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FOR INTENSIVE CARE TRAINING

Traumatic brain injury

Organ specific problems

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LEARNING OBJECTIVES

After studying this module on Traumatic brain injury, you should be able to:

1. Undertake early management of head injury
2. Define and detect secondary brain injury
3. Assess severe head injury
4. Treat severe head injury
5. Identify complications and outcome from severe head injury
# Introduction

## 1/ Early management of head injury

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- **Immediate assessment and treatment**
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  - Circulation
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- **Criteria guiding referral to neurosurgical hospital**

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- **Epidural haematoma**
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- **Cerebral oedema**
- **Diffuse cerebral oedema**
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INTRODUCTION

Traumatic brain injury (TBI) is one of the leading causes of mortality and disability in industrialised countries. Relevant yearly statistics include:

- TBI accounts for 150-200 hospital admissions per 100,000 of the population.
- The death rate from TBI is 14-30 per 100,000 of the population.
- The age group 15-35 years is at highest risk.
- The male/female ratio is 2.5:1.

TBI is a major cost to society in both human and economic terms.


Intensive care for patients with head injuries is a dynamic process starting at the scene of the accident. During the early stages of hospital care, the patient may require to be managed in a variety of locations including the emergency room (ER), the operating room (OR), the radiology department and the intensive care unit (ICU). It is in the best interests of patient care that the intensive care practitioner is thoroughly conversant with the practice of pre-hospital and immediate emergency care.
1/ EARLY MANAGEMENT OF HEAD INJURY

The main aim in the early stages of management should be to prevent or minimise the risk of secondary brain injury. The effectiveness of management will be optimised if you develop and practise a routine along the lines indicated below. You should, however, be prepared for the unexpected.

Clinical history

The importance of taking a complete history in a patient with acute head injury cannot be over exaggerated. You may obtain the details from a variety of sources and assemble the story on a piecemeal basis.

Of critical importance are eye-witness descriptions of the mechanism of injury, the level of consciousness when the patient is first found and reports from the ambulance crew and emergency doctors. Your history taking should include:

- Neurological state (consciousness, pupillary response) and vital parameters at the scene and during transport
- Estimated blood loss
- Nature of treatment at scene and in transit
- Use of airbags, seat-belts, crash-helmets
- Time, place, mechanism, vehicle speed (in relation to the accident)
- Past medical history
- Suspected influence of drugs or alcohol
- Possible medical reason for the accident (e.g. fit, heart attack, spontaneous intracranial haemorrhage, stroke)
- Use of anticoagulants or antiplatelet therapy

It will help you to appreciate the importance of obtaining as complete a history as possible if you prepare your own checklist with the items prioritised. In the next six patients with TBI you see, determine how relevant and useful your list proves to be.

There is a lot of evidence of the protective effect of, for example, airbags, seat-belts and crash-helmets. For further information refer to:

Promoting road safety measures contributes to healthcare
Q. In what way might information about level of consciousness at the scene of the accident prove to be of value in the subsequent care of a head injury patient?

A. This serves as an invaluable baseline for further observation. A falling level of consciousness indicates a problem that demands immediate attention. This is illustrated in the first patient in the ‘Patient Challenges’.

Q. At the scene of an accident tracheal intubation may have been carried out. What evidence is there that this benefits head injury patients?

A. Clear evidence is elusive. A widely accepted view is that intubation should be carried out ‘as soon as safely possible’ in selected patients. When transport times are long and trained personnel are available such management seems logical. In this context the adverse effects of transporting a severely head-injured patient with a compromised airway outweigh the possible complications of intubation in the field.

Recording of on-site observations is vital

‘Scoop and run’ versus ‘stay and play’

Immediate assessment and treatment

The ‘first line’ assessment (including physical examination) and treatment of any head-injured patient are closely inter-related and include the following:

- Secure (or maintain) the patient’s airway.
- Optimise oxygenation and ventilation.
- Initiate haemodynamic resuscitation and fluid administration.
- Identify both intracranial and extracranial injuries.
- Prioritisation of injuries (see management algorithm). Collate information about the mechanism of the injury and relevant past medical history.
- Continue to (re)assess level of consciousness.
Airway, breathing

Hypoxia is associated with higher mortality and increased morbidity following TBI.

Hypercapnia is not commonly detected but may contribute to vascular engorgement and induce increases of intracranial pressure.

The following manoeuvres should be performed without delay.

- Give high-flow oxygen (by face mask) to all patients with traumatic brain injury regardless of its severity.
- Presuming the presence of appropriate skills, intubate and ventilate patients with any impairment of airway reflexes or with abnormal pupillary response to light.
- Adopt strategies to minimise any immediate hypotension after intubation and ventilation.
- Avoid and/or treat aspiration.
- Check that arterial saturation is >95% except in patients with impaired lung mechanics or profound hypoxia in whom lower SpO₂ values (90%) may be preferable to the potentially injurious effect of recruitment and high PEEP levels.
- Avoid hyperventilation in the early phase of the injury. Early use of end-tidal CO₂ is strongly encouraged and pCO₂ should be kept between 30-35 mmHg (4-4.5 kPa) in normotensive patients. Avoid hypercapnia.
• Apply hyperventilation in patients with abnormal papillary light reflexes not due to direct iridis effects. If hyperventilation leads to hypotension then discontinue.


Q. With reference to the second bullet point above, will intubation and ventilation in these circumstances require the patient to be anaesthetised? If so, what techniques and drugs would you use and why?

A. The patient will require to be anaesthetised, even when consciousness is already impaired, to minimise the risk of secondary brain damage due to induced raised ICP (intracranial pressure). Rapid sequence induction and intubation is the recommended technique using a combination of sedative with low cardiopressant effects (e.g. midazolam, ketamine), analgesic (fentanyl) and muscle relaxant (e.g. succinylcholine) agents.

⚠️ When intubating a patient, think of possible injuries to the cervical spine and minimise the risk of possible further neurological insult.

NOTE Consider intubation and ventilation in patients with higher (Glasgow Coma Scale; GCS) motor scores (see below) but with associated injuries which place the patient at risk from acute hypoxia, hypercapnia and/or progressive hypotension. This is a complex situation where your clinical judgment matters. Complications such as crushed chest injury, pneumothorax, and ongoing bleeding reduce tissue oxygen availability.

Circulation

Hypovolaemia and hypotension are also associated with more mortality and morbidity following TBI. Once you have dealt with the airway and breathing you should rapidly turn your attention to the patient’s circulation.

• Insert at least two large bore peripheral intravenous cannulae.
• If hypotension occurs: first check for extracranial injuries.
• In patients without penetrating injuries and not suspected of having internal bleeding, establish a normal arterial blood pressure for patients age to obtain an adequate cerebral perfusion pressure. For adults consider a systolic pressure >110 mmHg and for older patients >130 mmHg.
• In patients with TBI and active bleeding accept a systolic pressure of 90 mmHg and rapidly transfer the patients to the closest trauma centre.
Q. What pathophysiological process makes it unlikely that isolated intracranial injury would cause hypotension?

A. It takes only 100 to 150 mL of intracranial blood loss to cause brain death by herniation. Thus hypotension normally signifies extracranial injury.

