HEAD INJURY

After reading this chapter you will be able to answer the following questions:

- What is the importance of head injury?
- What is the pathophysiology of head injury?
- How is the severity of head injury assessed?
- What is the treatment of head injury?

Importance

Head injury is common following blunt trauma such as a motor vehicle accident or fall. Indeed, head injury is the commonest cause of death in those who survive to reach hospital following serious blunt trauma. About 50% of blunt trauma deaths result from head injury.

In those who die at scene hypoxia is a common avoidable cause (Royal College of Surgeons of England, 1988) and up to 39% of pre-hospital deaths following trauma may be avoidable (Hussain and Redmond, 1994). Head injury is a significant contributing factor, as even a mild 'concussion' may result in temporary airway compromise and death from hypoxia.

Pathophysiology

Head injury is caused by:

- blunt trauma, or
- penetrating trauma

Blunt trauma results in **direct (primary)** injury in the brain underneath the area of impact ('coup' injury), and opposite the area of impact ('contra coup" injury). The brain has a degree of mobility and will accelerate away from the impact. causing contusion where the brain on the opposite side then impacts with the skull.

Penetrating trauma results in **direct** injury in the brain along with path of the missile. This is the **permanent cavity.** If a wound is associated with a high energy transfer (as is common when a missile strikes bone) then there may be an additional phenomenon of **temporary cavitation,** when the tissues continue to move away from the permanent cavity for a few milliseconds after the missile has passed through. This produces severe disruption remote from the wound track. The brain is particularly susceptible to temporary cavitation.

Indirect (secondary) injury occurs after the initial blunt or penetrating insult. This will often be a result of hypoxia, which in turn may be because of an untreated airway obstruction, an uncorrected problem of ventilation, or uncorrected hypovolaemia. Hyperthermia and hypoglycaemia are also important causes of secondary injury.

The final common pathway for hypoxia is **raised intracranial pressure,** as a result of cerebral oedema. Compensatory mechanisms exist to combat early rises in the intracranial pressure which include a reduction in cerebral vascular and cerebrospinal fluid volume. Once these mechanisms fail a small rise in volume will produce a rapid rise in pressure.

The brain is surrounded by a rigid box and there is no flexibility (except in infants less than 12-18 months who have patent sutures or fontanelles) for the expanding brain. Brain is therefore extruded, or "herniated", through any available opening. The cerebral hemispheres are separated from the cerebellum by a fibrous sheet (the faix cerebri) which is pierced centrally by the brainstem. The small gap that surrounds the brainstem is an area for brain herniation which first occurs on the same side as the expanding intracranial haematoma. it is the uncus of the temporal lobe that herniates and it compresses the brainstem at the level of the third cranial nerve nucleus (CN III controls pupillary constriction) which results in a dilated pupil on the **SAME** side. Motor signs may occur on the **OPPOSITE** side (because motor fibres cross over in the brainstem).

Assessment

The hallmark of brain injury is an altered level of consciousness. However, an altered level of consciousness does not necessarily imply serious head injury as hypoxia from airway, breathing or circulatory compromise will all result in a reduced level of consciousness.

The level of consciousness is assessed in the primary and secondary surveys. In the primary survey the "AVPU' system is used:

A	Alert
V	Responds to Voice
P	Responds to Pain
U	Unresponsive

In addition an assessment is made of pupillary size, and in children particularly it is useful to look for changes in posture in response to painful or noise stimuli. There is little need to be able to differentiate between the 'decerebrate' or decorticate' postures as both indicate the presence of severe brain injury.

In the secondary survey the level of consciousness is assessed using the Glasgow Coma Scale, first described by Teasedale and Jennett in 1974. This is a reproducible scale (one operator will get the same result as another on the same patient) that equates with the severity of brain injury. The scale has three components; best motor response, best verbal response, and best ocular response. The maximum score is 15, which is 'normal' and the minimum score is 3. The relationship to injury severity is a follows.

