Anaesthesia for extra-cranial surgery in patients with traumatic brain injury

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Key points

Patients with blunt trauma have a high incidence of head injury.

Avoid hypotension, hypoxaemia and raised intracranial pressure (ICP) when anaesthetizing a trauma patient for extra-cranial surgery.

An ICP monitor is important in all patients with severe head injury.

During persistent hypotension in a trauma patient with head injury in whom other causes of hypotension have been excluded, a laparotomy is as important as a craniotomy.

Fixation of long bone and maxillofacial fractures can be delayed until the head injury has been stabilized.

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Severe head injury remains common and patients who survive the initial insult are often left with a major neurological deficit. This has serious economic implications both for health care resources and for society, as most of the patients affected are young adults early in their working lives. Intensive care of patients who suffer traumatic brain injury aims to prevent secondary neuronal damage. Up to 75% of blunt trauma victims suffer some degree of head injury, which has been linked to higher mortality. Despite widespread compliance with published guidelines regarding the management of the head injury per se, the optimal timing for dealing with extra-cranial injury in patients with associated brain trauma remains unclear and is discussed in this article.

Head injured patients who, in addition, suffer an extra-cranial injury fall into two broad categories:

- (i) those with an associated life-threatening extra-cranial injury (e.g. ruptured spleen).
- (ii) those with less serious, non life threatening extra-cranial injury (e.g. long bone or maxillo-facial fractures).

The timing of surgery for associated injuries will therefore depend on the severity and the effect of that injury on the ability to maintain adequate brain oxygenation and cerebral perfusion.

General management of a head-injured patient

Initial resuscitation

Irrespective of other injuries, the importance of securing the airway and maintaining adequate oxygenation and blood pressure in the severely head-injured patient cannot be overemphasized. Hypoxaemia and hypotension double the mortality from severe head injury. Endotracheal intubation is necessary in severely head-injured patients (Glasgow Coma Score ≤ 8) as they are unlikely to be able to protect their airway and often have impaired gas exchange. It is important to remember that a significant proportion of severe head injuries are associated with injuries to the cervical spine; therefore, manual in-line stabilization of the neck during induction and tracheal intubation is essential. Nasal intubation is best avoided in the patient with fracture of base of skull because of the risk of passing the endotracheal tube into the brain through the skull defect and risk of infection.

Severely head-injured patients must be assumed to have a full stomach. Therefore, a rapid sequence induction with a small dose of induction agent followed by succinylcholine 1 mg kg^{-1} is mandatory. Apart from ketamine, which is contraindicated because of concerns about its effects on intracranial pressure (ICP), the choice of induction agent is not important as long as it is administered with care and large variations in blood pressure or significant hypotension are avoided. Thiopental and propofol are the main induction agents used at our centre. Lidocaine 1 mg kg^{-1} may be given as a useful adjunct in attenuating the cerebrovascular response to laryngoscopy and tracheal intubation. Succinvlcholine may result in a transient rise in ICP from increased carbon dioxide production and cerebral stimulation via afferent muscle activity. However, the potential risk of hypoxaemia and hypercapnia far outweighs the risk of this transient increase in ICP.

Once the airway is secured, the lungs are mechanically ventilated to maintain mild hypocapnia (not <4 kPa) and adequate Pa_{o_2} . The patient is best sedated and paralysed. There is no excuse for having the patient coughing or straining on the endotracheal tube. Except for young children, in whom blood loss from a scalp wound is sufficient to cause hypotension, hypotension should prompt an investigation of sites of blood loss with immediate laparotomy or thoracotomy if necessary.

The choice of fluid used for resuscitation is less important than the amount given. The use of glucose containing solutions is discouraged unless hypoglycaemia is suspected, as hyperglycaemia (leading to lactic acidosis) has been shown to correlate with poor outcome. Hyperglycaemia should be actively treated

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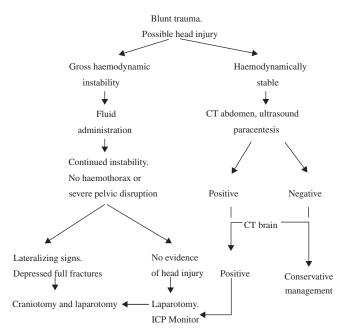


Fig. I Flow chart for the management of blunt abdominal trauma with severe head injury.

and blood glucose concentrations controlled with an infusion of insulin. Because the majority of head-injured patients receives mannitol, an adequate urine output is often a poor indicator of volume status in these patients. Central venous pressure monitoring is very useful as an aid to assessing intravascular fluid volumes and effectiveness of resuscitation, and may be combined with the use of a pulmonary artery flotation catheter in the elderly, patients with heart disease and in those patients requiring the use of inotropic support.

Definitive treatment

Adequate resuscitation and a thorough re-examination of the patient must be completed before making decisions about further treatment priorities. Quality radiographs are taken of suspicious areas and CT scans arranged. Once the patient has been stabilized, a decision can be made regarding transfer to a regional neurosurgical unit for further treatment.

