

Assessment and management of the head-injured patient

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Head injury is one of the most important causes of serious morbidity and mortality in young adults. Each year in Britain, there are approximately 5000 deaths from serious head injuries. Appropriate multidisciplinary assessment and management of systemic and intracranial pathology can significantly improve the outcome.

Each year approximately one million people in the UK sustain a head injury severe enough for them to attend accident and emergency departments (Royal College of Surgeons of England, 1999). The majority of these are discharged without any treatment. However, approximately 180 000 people are admitted to hospital (Lindsay and Bone, 1997) (Table 1). In most cases, a period of observation to exclude delayed complications will suffice, but some will require neurosurgical management. Many of these patients are young adults, and any long-term disability arising from the head injury will therefore have devastating personal, social and economic consequences.

In the UK, the most common causes of serious head injuries are road traffic accidents (60% of all deaths), assault, falls, sports injuries and accidents at work or home. The relative frequency will vary with occupation, gender and age, but alcohol is commonly implicated (Lindsay and Bone, 1997). Enforcing use of seatbelts and crash helmets, and safety at work legislation have played a major part in the prevention of severe head injuries.

Brain injury can be primary (irreversible changes occurring at the time of injury) or secondary (cerebral damage as a result of systemic or intracranial pathological derangement occurring after the initial trauma) (Table 2). The management of head-injured patients aims to minimize secondary damage caused by either intracranial pathology or systemic problems, such as hypoxia or hypotension. In this article, the management of more serious adult head injuries is discussed.

PATHOLOGY

Most head injuries are closed, although open injuries can occur as a result of penetrating objects, in conjunction with depressed skull

fractures or associated with skull base fractures. Primary brain injury may result from a number of pathological mechanisms, often occurring together.

Direct injury may be a result of cerebral contusion, which often occurs opposite the site of impact because of a *contre-coup* effect. More severe contusions are often associated with lacerations, which may be caused by movement of the brain across bony ridges on the inner aspect of the skull. A contused, lacerated lobe is often referred to as a burst lobe. The brain may also be directly injured by a penetrating object, but although the clinical and radiological picture may be dramatic, the degree of brain injury is usually less significant than with blunt trauma, as less energy is dissipated within the brain.

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TABLE 1.
Indications for hospital admission and for computed tomography scanning of head-injured patients

Action	Indication
Admission	Transient or persisting loss of consciousness
	Post-traumatic amnesia
	Persisting nausea or vomiting
	Difficulty with assessment, e.g. alcohol
	Other medical conditions, e.g. coagulopathy
	Lack of responsible adult at home
	If in doubt, admit
Computed tomography scan	Reduced score on the Glasgow Coma Scale, including confusion
	Neurological deficit
	Epileptic seizure
	Skull fracture
	Cerebrospinal fluid leak

Rapid deceleration of the head at the time of impact will cause the brain to move within the head causing shearing and tearing of the long axon tracts. Although the radiological changes associated with diffuse axonal injury may be quite minor, there are often severe functional effects.

Over the following days, cerebral swelling will often occur as a result of vascular engorgement and oedema (because of accumulation of intracellular or extracellular fluid). Localized oedema will be manifest radiologically as an area of hypodensity.

INITIAL MANAGEMENT OF SEVERE HEAD INJURY

The diagnosis of head injury will normally be evident, and care must be taken, at all stages of management, not to ignore systemic problems that might require urgent treatment. The patient should be assessed immediately for evidence of cardiorespiratory insufficiency, and this should be dealt with as a priority. At the first opportunity, pupillary reactions and limb movements should be recorded. Conscious level should be assessed using the Glasgow Coma Scale (GCS) (Table 3). A survey for systemic injuries should also be performed, including radiological imaging of the cervical spine (if there is any doubt as to the stability of the spine, it should be immobilized). If the airway or breathing is compromised, or if the GCS is eight or less, the patient should be intubated, with sedation if necessary.

