nerve and nuclei.
(v) Absent respiratory effort in the presence of hypercarbia (apnoea test)

During the examination spinal reflexes may be present. Viable cells in the spinal cord can also result in natural movements (known as Lazarus sign or Lazarus reflex) in otherwise brain-dead patients whose organs have been kept functioning by life support. Sadly, these body movements can cause false hope for family members.

8.34.1 Assessment of Brainstem Reflexes

(i) Pupils—no response to bright light (cranial nerve II and III)
(ii) No oculocephalic reflex (testing only when no fracture or instability of the cervical spine or skull base is apparent)
(iii) No deviation of the eyes to irrigation ice water in each ear
(iv) No corneal reflex

(v) No jaw reflex
(vi) No grimacing to deep pressure on nail bed, supraorbital ridge, or temporomandibular joint
(vii) No response after stimulation of the posterior pharynx
(viii) No cough response to tracheobronchial suctioning

After the first examination, the patient should be observed for a defined period of time (usually 6 hours). Longer intervals are advisable in young children. The examination should then be repeated. In some patients, skull or cervical injuries, cardiovascular instability, or other factors may make it impossible to complete all parts of the assessment. Confirmatory tests are then required. These may include Angiography, Electroencephalography, Nuclear brain scanning, Somatosensory evoked potentials, Transcranial doppler ultrasonography. Determination of brain death in children less than one year of age is different. This is because the brains of infants and young children have increased resistance to damage and can recover significant functions even after exhibiting prolonged unresponsiveness. Therefore a longer observation period is required. The patient must not be significantly hypothermic or hypotensive.

8.34.2 Vegetative State

This is different from brain death and can occur after extensive brain damage.

A patient in a vegetative state can show signs of wakefulness—they may open their eyes, but not respond to their surroundings. The important difference is that someone in a vegetative state still has a functioning brain stem. This means that (i) some form of consciousness may exist, (ii) spontaneous breathing is usually possible, (iii) there is a slim chance of recovery. A person who is brain dead has no chance of recovery, because their body is unable to survive without artificial support.