In addition to the minimum mandatory monitors the following additional monitoring should be instituted wherever possible:

- (i) Arterial line—provides beat-to-beat measures of mean arterial pressure (MAP) and is a source for blood gas analysis.
- (ii) Central line—this maybe particularly useful in the context of resuscitation.
- (iii) Pupillary reactivity—in a sedated and ventilated patient, the signs of tentorial herniation are limited to pupillary activity. Hence, it would be useful to monitor pupil size and reactivity wherever possible.
- (iv) ICP monitor—the advantages of ICP monitoring include:(a) earlier detection of an intracranial haematoma,

(b) limiting indiscriminate use of therapy for control of ICP, (c) CSF drainage, (d) determination of prognosis and (e) possible improvement in outcome.

It is generally accepted that patients with an admission Glasgow Coma Scale (GCS) of 9–15 have a low risk of developing intracranial hypertension. Less than 3% with mild head injury (GCS 14–15) and 10–20% with moderate head injury (GCS 9–13) progresses to coma and classification as severe head injury. About 25% of patients with severe head injury (GCS < 9), in the absence of surgical lesions, have evidence of raised ICP and suffer morbidity and mortality similar to those with surgical lesions. Hence, ICP monitoring would be beneficial in all severe head injuries and maybe of benefit in patients who are at risk of secondary brain injury (e.g. hypotension, hypoxaemia). Patients >40 yr old are also more likely to progress to raised ICP.

Patient position and tourniquets

A $10-15^{\circ}$ head up position should be assumed wherever possible. Avoid the head down position. Tourniquets have a two-fold effect. Initially they cause an increase in MAP attributable to increased venous return. This may have a variable effect on ICP depending on autoregulation. Care should be taken when the tourniquet is released as an increase in carbon dioxide along with a drop in MAP can affect cerebral blood flow (CBF).

Role of hyperventilation

Hyperventilation causes cerebral vasoconstriction and hence reduces cerebral blood flow. However, in head injury CBF may be 50% of normal value. Hence, aggressive hyperventilation may cause critical reduction of CBF, worsening cerebral ischemia. This may be compounded by no change in the ICP and there may be a loss of autoregulation. It would be advisable to maintain P_{co_2} within normal limits. However, if there are signs of tentorial herniation (i.e. unilateral or bilateral pupillary dilatation or asymmetric pupillary reactivity), hyperventilation should be instituted as a temporary measure until definitive treatment (e.g. decompressive craniotomy) is available.

Mannitol and barbiturates

Clinically, mannitol is used to lower ICP. Its action is unpredictable in both duration and extent. Its osmotic diuretic effect causes hypovolaemia and hypotension causing secondary brain injury. Hence it should only be used where there is evidence of tentorial herniation or a rise in ICP.

Barbiturates are effective in controlling ICP when all other measures have failed. However, this measure is usually taken on ICU as part of a treatment protocol.

Temperature control

It has been suggested that hypothermia may be beneficial in head-injured patients. However, in the absence of evidence

from randomized controlled trials, only moderate hypothermia should be employed as a last resort, and as part of an algorithm for treatment of raised ICP. Hyperthermia exacerbates brain injury and can lead to a worse outcome. Hence, patients who are febrile should be actively cooled.

Head injury with a life-threatening extra-cranial injury

A 17-yr-old boy meets with a road traffic accident. His GCS is 12 at the scene of the accident but deteriorates to 8 when he is intubated and ventilated. He is persistently hypotensive despite aggressive fluid resuscitation.

The initial assessment of the patient should be to evaluate the cause of persistent hypotension. Common causes of concealed haemorrhage are:

- (i) blunt injuries to the chest,
- (ii) long bone and pelvic fractures, and
- (iii) abdominal trauma.

A chest x-ray reveals most causes of concealed haemorrhage in the chest and long bone fractures are usually obvious. The diagnosis of abdominal bleeding is more difficult, especially in an intubated and ventilated patient.

With limited initial information, diagnostic studies treatment choices must be prioritized. They must be based on knowledge of the possible mechanism of injury and the time taken for each investigation. Further, the morbidity and the mortality of delaying operative intervention either for the abdominal injury or for the associated head injury needs to be considered.

Hypotension at anytime after the injury is a well known poor prognostic indicator in head injury. In patients with severe head injury and blunt trauma, up to half of preventable deaths can be caused by failure to stop ongoing haemorrhage and inadequate resuscitation. Of the patients who die from head injury, most deaths are caused by delay in craniotomy. Failure to evacuate traumatic subdural haematomas within 4 h is associated with a poor outcome. Hence, it is important to decide which operation to do first.

As many as 70% of blunt trauma victims suffer some degree of head injury so concerns about missing surgically treatable head injury are very real. Retrospective studies have shown that the abdominal trauma is more likely in younger patients involved in road traffic accident, especially in the presence of hypotension and tachycardia. Victims of motor vehicle accidents are less likely to have intracranial lesions. However, injury resulting from a fall, old age, and a low GCS score are more predictive of head injury. Further, retrospective studies have shown that laparotomies are more commonly performed than craniotomies in severe head injury with blunt trauma. The likelihood of a patient requiring both a craniotomy and laparotomy at the same time is small.