Once the patient has been stabilized, a computed tomography (CT) scan of the brain should be performed (Bartlett et al, 1998) (Table 4). By this stage, if the scans show significant pathology, the neurosurgical team will have become involved, and further management will depend on the specific situation. Transfer to a distant

neurosurgical unit carries risks for the ventilated patient, who should therefore be accompanied by an experienced anaesthetist, with adequate monitoring (Association of Anaesthetists of Great Britain and Ireland, 1996). If possible, any scalp laceration should be sutured in the accident and emergency department to reduce the risks of infection and blood loss.

NEUROSURGICAL MANAGEMENT: GENERAL ISSUES

Each patient is unique and will need individual consideration, but as a minimum, all neurosurgical patients require careful monitoring and supportive therapy to maintain appropriate blood pressure, blood gases, fluid balance and electrolyte levels. Table 5 outlines a basic list of parameters that should be monitored. Time must be made to talk to the relatives to try to explain the problems, aims of treatment and prognosis.

NEUROSURGICAL MANAGEMENT: SPECIFIC SITUATIONS

Unventilated, non-surgical patients

Patients with a mild impairment of GCS and with normal CT scans will usually be managed conservatively. Regular neurological observations should be undertaken until the patient recovers. Sedative drugs should be used extremely cautiously. In a few cases, a CSF leak may occur, either from the nose (rhinorrhoea) or ear (otorrhoea), caused by a basal skull fracture. In most cases, the leak will settle after a few

TABLE 2.
Causes of secondary brain injury

Category	Specific mechanism	Comment
Cerebral tissue changes	Oedema	Causes raised intracranial pressure
	Local ischaemia	
	Free radicals	
	Excitotoxicity	
Intracranial pathology	Haematoma	Extradural, subdural, intracerebral
	Infection	
	Hydrocephalus	
	Cerebral herniation	
Systemic problems	Hypotension	
	Hypoxia	
	Pyrexia	

Note: these mechanisms can occur simultaneously and often interact in a complex fashion

TABLE 3.
Glasgow Coma Scale

Category	Response	Score
Eye opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Speech	Orientated	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
	None	1
Motor	Obeys commands	6
	Localizes pain	5
	Flexion to pain, normal	4
	Flexion to pain, abnormal	3
	Extension to pain	2
	None	1
Total	Minimum-maximum	3-15

days of observation, but in some cases, a lumbar drain may be helpful. If the leak persists, a surgical repair of the dural defect may be needed.

Ventilated, non-surgical patients

Significant and persistent depression in conscious level with a CT scan showing no mass lesion requires continued ventilation. The patient will normally be sedated, and intracranial pressure (ICP) monitoring provides fundamental management information. The intracranial pressure follows a wave pattern related to the intravascular pattern. Monitors will integrate this information to give a single, average ICP reading. Normal ICP readings will range up to approximately 15 mmHg, but a prolonged rise can be an indication of evolving intracranial pathology. ICP monitoring can therefore provide early warning of a treatable lesion, such as an intracranial haematoma, which might otherwise remain undetected. In addition, there is evidence that the maintenance of ICP below 20 mmHg and a cerebral perfusion pressure (CPP) above 70 mmHg will improve prognosis.

ICP can be optimized by careful fluid and electrolyte management and by the avoidance of hypercapnoea, fever or pain. In cases where the ICP rises persistently, in spite of all efforts, mannitol may be of benefit. Mannitol acts as an osmotic diuretic and improves local cerebral blood flow, but if given too frequently, it can cause dehydration and ultimately renal failure. In certain defined situations, a raised ICP can be treated by artificially lowering the PCO_2 . If necessary, the CPP can be raised by the administration of inotropic drugs. Barbiturates or induced hypothermia are advocated by some, but are not in general use. If all medical methods are exhausted and the ICP remains critical, consideration may be given to either inserting an external ventricular drain to reduce the volume of intracranial CSF or performing a lobectomy or craniectomy.

At present, there is no clear evidence of benefit from neuroprotective drugs, but trials are underway to assess the role of steroids and a number of other agents (Yates and Roberts, 2000).

