
The Acute Management of Head Injuries

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Cerebral Perfusion

The brain weighs 1500 g (2% body weight) and receives 15% of the cardiac output. It also uses 20% of the body's total oxygen consumption. Unlike other organs, there are minimal stores of oxygen and high energy phosphates, and these must be provided to the brain via the bloodstream. Hence, cerebral blood flow must be maintained. It can be described as a general equation of flow:

$$\text{Cerebral blood flow (CBF)} = \frac{\text{cerebral perfusion pressure (CPP)}}{\text{cerebrovascular resistance}}$$

The cerebral blood vessels are able to autoregulate, such that changes in CPP are matched by changes in cerebrovascular resistance, resulting in a constant cerebral blood flow. This occurs at the level of the arterioles. If CPP drops, these vessels dilate so that CVR drops proportionally, and CBF remains constant.

Cerebral perfusion can be affected if the arterial pressure is low and/or the intracranial pressure is high.

Cerebral perfusion pressure

$$= \text{mean arterial pressure} - \text{intracranial pressure}$$

Mean arterial pressure

$$= 1/3(\text{systolic pressure} - \text{diastolic pressure}) + \text{diastolic pressure}$$

Autoregulation maintains CPP over a range of mean arterial pressure levels between 60 and 150 mmHg.

The autonomic nervous system affects the range of CPP over which autoregulation works. Patients with chronic hypertension have the range set upwards, so care must be taken when lowering the blood pressure of a patient with chronic hypertension.

The maintenance of cerebral perfusion underlies the acute management of head injuries. Primary damage (i.e. damage occurring at the scene of the accident) has already occurred, and cannot be influenced by the clinician. The prevention of secondary damage, from hypoxia, hypotension and raised intracranial pressure, gives the patient the best chance of recovery.

Raised ICP

Raised intracranial pressure can occur from an expanding haematoma (extradural, subdural, intracerebral) or from brain swelling.

The skull can be considered as a “box” containing the intracranial contents. These include the meninges, the neurones, the glial cells, the blood vessels, the extracellular fluid (ECF) and the cerebrospinal fluid (CSF). The Monro-Kellie doctrine says that if a space-occupying lesion (such as a haematoma) is added to this “box”, the pressure will rise unless an equivalent volume is removed. However, this “box” opens into the spinal canal via the foramen magnum, and the initial displacement of an equivalent volume of CSF from the cranial cavity to the spinal canal allows compensation to occur. The intracranial pressure (ICP) remains normal until no more CSF can be displaced. Decompensation then occurs; the ICP rises rapidly, then cerebral blood flow decreases, and the patient loses consciousness.

Unfortunately, there is no easy method of determining when a patient is about to decompensate. Hence clinicians must be acutely aware of the possibility of the rapid deterioration of an alert patient with a space-occupying lesion.

Brain Herniation

Raised ICP can affect the brain by affecting cerebral blood flow or by causing brain herniation.

Brain tissue will herniate across an opening if there is a pressure gradient across this opening. This can occur in four situations:

(1) Cingulate or subfalcine herniation

The cingulate gyrus is part of the limbic system, and clinical effects cannot be seen easily. But the distal part of the anterior cerebral artery can be compressed. This supplies the part of the motor cortex that controls the leg, and hence the patient develops contralateral leg weakness.

(2) Unilateral transtentorial or uncal herniation

The most medial part of the temporal lobe, the uncus, can herniate across the tentorial hiatus. Compression of the parasympathetic fibres that lie on the surface of the oculomotor nerve, results in an ipsilateral fixed dilated pupil. Compression of the midbrain leads to loss of consciousness. The posterior cerebral artery runs through the tentorial hiatus, and kinking of this vessel results in an occipital lobe infarct. The patient later notices a contralateral homonymous hemianopia that is usually macular sparing. The cerebral peduncle of the same side can be compressed, leading to a contralateral hemiplegia. However, the cerebral peduncle of the opposite side can also be compressed by the free edge of the tentorium, producing an ipsilateral hemiplegia. Post-mortem studies can show the groove on the peduncle, known as Kernohan's notch. This means that the side of the paralysis is less useful to localise the side of the problem. The side of the blown pupil is better.

(3) *Central transtentorial herniation*

This occurs when the cerebral hemispheres are so swollen that both temporal lobes herniate into the posterior fossa. In addition to bilateral fixed dilated pupils, coma, no limb movement and occipital lobe infarction, the perforators coming off the top of the basilar artery are stretched. These supply the hypothalamus, which consequently becomes ischaemic. Diabetes insipidus results, and this is often seen in the terminal stages of a severe head injury.