Q. Do you know of the commonly quoted exception to the above rule?

A. Only in newborn infants and babies can intracranial haemorrhage result in significant hypotension.

- Use intravenous isotonic solutions (e.g. Ringer’s-Solution, NaCl 0.9%) and colloids for volume resuscitation.
- Hypertonic saline has been used in the resuscitation of injured patients with TBI. Its efficacy in regard to survival and neurological outcome in TBI has yet to be proved.
- Theoretically, in patients with suspected increased intracranial pressure and with hypotension due to bleeding, hypertonic saline should be appropriate.
- In patients with clinical signs of transtentorial herniation high-dose mannitol (1-2 gr/kg) or hypertonic saline are potentially useful to reverse herniation.
- The routine use of colloids for patients with TBI is controversial. Results from the SAFE study showed a significantly higher mortality rate (24.6% vs 16%) in those patients with TBI who were volume resuscitated with albumin vs saline. For this reason, many practitioners avoid the use of colloids in these patients.

You will find useful reviews of this topic in:


THINK about the practice in your own region in relation to the choice of fluids for resuscitation in TBI - although efficacy in regard to neurological outcome in TBI has yet to be proven.

- Give vasopressor agents if adequate volume replacement fails to produce an adequate systolic blood pressure within minutes. In the context of TBI no one vasopressor has been shown to be superior to the other.
• In patients with penetrating injuries and in those with internal bleeding, increase systolic pressure with catecholamine to no more than 90 mmHg.
• Requirement for catecholamine during transfer is not uncommon and requires infusion via syringe pumps.
• Use catecholamine if hypotension appears to be due to sedation and ventilation.


Tako-tsubo cardiomyopathy and neurogenic pulmonary oedema

Some patients present to the emergency room with overt pulmonary oedema, and peripheral vasoconstriction. They may have an abnormal ECG, lactic acidaemia and a low pH. Arterial pressure may be elevated. Central venous saturation may be low. ECG shows prolonged ST segment elevation. Echocardiography shows transient left ventricular apical ballooning. This syndrome, previously named ‘neurogenic oedema’ or ‘stunned myocardium’ is now termed Tako-tsubo cardiomyopathy. It is the result of catecholamine storm in response to the initial acute increase of intracranial pressure after trauma. With supportive management the condition often resolves within hours or days but it ultimately requires a negative coronary angiogram for conclusive diagnosis.

You will find a useful review of this topic in:


Monitoring

Optimal resuscitation is facilitated by establishing accurate and reliable monitoring rapidly. Basic monitoring is indicated in all patients with significant head injury while more advanced monitoring is appropriate in selected instances.

Basic monitoring

Basic monitoring of patients with isolated head injuries should be initiated, where possible, at the site of the accident and include:
- 3-lead-ECG (all patients).
- Pulse rate and arterial blood pressure, non-invasive (all patients).
- Pulse oximetry (all patients).
- Capnography (ventilated patients). You will find of value the section on the relationship between PaCO2 and Pet CO2 in the following reference.
- Capnography is strongly suggested in the extrahospital setting where hypoventilation or inadvertent therapeutic hyperventilation is common.


Q. How frequently should such monitoring be performed?

A. As frequently as indicated by the patient’s condition. In a recently admitted or unstable patient, recordings every 15 minutes (or more frequently) are desirable.

Q. In the ‘Patient Challenges’, the clinical condition of the first of the two patients deteriorated. In such a situation, could any of the above parameters have changed and why?

A. Patients with severe head injuries show a marked variation in pulse rate and arterial blood pressure. An increase in blood pressure and a drop in pulse rate (Cushing’s response) are often found in patients with transtentorial herniation due to an increase in ICP and indicate an acutely critical state.

Neurological monitoring

Neurological monitoring may be initiated either in the emergency room or in the operating room (ICP-device) and after admission to the ICU (EEG, evoked potentials, transcranial Doppler, jugular bulb catheterisation).

**NOTE** Sedation +/- paralysis for intubation and then ventilation interferes with using the GCS as a monitor.

Further monitoring

After initial resuscitation, involving airway, breathing, and circulation, additional practical procedures include:
- central venous catheter (moderate/severe injuries; multiple trauma)

Q. What modification in the procedure of CVP line insertion would be appropriate in head-injured patients?
A. If the patient is haemodynamically stable, the patient’s head should be maintained at an elevation of 15-30° (head-down tilt may cause ICP to increase).

- arterial catheter (moderate/severe injuries; multiple trauma)
- nasogastric tube (or orogastric tube in presence of bad facial injuries)

In patients with fronto-basal injuries nasogastric tubes may perforate the base of the skull.

NOTE Consider bladder catheter with temperature measuring capacity as a monitor of core temperature (moderate/ severe injuries; multiple trauma).

- Pulmonary Artery Flotation Catheter (if otherwise indicated only)

Q. Give two ways in which misplacement or a complication of a central venous catheter placement may be a particular problem in a head-injured patient?

A. Either by causing bleeding in the neck region and therefore obstructing venous return or by producing a pneumothorax leading to hypotension or hypoxia or both.

Patient investigation

Laboratory

During the course of resuscitation, blood should be drawn for laboratory analyses. The sooner this is done the more valuable are the results in assessing the patient’s subsequent progress.

After admission to the emergency room the following laboratory values are obtained in all patients:

- Haemoglobin (haematocrit), leukocytes, platelets
- Sodium, potassium, blood glucose
- Coagulation parameters
- Blood type
- Blood urea, creatinine and electrolytes
- Liver enzymes
- Pregnancy test (where appropriate).

If available, more complex analysis of coagulation physiopathology should be indicated especially in patients with known or suspected history of anticoagulants or antiplatelets use and in patients with multiple injuries.

Patients with moderate or severe head injuries also require:

- Cross-matching of at least four units of blood
- Arterial blood gases.
Coagulation

Coagulopathy after TBI involves all components of haemostatic system, has a prevalence between 22 to 33% and a strong correlation with worse prognosis. Coagulopathy is particularly common in the presence of significant extracranial injuries. Tissue injury may induce consumption of clotting factors and fibrinolysis. Co-existing hypothermia, and acidosis due to hypoperfusion, may impair (i) the chemical reactions on which coagulation depends and (ii) the activity of platelets. In addition, crystalloid transfusion may dilute coagulation factors. Thus a vicious circle may be created in which haemorrhage is exacerbated and hypotension and anaemia (which further insult brain tissue) is exaggerated. We must also remember that an increasing proportion of the population are on long-term prescribed anticoagulants and antiplatelet agents.

All efforts should be made to stop bleeding as soon as possible; this may require compression of external bleeding sites on limbs, compression of the pelvis and if necessary angiography with embolisation, damage control abdominal surgery or embolisation for internal bleeding. In patients with TBI and active bleeding, it may be advantageous to maintain systolic pressure no higher than 90 mmHg - but with the intention of avoiding hypoperfusion. Hypothermia should be avoided with the infusion of warmed crystalloid fluids. Hypertonic saline may reduce the use of crystalloids and the risk of multiple compartment syndrome.

