Seminar

Traumatic brain injury

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The decrease in mortality and improved outcome for patients with severe traumatic brain injury over the past 25 years can be attributed to the approach of "squeezing oxygenated blood through a swollen brain". Quantification of cerebral perfusion by monitoring of intracranial pressure and treatment of cerebral hypoperfusion decrease secondary injury. Before the patient reaches hospital, an organised trauma system that allows rapid resuscitation and transport directly to an experienced trauma centre significantly lowers mortality and morbidity. Only the education of medical personnel and the institution of trauma hospital systems can achieve further improvements in outcome for patients with traumatic brain injuries.

Traumatic brain injury is the most common cause of death and disability in young people. There is much hope for improvement in early care and functional outcome by use of scientific evidence-based guidelines. Traumatic brain injury is graded as mild, moderate, or severe on the basis of the level of consciousness or Glasgow coma scale (GCS) score after resuscitation (panel). Mild traumatic brain injury (GCS 13–15) is in most cases a concussion and there is full neurological recovery, although many of these patients have short-term memory and concentration difficulties.[1] In moderate traumatic brain injury (GCS 9–13) the patient is lethargic or stuporous, and in severe injury (GCS 3–8) the patient is comatose, unable to open his or her eyes or follow commands.

Patients with severe traumatic brain injury (comatose) have a significant risk of hypotension, hypoxaemia, and brain swelling. If these sequelae are not prevented or treated properly, they can exacerbate brain damage and increase the risk of death. Major improvements in outcome can be achieved for such patients before they reach hospital by rapid resuscitation and direct transport to a major trauma facility, and in the hospital setting by monitoring of intracranial pressure and institution of adequate cerebral perfusion. Two scientific, evidence-based documents[2,3] support this position and are summarised in this seminar.

Epidemiology

In the USA, for example, each year about 1·6 million people sustain traumatic brain injuries, of whom 800 000 receive early outpatient care and 270 000 require hospital admission.[4] Each year about 52 000 deaths and 80 000 permanent severe neurological disabilities result from severe traumatic brain injury.[4] The financial burden is enormous. Worldwide, injury is the cause of the largest number of disability-adjusted life years lost, which includes years lost to death and to varying degrees of disability.[5] In both more and less developed countries, motor vehicles are the major cause of deaths and disabilities, particularly in young people.[5] Falls are the leading cause of death and disability from traumatic brain injury in people older than 65 years.[6]

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Glasgow coma scale

Eye opening		Motor response		Verbal response	
Spontaneous	4	Obeys	6	Oriented	5
To speech	3	Localises	5	Confused	4
To pain	2	Withdraws	4	Inappropriate	3
None	1	Abnormal flexion	3	Incomprehensible	2
		Extensor response	2	None	1
		None	1		

Secondary injury

Neurological damage does not all occur immediately at the moment of impact (primary injury) but evolves afterwards (secondary injury). Secondary brain injury is the leading cause of inhospital deaths after traumatic brain injury.[7] Most secondary brain injury is caused by brain swelling, with an increase in intracranial pressure and a subsequent decrease in cerebral perfusion leading to ischaemia.[8] Within hours of traumatic brain injury, vasogenic fluid accumulating in brain causes cerebral oedema, raises intracranial pressure, and lowers the threshold of systemic blood pressure for cerebral ischaemia.[9] A reduction in cerebral blood flow or oxygenation below a threshold value or increased intracranial pressure leading to cerebral herniation increases brain damage and morbidity. Several pharmacological agents, such as free-radical scavengers, antagonists of N-methyl-D-aspartate, and calcium-channel blockers, have been investigated in an attempt to prevent the secondary injury associated with traumatic brain injury, but none has proven effective.[10]