(4) *Tonsillar herniation*

If the posterior fossa pressure is high, the cerebellar tonsils can herniate through the foramen magnum, compressing the medulla. In addition to loss of consciousness, the cardiovascular and respiratory centres are affected, leading to a labile blood pressure and irregular breathing.

Cushing Reflex

When the hypothalamus is ischaemic due to poor brain perfusion, the sympathetic centre becomes more active, acting on the heart to increase cardiac output, and on the arterioles to increase peripheral resistance. Blood pressure rises as a protective reflex to increase cerebral perfusion. This rise in blood pressure activates the stretch receptors in the carotid sinuses, causing the normal reflex activation of the parasympathetic nervous system, with a bradycardia. Hence the Cushing reflex results in a rising blood pressure associated with a bradycardia. This is seen in the terminal stages of an acute head injury.

The Immediate Management of a Head Injured Patient

To prevent secondary damage from hypoxia and hypotension, resuscitate patient first, according to generally accepted principles, i.e.

- Airway
- Breathing
- Circulation

Thereafter a rapid neurological assessment is made, including:

- Glasgow coma score, which is an assessment of conscious level (see Fig. 1).
- Pupillary response.
- Pattern of limb movements (posturing, asymmetry).
- Brainstem reflexes (corneal, oculocephalic, gag).

The cervical spine is also assessed for possible injuries (see Figs. 2 and 3), because trauma that is sufficient to produce a severe head injury may also cause a spinal injury.

EYE OPENING

- 4 spontaneous
- 3 to speech
- 2 to pain
- 1 none

SPEECH

- 5 oriented
- 4 confused
- 3 inappropriate
- 2 incomprehensible
- 1 none

BEST MOTOR RESPONSE

- 6 obeys commands
- 5 localises to pain
- 4 withdrawal to pain
- 3 abnormal flexion (decorticate)
- 2 extension (decerebrate)
- 1 no movement

Total: 3 to 15

Fig. 1 Glasgow coma scale.

- Antero-posterior and lateral views.
- Open mouth view if C1 and C2 cannot be seen on a-p view.
- In multisystem trauma, lateral view as an initial screening investigation (but needs review after resuscitation and stabilisation as it does not exclude a cervical spine injury).⁴
- GCS 15 if:
 - neck pain or tenderness
 - neurological deficit not due to an intracranial or peripheral problem
 - mechanism of injury suggests a spinal injury
- GCS 14 or less if:
 - normal X-rays, but significant neck pain, lateral flexion and extension views done by the patient^{3,4}
 - normal X-rays but neurological deficit that might be caused by the cervical spine, X-ray entire spinal column (a-p and lateral views of thoracic and lumbosacral spine) and refer to specialist for admission^{3,4}

Fig. 2 Indications for cervical spine X-rays.²⁻⁴

- C7/T1 if not seen on plain X-rays (despite optimisation of shoulder position).
- C1 and C2 if GCS 8 or less, or if cannot be seen on a-p or open mouth view, to be done at time of CT head scan (need to specify on CT request form).

Fig. 3 Indications for CT scan of the cervical spine.^{2,3}

Any further investigations, including skull X-rays and CT brain scans, are ordered if there is a clinical suspicion of an intracranial injury, to confirm an intracranial haematoma(s) or brain swelling that may require specific medical and/or surgical treatment, in order to preserve cerebral perfusion. The indications for these tests depend on the likelihood of finding significant abnormalities.

Indications for Investigations and Admission

The following questions are most commonly asked:

- (1) When to do skull X-rays?
- (2) When to order a CT head scan?
- (3) When to order a CT head scan for epileptics or drunks?

- (4) When to allow home?
- (5) When to admit to the Observation Ward (if available)?
- (6) When to admit to the General Ward?
- (7) When to admit to the High Dependency Unit?
- (8) When to admit to the Intensive Care Unit?
- (9) When to intubate?
- (10) When to give mannitol?
- (11) When to hyperventilate?
- (12) When to give phenytoin?

When should skull X-rays be done?^{2,5}

A skull fracture increases the chance of an intracranial haematoma by 400 times, and historically, this has led to hospital admission to observe all patients with skull fractures. But the significance of a skull fracture, without looking at the clinical picture, is less useful because there will be subgroups where the clinical picture will make the chance of an intracranial injury low or high. Guidelines for skull X-rays taking into account the clinical presentation exist⁵ but with the widespread availability of CT scanners, these have been superceded by newer recommendations for CT brain scans.

