Traumatic brain injury: from impact to rehabilitation

Traumatic brain injury is a significant cause of mortality and morbidity in our society, particularly among the young. This review discusses the pathophysiology of traumatic brain injury, and current management from the acute phase through to rehabilitation of the traumatic brain injury patient.

Traumatic brain injury is defined as 'brain injury caused by trauma to the head, including the effects of possible complications of injury, notably hypoxaemia and hypotension, and intracerebral haematoma' (Medical Disability Society, 1988). Of all the injuries sustained by victims of trauma, head injury is most frequently associated with adverse outcomes, such as death, or in those that survive, major neurological deficits. The consequences of head injury are extensive, not only for victims and their families, but also for society as a whole. The economic costs of head injuries are high, as most traumatic head injuries occur in young people during their working years (Jennett and MacMillan, 1981). This review discusses traumatic brain injury from its pathological basis to its acute and chronic management, with particular focus on future advances.

Epidemiology

Approximately 1.4 million people suffer a head injury in the UK each year, with about 2500 of these suffering a severe head injury. The main causes of head injuries are falls, assaults and road traffic accidents, with the latter being associated with the greatest mortality. The outcome after traumatic brain injury ranges from death before hospital admission to minor injuries not resulting in presentation to a hospital. For those patients who do present to hospital, the type and severity of their injuries will determine immediate management and any subsequent intervention.

The Glasgow Coma Scale (GCS) (*Table 1*) is a widely accepted clinical method of assessing neurological status. In the context of traumatic brain injury the score is a useful method of assessing severity of injury. Head injuries have thus been classified as mild (GCS 13–15), moderate (GCS 9–12) or severe (GCS ≤ 8). The trend in GCS provides useful information – for example a sudden decrease after a lucid interval may indicate compression of vital structures in a patient with an extradural haematoma (the so-called 'talk then die' scenario), indicating the need for urgent surgical intervention. The GCS can also provide a rapid and accurate method of predicting outcome. When early GCS scores are used to classify injury severity or predict outcome, it is important to use post-resuscitation scores, since physiological derangements such as hypoxia and hypovolaemia may cause reversible exacerbations in neurological dysfunction.

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Normal physiology

The normal cerebral perfusion pressure is the difference between inflow and outflow pressures – mean arterial pressure and jugular venous pressure respectively. When intracranial pressure exceeds the jugular venous pressure, then the cerebral perfusion pressure is the difference between mean arterial pressure and intracranial pressure. Autoregulation maintains a constant cerebral blood flow between a cerebral perfusion pressure of 50 and 150 mmHg, achieved by alterations in cerebrovascular resistance. Outside this range cerebral blood flow is proportional to cerebral perfusion pressure. Under normal circumstances intracranial pressure is ≤10 mmHg, and

Table 1. Glasgow Coma Scale

Eye opening	4	Spontaneously
	3	To speech
	2	To pain
	1	None
Verbal response	5	Oriented
	4	Confused
	3	Inappropriate
	2	Incomprehensible sounds
	1	No response
Motor response	6	Obeys commands
	5	Localizes to pain
	4	Normal (flexion) withdrawal
	3	Abnormal flexion to pain
	2	Extension to pain
	1	None

Total score is the sum of the scores for each of the three domains

small increases in the volume of any of the constituents of the cranial cavity can be compensated for by subtle reductions in the volume of intracranial blood or CSF.

Pathophysiology

Primary injury

Primary injury describes the (preventable, but untreatable) damage that occurs at impact; its severity depends on the degree of force applied to the brain. It can produce focal injuries such as skull fractures, axonal lacerations, intracranial haematomas and brain contusions or it can be diffuse, as in diffuse axonal injury.

Secondary injury

Secondary injuries are the injuries occurring as a result of the primary injury. They generally result in cerebral ischaemia and significantly affect outcome (Chesnut et al, 1993). Secondary injuries include micro- and macroscopic changes such as astrocyte swelling, that generate microvascular compromise, blood–brain barrier disruption and inflammatory cell recruitment mediated through a variety of neurochemical mediators such as excitatory amino acids (Nortje and Menon, 2004).

Several factors can exacerbate the severity of the secondary injury. These include:

- Systemic hypotension (systolic blood pressure ≤90 mmHg)
- Hypoxaemia (SpO_2 ≤90%)
- Hypercarbia ($PaCO_2$ >5.3 kPa)
- Hyperventilation (causing hypocapnia leading to cerebral vasoconstriction; *Figure 1*)
- Pyrexia (core temperature ≥38°C)
- Intracranial hypertension (intracranial pressure >30 mmHg)
- Arterial vasospasm (caused by blood in the subarachnoid space).