Extradural haematoma

A haematoma arising in the space between the dura and the skull is a neurosurgical emergency, and with prompt surgical evacuation, the outcome can be excellent. The classical clinical pattern, often absent, is of a brief initial loss of consciousness, followed by progressive deterioration in the GCS. This pattern can be understood in terms of the pathology of extradural haematomas. There is an initial concussion, but as the brain injury may

TABLE 4.
Initial investigations following a severe head injury

	Investigation	Comments
Mandatory radiological investigations	Head computed tomography	
	X-rays	Lateral C-spine
		Chest X-ray
		Pelvis
Mandatory blood tests	Full blood count	
	Urea and electrolytes	
	Clotting	
	Group and save	
	Blood gases	
Discretionary radiological investigations	X-rays of spine, limbs or face	According to clinical findings
	Computed tomography of spine	If plain X-rays are inconclusive or if further information about a fracture is required
Discretionary blood tests	Alcohol	
	Drugs	
	Anticonvulsant levels	
	Calcium	
	Liver function tests	

TABLE 5.
Monitoring a head-injured patient

Parameter	Comments
Pupils	Pupillary size and reactions Fixed and dilated pupils may be caused by transtentorial herniation
Glasgow Coma Score	Should be assessed even in ventilated and sedated patients
Limb function	Unilateral weakness is usually a result of injury of the opposite cerebral hemisphere, but in some cases, the weakness may be ipsilateral because of pressure at Kernohan's notch. Therefore, hemiparesis is not a reliable indicator of the location of pathology
Pulse and BP	In some cases, intermittent non-invasive monitoring will be adequate, but in others arterial monitoring may be required. A rise in BP with falling pulse rate was recognized by Harvey Cushing (the pioneering early 20th century American neurosurgeon) as being an indicator of rising ICP. However, this sign is unreliable and undue significance should not be attributed to either its presence or absence
Cerebrovascular pressure	Advisable where intensive management is undertaken
Blood gases	Monitor regularly in ventilated patients
Electrolytes	Hyponatraemia is a common complication in neurosurgical patients and electrolytes should therefore be checked daily
FBC	Check frequently at the time of admission or after surgery
CT scan	In ventilated patients, the transfer to the CT scanner involves some risks and should be undertaken only when there is a specific indication
ICP	Important in ventilated patients (see text)
CPP	CPP = BP-ICP
Others (new types of monitoring)	Jugular bulb O_2 saturation (SjvO ₂) can provide useful information to help guide decisions on ventilation
	Intraparenchymal O_2 saturation
	Intraparenchymal temperature

BP = blood pressure; CPP = cerebral perfusion pressure; CT = computed tomography; FBC = full blood count; ICP = intracranial pressure

be insignificant the conscious level quickly recovers. Most extradural haematomas involve a skull fracture that has torn an extradural artery, classically the middle meningeal, and an expanding blood clot will therefore cause a progressive increase in the ICP and hence the delayed clinical deterioration. A CT scan will show a lentiform haematoma (*Figure 1*). If the clot is removed promptly by a craniotomy, the patient can often be extubated soon after surgery. Of course, if surgery is delayed or there was significant direct brain injury at the time of trauma, the outlook is more variable. In a few cases, where the clot is small and amounts to no more than a fracture haematoma, the patient may be managed conservatively providing he or she is carefully monitored.



Figure 1. Computed tomography scan showing a lentiform extradural haematoma with midline shift.



Figure 2. Computed tomography scan demonstrates a large, acute subdural haematoma with severe midline shift.

Subdural haematoma

This is often associated with significant brain injury and sometimes intracerebral haematoma. The bleeding is usually venous and tends to spread further over the surface of the brain than extradural haematomas (*Figure 2*). If the haematoma is causing compression of the brain, urgent craniotomy is required. Since there is usually associated cerebral injury, these patients will usually be kept sedated and ventilated post-operatively until the ICP has settled.

Intracerebral haematoma

Post-traumatic intracerebral blood can vary from small contusions through to large, well-defined haematomas (*Figure 3*). Contusions may mature into larger blood clots in the first few days after injury, and there should therefore be a low threshold for rescanning. In cases where the haematoma is small and neurological function good, management will usually be conservative. In other cases, initial management will be with ventilation and ICP monitoring, but if there is a deterioration, the haematoma may need surgical evacuation.