Skull X-rays should include antero-posterior and lateral views, with the Towne's view for occipital trauma, and an oblique view for a suspected depressed fracture. It should be done for all patients with GCS 13 and 14, and those with GCS 15 if the following are present:

- (1) Mechanism of injury suggests a severe blow.
- (2) Full thickness scalp laceration or boggy haematoma.
- (3) Loss of consciousness (any period of time).
- (4) Loss of memory.
- (5) Vomiting.
- (6) Inadequate history.
- (7) Difficulty in clinical assessment, for example, alcohol intoxication, epilepsy, uncommunicative children.
- (8) Depressed fracture or foreign body suspected.

When should CT head scans be done?^{2,6,7}

CT scans have revolutionised the management of head injuries by identifying collections of blood that cannot be seen on plain X-rays and cerebral angiograms. The indications include:

- (1) All skull fractures.
- (2) Signs of skull base fracture.^a
- (3) Deteriorating conscious level.
- (4) Neurological signs.
- (5) Seizure.
- (6) Patients with GCS 15 with a persistent severe headache, persistent vomiting, and/or neurological signs.
- (7) Patients with GCS 13 to 14 and who fail to improve after four hours of observation.
- (8) Patients with GCS 13 to 14 who need a general anaesthetic for another reason, e.g. orthopaedic injury.
- (9) All patients with GCS 12 or lower.

CT head scans in an intoxicated or post-seizure patient²

This is a difficult group of patients to manage, as they would already be drowsy. They should be scanned if they have a GCS 13 to 14, and fail to improve after four hours of observation, or if any of the above indications are found.

Criteria for discharge from Emergency Department

All patients with GCS 15 with no indications for observation or admission, and all patients with GCS 15 with no neurological symptoms, can be allowed home. They should be told to return if they develop a severe headache, drowsiness, vomiting, seizure or a neurological

^aSigns of a skull base fracture include periorbital bruising, subconjunctival haemorrhage, epistaxis or CSF rhinorrhoea, mastoid bruising (Battle's sign), bloody or CSF otorrhoea.

deficit. Ideally, an “advice sheet” is given to a responsible second person, to watch for these warning signs.

Indications for admission to emergency observation ward or general ward

Patients are observed in order to detect evolving intracranial events, such as an expanding haematoma, that may require treatment. Such patients include those with:

- (1) Headache.
- (2) Non-specific dizziness.
- (3) Scalp haematoma, laceration, contusion, abrasion.
- (4) Loss of consciousness.
- (5) Soft tissue facial injury.
- (6) Vomiting.
- (7) Alcohol or drug intoxication.
- (8) Unreliable or inadequate history.
- (9) Age less than 3 (unless injury is very trivial).
- (10) Patients with bleeding tendencies e.g. anticoagulation, thrombocytopenia.

In the observation ward, the management should include:

- (1) Monitoring the GCS half hourly for two hours, then every hour thereafter.
- (2) Review at four hours (extra care of patients with bleeding tendencies).
- (3) Nil by mouth.
- (4) Intravenous hydration (if clinically indicated).

Indications for admission to general ward

Head injured patients with any of the following should be admitted for observation in the general ward:

- (1) Fracture on skull X-ray.
- (2) High speed injury.
- (3) Signs of skull base fracture.
- (4) Possible skull penetration or depressed skull fracture.
- (5) Suspected child abuse.
- (6) Patients on emergency observation ward who have been assessed by the senior doctor as requiring admission.

A CT brain scan should be ordered if indicated.

On the ward, the patient should be managed as follows:

- (1) Blood samples are taken for full blood count, and analysis of serum urea and electrolyte concentrations, prothrombin time and partial thromboplastin time.
- (2) Blood is taken for “Group and Save”.
- (3) The GCS is assessed half hourly for two hours, then every hour thereafter.
- (4) Hourly blood pressure (BP) and pulse rate.
- (5) Nil by mouth.
- (6) Intravenous infusion (if clinically indicated).