Hypoxaemia and hypotension occur commonly before the patient reaches hospital and significantly increase the risk of secondary brain injury and the likelihood of a poor outcome.[11,12] In a study of children with traumatic brain injury, 13% had a documented hypoxaemic episode and 6% had hypercapnia. Various studies have reported that 27% to 55% of patients with traumatic brain injury were hypoxaemic (arterial oxygen saturation <90%) at the scene, in the ambulance, or on arrival at the emergency department. Intubation at the scene of the accident or in the emergency department was required for all patients if the GCS score was 3–5, 73% if the GCS was 6–7, and 62% if the GCS was 8–9.[13]

In adults, hypotension is defined as a single measure-

Age (years)	Systolic blood pressure (mm Hg)
<1	65
1–5	70–75
5–12	75–80
12–15	80–90

Systolic blood pressure associated with a poor outcome in children with traumatic brain injury

ment of a systolic blood pressure below 90 mm Hg. In two US studies, hypotensive episodes were observed in 16%[11] and 32%[14] of patients with severe traumatic brain injury at the time of hospital arrival and during surgical procedures, respectively. A single episode of hypotension was associated with increased morbidity and doubling of mortality. An Australian study reported similar findings.[12] In children, a low systolic blood pressure, sustained for at least 5 min, is associated with a poor outcome (table).

Prehospital guidelines

Early identification of severe traumatic brain injury at an accident scene, with proper assessment, treatment, and transport destinations can lower the risk of secondary injury and subsequent long-term care costs. The *Guidelines for the Prehospital Management of Traumatic Brain Injury*[3] address the assessment, treatment, and transport decisions based on current scientific evidence (figure 1).

Oxygenation and blood-pressure treatment

Endotracheal intubation decreases the risk of death for patients with isolated severe traumatic brain injury from 50% to 23% and that for all trauma patients from 36% to 26%.[15] Prehospital neuromuscular blockade for endotracheal intubation has also been successful, with studies demonstrating the safety of short-acting neuromuscular blockade used at the scene of the accident to facilitate endotracheal intubation by paramedics. In the absence of signs of cerebral herniation, ventilatory assistance after endotracheal intubation should be provided (respiratory rate about 10 breaths per min for adults, 20 breaths per min for children, and 25 breaths per min for infants) until arterial blood-gas analysis is available to guide the minute ventilation rate.

Shock (systolic blood pressure <90 mm Hg) should be prevented, rapidly diagnosed, and treated.[11,16,17] The underlying cause of hypotension in trauma patients is most commonly haemorrhage; therefore, intravascular fluid is intuitively the most effective way to restore blood pressure. Adult resuscitation protocols involve the rapid infusion of 2 L Ringer's lactate or normal saline as an initial crystalloid bolus.[18] However, there is evidence that early restoration of blood pressure may worsen outcome in penetrating thoracic injuries.[19] Trials have also shown both higher systolic blood pressure and better survival in trauma patients resuscitated with hypertonic saline instead of crystalloid. Meta-analysis found that patients who received hypertonic saline and dextran were about twice as likely to survive as those who received standard therapy.[20] Furthermore, hypertonic saline may decrease intracranial pressure in patients with intracranial