The effects of coincident and repeated insults are additive. Via complex pathophysiological mechanisms secondary injuries limit the ability of the injured brain to cope with minor variations in physiology and maintain cerebral blood flow. Autoregulation is often impaired, so that decreases in cerebral perfusion pressure commonly result in ischaemia while increases in cerebral perfusion pressure may increase cerebral blood flow and result in increased cerebral blood volume and intracranial pressure. In some injured areas excitotoxic changes will increase cerebral metabolism, so that ischaemia will be likely in the face of normal or even increased cerebral blood flow. While the cerebral blood flow thresholds below which irreversible ischaemia occurs are unknown, the association between the volume of ischaemic brain tissue and morbidity following traumatic brain injury is well documented. Accurate identification of the extent of true ischaemia requires combined measurements of cerebral blood flow and metabolism using advanced techniques such as positron emission tomography (Coles, 2007).

The sequential activation of injury processes in acute head injury is shown in *Figure 2*.

Management

The severity of secondary injury is a major determinant of traumatic brain injury outcome (Jones et al, 1994). Thus the focus of care is attenuation of these injuries and avoidance of preventable additional insults that exacerbate secondary injuries and adversely affect outcome (see Brain Trauma Foundation (2007) guidelines).

Initial resuscitation

This should follow the Advanced Trauma Life Support (ATLS) protocols to ensure airway patency, maintenance of good oxygenation and tissue perfusion, and stability of the cervical spine. This initial resuscitation is a crucial part of minimizing secondary brain injury. Pupil size and reaction to light should be assessed, since asymmetrical pupil sizes or impaired light reaction may indicate a surgically remediable intracranial haematoma. Monitoring of blood pressure, pulse and respiratory rate are essential,

Figure 1. Influence of hyperventilation on cerebral perfusion in a patient 6 hours after traumatic brain injury. Gray scale positron emission tomography cerebral blood flow images obtained from a head injury patient (a) at relative normocapnia and (b) hypocapnia. Red areas indicate regions with a cerebral blood flow less than 20 ml/100 g/min. ICP = intracranial pressure.

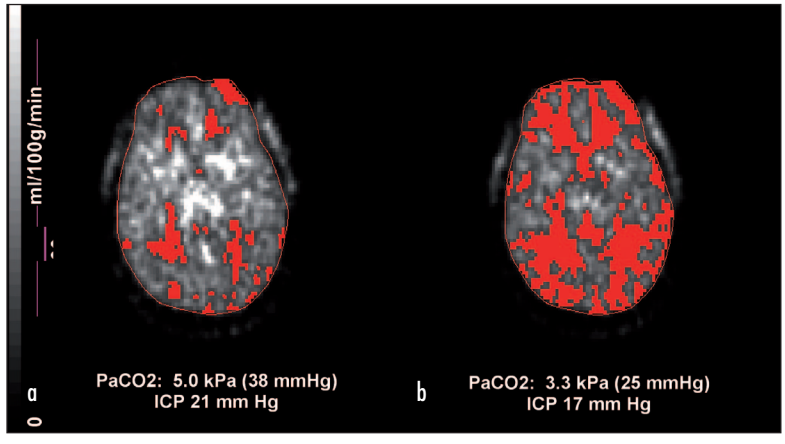
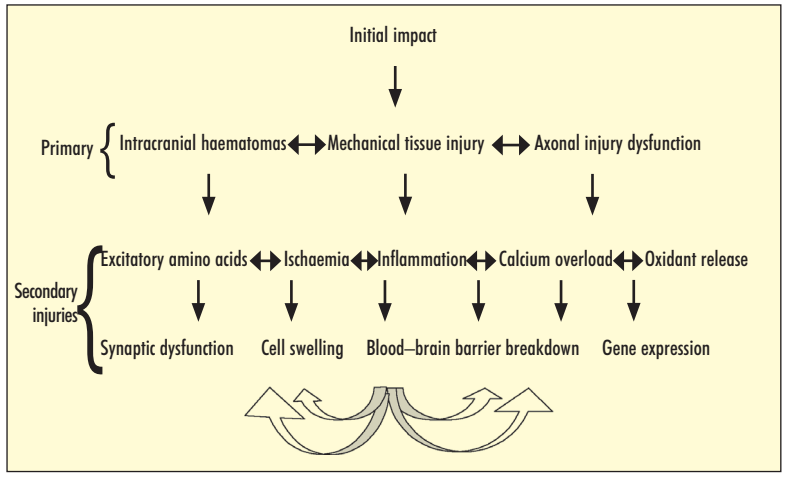


Figure 2. Sequelae of injury processes in traumatic brain injury.



not only to ensure cardiorespiratory stability, but also to indicate possible brainstem dysfunction.