TBI patients may also develop endogenous acute coagulopathy (EAC) due to activation of the protein C pathway. This is thought to have occurred in about 25% of major trauma patients on arrival in the ED. EAC is characterised by anticoagulation derangement and hyperfibrinolysis but is not reflected by standard coagulation tests (aPTT and PT/INR). Therefore, once multiple injuries are found and the patient is unstable despite crystalloid infusion, balanced transfusion of blood products should be considered early, possibly without waiting for lab results. Balanced transfusion involves the use of plasma, platelets and packed red blood cells (1:1:1), with the aim to effectively reconstitute whole blood.

Point of care analysis such as thromboelastography (TEG) and rotational thromboelastometry (ROTEM) is improving knowledge on the pathophysiology of coagulation disorders and appears to be useful in the management of bleeding trauma patients. Early haemostatic monitoring including TEG or ROTEM, if accessible, should be performed during the first 72 hours after trauma.

In TBI patients with no extracranial injury, who have been taking oral anticoagulants (Vit K antagonists), guidelines suggest reversing the effects by means of prothrombin complex (concentrated factors) and vitamin K infusion once intracranial bleeding is detected at brain CT. The strength of the indication for anticoagulation in individual patients should be fully evaluated. The doses of prothrombin complex concentrates should be based on weight and guided by initial PT/INR values. In multiply injured, haemodynamically unstable patients plasma transfusion may be more appropriate since it adds both volume and coagulation factors.

In isolated TBI patients with intracranial bleeding who are on antiplatelet therapy, there is no consensus on management. Platelet function is more complex, difficult to measure and the targets of therapy remain uncertain.

You may find the reviews below of interest:


**Radiological**

Initial investigations should include the following:

In emergency room

- X-rays, Chest, pelvis
- Abdominal ‘FAST’ scanning

Isolated TBI

- CT scan of the head
- CT scan of cervical spine (C1 to T1)

TBI and multiple injuries

- CT scan thorax, abdomen, pelvis
- Reconstruction of thoracic and lumbar spine
- Angio-CT, arterial, venous and delayed phase

In the last few years, the improvement of time acquisition for CT scanning, suggests that whole body CT scan should be considered more in multiple injured patients as the first radiologic examination.

In TBI patients, this approach will be less time consuming than screening X-rays of chest and pelvis done in ER. The immediate use of whole body CT should be associated with a continuation of resuscitation care by means of the transfer of damage control strategies into the CT room.

Communication

At an appropriate stage during initial resuscitation, communication with the patient’s relatives is important. Consider who should undertake this task and in what way this might be conducted.

- One of the medical team involved in the patient’s care should undertake to speak with the relatives as soon as appropriate. (If the initial communication is by telephone caution should be exercised in providing information.)
- The relatives should preferably be seen by a senior member of staff but it can be useful to all if junior medical staff also attend. Supplementary important clinical information e.g. on medications being taken by the patient may only become available at this stage. Prognostication should be guarded at this stage since the situation may change rapidly.
- Comments as to the patient’s condition at this time should be limited and focused on the immediate plans for treatment.
- Provide reassurance that the patient is in good hands and that regular updates on progress will be forthcoming.

For further information see the PACT module on Communication.

Further and more detailed physical examination (secondary survey)

Upon completion of initial resuscitation and stabilisation of the patient’s vital signs, you should undertake a careful head-to-toe examination.

Inspect and feel the entire scalp. Wounds may be hidden by blood-clotted hair and fractures may be palpable. Look for

- Periorbital haematomas, bruising behind the ear (Battle’s sign) as shown in this illustration; cerebrospinal fluid (CSF) rhinorrhoea or otorrhoea may indicate basal skull fractures.
- Open injuries of the cranial vault with obvious CSF leakage or cerebral debris including gunshot wounds and stabbing.
- Foreign bodies (glass or metal splinters).
- Maxillofacial injuries and injuries to the eyes.
Carry out a comprehensive general examination to identify extracranial injuries.

- Observe the spontaneous breathing pattern of the patient. Before intubation abnormal respiratory patterns may indicate severe neurological damage. Diaphragmatic breathing, for example, may indicate an injury to the lower cervical spinal cord.
- In males, priapism may also indicate spinal cord injury.
- Assume all patients with head injuries have fractures of the spine (especially cervical spine) and treat them accordingly until such fractures have been ruled out radiographically. Road-traffic accidents and falls from great heights are associated with the highest risk of associated spinal injuries.

**Neurological examination**

The key aims of neurological assessment in the early stages of TBI are to determine the level of consciousness and to note the presence or absence of focal or lateralising neurological signs. In carrying out a neurological assessment:

- Determine the level of consciousness according to the Glasgow Coma Scale
- Examine the pupils for size, symmetry, and reaction to light.

The response of the pupils to light is dependent on intact afferent (optic nerve) and efferent (oculomotor nerve) function transmitting the light impulse from the retina to the midbrain and hence the pupillary musculature. Pupillary abnormalities are very common in patients in traumatic coma.

Assess the size and shape of each pupil both separately and compared to the other.

Assess the response of the pupils to light, both directly and indirectly.

Q. In the ‘Patient Challenges’ scenario the first patient’s sequential neurological examination revealed the rapid onset of left pupillary dilatation on the side of the intracranial bleed. What is the pathophysiological significance of this observation and its relevance to early management?
A. This indicates transtentorial herniation due to the developing haematoma, probably associated with elevated intracranial pressure.

Q. Outline the approach to the immediate management of this acute situation.

A. High-dose mannitol (1-2 gr/kg infused over minutes) or hypertonic saline should be administered to reduce raised intracranial pressure temporarily in order to gain time to get the patient to the CT scanner and to the operating room (OR). An alternative or combined strategy would be the use of hyperventilation acutely pending specific evacuative therapy.

- Examine the most meaningful brain-stem reflexes (corneal reflex, abnormal eye movements, cough and gag reflexes).
- Examine motor responses (arms and legs).

In conscious patients, begin with commands such as ‘Lift up your right arm’. In unconscious patients noxious stimuli such as pinching the ear lobe or cheek, or pressing firmly on the fingernail bed are used to elicit a response. All four limbs should be checked independently to detect focal deficits.

Major findings from carrying out this mini-neurological examination may include:

- Hemiparesis caused, in most cases, by an intracranial injury.
- Monoparesis of one limb, which may either be the result of a direct limb injury or the consequence of peripheral nerve damage (e.g. monoparesis of one arm caused by an injury to the brachial plexus).
- Paraparesis is usually the result of a spinal cord injury.

Check for respiratory abnormalities, see above.

Consider, in restless agitated patients, that patients with frontal lobe contusions may be without motor deficit.

Regular observation and careful documentation is critical to the detection of neurological changes.

A relevant, neurological deterioration is considered a decline of GCS of two points, or an evolution of CT scan, or a new abnormality in pupil reactivity to light.


Repeated GCS measurement is indicated even when the initial GCS value is high. In such case a decline of GCS is a useful guide for further CT and clinical decision-making e.g. as an indication for surgery or ICP monitoring. Conversely, patients presenting with low GCS values, abnormal pupil reactivity and with neurosurgical emergencies do not get the same benefit from further neurological examination as...
intubation and ventilation is implemented as soon as possible and, in such cases, repeated GCS evaluation may well conflict with appropriate care.