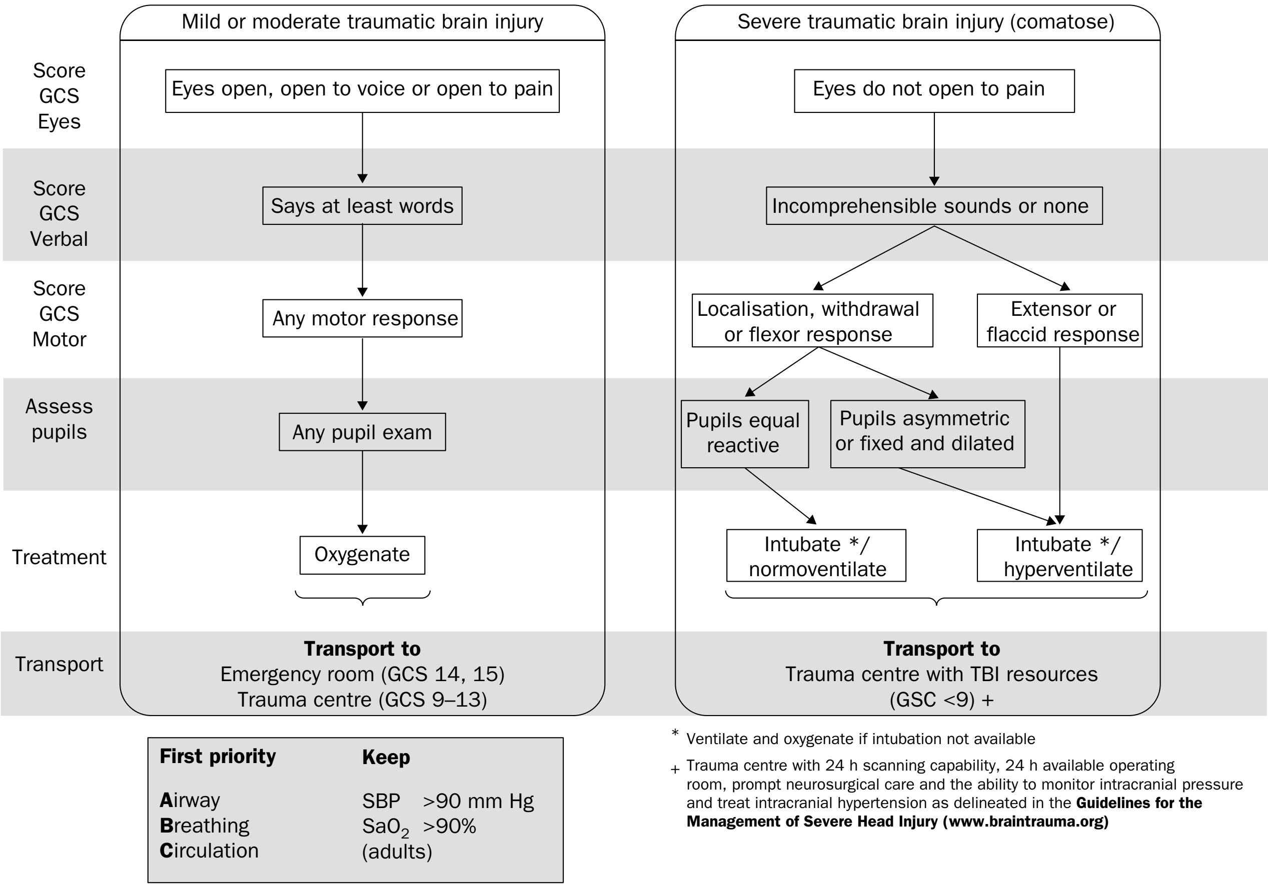

Figure 1: **Prehospital triage for patients with traumatic brain injury**

SBP=systolic blood pressure; SaO_2=oxygen saturation; TBI=traumatic brain injury.

*Ventilate and oxygenate if intubation is not available. †With 24 h scanning capability, 24 h operating facilities, prompt neurosurgical care, ability to monitor intracranial pressure and treat intracranial hypertension (www.braintrauma.org: reprinted with permission from the Brain Trauma Foundation).[3]

hypertension. Other studies have shown no difference in survival or an improvement with use of hypertonic saline with or without dextran over isotonic saline for fluid resuscitation; most benefit was seen in the subgroup of patients with GCS scores below 9. Hypertonic saline may offer a distinct survival advantage in patients with severe traumatic brain injury, but definitive prospective clinical trials have not yet been done.