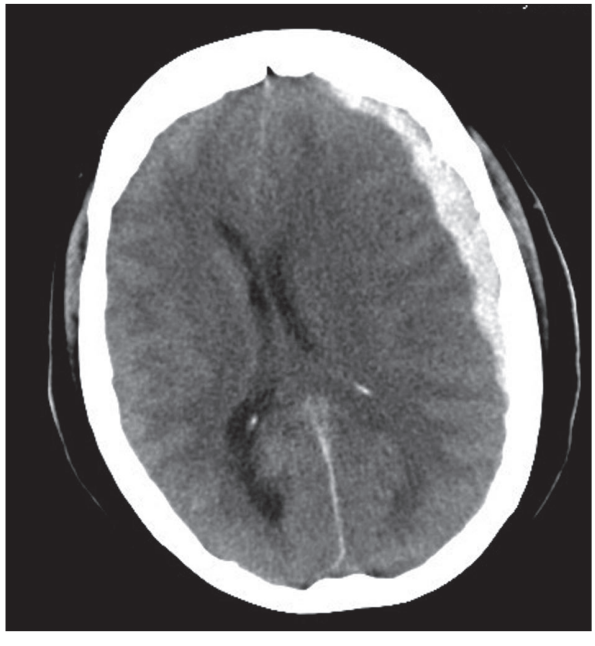
Inter-hospital referral and transfer

Many patients with traumatic brain injury will first present at a non-neurosurgical centre. The Association of Anaesthetists (2006) have published a booklet summarizing which cases should be discussed with a neurosurgeon, the indications for transfer to a neurosurgical centre, and guidelines for safe transport. It is well recognized that good quality, safe transfer improves patient outcome. Senior (consultant) involvement at the sending and receiving units, adequate pre-transfer resuscitation and investigation, and provision of an experienced nurse and senior anaesthetic trainee during transfer are essential. The patient requires mobile critical care, with close monitoring, and ongoing active treatment to maintain normoxia (PaO_2 >13 kPa), normocarbia ($PaCO_2$ 4.5–5.0 kPa), normovolaemia and normothermia or mild hypothermia.

Surgical intervention

Several sequelae of traumatic brain injury require urgent neurosurgical intervention. Unless acute subdural (*Figure 3*) and extradural (*Figure 4*) haematomas are evacuated within 2–4 hours, unfavourable outcomes are inevitable. The decision to evacuate intracranial haematomas depends on the neurological status and intracranial pressure. Posterior fossa collections and large temporal lobe lesions should be treated urgently (to avoid the risks of brainstem compression and transtentorial herniation respectively). Intracranial mass lesions that generate >5 mm midline shift, or basal cistern compression (a sign of imminent transtentorial herniation), require urgent surgical intervention.

Figure 3. Computed tomography scan showing left traumatic acute subdural haematoma, resulting in midline shift.



Not all traumatic brain injury patients require surgical intervention. In some circumstances, small acute subdural or extradural haematomas can be managed conservatively. Operative intervention may also be inappropriate in patients with injuries so severe that survival is impossible.

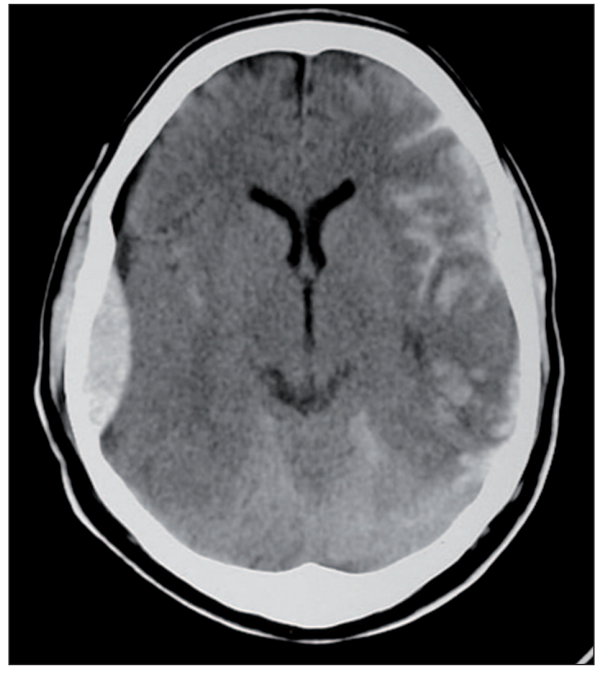
Neurological intensive care

The primary role of the neurosurgical critical care unit is the prevention, detection and attenuation of secondary neuronal injury, and the maintenance of cerebral blood flow and oxygenation.