Patients who are not at high risk of intracranial hypertension, should be reevaluated when they have been stabilised. Younger patients, those who have had transient (but not severe) hypoxia and/or hypotension and/or anaemia, those with diffuse injury at CT without swelling or those presenting with intact eye opening, could be less severely affected than initially expected. Repeated neurological examination may help in these patients to detect those who may have been initially, mistakenly classified as severe TBI patients.

You should practise using the Glasgow Coma Scale and performing the associated neurological tests as often as possible in relevant patients. Note any difficulties you have and consult with senior/specialist colleagues. Try to formulate a ‘working diagnosis’ (e.g. ‘injury to the left hemisphere’, ‘spinal cord injury at a lower cervical level’) in each relevant patient and compare it with the radiological results. If you want to obtain more information on the clinical approach to the unconscious patient, see the following reference.


See the PACT module on Coma and altered consciousness where the Glasgow Coma Scale is addressed in detail.

We have now completed the early management of the patient with TBI. The importance of the initial ABC(DE) approach has been emphasised together with regular monitoring to allow early and effective therapeutic intervention. We now move to the important task of defining and detecting secondary brain injury. Initially however, we’ll review the important consideration of when to refer a patient with overt or suspected brain injury to a specialist centre.

Criteria guiding referral to neurosurgical hospital

Mortality and morbidity after a head injury are influenced by both primary and secondary damage. While there is no effective treatment of primary brain damage, prevention and/or effective treatment of secondary insults to the brain are the key issues in the treatment of patients with traumatic brain injury.

Trauma systems are differently organised within different countries. In the USA using an ‘exclusive’ system most patients are directly admitted to a trauma centre. In such systems, overtriage is frequent. Conversely in Europe the ‘inclusive’ Trauma system is usually utilised so most cases of minor trauma are admitted to a non-trauma centre hospital. In the European system telemedicine is useful to select those patients who could potentially benefit by referral to a neurosurgical hospital and trauma centre. Resource limitation tends to determine that secondary referral is confined to patients with potentially treatable lesions.
TBI patients affected by severe comorbidities should be individually evaluated, before consideration for secondary referral. Patients who certainly would be admitted secondarily to a neurosurgical hospital are those with mass lesions, those with abnormal pupillary reactivity to light, and those patients with diffuse swelling. Also patients with potentially evolving lesions, early traumatic brain contusion, vault or sylvian traumatic subarachnoid haemorrhage, or some kind of bone fracture, should be referred to a trauma centre to ensure a prompt intervention in case of further evolution.

For patients with diffuse injury without swelling, it is unclear whether transfer is appropriate, but a neurointensive approach is suggested for comatose patients with lesions in corpus callosum, diencephalon and rostral forebrain. Patients with extracranial injuries and severe TBI should be referred to a trauma centre as soon as possible once any cardiovascular or respiratory instability is resolved. Patients simultaneously affected by potentially evolving intracranial as well extracranial lesions should be referred centrally to have the best care in case of further evolution of intracranial lesion and/or extracranial bleeding.


Mortality and morbidity after a head injury are influenced by both primary and secondary damage. While there is no effective treatment for primary brain damage, prevention and/or effective treatment of secondary insults to the brain are the key issues in treatment.

Having ascertained the severity of brain injury at the earliest possible opportunity, this is the best guide to the extent of primary brain injury which has resulted from the direct mechanical trauma at the scene of the accident. We have seen in Task 1 how this may be achieved clinically using the Glasgow Coma Scale and we shall examine additional methods later in this Task. Changes in these parameters with time may assist you in detecting secondary brain injury, determining the effects of treatment and predicting outcome.

Primary and secondary brain injury often cannot however be clearly distinguished from each other.

Q. Another clinical description of head injury is ‘open’ or ‘closed’. How would you define an open head injury and how may it be caused?

A. An open injury involves direct communication between intra- and extracranial spaces. The integrity of the dura mater may be disrupted by bullets, sharp instruments or skull fractures.

Q. What is the relevance of the distinction between the ‘open’ vs ‘closed’ mode of head injury?

A. Patients with open head injuries are more likely to develop intracranial infection (meningitis, subdural empyema, brain abscess) or post-traumatic epilepsy and therefore require neurosurgical intervention (best done within the first eight hours).

Primary and secondary brain injury

Primary brain injury includes disruption of brain vessels, haemorrhagic contusion and diffuse axonal injury (DAI).


Secondary brain injury may have extra- or intracranial causes. Extracranial causes include systemic hypotension, hypoxaemia, hypercarbia, disturbances of blood coagulation and infection.

At macroscopic level intracranial causes include intracranial haematoma, brain swelling and cerebral oedema, intracranial infection and uncontrolled fits. At cellular level, a cascade of events contributes to secondary injury, and the inter-
relationship between primary and secondary brain insults and their potential consequences may result in ultimate cerebral cell ischaemia and cell death.

**NOTE**  Secondary insults are more commonly associated with cardiorespiratory disturbances and are independent predictors of poor outcome. They are often avoidable.


Identify the presence, severity and duration of secondary insults in the next ten patients with TBI. Determine to what extent more effective prevention and treatment of these secondary insults might have been achieved. Discuss these findings with your colleagues.

**Extra- and intracranial causes**

**Extracranial causes** of secondary insults to the damaged brain, e.g. hypotension and hypoxia, require prompt and efficient management. Following on from the section on resuscitation in the previous Task, subsequent maintenance of optimal cerebral oxygen delivery remains of paramount importance.

The more severe the extracranial injury the higher the risk of mortality. This association may be due to several factors beyond hypotension, hypoxia and anaemia, including multiorgan failure, infections and medical complications. However from a practical standpoint, the initial management should be directed to minimise any source of bleeding due to extracranial injury.

**NOTE**  One of the problems with the concept of optimising cerebral oxygen delivery is that traumatised tissue may not respond physiologically.

‘Autoregulatory impairment’ in TBI is a hotly debated topic, however in the early acute phase it has not been extensively studied. Patients with un-evacuated subdural hematoma are affected by elevated ICP and low CPP (Cerebral perfusion pressure). As a consequence, global cerebral blood flow (CBF) is reduced in the early phases corresponding with low jugular bulb oxygen saturation (SjO₂) values. Since deranged autoregulation is associated with ischaemia, it is likely that, in the acute phase, severely brain-injured patients have cerebral perfusion which is linearly dependent on arterial pressure.

Intracranial causes of secondary brain insult will be the focus of the remainder of this Task.

Traumatic brain injury is a heterogenous disease but the traditional classification, based on GCS, takes into account only the neurological consequences of trauma. Clinicians use at least computed tomography (CT) scan and the pupillary reactivity to light to define the severity of TBI. CT scan is by far the most informative tool to investigate the heterogeneity of TBI. At present a working group is defining a new classification of CT scan and pupil evaluation as a better guide for both clinicians and epidemiologists. In the meantime, CT scan lesions are currently classified by a classification of brain injury (see reference below) which distinguishes focal from diffuse lesions.


Before defining these lesions in more detail, you may wish to refer to the following summary of the main elements of CT interpretation.

**Interpretation of CT images**

You should start by reminding yourself of the normal anatomy as presented on a CT scan. Useful web addresses in this connection are:

http://rad.usuhs.mil/rad/iong/homepage.html
http://www.med.harvard.edu/AANLIB/

Before examining any CT scan always check the patient’s details, the date and time, and the anatomical orientation (i.e. the patient’s right is on the left side of the image as you examine it and vice versa).