Hyperventilation

Hyperventilation can lower acutely increased intracranial pressure by the hypocapnic induction of cerebral vasoconstriction with a subsequent reduction in cerebral blood flow. The rapid action of hyperventilation led medical personnel to administer it prophylactically to comatose patients with the aim of preventing potential intracranial hypertension. However, there is no evidence that prophylactic hyperventilation improves outcome. In the first few hours after traumatic brain injury, cerebral blood flow is very low, and the decrease may be exacerbated by hypocapnia.[21] Sustained prophylactic hyperventilation retards recovery from severe traumatic brain injury and is not recommended.[22] Hyperventilation may be useful transiently if the patient shows any obvious signs of cerebral herniation after correction of hypoxaemia or hypotension.[3] Signs of cerebral herniation include fixed, dilated, or asymmetric pupils and motor responses of extensor posturing or no movement when an unpleasant stimulus is applied. When these signs are present in a comatose patient with traumatic brain injury, hyperventilation (about 20 breaths per min for adults, 30 breaths per min for children, and 35 breaths per min for infants) may be used until arrival at hospital, where blood-gas analysis can guide the rate of ventilation.

Hospital transport decisions

An organised emergency medical services system improves outcome for patients with severe traumatic brain injury if they are directly transported to designated trauma hospitals with the requisite resources. When a call is made to the emergency medical services, information is obtained so that the probability of traumatic brain injury can be assessed. If head injury is likely, the highest-level available provider of emergency medical services, with the greatest ability to minimise secondary injury, should be dispatched. The value of the emergency medical services system is suggested by a study that compared the risks of death in India and in a US cohort; the risk of death was two times higher in India.[23] Ambulances were called in 0·5% of cases in India and 84% of cases in the USA, and patients took longer to arrive at the emergency department in India. Results of other similar comparisons were the same. Lack of an emergency medical services system and delay in presentation were significant factors in the difference in outcome.

The field transport choice for hospital destination for patients with traumatic brain injury is one of the most important decisions affecting outcome. Individual outcomes improve when prehospital care, triage, and admission to designated trauma centres are coordinated within US regional trauma systems. Outcome after the implementation of a trauma system in Oregon improved survival for patients with traumatic brain injury.[24] Before implementation of a trauma system in Quebec, Canada, mortality for all trauma patients was 20%; after implementation mortality decreased to 10%.[25] In the UK, only 33% of patients with major trauma were taken to a trauma centre in 1990; the proportion increased only to 39% by 1993.[26] In all comparisons between organised and non-organised emergency medical services and trauma systems, outcome was better with organisation.[27] However, for the opportunity of the best outcomes, patients with traumatic brain injury require direct transport to a trauma centre. In rural areas, a helicopter should be used.

Patients with major trauma should be transported directly to a trauma centre whenever possible. Survival is better if patients are transported directly to a trauma centre than if they are treated at a local hospital before transfer.[28] A UK survey reported a 75% frequency of secondary transfer.[29] If direct transport is not available, restoration of oxygenation and blood pressure should be ensured at a local facility before transfer to a definitive care facility. Providers of emergency medical services involved in transport of extended duration should be trained to carry out endotracheal intubation, resuscitation, and continuing neurological assessments so that changes in the patients' neurological status can be recognised and treatment adjusted accordingly.

A trauma centre has the facilities, staff, and equipment for immediate care of critically injured patients. Designated trauma centres must have appropriate and available medical personnel, and 24 h availability of a computed tomography scanner, operating room, neurosurgical specialists, intracranial pressure monitoring, and experienced critical care management of intracranial pressure. A 1999 survey of trauma centres in England found that only 79% had computed tomography available 24 h, 70% lacked on-site neurological coverage, and a minority of inpatient-care surgeons were trained in management of traumatic brain injury.[30] The Royal College of Surgeons of England has recently issued recommendations on the management of patients with head injuries. The non-availability of computed tomography capabilities or neurosurgeons delays diagnosis and treatment and can result in a poor outcome.

