Part X: Trauma

Chapter 67: Severe Head Injuries

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Head injuries are a major medical and social problem in developed countries. Neurotrauma is the leading cause of mortality and morbidity in the 15-24 year age group. The death rate per 100,000 from neurotrauma in New South Wales in 1977 was 28, and in South Australia, 25. Statistics are often difficult to compare, because of differences in definition, coding, and data collection. Mortality from head injuries in the USA in the late 1970s was estimated to be 22-25 per 100,000, while in Britain it was 9 per 100,000. Motor vehicle accidents are responsible for approximately 60% of the deaths due to head injuries.

Hospital admission rates for head injuries in the USA and Britain are approximately 200-300 per 100,000 (ie, over 400,000 admissions in the USA per year). Most of these are admitted only for observation over 24-48 hours.

In general, only patients with severe head injuries, or who have associated multiple trauma or medical complications are admitted to the ICU. Severe head injuries form approximately 9-11% of the total head injury population. This is the group of hospital patients exposed to the highest mortality and morbidity. Aggressive ICU treatment has been shown to improve the outcome of severe head injuries, without increasing the number of severely disabled or vegetative survivors.

Initial Assessment

The severely injured patient must have a rapid but complete initial assessment. General measures are instituted to reduce the incidence of secondary insults, particularly hypoxia, hypercarbia and hypotension. These include:

1. Establishing Airway and Ventilation

Intubation and continuous mechanical ventilation (CMV) are required if ventilation or gas exchange are inadequate, and/or if the patient is incapable of protecting his airway. About 30% of severe head injuries, particularly those associated with multiple trauma, are hypoxaemic and should be intubated promptly. Adequate ventilation should produce a $PaCO_2$ of 25-30 mmHg (3.3-4.0 kPa) and PaO_2 greater than 70 mmHg (9.3 kPa). Until proven otherwise with cervical X-rays, these patients should be considered to have a cervical spine injury and handled appropriately.

2. Treatment of Shock

Hypovolaemia is a common finding in multiple trauma. However, shock is uncommon in isolated head injuries, except those involving young children, medullary injuries or large scalp lacerations. Prompt and effective resuscitation is required.

3. Clinical Examination

A full examination helps to determine priorities of treatment and ascertain other injuries.

4. Complete Neurological Examination

(a) *Conscious state* - The Glasgow Coma Scale (GCS) is advocated for all head injuries and has some prognostic significance by itself and in conjunction with other clinical and laboratory findings. It is useful for comparing treatment regimens from different centres, but is not a complete neurological assessment. Drug or alcohol intoxication will make assessment of conscious state difficult. Unfortunately, intoxicated patients often have an associated head injury.

(b) *Pupils* - The pupil size and reactivity are especially important. Abnormalities indicate compression and compromise of the third cranial nerve. This may help to localize supratentorial lesions. Signs of third nerve compression, depression of conscious state plus asymmetrical motor responses, are the triad of signs for transtentorial herniation. Extraocular movements (doll's eyes and reponse to aural caloric testing) are also important in the assessment of the mid brain and pons. The doll's eye manouevre should not be performed until fractures of the cervical spine have been excluded. Papilloedema is uncommon in the acute phase of head injuries.

(c) *Motor function* - Evidence of motor function, especially decerebrate rigidity, hemiplegia or other localizing signs are sought.

(d) *Other assessment* - These include the gag and cough reflex, cardiac status, particularly arrhythmias, ventilatory patterns, and examination of the remaining cranial nerves.

If the patient is repsonsive to commands or questions then a more detailed neurological assessment must be performed.

5. Further History

This should be obtained with particular reference to the circumstances of the injury and retrieval, seizures, intoxications, pre-existing medical problems and medications.

Patients with severe head injuries should be seen early by a neurosurgeon. Those with a GCS less than 8 or with progressive deterioration of neurological status, should be seen immediately. Early CT scanning should be performed. Intracranial mass lesions occur in only 6-7% of total head injury admissions, but in 40-60% of severe head injuries.

Further Diagnostic Measures

1. Computerised Tomography (CT)

The CT scan is the procedure of choice to determine the presence or absence of mass lesions. It will also indicate areas of oedema, infarction, contusion, intracranial air, and size

of the ventricular system. In addition, the CT scan may help to decide on intracranial pressure (ICP) monitoring and may be of some prognostic value. It has replaced angiography, ventriculography and the exploratory burr hole in the emergency assessment and management of head injuries. If time permits and there are indications, views of the cervical spines are also obtained.