Indications for admission to high dependency area

Head injured patients with any of the following should be admitted to the High Dependency Unit (HDU) where closer monitoring is present:

- (1) Depressed (GCS 12 or less) or decreasing conscious level.
- (2) Multiple fractures.
- (3) Serious facial injury.
- (4) Post-traumatic seizure.
- (5) Severe co-morbidities.
- (6) CT brain scan findings of intracranial pathology with significant mass effect and/or signs of brain swelling (loss of grey/white differentiation, loss of perimesencephalic cisterns, loss of cortical sulci).

The management in the High Dependency Unit should include:

- (1) Obtaining blood samples for full blood count and the analysis of serum urea and electrolyte concentrations, prothrombin time and partial thromboplastin time.
- (2) Obtaining blood for “Group and Save”.
- (3) Obtaining a chest X-ray and a 12-lead electrocardiograph (ECG).
- (4) Monitoring the GCS half hourly for two hours, then every hour thereafter.
- (5) Hourly measurements of fluid input and urine output.
- (6) Continuous ECG, BP, pulse, arterial oxygen saturation monitoring.
- (7) Nil by mouth.

Indications for admission to intensive care unit

Head injured patients who require mechanical ventilatory support or who have cardiovascular instability requiring support, should be admitted to the intensive care unit (ICU).

The management in the ICU should include:

- (1) Obtaining blood samples for full blood count and analysis of serum urea and electrolyte concentrations, prothrombin time and partial thromboplastin time.
- (2) Obtaining blood for “Group and Save”.
- (3) Obtaining a chest X-ray and a 12-lead electrocardiograph (ECG).
- (4) Monitoring the GCS half hourly for two hours, then every hour thereafter.
- (5) Hourly measurements of fluid input.
- (6) Inserting a urinary catheter for hourly measurements of urine output.
- (7) Continuous monitoring of the ECG, arterial oxygen, and capnography.

- (8) Invasive monitoring of BP and pulse via an arterial line.
- (9) Nil by mouth.
- (10) Naso-gastric tube (oro-gastric if a skull base fracture is suspected).
- (11) Monitoring of peripheral temperature.
- (12) Insertion of central venous catheter and monitoring of central venous pressures (CVP).
- (13) Fluid replacement with normal saline (avoid 5% glucose).
- (14) Monitoring of blood glucose concentration every six hours.

The management of these patients should be adjusted judiciously to achieve the following:

- (1) Arterial oxygen partial pressure (PaO_2) greater than 60 mmHg¹.
- (2) Arterial carbon dioxide partial pressure (PaCO_2) at chosen range but not less than 30 mmHg¹.
- (3) Systolic BP greater than 90 mmHg¹.
- (4) Intracranial pressures (if measured) less than 20 mmHg¹.
- (5) Cerebral perfusion pressures more than 60 mmHg (if ICP is measured).
- (6) Blood glucose levels of 4 to 10 mmol/l.
- (7) Maintaining core temperature between 36 and 37°C.
- (8) No seizures.

Indications for intubation⁴

Head injured patients with any of the following should be intubated:

- (1) Severe facial fractures.
- (2) A GCS between 3 and 8.
- (3) Arterial oxygen partial pressures less than 60 mmHg.
- (4) Systolic blood pressure less than 90 mmHg.
- (5) Signs of transtentorial herniation (see below).
- (6) Suspicion of neurological deterioration not due to extra-cranial explanations.

Indications for mannitol¹

Mannitol is a high-molecular-weight sugar that can be administered to lower the ICP. By exerting an osmotic effect, extracellular fluid is removed from the brain.

Mannitol is indicated if there is evidence of transtentorial herniation or progressive neurological deterioration. The signs of transtentorial herniation include unilateral or bilateral pupillary dilatation, asymmetric pupillary reactivity, and abnormal motor posturing (decorticate, decerebrate or no movement).

If intracranial pathology with significant mass effect, and/or signs of brain swelling (loss of grey/white differentiation, loss of perimesencephalic cisterns, loss of cortical sulci) have been identified after a CT scan of the brain, mannitol therapy should also be instituted.

Mannitol should not be routinely administered for extradural haematomas (as the volume of haematoma may enlarge) unless signs of herniation are detected and the conscious level has deteriorated and the patient requires airway maintenance and ventilatory support, prior to surgical evacuation of the haematoma.

Mannitol is available as a 20% (20 g per 100 ml or 5 ml per g) solution and is administered at 0.5 g per kg per dose. A 70 kg patient would require 35 g (175 ml) of mannitol. As mannitol therapy will result in an osmotic diuresis, a urinary catheter should be inserted prior to starting mannitol.