Hospital guidelines

Management variability

Within countries there are large variations in practical care of patients with traumatic brain injury from the accident scene through the completion of intensive care that could adversely affect outcome. In a US survey of trauma centres,[31] intracranial-pressure monitoring was used routinely in only 28% of hospitals, hyperventilation was used routinely in 83%, and steroids in 64% of patients. Similar results are reported in the UK and in a survey by the European Brain Injury Consortium[29] which showed that the frequency of intracranial-pressure monitoring in comatose patients averaged 43% and ranged from 5% to 53% in various countries.[29] Do differences in care adversely affect outcome? There is good scientific evidence that traumatic brain injury has both a primary injury component incurred at the time of the trauma and a secondary injury component that evolves over time (usually within the week after the injury) and is amenable to medical intervention.

Guidelines for the management of severe head injury[2] were developed in response to the variability in critical care management of severe traumatic brain injury in the USA.[31] The European Brain Injury Consortium has

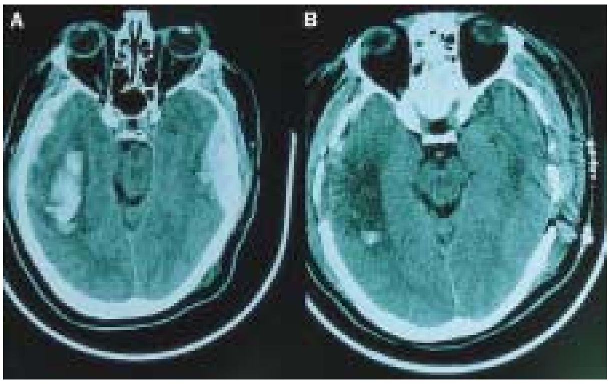

Figure 2: **Computed tomography of a comatose patient with a left temporal epidural haematoma, right parenchymal temporal lobe haematoma, and a right convexity subdural haematoma before and after craniotomy and evacuation of haematomas**

published similar guidelines.[32] Scientific evidence was reviewed on treatment topics deemed to have significant impact on outcome. The sections after that on neurosurgical operations are derived from these guidelines.

Neurosurgical operations

Patients with traumatic brain injury who have been stabilised should be examined by computed tomography of the head so that mass lesions such as subdural or epidural haematomas that need surgical evacuation can be identified (figure 2). In addition, parenchymal haematomas in the temporal and frontal regions should be removed prophylactically if there is significant mass effect (mainly for temporal-lobe haemorrhagic contusions), and if there is persistent intracranial hypertension. In most large studies of severe traumatic brain injury, only a third of patients need craniotomy. Acute subdural haematomas in patients with severe traumatic brain injury are associated with 90% mortality if evacuated more than 4 h after injury and only 30% mortality if evacuated earlier.[33] If subdural evacuation is done within 2 h after injury, one study reported a 70% decrease in mortality.[34] This evidence reinforces rapid transport of patients with severe traumatic brain injury to a facility with computed tomography and neurosurgical capabilities.

Monitoring of intracranial pressure

Raised intracranial pressure can cause a reduction in cerebral perfusion, and therefore, is a significant factor in secondary brain injury. Monitoring and treatment of intracranial pressure increase the likelihood of a favourable outcome.[35,36] Patients with severe traumatic brain injury and abnormalities shown by computed tomography on admission have a greater than 50% chance of intracranial hypertension. This complication is also noted in patients with severe traumatic brain injury whose computed tomography scans are of normal appearance if two of the following three features are present: age over 40 years, unilateral or bilateral motor posturing, or systolic blood pressure below 90 mm Hg.[37] Since there is a high likelihood of intracranial hypertension in these patients, monitoring of intracranial pressure is recommended in adults and children to direct specific diagnostic tests and treatment to maintain cerebral perfusion and also to give prognostic information.