According to their density, the various structures of the head absorb radiation to a different degree:
Interpretation of a CT scan involves two tasks: firstly to detect (what do I see?), … and secondly to analyse (what does this mean?). Things to look for are shown below:

<table>
<thead>
<tr>
<th>Scalp</th>
<th>Swelling</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skull</td>
<td>Fracture (CT better for skull base and midface)</td>
</tr>
<tr>
<td>Intracranial</td>
<td></td>
</tr>
<tr>
<td>Hyperdense</td>
<td>Blood</td>
</tr>
<tr>
<td>Mixed density</td>
<td>Blood and cerebral oedema</td>
</tr>
<tr>
<td>Hypodense</td>
<td>Cerebral oedema</td>
</tr>
<tr>
<td>Foreign bodies</td>
<td>metal/bone hyperdense; wood/glass etc. hypodense</td>
</tr>
</tbody>
</table>

The intensivist should be able to rapidly determine the following:

- Is there a focal lesion?
- Is it associated with a mass effect (see ‘mass effect’ heading below)?
- Would the patient benefit from an urgent evacuation or would it be more appropriate to monitor neurological status and/or ICP?
- Is there a diffuse lesion?
- Is it associated with brain swelling, and probably intracranial hypertension?
- Is it associated with petechiae, and where are they located?
- Are there both diffuse and focal lesions?
- Are there lesions warning of potential evolution toward a new mass or the enlargement of a previous detected mass?

The Marshall classification is extremely useful in that following these priorities, when looking at the CT, will automatically generate the CT score:

1. If there is a large mass (roughly above 25 mL), it is helpful to picture and determine whether there is ‘mass effect’
2. Does this mass object require an urgent evacuation? This indirectly suggests that the mass has a ‘mass effect’
3. Is it associated with a midline shift toward the opposite side where it is located? This is a quantitative indicator of a ‘mass effect’
4. Are the basal cisterns distorted or compressed on the same side of the mass lesion; this is a further sign of ‘mass effect’
5. Are the basal cisterns bilaterally compressed or absent? This is a sign of intracranial hypertension and coning due to the ‘mass effect’ or a sign of diffuse brain swelling.

A look to these five points focuses on intracranial emergencies and gives the best possible estimate of ICP before its measurement.

Secondarily it is important to evaluate:

1. The nature of the mass lesion: the physiology, evolution, management and prognosis is substantially different between the different lesions. Isolated extradural haematoma (EDH) needs emergent management and may have a relatively good outcome.
2. A subdural haematoma (SDH) will exert a mass effect and most are associated with cortical laceration. Mass effect together with simultaneous primary brain damage means that SDH is the haematoma with the worst prognosis.
3. Intraparenchymal laceration/contusion may sometimes need immediate surgery (as a consequence of the volume, or the location) but most are observed over hours and days. They may enlarge in the core or more frequently result in perilesional oedema. Lesions with a dense and large haemorrhagic core are called intraparenchymal haematomas. They are less frequent but need urgent evacuation, except in unsalvageable patients.

Once intracranial emergencies have been excluded, it is important to anticipate the evolution of mass lesion in the next hours.

1. Is there thin layer EDH or SDH in temporal fossa or posterior fossa? These can enlarge over time and rapidly cause regional hypertension.
2. Is there vault or scissural subarachnoid haemorrhage? This may be suggestive of an evolving intraparenchymal CT lesion.
3. Are there intraparenchymal lesions? These can develop over days and result in peri-haemorrhagic oedema.

Finally, once the warning signs of potentially evolving lesions have been excluded, the focus should be on the severity of diffuse lesions:

1. Are there multiple petechiae, or are there gliding petechiae, on the corpus callosum, on thalami, or on the midbrain? If present, an MRI is needed to improve the diagnostic specificity.
2. If the CT is negative, is an MRI indicated?
3. Are there risk factors for an extracerebral arterial lesion, e.g. dissecting lesion of extracranial carotid? If so, a CT-Angiogram may be needed.

Mass effect

A developing haematoma requires space. It therefore compresses other intracranial structures. Ultimately, the brain itself is compressed and displaced - the so-called space-occupying or mass effect. The larger the haematoma the more pronounced the mass effect. Patients with atrophic brains (alcoholics and the elderly) tolerate haematomas better than younger patients - they have more CSF.
In the case of supratentorial haematoma, the following sequence occurs:

- narrowing of the ipsilateral subarachnoid space and the ipsilateral ventricle
- shifting of the ventricle to the opposite side
- compression and displacement of the third ventricle
- transtentorial herniation of the medial portion of the temporal lobe
- compression of the paramesencephalic cisterns (pupillary dilation on the side of the haematoma).

A series of helpful diagrams illustrating various forms of herniation is available in the reference below. Although these mass effects are closely correlated with raised intracranial pressure, CT scans are not a substitute for continuous monitoring of ICP.


Focal lesions (intracranial mass lesions)

Approximately a quarter of all patients with severe head injury have an ‘operable’ intracranial haematoma.

Extra-axial lesions are found outside the brain parenchyma and include subdural and epidural haematoma. Intra-axial lesions include haemorrhagic contusions and traumatic intracerebral haematoma.

The localisation of a focal lesion in the posterior fossa deserves special and more urgent consideration.

The final decision as to whether and when to operate on an intracranial mass rests with the neurosurgeon and is based on a variety of factors in the individual patient (including space-occupying effect, neurological state and general condition of the patient). Over time, however, and with tuition in CT interpretation, the critical care doctor will become increasingly familiar with the haematomas for which immediate evacuation is indicated and for those which are not so urgent.

You should take every opportunity to participate in the case conferences and more acute decision-making processes. As your experience grows your input will be increasingly valued. Note where decisions are based on ‘solid information’ and where on ‘clinical judgment’. Ask questions about the latter and remember a previous example we noted in Task 1.

Skull fractures

You may be confused about the significance of skull fractures which are a common finding in head injury. If the fracture is closed and not depressed, specific treatment is rarely required and healing occurs spontaneously. Open and depressed fractures have to be treated by the neurosurgeon.

Normally, skull fractures are not palpable through the intact galea. Bruises to the skin may indicate an underlying fracture; periorbital (raccoon eyes), and retroauricular haematomas (Battle’s sign) may indicate basal skull fractures (look for CSF fistula). Patients with skull fractures have a high incidence of intracranial haematomas (see below).

<table>
<thead>
<tr>
<th>Severity of Head Injury</th>
<th>Haematoma on CT (%)</th>
<th>No Haematoma (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Severe (GCS 3-8)</td>
<td>with fracture</td>
<td>44</td>
</tr>
<tr>
<td></td>
<td>without fracture</td>
<td>32</td>
</tr>
<tr>
<td>Moderate (GCS 9-12)</td>
<td>with fracture</td>
<td>29</td>
</tr>
<tr>
<td></td>
<td>without fracture</td>
<td>8</td>
</tr>
<tr>
<td>Mild (GCS 13-15)</td>
<td>with fracture</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>without fracture</td>
<td>1</td>
</tr>
</tbody>
</table>

Incidence of intracranial haematomas depending on the severity, and the presence of a skull fracture in 3802 patients (Miller JD, 1995)

Detection of a skull fracture is important and is usually detected by means of emergent CT.