It has long been known that among patients who die following head injury, evidence of cerebral ischaemia in post-mortem studies is almost invariable (Graham et al, 1978) and that uncontrolled intracranial hypertension is the primary cause of death in ~50% of patients with severe traumatic brain injury. Chesnut et al (1993) found that hypotension (systolic blood pressure <90 mmHg) was associated with a 150% increase in mortality, and Marmarou et al (1991) that the worst outcomes occur in patients with intracranial pressure >20 mmHg and/or cerebral perfusion pressure <60 mmHg. Thus in most neurosurgical critical care units the treatment goal is to maintain intracranial pressure <20–25 mmHg using a combination of interventions that include nursing the patient 10–15° head up, sedation, CSF drainage, osmotherapy (using mannitol), muscle paralysis, hypothermia, barbiturate-induced coma and decompressive craniectomy. These interventions are typically used in a stepwise manner.

Cerebral perfusion pressure is maintained by fluid therapy targeted to normovolaemia, supplemented by

Figure 4. Right extradural haematoma, with left-sided traumatic subarachnoid haemorrhage and contusions.



vasopressor and inotrope administration when required. The optimal cerebral perfusion pressure threshold in traumatic brain injury patients is controversial. Current Brain Trauma Foundation (2007) guidelines recommend a cerebral perfusion pressure target of ≥60 mmHg in the first instance. In Cambridge an initial target of 70 mmHg is applied. Where no intracranial pressure monitor is present, some units will generally aim for a mean arterial pressure of 90 mmHg. Evidence suggests that there is considerable within-patient spatial and temporal variation in pathophysiology, that routine elevation of cerebral perfusion pressure > 70 mmHg may be associated with cardiorespiratory complications and that the impact of cerebral perfusion pressure elevation depends on the volume of critically ischaemic brain (Coles et al, 2004; Warner and Borel, 2004). Although a single cerebral perfusion pressure target is not ideal, further research is required to show that definition and application of individual cerebral perfusion pressure goals is feasible and beneficial.

Measurement of cerebral perfusion pressure requires continuous invasive monitoring of intracranial pressure and arterial blood pressure. Intracranial pressure is commonly monitored by placing a catheter, connected to a manometer or an electronic pressure transducer, in a lateral ventricle. Increasingly, ventricular catheters are being replaced by intraparenchymal micro-manometers and fibreoptic probes, which are much easier to use and carry a significantly lower infection risk. Their disadvantage is that they are expensive, do not allow for drainage of CSF to reduce intracranial pressure, and sometimes suffer from zero drift.

More specialized monitoring techniques are needed to assess local or regional changes in CNS physiology. These include transcranial Doppler ultrasonography for the non-invasive estimation of cerebral blood flow, electroencephalography, and jugular venous saturation monitoring for an estimation of the adequacy of oxygen delivery to the brain.

Although few individual interventions have been shown to improve outcome, provision of a package of care, involving neuromonitoring and protocol-driven care on a dedicated neurosurgical critical care unit, is probably associated with a significantly improved outcome (Patel et al, 2002). Unfortunately the current provision of neurocritical care in the UK is far from optimal. Matta and Menon (1996) found that of the 44 neurosurgical referral units identified from the UK Medical Directory, only 66% managed their patients in specialized neurosurgical critical care units. They also found large discrepancies in the monitoring and therapies used across centres in the UK.