CT scans of the head and abdomen are useful in diagnosis and help in deciding which lesion should be dealt with first. Because of transport times and the need for administration of contrast, a CT scan can take 30–60 min. Therefore, a CT scan is only an option in a patient who is stable with on-going resuscitation. Other options for diagnosis of abdominal bleeding include diagnostic peritoneal lavage and ultrasound. A diagnostic peritoneal lavage has a high sensitivity but may lead to an excess of unnecessary laparotomies. An ultrasound is a useful bedside investigation but is unable to determine the exact sites of bleeding, especially in the presence of a haemoperitoneum.

Patients with marked haemodynamic instability not responsive to resuscitation, and suspected to be the result of intra-abdominal pathology should have an immediate laparotomy. If there is evidence of lateralizing signs or a depressed skull fracture, they should have a craniotomy simultaneously. If there is suspicion of a head injury, an ICP monitor should be placed intraoperatively and further management of the head injury should be guided by the ICP.

Occasionally, after aggressive fluid resuscitation, the patient may be haemodynamically stable in the presence of ongoing resuscitation. In this scenario, there is adequate time to perform a CT of the head and abdomen, if necessary. Once CT scans are available, laparotomy, craniotomy or conservative management can be considered.

There is a trend towards conservative management of liver and splenic trauma, especially if the patients remains haemodynamically stable. There are several obvious advantages of this. However, maintaining a CPP of >70 mm Hg may precipitate further bleeding; therefore a CPP of 50 mm Hg may have to be accepted.

Head injury with a non-life-threatening extra-cranial injury

A 20-yr-old man is admitted to Accident & Emergency, intubated and ventilated after a road traffic accident. His GCS was 9 at the scene. He has a femoral fracture that needs intramedullary nailing.

In the past, management of patients with long bone fractures was traction and delayed fixation. However, evidence now suggests that early (<24 h) fixation is associated with improved outcome. Advantages of early long bone stabilization include early patient mobilization attributable to elimination of the need for traction. Thus patients have less pain and blood loss may be reduced. Furthermore, there was significant reduction of pulmonary complication (e.g. acute respiratory distress syndome, pneumonia and fat emboli syndrome). As a result, patients required fewer days of mechanical ventilation and reduced length of ICU and hospital stay. Overall mortality was also reduced. Additionally, it seems that there may be a physiological advantage attributable to the reduction of locally produced cytokines with subsequent amelioration of the systemic inflammatory response. However, most of these studies were retrospective and had several methodological problems. Despite this, early fracture fixation has become the norm. There is certainly no evidence to suggest that this is detrimental to patients.

However, early long bone fracture fixation may not be beneficial in all patients, for example blunt chest trauma, associated injuries, and significant head injury. It may also not be suitable in patients who have had 'prolonged' resuscitation, for example requirement for blood or blood products for resuscitation, hypothermia, coagulopathy and prolonged surgery for other injuries. This is probably related to increased episodes of hypoxaemia and hypotension during the perioperative period.

In the head-injured patient, the avoidance of secondary brain injury is of paramount importance. Hypoxaemia, hypotension and raised intracranial pressures worsen outcome. Several studies have shown that timing of surgery is predictive of the development of perioperative hypotension. Patients are more likely to suffer hypotensive and hypoxaemic episodes if operated upon within the first 24 h. Treatment of hypotension usually involves further administration of fluid. Furthermore, cerebral oedema often increases 48–72 h after injury. Early fluid administration may be harmful in this group of patients.

Clinical factors that may be relevant in assessing the appropriateness of early long bone stabilization include:

- (i) severity of the brain injury (GCS, CT scan, ICP),
- (ii) severity of pulmonary dysfunction (PaO₂/FIO₂, lung
- compliance, positive end-expiratory pressure), and (iii) evidence of hypotension.

Most of the literature concentrates on long bone fracture fixation. However, trauma patients may have several other associated injuries. For example, facial injuries are common and early fixation is associated with better aesthetic results. However, principles similar to those with long bone fractures apply in this situation and maxillofacial surgery can be delayed for up to 72 h.

The timing of operative intervention should be determined by the response to initial stabilization, extent of intracranial injury and risks of delaying the operative repair. The priority has to be management of the head injury. Fixation of the long bone fracture can be delayed until physiological parameters are stabilized. Preferably, long bone fixation should be undertaken after 48–72 h. Compound fractures can be debrided and cleaned during resuscitation and stabilization. Skeletal traction and Thomas splint improves anatomical alignment and functional results. These can be used in the interim period until fracture fixation is undertaken.

Conclusion

The priorities for anaesthetic management are straightforward. Avoidance of hypoxaemia and hypotension with control of intracranial pressure avoids secondary brain damage; this is the cornerstone of anaesthetic management. Surgical intervention in the trauma patient is guided by the principles discussed in this article. Any injury causing persistent hypotension should be treated immediately. Space-occupying lesions in the cranial vault should be evacuated simultaneously. Limb-saving peripheral vascular surgery should be performed urgently. However, operative treatment of long bone and other fractures can be delayed until the head injury has been stabilized.

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See multiple choice questions 115–117.