Traumatic subarchnoid haemorrhage (tSAH)

This is the presence of blood in the subarachnoid space. Its distribution varies greatly, but it may be located
- in the cortical vault and/or within cortical scissurae, and/or
- at the base of the brain, in the basal cisterns, over the tentorium, and/or
- in the interpeduncle space.

Most evidence suggests that subarachnoid blood in TBI is not associated with vasospasm, but is a landmark of CT severity. Furthermore traumatic subarachnoid haemorrhage (tSAH), especially if located above the cortex or within scissurae could be a warning for potentially evolving cortical lesions which bleed into the subarachnoid space.
Normal neurological status on admission does not rule out significant brain pathology. An epidural haematoma (EDH) is an accumulation of blood in the epidural (also known as ‘extradural’) space between the inner side of the skull and the dura mater. In most cases the cause is a skull fracture crossing the middle meningeal artery or its branches in the fronto-temporal region. Rarely, a fracture may be associated with tearing of large veins at the vertex of the skull or of the venous sinuses of the brain itself.

You will find further images in the appendix in the interactive version.

Patients with a skull fracture may be neurologically intact on admission and later deteriorate as the EDH develops. More often, however, primary brain damage has caused some disturbance of consciousness and the developing haematoma results in rapid neurological deterioration. This sequence of events occurs in the first of the patients in the ‘Patient Challenges’.

Q. Epidural (extradural) haematoma (EDH) is quite uncommon in the young (<5 years) and the elderly (>65 years). Why do you think this might be?

A. The dura is tightly adherent to the skull in these age groups and does not tear easily.

The management is described by ABC guidelines. EDH is a medical emergency and the final outcome is definitively affected by a prompt critical care and surgical intervention. Once the patient is declining neurologically and surgery has been
decided, especially for patients requiring transfer from a hospital which is far from the neurosurgical centre, it is preferable to intubate, sedate and ventilate the patient to transfer him/her to OR in the best condition possible - having taken and titrated concurrent, acute, temporising measures to reduce the elevated intracranial pressure.


**Subdural haematoma**

Subdural haematoma (SDH) is an accumulation of blood between the inner side of the dura and the arachnoid layer of the brain. It occurs if a cortical vessel is torn. In most cases a large contusion at the frontal or temporal surface of the brain is found. Subdural haematomas are called acute if they develop during the first 24 hours, subacute if they develop between 1-7 days, and chronic thereafter.

As most patients with acute SDHs have some kind of accompanying brain injury, their prognosis is worse than that of patients with EDHs.

Most patients harbouring an acute SDH are unconscious immediately after the trauma. The expanding haematoma then causes additional neurological deterioration. You will find further images in the appendix in the interactive version.

Subacute subdural haematoma is more common in patients with an atrophic brain. Most of them have minor or moderate disturbances of consciousness level at first and deteriorate during the first two to four days.

The management is described by ABC guidelines. Intensivists and anaesthetists should consider that before SDH evacuation, ICP can be extremely elevated and every effort should be done to minimise intracranial hypertension even if not measured.

In the forthcoming period, analyse patients that are diagnosed with either epidural or subdural haematomata. List how you differentiate between these two conditions. Compare your answer with the table below.

<table>
<thead>
<tr>
<th></th>
<th>Epidural</th>
<th>Subdural</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Incidence</strong></td>
<td>Approximately 10% of all patients with severe head injuries.</td>
<td>Most frequent extra-axial haematomas in patients with TBI.</td>
</tr>
<tr>
<td><strong>Onset</strong></td>
<td>Most develop during first 8 hours after the injury.</td>
<td>Occur either during the first 24 hours (acute) or during day two to four (subacute) after the injury.</td>
</tr>
<tr>
<td><strong>Clinical features</strong></td>
<td>Risk of rapid deterioration of conscious level (GCS) with focal neurological signs (ipsilateral dilating pupil, contralateral hemiparesis).</td>
<td>Rapid neurological deterioration (GCS) and focal neurological signs (ipsilateral dilating pupil, contra lateral hemiparesis) in an already deeply comatose patient in acute cases. More gradual neurological deterioration and focal neurological signs in subacute or chronic cases.</td>
</tr>
</tbody>
</table>
Traumatic contusion/laceration and intracerebral haematoma:

Parenchymal lesions are by far the most frequent lesion in TBI. They cover a wide range, moving from hypo-attenuated lesions without bleeding to lesions with a dense haemorrhagic core.

Traumatic contusion core is affected by an irreversible damage of the tissue. Bleeding can occur within this area in a variable manner, from none to ‘salt and pepper’ appearance on CT, to larger lesions. In the case of larger lesions they are more appropriately defined as a traumatic haematoma. In such lesions, a core bleeding (hyperdense lesion) predominates at CT. Traumatic haematomata are frequently larger than traumatic contusions but there is no clear cut-off defined. The initial CT scan often does not represent the definitive size of the contusion or haematoma since these lesions can enlarge even days after the original trauma. The enlargement of the core is unlikely after the first days.

However, a further evolution can involve an increase of the intraparenchymal lesion via growth of perilesional oedema. This usually peaks at the end of the first week post injury.

Space-occupying (mass) effect, neurological state and general condition of the patient are all variables influencing the management. Relevant for surgical decision is also the location of mass lesion in an eloquent area (e.g. left temporal lobe). A conservative approach, applying a higher level of medical therapy, is suggested in such cases. The previous general medical condition of the patient and complications during ICU stay may argue in favour of surgery to decrease the risk of further functional impairment.

Q. In relation to head injury, arrange intra- and extra-axial haematoma e.g. intracerebral, subdural and extradural haematomata in order of frequency?

A. Intracerebral, subdural, extradural.

Remain vigilant particularly in the early stages of head injury. Do not consider ICP a surrogate for CT at least in the first phases. Any progressive increase of ICP would
indicate a need to repeat the CT to disclose any potential mass evolution.

Q. We have emphasised the importance of early assessment in TBI. Is a negative CT scan on admission sufficient to rule out intracranial haemorrhage?

A. Most are diagnosed on the first CT, but they can arise or enlarge even days after the injury. Therefore a repeat CT scan is suggested in all patients with a severe or moderate injury within 6 hours after the initial CT scan.

In patients in coma, with traumatic subarachnoid haemorrhage (tSAH), fractures or a known history of antiplatelet or anticoagulant therapy, it is suggested to repeat CT even earlier, 3 hours after the first CT.

Although at a poor grade of evidence, surgical guidelines suggest some strong recommendations.


Penetrating injuries

Penetrating injuries are caused by gunshots, knives, and other objects that penetrate the skull and the brain tissue. External evidence is a scalp wound (sometimes quite minor).

The associated fracture is usually depressed and can be diagnosed on plain skull X-rays or CT scan. Depending on the site and force of the injury, patients with stabbing injuries may be neurologically intact or may be awake with only focal neurological signs.

Patients with gunshot wounds tend to present at both ends of the GCS spectrum i.e., with scores of 3-5 or 12-15. The prognosis in patients with a GCS of 3-5 is extremely poor. You will find further images in the appendix in the interactive version.
Although patients with penetrating injuries may sometimes present in
dramatic fashion, they should be treated by the same algorithm used in all other
patients with head injuries.