Outcomes of traumatic brain injury

Progress in neurocritical care has led to an increase in survival rates among patients suffering severe traumatic brain injury. Functional outcome after traumatic brain injury is difficult to predict from early structural or anatomical computed tomography (CT) and magnetic resonance imaging (MRI) scans, and varies from permanent coma through to recovery of consciousness and a return to independent life in the community. Intermediate outcomes include the vegetative state, minimally conscious state and locked-in syndrome. Patients with the latter show both awareness and wakefulness but are generally aphonic and quadriplegic as a result of brainstem injuries. Patients with vegetative state are sometimes described as having 'eyes open coma', a state in which they appear to be awake but are unaware of themselves or their environment; whereas patients with minimally conscious state show limited but clear, reproducible evidence of awareness of themselves or their environment but are unable to communicate consistently (Laureys et al, 2004). For research purposes outcomes are usually classified according to the Glasgow Outcome Score (Jennett and Bond, 1975): outcomes and their scores are classified as either death (1), persistent vegetative state (2), severe disability (3), moderate disability (4) or good recovery (5).

Assessment

At present clinicians are generally only able to infer the presence or absence of conscious experience from simple clinical signs. Since these are somewhat subjective, an accurate diagnosis is difficult and depends heavily on the skill of the clinician. Zeeman (1997) reports that one in three persistent vegetative state patients are conscious when a diagnosis is made with 'insufficient care'. Childs et al (1993) studied 49 patients admitted to a rehabilitation centre with a diagnosis of persistent vegetative state and found evidence for awareness in 37% within 1 week of admission. Three promising new assessment tools have been proposed – the Coma Recovery Scale, the Wessex Head Injury Matrix and the Western Neurosensory Stimulation Profile (Majerus et al, 2005). Further studies are required, but it appears that effective implementation of these tools will improve diagnostic accuracy.

Neuroimaging

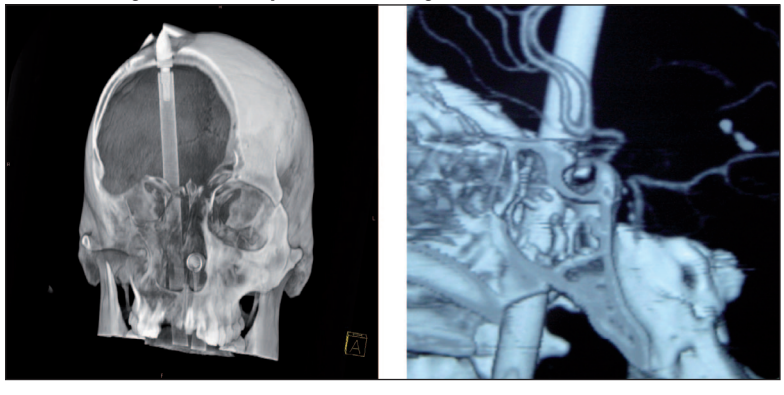
Structural imaging using CT and MRI scans plays an important role in the acute management of traumatic brain injury to determine the extent of injury and guide surgical interventions. Modern CT scanners acquire images very quickly and can also rapidly produce three-dimensional images where these may assist in management (*Figure 5*). There is growing interest in the role of functional neuroimaging – functional MRI (fMRI) and positron emission tomography (PET) – in traumatic brain injury. In the acute stages PET can more accurately identify pathophysiological changes such as critical ischaemia, leading to better selection and application of therapies to attenuate secondary injury. Early, accurate identification of the volume of critically ischaemic or metabolically inactive (necrotic) brain tissue may provide better prediction of final outcome than early structural images.

Functional imaging has several potential roles in the chronic management of traumatic brain injury patients. It may provide information on underlying functional abnormalities in the different states of consciousness, aid in their diagnosis, and guide rehabilitation. At present the number of neuroimaging studies of traumatic brain injury patients in the chronic phase is limited, but further work may produce models to distinguish the pathophysiological basis of the different disorders of consciousness.

Since the early 1990s fMRI has been used extensively to infer brain activity in studies of human cognition, and more recently there has been emerging evidence that it can be used to provide evaluations of cognitive functions not amenable to routine clinical testing. Schiff et al (2004) were the first to use it to study neural activity in minimally conscious state patients. They discovered language-related cortical activation with auditory stimulation, suggesting that some minimally conscious state patients may retain potential for cognitive and sensory function despite their inability to communicate reliably. More recently Owen et al (2006) reported the results of an fMRI study of a young woman who had been diagnosed as having persistent vegetative state 5 months after sustaining a severe traumatic brain injury in 2005. When instructed to imagine playing tennis or imagine the layout of her house, her fMRI images showed a similar neural activation pattern to those of healthy people performing the same task, indicating that although she was unable to mount a motor response at the time, she was able to hear, understand and follow instructions.