The normal range for intracranial pressure is 0–10 mm Hg. 20–25 mm Hg is the upper limit of normal at which treatment should be initiated (note that this is an upper limit, and many investigators use 15 mm Hg as a threshold to start treatment).[38] A catheter placed within the ventricles and connected to an external pressure transducer is recommended (figure 3). This monitoring system is the most accurate and cost-effective method of measuring intracranial pressure.[2] It additionally provides a means to drain cerebrospinal fluid, which decreases the intracranial pressure. This therapeutic manoeuvre is the first approach used to lower intracranial pressure.

The benefit from placement of an intracranial-pressure monitor far outweighs the risks of bacterial colonisation (about 6%) and significant haemorrhage (less than 1%).[2] These infrequent complications rarely have long-term consequences. Ventricular catheter placement in slit ventricles can be achieved by use of a 90° trajectory to the surface of the skull or scalp at a point in the mid-pupillary line just anterior to the coronal suture in adults and children. Parenchymal monitoring gives similar readings of intracranial pressure to those obtained in the ventricles, but these devices can have measurement drift and cerebrospinal fluid cannot be drained. These devices, despite the significantly higher cost, are popular because of ease of placement and capacity to measure intracranial pressure irrespective of head position. Intracranial-pressure monitoring in epidural, subdural, or subarachnoid spaces is not accurate compared with ventricular monitoring, and is therefore not recommended.

Management of cerebral perfusion pressure

Cerebral perfusion pressure is defined as the difference between mean arterial pressure and intracranial pressure. If the cerebral perfusion pressure is maintained above 70 mm Hg, mortality can be significantly reduced in patients with traumatic brain injury.[36]

Intracranial pressure per se is prognostic when time with values above 20 mm Hg is examined. Therapy

Figure 3: **Ventricular catheter placement for monitoring of intracranial pressure**
The catheter was placed into a slit ventricle with a 90° trajectory.

should target reduction of intracranial pressure when it exceeds 20 mm Hg and maintenance of mean arterial pressure at or above 90 mm Hg.

Before management of cerebral perfusion pressure, a normal blood volume should be assured by placement of a central line and maintenance of central venous pressure at 5–10 mm Hg. Hypervolaemia and a positive fluid balance in patients receiving vasopressor treatment to keep the cerebral perfusion pressure above 70 mm Hg increases the risk of pulmonary complications.[39] If the cerebral perfusion pressure is below 70 mm Hg in an adult (threshold levels have not been determined for children) alpha agonists such as norepinephrine can be used to raise mean arterial blood pressure and thereby increase cerebral perfusion pressure. There is currently no evidence that maintenance of cerebral perfusion pressure at levels greater than 70 mm Hg improves outcome.[40]

Treatments to lower intracranial pressure

Ventricular drainage of cerebrospinal fluid is used continuously for patients with intracranial hypertension. Persistent hypertension (intracranial pressure >25 mm Hg) requires mild hyperventilation and possibly use of diuretics. However, hypocapnic vasoconstriction and hypovolaemia, respectively, can be detrimental side-effects of hypocapnic and diuretic therapies. If the intracranial pressure exceeds 25 mm Hg, repeat computed tomography is recommended to ascertain expanding or new intracranial lesions. These lesions are common in comatose patients with coagulopathy and those who present with significant other injuries or hypotension.[41]

Hyperventilation decreases the arterial carbon dioxide concentration, causes cerebral vasoconstriction, reduces cerebral blood flow and subsequently results in decreased intracranial pressure. If used prophylactically and for a long time, hyperventilation worsens outcome.[22] Furthermore, aggressive hyperventilation can cause vasoconstriction to the point of cerebral ischaemia. Children also have reduced cerebral blood flow after traumatic brain injury and are at risk of hyperventilation-induced ischaemia.[42] About 20% of patients with intracranial hypertension have a mismatch of cerebral blood flow to metabolism (brain oxygen consumption) and blood flow seems to be more than is required for metabolism. This increased flow can be lowered by hyperventilation, thus reducing blood volume and subsequently intracranial pressure. However, there is no evidence that hyperventilation in selected patients who show increased blood flow improves outcome. Increased cerebral blood flow may be a consequence of impaired aerobic glycolysis leading to a need for increased glucose delivery for anaerobic metabolism.