Mannitol should be administered over 30 minutes. The onset of action is approximately 15 to 30 minutes, with maximal effect seen at 90 minutes. As the effect of each dose lasts for up to four hours, the administration of mannitol can be repeated every four hourly.

As mannitol therapy can result in a hyperosmolar state leading to renal hypoperfusion and renal dysfunction, fluid input and output should be monitored and balanced with the use of normal saline solution to avoid hypovolaemia. Daily serum urea and electrolytes concentrations should be monitored, and the serum osmolarity kept below 320 mOsm. The serum osmolarity should be routinely monitored after two days of therapy, and sooner if renal impairment is suspected.

Mannitol is contraindicated if there is congestive cardiac failure or pulmonary oedema.

Indications for hyperventilation (PaCO₂ 30 to 35 mmHg)¹

The cerebral blood vessels constrict when the arterial CO₂ is lowered, leading to decreased cerebral blood volume in the cranium, lowering ICP.

After intubation, ventilate with tidal volume of 8 ml per kg, 10 breaths per minute, 100% oxygen. Arterial blood gases should be measured after 15 minutes of initiating mechanical ventilation, and the PaCO₂ should be maintained between 35 to 40 mmHg.

With transtentorial herniation or progressive neurological deterioration, the minute ventilation should be adjusted to maintain PaCO₂ at between 30 to 35 mmHg. Hyperventilation should also be done if the ICP is higher than 20 mmHg, despite mannitol therapy (and surgical decompression or a repeat CT scan of the brain should be considered if this is unsuccessful).

Indications for phenytoin¹

Phenytoin therapy is indicated in head injured patients with increased risk of developing early (within first week) post-traumatic seizures. These patients may have any of the following:

- (1) Seizure within 24 hours of injury.
- (2) A GCS of less than 10.
- (3) Penetrating head wound.
- (4) Depressed skull fracture.
- (5) Extradural haematoma.
- (6) Acute subdural haematoma.
- (7) Cerebral contusion.

The adult loading dose is 15 mg/kg (1050 mg for a 70 kg patient). The physician should administer the initial intravenous dose of 500 mg

at a rate of not more than 50 mg per minute with ECG and BP monitoring. The dose is repeated after 12 hours. The side effects to expect include cardiovascular and central nervous system depression, with arrhythmias, hypotension, cardiovascular collapse and respiratory arrest.

The adult maintenance dose thereafter is at 4 mg/kg/day (280 mg for a 70 kg patient). Monitor the serum phenytoin concentrations after two days. If the dose is 300 mg or more per day, increase at 30 mg intervals. The therapeutic serum level is between 10 and 20 $\mu\text{g/ml}$, and at this level, a small dosage change can result in a large level change so that any increments should be small.

Neurosurgical Treatment

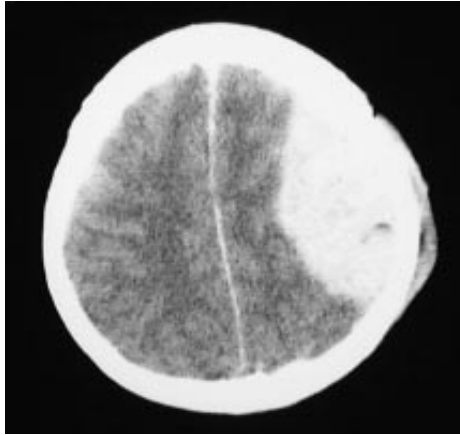
Medical treatment options include:

- (1) Mannitol therapy (see above).
- (2) Hyperventilation (see above).
- (3) Hypertensive therapy to maintain cerebral perfusion pressure above 60 mmHg if other measures to lower ICP are unsuccessful.
- (4) Barbiturate therapy to suppress cerebral metabolism. However, there are potential problems with hypotension, and its efficacy is not clearly established.

Surgical options include:

- (1) The insertion of an ICP monitor (to allow measurement of cerebral perfusion pressure).
- (2) The insertion of an external ventricular drain (to allow drainage of CSF to lower ICP).
- (3) Surgical evacuation of haematomas (extradural, acute subdural, intracerebral contusions).
- (4) Decompressive craniectomy.

This is best left to the neurosurgeons to manage, and is outside the scope of this book.



CT scan of an extradural haematoma.