It is important to plan for all the logistical problems involved in moving an acutely injured patient to the CT scanner. The patient is required to lie completely still during the scan, otherwise an inadequate scan will result. Early generation scanners took approximately 20 minutes for a complete scan but newer scanners are much faster. Thus, many patients, if not already intubated at this stage, will require intubation and anaesthesia for the CT scan.

All routine preparations should be made for emergency neuroanaesthesia in cases with a potential intracranial collection. If such a collection shows on CT, the patient is kep anaesthetized and ventilated, prior to transfer to the operating theatre. Should the patient's neurological status deteriorate rapidly, a single cut CT scan through the mid-ventricular region may provide sufficient information for surgery. Such a patient should also receive mannitol in an IV dose of 0.5-1 g/kg before surgery.

2. Skull X-Rays

The presence of a fracture increases the likelihood of complications (ie, intracranial haematoma), and may help to localize an extradural haemorrhage. The position of a calcified pineal gland, if seen, may indicate shift of intracranial contents relative to the midline. Fractures of the base of the skull may be seen on X-ray without prior clinical indication of their presence. Skull X-rays are also important in the assessment and future planning of reconstructive surgery. However, skull X-rays are not generally as helpful as CT scanning, and their routine use in the emergency evaluation of head injured patients has been questioned.

The importance of adequate X-rays, if a fracture of the cervical spine is suspected has been stated.

3. Cerebral Angiography

This is now rarely indicated when there is ready access to a CT scanner. However, if an isodense traumatic lesion is seen on the CT scan, the clinical condition is not consistent with the CT finding, or a vascular lesion is suspected, then cerebral angiography should be performed. In the absence of a CT scanner, cerebral angiography can be used in the diagnosis of intracranial haematoma.

4. Ventriculography

Ventriculography has been to a large extent superseded by CT scanning. It will provide information on the degree of midline shift and allow measurement of the intracranial pressure.

5. Echo Encephalography

This has been superseded by the CT scanner.

6. Radio Isotope Scan

This is of little benfit compared to a CT, but it does give some information on cerebral vascularity. Newer imaging techniques, ie, positron emission tomography (PET) will provide additional information on cerebral blood flow and neuronal function.

7. Magnetic Resonance Imaging (MRI)

MRI of head injuries may have some advantages over CT scanning in:

(a) diagnosing and estimating the size of extracerebral fluid (especially small) collections;

(b) distinguishing chronic subdural haematomas from hygromas;

(c) displaying non haemorrhagic contusions.

However, there are many logistical problems in supporting and monitoring injured patients during MRI, and CT scanning remains superior for diagnosis of acute parenchymal and subarachnoid haemorrhages. CT thus remains the procedure of choice for acute head injuries and MRI has yet to find its place, particularly for severe head injuries. It has been suggested that MRI may be of prognostic value in the management of mild and moderate head injuries.

8. Intracranial Pressure (ICP) Monitoring

Surgically amenable intracranial mass lesions should be diagnosed and treated early in head injured patients. The continuous measurement of ICP is of great value, particularly in a patient who is comatose or on a ventilator, when assessment of neurological function is difficult. The value of ICP monitoring has been established. Prolonged levels of ICP over 25 mmHg (3.3 kPa) are associated with a very poor prognosis. It is reported that the outcome of head injuries may be improved if ICP over 15-20 mmHg (2.0-2.7 kPa) is treated with aggressive therapy. However, it has also been suggested that early evacuation of intracranial haematoma without ICP measurement can produce comparable results.

9. Recordings of Cerebral Activity, ie, Multimodality Evoked Potentials (MEP) and the Electroencephalogram (EEG)

These may be useful after the initial stabilization of the patient. They can aid in the monitoring of the patient's clinical course and specific neurological function.

Management

The aims of ICU management for head injuries are:

1. Early detection of changes in neurological status through constant observation and monitoring.

2. Prevention of secondary cerebral insults, especially those related to hyponatraemia, hypotension, hypoxaemia, hypercarbia and raised intracranial pressure.

3. Early diagnosis and treatment of medical and surgical problems, particularly intracranial mass lesions, cerebral oedema and epilepsy, which may be intercurrent or in the process of developing.

The main principles of management are as follows:

1. Constant Observation

Nursing observations as per GCS is extremely important. If deterioration occurs the cause must be sought.

2. Patient Position

If possible, the patient should be nursed in the head-up position (approximately 30-45°) with the head in a neutral plane relative to the body, in order to facilitate ventilation and reduce ICP.