If intracranial pressure remains above 20–25 mm Hg after drainage of cerebrospinal fluid, hyperventilation can be used. Possible approaches are hyperventilation to a $PaCO_2$ of 30–35 mm Hg and treatment with mannitol. This agent is effective in the reduction of intracranial pressure in head-injured patients.[43] The effective dose range of mannitol is 0·25–1·00 g/kg intravenously. Intermittent boluses may be more effective than a continuous infusion, and the serum osmolarity should not exceed 320 mmol/L. If the intracranial pressure falls below 20 mm Hg, these therapies can be carefully withdrawn. However, if intracranial hypertension persists, repeat head CT scan is recommended to assess for the presence of a new or expanding mass lesion.

	Euvolaemia (CVP 5–10mm Hg)	MABP (≥90 mm Hg)	CPP (≧70 mm Hg)	CSF drainage	$PaCO_2$	Mannitol	Paralytics sedation
ICP <25 mm Hg	+	+	+	+	35	–	–
ICP >25 mm Hg	+	+	+	+	30–35	±	+
Herniation	+	+	+	+	25–30	+	+

· Evacuate significant accessible intracranial mass lesions
· Other treatments for steps 2 and 3 include decompressive craniotomy and anaesthetic drugs
* Pressures are in mm Hg

Figure 4: **Management of intracranial pressure and cerebral perfusion pressure**
MABP=mean arterial blood pressure; CPP=cerebral perfusion pressure; CSF=cerebrospinal fluid; ICP=intracranial pressure.

If intracranial hypertension is refractory to the strongest medical and surgical treatments, high-dose barbiturates can be used in patients who are haemodynamically stable, with injuries compatible with recovery.[44] However, prophylactic administration of barbiturates has shown no benefit and may be harmful in some patients. The loading dose of pentobarbital is 10 mg/kg over 30 min or 5 g/kg per h for 3 h, and the maintenance dose is 1 mg/kg per h. Barbiturates produce a dose-dependent decrease in arterial blood pressure and cardiac output and therefore intensive cardiac monitoring and blood-pressure support are necessary if these drugs are used to treat persistent intracranial hypertension. Another second-tier therapy is hyperventilation to a $PaCO_2$ of less than 30 mm Hg. With this approach, assessment of jugular venous oxygenation or cerebral blood flow is recommended to monitor ischaemia. Hypothermia, a potential early therapeutic tool, has not proven effective in a US multicentre trial. A final effort includes decompressive craniotomy for progressive, therapy-resistant intracranial hypertension with associated pupillary dilatation or decerebrate posturing. A substantial portion of the cranium is removed and the dura is opened to allow the brain's volume to increase with a concomitant pressure reduction. One series reports "surprisingly good outcomes" with this technique.[45] Figure 4 summarises the management of intracranial pressure and cerebral perfusion pressure.

Seizures can cause intracranial pressure to rise and increase metabolism, which can be detrimental to the injured brain. Post-traumatic seizures are classified as early (within 7 days of injury) or late (occurring after 7 days). A Cochrane library review of randomised trials of antiepileptic agents found that prevention of early seizures did not have any effect on death or neurological disability.[46] Prophylactic use of antiepileptic agents is not recommended for preventing late post-traumatic seizures.[2]

Although traditionally used in the treatment of brain tumour oedema, glucocorticoids are not recommended for the treatment of intracranial hypertension resulting from traumatic brain injury. In many studies, steroid therapy did not significantly improve intracranial pressure or clinical outcome. A Cochrane library review of 13 pooled steroid trials showed a 1·9% non-significant reduction in deaths.[47] Future clinical trials on selected subgroups of patients, such as those with focal contusions, may be more revealing scientifically. Steroid administration may also adversely effect the nutritional, metabolic, and glycaemic status of patients with traumatic brain injury.