Follow-up studies will show whether such evidence of covert cognitive function can predict which patients are likely to recover some function and possibly identify those who will benefit most from intensive neurorehabilitation programmes. Additionally, as pointed out by Schiff et al (2004), fMRI could be used to customize rehabilitation based on which areas of a patient's brain are working. It could also provide a novel way of testing the efficacy of novel neuroprotective agents such as excitatory amino acid antagonists, calcium-channel blockers and antioxidants.

Figure 5. Examples of three-dimensional reconstructed computed tomography images showing the path of a crossbow arrow lodged in the head of a young man. Information from these images was used to plan further management.



Rehabilitation

Rehabilitation should begin as soon as possible. In the acute stages interventions should focus on reducing neurological and physical impairments and preventing secondary complications such as contractures, pressure ulcers and pneumonia. Provision of such acute rehabilitation is not only important in terms of minimizing these secondary complications, but for the perpetuation and improvement of rehabilitation following discharge. It has been suggested (Greenwood et al, 2004) that acute inpatient rehabilitation allows proper assessment of patient needs before discharge and improves the likelihood of patients receiving appropriate rehabilitation in the community. They found that 60% of patients discharged following acute inpatient rehabilitation received community rehabilitation, whereas none of the patients who had received standard care in hospital received community care. Provision of such acute inpatient rehabilitation is severely lacking in the UK, however, and this issue needs to be addressed to improve patient outcomes after traumatic brain injury.

After acute inpatient rehabilitation, ongoing, longer term neurorehabilitation occurs in the community. For the most severely disabled traumatic brain injury patients this is typically occurs in 'neurological' care homes, of which there are several in the UK. Neurorehabilitation is a multidisciplinary medical field with the goal of restoring impaired brain activity by implementing the research results of the structural neurosciences (e.g. neurobiology, neurochemistry, neuroradiology, neurophysiology) to the field of the functional sciences (e.g. rehabilitation, biomechanics, kinematics, kinetics). It should involve a holistic therapeutic process, structured as an individualized programme specifically developed to meet the needs of the patient.

Deficit-specific and goal-oriented neuropsychological treatment strategies are used to encourage recovery of disabled functions and skills. In 2005 Cicerone et al published an extensive literature review on cognitive rehabilitation and concluded that cognitive rehabilitation is of significant benefit compared with alternative treatments.

Interest in the role of rehabilitation in improving outcomes after traumatic brain injury was sparked by the work of Luria in 1969 who suggested that practice, applied systematically, can lead to changes in brain organization. This hypothesis has been supported by animal studies which provide clear evidence that activity does lead to changes in brain organization and that enriched environments enhance brain cell recovery after brain damage (for an example see van Praag et al, 2000). The principles of current neurotraining programmes are summarized in *Table 2*. For further information consult the national clinical guidelines on rehabilitation following brain injury (British Society of Rehabilitation Medicine and the Royal College of Physicians, 2003).

Rehabilitation centres represent an as-yet untapped wealth of information from which we can learn much more about brain plasticity, the success of various neurorehabilitative approaches and attempt to understand why neurorehabilitation is so successful in some patients and not in others.

Conclusions

Although there have been a great many advances in the provision of care for traumatic brain injury patients, and vast improvements in the very poor outlook that was previously associated with head trauma in the last 30 or so years, much work remains to be done. Inevitably, as our understanding of the pathophysiology of brain injury improves, along with improvements in the provision of neurointensive care and neurorehabilitation for brain injured patients, patient outcomes following traumatic brain injury will improve. BJHM

Figure 1 *is reproduced from Coles (2007) by kind permission of the* British Journal of Anaesthesia.

Conflict of interest: none.

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Table 2. Principles of current neurotraining programmes

Recapitulation of normal growth and development
Personal attention – training on a one-to-one basis
Provision of constant and systematic feedback to the patient
Prolonged and intensive multimodal stimulation through repetitive practice
Entering training at the proper functional level
Increasing difficulty in small increments
Ensure successful endeavours
Insist upon overlearning

KEY POINTS

- Traumatic brain injury is a significant cause of mortality and morbidity in our society.
- Injuries can be divided into primary and secondary insults.
- Outcomes from traumatic brain injury can be significantly improved through use of a thorough, protocol-led management of the traumatic brain injury patient from the moment of impact.
- Such management includes thorough monitoring and appropriate interventions, whose use is driven through an understanding of the pathophysiology of traumatic brain injury.
- Outcomes can be significantly improved by following a specialized neurorehabilitative approach to the traumatic brain injury patient, once he/she is medically stable.
- There is a growing role for the use of neuroimaging in traumatic brain injury.