3. Respiratory Care

Hypoxia, hypercarbia or respiratory obstruction must be avoided. The inspired oxygen should be adjusted to maintain a PaO_2 greater than 70 mmHg (9.3 kPa) and the ventilation adjusted to produce a $PaCO_2$ of 25-30 mmHg (3.3-4.0 kPa). Endotracheal suction and physiotherapy will increase the ICP, and that such manoeuvres should be preceded by adequate sedation and analgesia.

4. Blood Pressure Control

Control of the blood pressure (BP) to keep it within its normal limits (ie, a systolic BP of 100-160 mmHg (13.3-21.3 kPa). There are varying degrees of loss of autoregulation following head injuries. It is therefore important to prevent BP being in the ranges where the cerebral blood flow is pressure dependent.

In acute severe head injuries, vascular factors probably account for a greater proportion of the increase in ICP than cerebrospinal fluid (CSF) factors.

5. Surgery

Operative treatment is indicated for complications such as intracranial mass lesions, hydrocephalus and depressed skull fractures.

6. Treatment of Raised Intracranial Pressure

If intracranial mass lesions and hydrocephalus are excluded, then raised ICP in head injuries is either due to cerebral vasodilation, cerebral oedema or varying combinations of the two. In the acute stages, the vascular dilatation may be more important in the genesis of raised ICP, whereas in the later stages cerebral oedema may become more important. To date, it has been clinically difficult to separate these two and hence varying combinations of the following are used:

(a) *Controlled Ventilation*

It is well accepted that reducing the arterial $PaCO_2$ will result in a reduction in ICP. Hyperventilation is used extensively for this purpose. The arterial $PaCO_2$ should be maintained around 30 mmHg (4.0 kPa). In addition to reducing the arterial $PaCO_2$, controlled ventilation will facilitate optimal airway management and oxygenation. Duration of ventilation is generally 48-72 hours in the first instance, followed by an attempt to wean the patient off the ventilator provided ICP is controlled. If ICP rises during weaning, then ventilation is continued for a further 24-48 hours. Increases in ICP during controlled ventilation necessitate the checking of arterial blood gases, re-assessment of ventilation and CT scanning.

(b) *Osmotic Diuretics*

If the blood-brain barrier is intact, osmotic diuretics such as mannitol and urea will lower ICP by drawing fluid across the blood-brain barrier (thus reducing the bulk of the normal brain). Mannitol is generally used, as there is less rebound with mannitol than urea. If a patient deteriorates rapidly in the acute stage of a head injury, mannitol in a dose of 0.3-1.0 g/kg is used. The subsequent dose of mannitol is 0.25-0.5 g/kg every 6 hours. The osmotic diuresis should not be pursued at the expense of cardiovascular stability. If a diuresis does not occur, mannitol should not be continued.

Serum osmolality is used as a guide to mannitol therapy. It should not rise above 310 mosm/kg as mannitol itself will enter the brain and interfere with the efficacy of the dehydration therapy. If the serum osmolality exceeds 350 mosm/kg, serious cellular damage may occur. Treatment with mannitol is continued for only 24-48 hours, as eventually, mannitol will cross into the brain and cause an increase in brain volume, ie, "rebound phenomenon". In one study, empirical mannitol therapy without ICP monitoring produced similar results to mannitol given for ICP elevation greater than 25 mmHg (3.3 kPa).

(c) *Steroids*

The value of steroids to treat cerebral oedema associated with intracranial tumours is well documented and accepted, but remains unproven for cerebral oedema associated with head injuries. Saul et al have suggested that there may be a subgroup of head injured patients,

who are early responders to overall treatment, whose outcome may be improved by steroids. However, several prospective double-blind studies have indicated that steroids do not significantly alter morbidity, mortality or ICP. As a result, the use of steroids in the management of head injuries has declined markedly.

(d) *Diuretics*

The reduction in brain oedema with frusemide may be due to mechanisms other than the diuresis per se. Use of both dexamethasone and diuretics has been shown to produce a greater reduction of brain oedema than the use of either agent alone. Frusemide is the diuretic of choice in patients with congestive heart failure plus cerebral oedema, and may produce less marked changes in serum electrolytes and osmolality than mannitol. Experimentally, frusemide can act synergistically with mannitol, thereby sustaining the osmotic gradient established with mannitol. Once-diuretic therapy (albumin plus frusemide) has similar cerebral effects to mannitol or frusemide.