Prognosis

Early indicators (within 24 h of injury) of prognosis in traumatic brain injury are useful to guide counselling of relatives and the use of limited resources. A recently completed evidence-based document[48] describes the most significant early features that are prognostic for a poor outcome. The GCS score measured after resuscitation shows a linear relation to a poor outcome (death, vegetative state, or severe neurological disability) in the range of 3–9, severe traumatic brain injury.

Although there is an increased risk of a poor outcome with advancing age, there is a sharp rise in risk over the age of about 60 years. Hypotension at admission is associated with a doubling of mortality risk.[11] Similarly, fixed and dilated (>4 mm) pupils are associated with 90% mortality.

Computed tomography can reveal intracranial pathology that is prognostic. Normally, the cisterns around the midbrain are visible, but with brain swelling and herniation these spaces are occluded and no longer visible and a significant predictor of poor prognosis.[49] Subarachnoid haemorrhage around the base of the brain increases the chance of vasospasm, poor perfusion, and subsequent death or significant disability. Midline shift of the brain is due to contusion or haemorrhage in most cases and is a poor prognostic indicator that strengthens with the addition of other computed tomographic features used in classification systems.[7]

Prediction models have been developed retrospectively from databases on traumatic brain injury,[50] but they have not proven useful prospectively, probably because treatment and unknown factors are not constant and because traumatic brain injury has heterogeneous pathology. Early indicators of prognosis are useful to describe, so they can be measured routinely and reliably and they can be included in research databases. These will yield more specific prediction information in the future when treatment is standardised and injury is categorised into homogeneous pathological entities.

Mortality from severe traumatic brain injury has fallen drastically over the past 30 years. Most of the deaths occurring in the first week are from intracranial hypertension. In the 1970s, a mortality rate of 55% was common in unmonitored patients. This rate improved to about 30% with the advent of critical care, routine computed tomography, and monitoring of intracranial pressure.[51] Publications over the past few years have reported mortality rates in the 20% range with management of intracranial pressure and cerebral perfusion pressure.

Fears that with the institution of intensive critical care, a decrease in death rates would lead to an increase in the numbers of patients left in a vegetative or severely disabled state are unfounded. There was an overall increase in good outcome (independent and possibly able to return to work or school) and the proportion of vegetative patients (5–10%) and severely disabled patients has remained stable. Large studies use the Glasgow outcome score at 6 months after injury to compare outcomes, since the majority of improvement occurs during this period. Recovery from severe traumatic brain injury depends on the severity of the initial injury, secondary injury, treatment effect, and possibly the patient's genotype.[52] The apparent lack of effect of intensive, inhospital treatment on vegetative-state outcome may be because the primary injury irreversibly damaged neural pathways involved in consciousness or, more likely, secondary injury such as hypoxia or hypotension occurred before the patient reached hospital. No case of good recovery has been observed in children and adults who were vegetative for 12 months. With advances in prehospital assessment and treatment of secondary injury, decreases in the frequency of vegetative state or severe neurological disability may be observed.

Conclusion

Advances in critical care, imaging, and the reorganisation of trauma systems have led to a pronounced reduction in deaths and disability resulting from traumatic brain injury. This improvement has resulted largely from early recognition and treatment of cerebral hypoperfusion. Variability in trauma systems and critical care led to the development of scientific, evidence-based guidelines for management[2] which serve as the basis for standardising inhospital acute care. The next advance in prevention of secondary brain damage will arrive with improved prehospital recognition and treatment of traumatic brain injury.[3] Prehospital and hospital evidence-based guidelines cannot be effective unless they are implemented. Prospective randomised trials of pharmaceuticals or treatment approaches undertaken in the setting of evidence-based practice will provide the future scientific evidence to strengthen guideline recommendations and close the loop from clinical research to bedside practice.

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