Examination of the whole scalp and body is essential in order not to overlook second
or third injuries.

Foreign bodies should not be removed in the resuscitation room or ICU as this may
cause massive intracranial haemorrhage.

[No authors listed] Surgical management of penetrating brain injury. J Trauma
2001; 51(2 Suppl): S16-25. PMID 11505195

Diffuse axonal injury

Diffuse axonal injury (DAI) without an intracranial mass occurs in
almost half of patients with severe head injury. Neuropathologically
severe DAI is a microscopically widespread damage to axons, often
associated with scattered small haemorrhages and mainly located
along or near the midline. It is classified in three subtypes. It may
predominate:

- At the junction between the cortex and the white matter and be
  suspected by the presence of small petecchiae - gliding
  contusion.(grade 1) or
- In the corpus callosum, often associated with traumatic intraventricular
  haemorrhage (grade 2) or
- In the thalamus and brain-stem (grade 3).

Adams JH, Doyle D, Ford I, Gennarelli TA, Graham DI, McLellan DR. Diffuse
axonal injury in head injury: definition, diagnosis and grading.
Patients with severe DAI are often in deep coma in contrast to a CT scan that often looks quite normal. In such cases but even in those with overt CT findings, an MRI during the acute phase is suggested. A T2-weighted MRI will reveal the cerebral damage to a greater extent than CT. A sequence called FAST Field Echo can emphasise haemorrhagic lesions more clearly, while the FLAIR sequence demonstrates damage to myelin.

The morphologic pictures are complex and a tight relationship between clinical patterns in acute phase and long-term outcome have not been yet established. However the early MRI (within few days) may help in the decision to monitor ICP, to estimate the expected duration of coma and the need for mechanical ventilation and tracheostomy. Furthermore it could ultimately be of help in planning the most appropriate rehabilitation care.

You will find a detailed discussion of this form of TBI in the following reference.

**Cerebral oedema**

Cerebral oedema is a consistent reaction of the brain to a variety of insults. It usually develops during the first three to five days and causes an increase in intracranial pressure, thus creating a potential vicious cycle (see diagram below).

![Diagram of cerebral oedema cycle](image)

e.g. hypotension, hypoxaemia, pneumonia, sepsis, coagulopathy

Cerebral oedema signifies an increase in the brain water content. There are three different types of cerebral oedema - vasogenic, cytotoxic, and interstitial.

Cerebral oedema should be distinguished from brain swelling, which is a more generic term describing just a homogeneous increase of brain volume independently from the cause. It may be associated with specific intraparenchymal lesions.

Patients with head injuries usually have a mixed type of oedema: vasogenic and cytotoxic.

**Diffuse cerebral oedema**

The most common imaging applied to TBI patients is CT but it is unable to detect diffuse cerebral oedema. The reduction of the difference between the density of the grey matter toward that of the white matter is a subjective, operator dependent, index of cortical oedema (hypoattenuation of the grey matter).

CT scan can suggest an increase of global cerebral volume and this picture is called ‘diffuse brain swelling’ to avoid any evaluation of the nature of this volume increase. However MRI studies suggest that in the early days this oedema is predominantly cytotoxic in origin. Magnetic Resonance Imaging has the potentiality to quantify diffuse oedema.
Focal cerebral oedema

Unlike diffuse cerebral oedema, CT can easily detect focal oedema. In trauma it is commonly appreciated in cases of traumatic contusion/laceration. It may appear immediately or evolve over days. Initial and central oedema is probably the expression of tissue disruption and cytotoxic oedema.

In contrast, the oedema that enlarges concentrically from the core over days is predominantly vasogenic oedema. A further subtype of oedema has been described, osmotic oedema. This type of oedema is located in the central area of tissue disruption where water accumulates driven by the high osmotic forces due to concentration of cellular debris.

Further oedematous lesions are those affecting post-traumatic infarction (predominantly cytotoxic), the non-contused cortex behind subdural haematoma, or the oedematous area of cerebral venous infarction (predominantly vasogenic).

The estimation of the subtype of the oedema by means of just CT is not possible and its interpretation is simply conjectural on the basis of clinico-anatomic knowledge. MRI can define case by case the respective relevance of vasogenic or cytotoxic oedema. This evaluation is not academic because vasogenic oedema is potentially reversible and should be spared by surgery. Furthermore in the near future the use of medical therapies may be guided by this knowledge.

Q. What is the relationship between the volume of cerebral oedema and the increase in ICP?

A. The ICP increases with increasing cerebral oedema but there is no direct quantitative relationship.

Q. Is the duration of the pathology a major determining factor in this relationship?

A. The duration of oedema/space-occupying lesion is an important determinant of how much the ICP rises - in more chronic lesions there is a greater volume reserve than in acute conditions.
Q. In a patient who has deteriorated neurologically, what are the CT scan features which raise the suspicion of increased ICP and how would you confirm this?

A. The third ventricle becomes obliterated and the basal cisterns compressed. In the case of mass lesion an enlargement of the lesion, an increase of shift, distortion of the ipsilateral perimesencephalic cisterns, compression of ventricular horns, or the presence of unilateral hydrocephalus.

Q. How good are these CT features in predicting elevated ICP. How would an elevated ICP be confirmed?

A. While these radiological parameters are associated with elevated ICP, their predictivity remains poor. In younger and paediatric patients, high ICP can be overestimated. Suspicion of raised ICP can be confirmed by measuring it.


Brain-stem lesions

Brain-stem lesions have been traditionally believed to be invariably associated with a poor outcome. Their pathogenesis is however heterogeneous and the ability to detect and understand this is easier now due to MRI. Recently a clinico-radiological classification which is MRI based has been proposed:

- Secondary to supratentorial herniation (mass lesion with shift)
- As a part of severe diffuse brain injury (the grey-white matter junction, corpus callosum, basal ganglia/internal capsule/thalamus)
- Isolated/remote brain-stem injury (without supratentorial lesion due to diffuse injury).

Post-traumatic cerebral ischaemia and infarction

Post-traumatic cerebral ischaemia (PTCI) includes functionally impaired yet still viable tissue, so-called ischaemic penumbra, and irreversible cerebral infarction. It is recognisable on CT as a new hypoattenuation area, without a perilesional distribution. Post-traumatic cerebral infarction can be present with three different patterns:

1. **Territorial cerebral infarction (complete or incomplete):** well circumscribed hypodense lesions within a defined cerebral vascular territory, involving the entire territory (complete) or only part of it (incomplete).

2. **Watershed cerebral infarction:** well circumscribed hypodense lesions located in boundary zones between the territories of anterior, middle and posterior cerebral artery (superficial or leptomeningeal border zones) or situated in terminal zones of perforating arteries within the deep white matter (deep or medullary border zones).

3. **Nonterritorial nonwatershed cerebral infarction:** single or multiple hypodense lesions, unilateral, bilateral, or multifocal with marked borders without a precise localisation in a vascular territory.

4. **Post-traumatic cerebral infarction is associated with more intracranial hypertension and with a poorer prognosis.**


Post-traumatic venous infarction

Thrombosis of a major intracerebral sinus and veins may occur frequently in association with fractures of the vault. It can be suspected in the presence of atypical haemorrhage with large volume oedema which evolves over the first hours.