Specific Traumatic Intracranial Haemorrhages

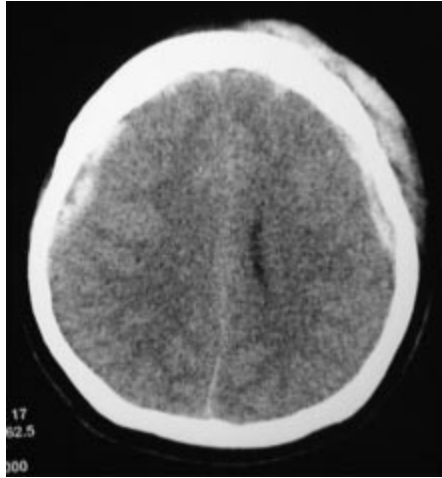
Extradural haematomas

This is usually associated with a skull fracture. Bleeding occurs into the potential space between the dura and the skull. This may take time to occur, and a lucid interval may be found, before clinical deterioration occurs. Because the pathology is essentially outside the brain, the prognosis is good provided the haematoma is evacuated soon enough.

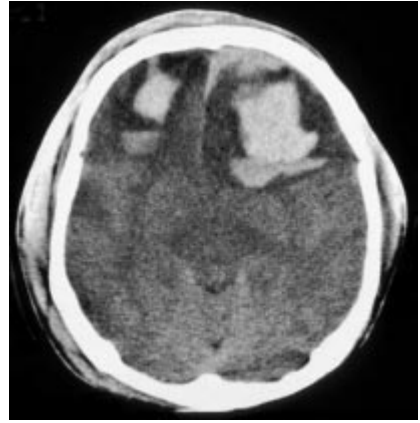
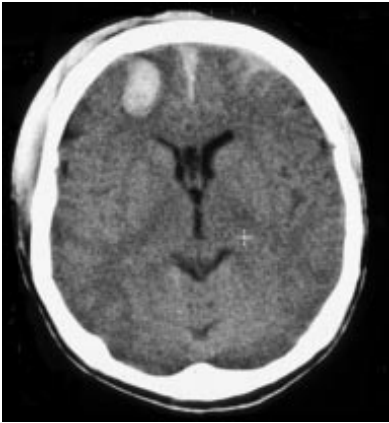
Acute subdural haematomas

Bleeding occurs into the subdural space, lying between the dura and the arachnoid. This can occur in two circumstances:

- (1) Bleeding from a torn bridging vein, especially in the elderly. This can sometimes occur slowly so that there is a lucid interval. Prognosis can be good if the clot is evacuated early.
- (2) Bleeding from underlying damaged brain matter. The prognosis depends on the underlying brain damage, as well as any delay in evacuating the clot.



CT scan of bilateral acute subdural haematomas.



CT scan of a brain contusion: on first day, and after 24 hours.

Intracerebral contusions

These typically occur in the frontal and temporal lobes, probably due to the bony prominences in the floor of the anterior and middle cranial fossae. They begin as small haemorrhages which coalesce into larger ones with mass effect. Together with the surrounding oedema that

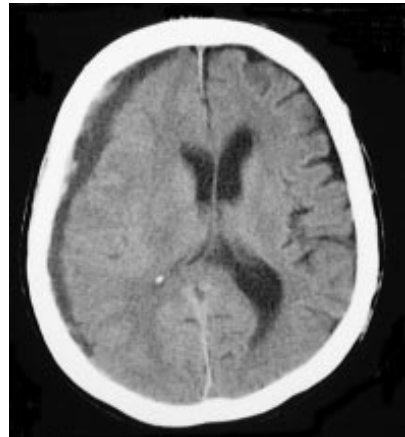
develops after 24 hours, delayed clinical deterioration can occur. The prognosis depends on the severity as well as the location of the haemorrhages.

Diffuse axonal injury

This refers to diffuse injury to the brain, with resultant generalised brain swelling without any large haemorrhages. The prognosis is variable as the damage to the brain cannot be localised. Long-term cognitive problems are common.

Chronic subdural haematomas

A subset of acute subdural haematomas begin to enlarge. A membrane surrounds these acute subdural clots, and recurrent bleeding from this membrane is the cause of the enlargement. It is postulated that the breakdown products of clot lysis inhibits platelet aggregation. Enlargement of the haematoma due to osmotic absorption of fluid may also contribute. Prognosis is usually good provided the haematoma is evacuated soon enough, usually via some burr holes.



CT scan of a small asymptomatic right acute subdural haematoma that became a symptomatic chronic subdural haematoma after 6 weeks.

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