15	Normal
13-14	minor head injury
9-12	moderate head injury
3- 8	severe head injury ("coma")

The components of the scale in detail are:

Motor response

Obeys commands	6
Localises to pain	5
Withdraws from pain (normal flexion)	4
Abnormal flexion to pain	3
Extension to pain	2
None	1

Verbal response

Normal speech	5
Confused speech	4
Inappropriate words	3
Incomprehensible sounds	2
None	1

Ocular response

Open spontaneously	4
Open to command	3
Open to pain	2
None	1

When localising signs (unilateral brain injury producing unilateral motor weakness) are present, the highest score obtainable for motor function is to be recorded.

The Glasgow Coma Scale is not suitable for small children whose speech is incompletely developed, and for this reason a Paediatric Coma Score has been developed with a separate verbal element. The PCS should be used for children of less than 4 years.

Verbal response

Smiles, interacts	5
Cries, but consolable	4
Cries, and occasionally consolable	3
Inconsolable crying	2
None	1

Further assessment in hospital will depend on local resources. A skull fracture seen on plain x-ray greatly increases the likelihood of an intracerebral haematoma (to 1:4 when there is an altered level of consciousness). Patients with a linear skull fracture should be admitted for observation and if there is an altered level of response early consideration should be given to obtaining a CT scan. patients with a depressed skull fracture should be discussed with a neurosurgeon. Where computed tomography is available this is the investigation of choice to exclude intracerebral pathology and to plan the patient's management. In general, CT scanning following head injury should follow these simple rules:

Fall in 2 or more points GCS	Immediate scan
GCS <9	Immediate scan
GCS 9-12	Early scan
Lateralising signs	Early scan
GCS 13-14	Scan after 4 hours observation, if still abnormal.

It should be remembered that the radiology department is a dangerous place, often remote from the emergency department and with poor resuscitation facilities. Careful consideration should be given to elective ventilation of patients who are vomiting, or who have reduced protective airway reflexes. Agitation from cerebral irritation is a relative indication for elective ventilation, if the patient is to remain still for the examination.

Treatment

Nothing can be done to reverse the primary brain injury. The main objective of treatment is to avoid secondary injury.

Hypoxia is treated by ensuring a clear, secure airway, providing supplemental oxygen, and by optimising ventilation. External blood loss must be stopped and hypovolaemia corrected. Quite simply, the priorities for treating a head injury are **airway, breathing and circulation.**

A patient with a GCS <9, that is a "comatose" patient, require early protection of the airway and elective ventilation. Ten minutes from arrival at hospital is a reasonable time in which to aim to achieve this.

Early evacuation of extradural and subdural haematoma will reduce mortality and morbidity. This is usually the province of the neurosurgeon who will raise a flap of skull to facilitate this. In a rural trauma setting there may only be surgeons with general surgical skills, and in this case burr holes may be performed. burr holes may fail to locate the haematoma, may not be life saving, and may cause further injury. but they may be the only option. A patient with a dilated pupil caused by an expanding intracranial haematoma will not survive transfer to a neurosurgical unit and requires an immediate surgical attempt to decompress the haematoma.

The common pathway for death following diffuse brain injury or an expanding haematoma is a rise in intracranial pressure. This can be minimised by careful positioning (keep the head in a neutral position, ideally 30 degrees head-up to aid venous drainage) and careful handling (avoid laryngoscopy without adequate anaesthesia). Fluids must be given to replace hypovolaemia, but should otherwise be infused judiciously. Induced hypocapnia by hyperventilation is recommended. Diuretics, and specifically mannitol 1g/kg IV, should only be given after discussion with the operating surgeon.

Summary

Head injury is common following blunt trauma, and is the leading cause of death following blunt trauma in those who survive to reach hospital. Injury may be blunt or penetrating, and direct (primary) or indirect (secondary).

The severity of head injury is assessed in the primary survey by AVPU + Pupils, and in the secondary survey by the Glasgow Coma Scale. Plain skull x-rays may be used to predict the likelihood of intracranial haematoma, but anatomical definition of injury can only be defined by CT scanning.

The aim of treatment is to avoid secondary injury. Initial priorities are to the airway, breathing and circulation.