(e) *Cerebral Metabolic Depression*

Treatment aims to reduce cerebral metabolic demand in tandem with reducing cerebral blood flow, thus preserving neuronal function. The agents generally used are the barbiturates, althesin (no longer available) or lignocaine. These agents may be used in patients with raised ICP, who are not responding to the above treatment, and who do not have surgically correctible mass lesion on repeat CT scanning. High doses of these agents are often required, and extensive cardiovascular monitoring and support are required together with ICP monitoring.

Barbiturate therapy is generally commenced at an ICP over 20-25 mmHg (2.7-3.3 kPa) with a closed skull, and over 15 mmHg (2.0 kPa) with a craniectomy. It reduces ICP, however, Ward failed to show that prophylactic pentobarbital improved outcome. Eisenberg recently reported that while high-dose barbiturates are only indicated in a small subset of patients, barbiturates are an effective adjuvant to "conventional therapy" for the control of ICP. In addition, there was a marked difference in the one-month survival between responders and non-responders to therapy directed at ICP control (ie, "conventional ± pentobarbital).

7. Fluid Balance

Following initial resusctiation and stabilization, strict countrol of fluid balance will help control cerebral oedema. However, fluid restriction should not be pursued at the expense of cardiovascular stability or renal function.

8. Electrolytes

Electrolyte disturbances are frequently seen in patients with head injuries as a result of the head injury, stress responses, osmotic diuresis, diabetes insipidus, fluid restriction, feeding regimens and medications. Regular monitoring of electrolytes, urea, creatinine, blood sugar and osmolalities are important in determining fluid and electrolyte therapy. It is important to prevent hyponatraemia and water overload.

9. Physiotherapy

Physiotherapy is important to remove lung secretions, prevent contractures, and in rehabilitation. Adequate sedation and blood pressure control prior to chest physiotherapy are required in order to prevent ICP elevation.

10. Antibiotics

These are used if there is a base of skull or compound fracture, or a fracture into a sinus. Antibiotics may also be used prophylactically following the insertion of an intracranial pressure monitoring device.

11. Treatment of Epilepsy

Epileptic seizures will markedly increase cerebral metabolic demands, and hence it appears logical to minimize the incidence of seizures. The routine use of phenytoin has been recommended for postoperative neurosurgical patients, including head injuries. However, the efficacy of prophylactic phenytoin to prevent early post traumatic seizures has yet to be firmly established. Acute seizures should be treated with a barbiturate or a benzodiazepine, and phenytoin commenced for longer term therapy.

12. Prophylaxis Against Gastric Ulceration

Gastroduodenal lesions, particularly erosive gastritis, are frequently seen on endoscopy in patients with severe head injuries, but significant haemorrhage only occurs in 10-14% of cases. Prophylaxis using antacids ± H2 receptor blockers should be considered pending the introduction of enteral feeding.

13. Feeding

Severe head injuries demonstrate markedly increased energy requirements, a negative nitrogen balance, weight loss and hypoalbuminaemia. Clifton recommended early enteral feeding while others have suggested that early parenteral feeding will improve outcome.

14. Other Additional Treatment

Other aspects of the management of head injuries deserve comment:

(a) *Temperature control* - Fever increases the metabolic demands of the brain and thus may exacerbate neuronal injury. It is therefore important to determine the cause and to treat appropriately. Although hypothermia has been shown experimentally to be protective to the brain in head injury and cerebral oedema, it has not become routine therapy.

(b) *Syndrome of inappropriate ADH* - This syndrome may be seen following a head injury and is managed as described in Chapter 79, Fluid and Electrolyte Therapy.

(c) *Diabetes insipidus* - may also follow a head injury.

(d) *Coagulopathy* - Coagulopathies are not uncommon in patients with severe head injuries, and must be looked for and treated promptly in order to reduce the occurrence of intracranial haemorrhage.

Prognosis of Head Injuries

A number of factors are important including the age of the patient, time-lag between injury and treatment, type of injury, Glasgow Coma Scale and severity of neurological deficit, plus the occurrence of complications, particularly hypoxaemia and hypotension. In general, poor motor function indicates a poor outcome, especially in the older age groups. Patients under 30 years of age have a better prognosis than those older with the same degree of head injury. However, it is important to avoid making a rash prognostic decision too early, as many head injured patients, particularly the very young, show a remarkable improvement with time.

Brain death, which is an indication for cessation of all active treatment is described in Chapter 42, Brain Stem Death.