Depressed skull fractures

In the absence of a significant intracranial haematoma, a depressed skull fracture (*Figure 4*) will only require neurosurgical intervention if there are cosmetic implications or, more commonly, if the fracture is compound. In this situation, an assessment is made as to whether the depressed fracture is more than the skull thickness, in which case the dura is likely to be

Figure 3. Computed tomography scan of a patient with a large, left temporal lobe haematoma and multiple contusions in both frontal lobes.



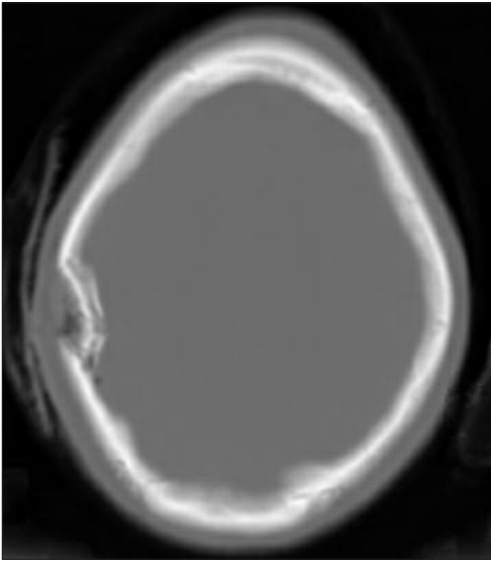


Figure 4. Computed tomography scan (bone window) showing a depressed skull fracture.

breached, increasing the risk of an intracerebral abscess. If the injury is less than 24 hours old, the skull fragments should be elevated to allow debridement of the underlying brain.

OUTCOME

A poor outcome following head injury is associated with:

- Older age
- Poor initial neurological status
- Systemic complications or illness
- Amnesia (particularly if post-traumatic amnesia is of more than 24 hours duration)
- The presence of subdural or intracerebral haematomas, rather than extradural haematomas.

In cases where no signs of cerebral activity can be detected after sedation is reversed, brainstem tests should be performed before the ventilatory support can be withdrawn.

Head injuries may be associated with post-traumatic hydrocephalus (which may require a shunt procedure) or epilepsy, particularly following compound depressed skull fractures. As well as neurological deficits, a variety of neuropsychological sequelae may occur including anxiety, poor memory, poor concentration and tiredness. Rehabilitation is important in helping patients to maximize their recovery.

CONCLUSIONS

Although head injury remains a potentially devastating condition, appropriate multidisciplinary management of systemic and intracranial pathology can significantly improve outcome. **HM**

Conflict of interest: none.

- Association of Anaesthetists of Great Britain and Ireland (1996) *Recommendations for the Transfer of Patients with Acute Head Injuries to Neurosurgical Units*. Association of Anaesthetists of Great Britain and Ireland, London
- Bartlett J, Kett-White R, Mendelow AD, Miller JD, Pickard J, Teasdale G (1998) Guidelines for the initial management of head injuries. *Br J Neurosurg* **12**: 349–52
- Lindsay KW, Bone I (1997) *Neurology and Neurosurgery Illustrated*. 3rd edn. Churchill Livingstone, Edinburgh
- Royal College of Surgeons of England (1999) *Report of the Working Party on the Management of Head Injuries*. The Royal College of Surgeons of England, London
- Yates D, Roberts I (2000) Corticosteroids in head injury. *Br Med J* **321**: 128–9

KEY POINTS

- Head injury is one of the most important causes of serious morbidity and mortality in young adults.
- Prompt resuscitation to avoid hypoxia or hypotension is vital.
- Neurosurgical advice should be sought at an early stage in the management of severe head injury.
- Surgery to remove intracranial mass lesions, such as haematomas, must be undertaken as quickly as possible.
- Ventilated patients require careful management, and intracranial pressure monitoring may provide valuable information to guide treatment.