Diagnosis should be confirmed by means of CT-Angiogram or MR-Angiogram, which is the preferred modality.

MRI is of specific interest for its capability to detect oedema in the territory of the occluded veins.

This condition is frequently associated with seizures, which may aggravate intracranial hypertension.

The treatment may be surgical, if secondary decompression seems appropriate, or medical. Treatment with heparin is usual.

**Blast injury**

In recent years, patients more frequently present with injuries not due to blunt or penetrating trauma. The current Iraq conflict and the prominent role of improvised explosive devices (IED) dramatically increased the fraction of war-associated TBI. This perhaps led to the well-publicised view that blast-induced traumatic brain injury (bTBI) is the signature brain injury for combat troops in today’s military. The Centers for Disease Control and Prevention (CDC) defines blast injury in four phases. However, the bulk of bTBI occurs in the first three phases: the primary injury phase is comprised of the response of brain tissue to the blast wave (an intense over-pressurisation impulse component of the blast). The secondary injury phase results from shrapnel penetration into the head. The tertiary injury phase results from head contact/acceleration forces as the body is moved by the ‘blast wind’ (a forced super-heated air flow). The quaternary injury phase incorporates any injury not covered in the other three phases such as some of the extracranial injuries or ‘polytrauma’ including haemorrhagic shock and chemical or thermal burn injuries that can occur. This quaternary phase of bTBI can significantly alter the timing and consequences of the primary damage occurring in the first three phases, and therefore can be a major contributor to overall brain pathology. This may be particularly true in mild bTBI, where there are either minor or no complicating factors.

See the reference for further information.


**Cellular and molecular events**

Recent research has shed light on changes at cellular and molecular level in TBI. Glutamate and other ‘excitotoxic’ transmitters released from damaged neurones generate unnecessary action potentials and squander cellular energy resources. Failing tissue perfusion can lead to the build up of metabolites such as lactate and highly reactive free radicals, which damage the membranes of neurones and their organelles. Compromise of structure and function can lead to distortions in transmembrane ion gradients, a catastrophic rise in the cytoplasmic calcium concentration, and cell death. These various changes are summarised in the diagram below.
In this Task we have discussed the various pathophysiological categories of secondary brain injury and considered how the condition manifests itself clinically and radiologically. The vicious cycle of cerebral ischaemia, cerebral oedema and raised ICP has been noted as a constant threat in TBI. Advances in neurobiology form the basis for future therapeutic strategies. In the next two tasks we shall examine this issue further in relation to the assessment and treatment of patients with severe head injury.

As always, prevention is better than cure
Inevitably, a proportion of patients have severe brain injury as a result of primary or secondary damage. These patients are amongst those normally transferred to a general ICU or a neuro-surgical ICU (see definition of severe head injury, GCS table) as a function of local hospital organisation and resources. Patients with moderate brain injury may also require an intensive care setting for a decline in neurological status and airway defence reflexes or to monitor ICP if there is a risk of evolution of a mass lesion.

**Neurological monitoring**

Some measurements are considered routine in specialised centres and units. Others are more experimental, more complex and their value in the management of individual patients is still uncertain.

**Standard measurements**

- Neurological status (GCS, pupils, motor signs, brain-stem reflexes)
- ICP
- Cerebral perfusion pressure.

**Advanced measurements**

- Brain tissue PO2
- Thermo dilution CBF measurement
- Jugular bulb oximetry
- Transcranial Doppler
- Evoked potentials
- Continuous EEG
- Microdialysis
- Cortical microelectrodes.

**ICP monitoring**

A good grasp of three physiological concepts will aid in your management of patients with TBI. To refresh:

- Intracranial volume is a constant in adults. An increase in any of the component volumes (blood/CSF/brain/pathological mass) must result in a decrease in one or more of the others (Monro-Kellie doctrine).
- The relationship between intracranial volume and ICP is exponential.
- Cerebral perfusion pressure (CPP) is defined as the difference between mean arterial pressure and mean ICP (i.e., \( CPP = MAP - ICP \)).

Raised ICP occurs in approximately 50% of all patients with mass lesions and 30% of all patients with severe diffuse injuries. If not controlled, it is a frequent cause of death in patients with head injuries. Mortality and morbidity in patients with head injuries is directly related to the duration of ICP above an arbitrary critical threshold of 20 mmHg. As we have already seen, raised ICP is associated with a reduction in cerebral perfusion as a result of decreased CPP.

The relevance of continuous ICP monitoring is highlighted once a digital continuous recording is obtained. Continuous digital monitoring is more accurate than nurse led hourly episodic measurement of ICP.

**Definitions and normal values**

ICP is the cerebrospinal fluid pressure measured via a catheter in the ventricular system with its tip at the level of the Foramen of Monro without loss of fluid from the system (equivalent to ventricular fluid pressure, VFP).

Normal values of VFP
- Newborn <7.5 mmHg
- Child <10.0 mmHg
- Adult <15.0 mmHg

ICP can be recorded at various sites in the cranial vault. Fluid pressures can be recorded in the ventricular system, the cisterna magna, and parenchymal pressures in the brain tissue itself at various locations (e.g. supra- or infra-tentorial). Pressures can also be recorded in the subdural, subarachnoid or epidural space. Subdural and epidural ICP recordings underestimate true ICP levels, especially at high levels of ICP; this is also true for parenchymal pressures at low levels of ICP.

It must also be kept in mind that, especially in cases with mass lesions, ICP gradients may occur which are the driving force for brain-shift and herniation. This is particularly clinically relevant for temporal and frontal masses.

**Why is intracranial volume constant?** Given that the relationship between intracranial volume and pressure is exponential, construct a graph of the relationship.

Intracranial haematomas in the infratentorial space are much more dangerous, as this is a very small compartment compared with the supratentorial space.
Q. In which situation does withdrawal of CSF via a ventricular catheter lower ICP most?

A. If overt hydrocephalus is present (rare in TBI) or a more subtle disturbance of CSF reabsorption has occurred.

Q. Even if no overt hydrocephalus is present, withdrawal of CSF may be beneficial if ICP is very high. Why?

A. In such cases the ICP value is in the ascendent portion of the intracranial pressure/volume curve and the withdrawal of small volume induces a relatively large reduction of ICP.

Q. Why, in the above circumstance, might the beneficial effect be unfortunately transient in nature?

A. Because accumulation of the cerebrospinal fluid was not the original cause of the elevated ICP, the effect in lowering ICP is transient.

Components of the basic ICP waveform

Under normal conditions cardiac and respiratory pulse are superimposed on the baseline level of ICP, resulting in a characteristic waveform. It remains unclear whether the pulsatile fluctuations of ICP are primarily arterial, venous, or both in origin. ICP values are also dependent on the intrathoracic pressure, which influences venous outflow from the intracranial space. In fully ventilated patients, inspiration increases ICP and expiration causes ICP to decrease.

ICP waveform disturbances

As ICP increases certain pathological waveforms can be observed on the ICP record. These abnormal waves are defined by their peak pressures and duration. A, B (see figure below) and C waves have been described but it is common to see a change in ICP which cannot easily be classified as one of these wave patterns. Individual patients may show a spectrum of events.
Indications for monitoring ICP in patients with head injury

While guidelines suggest that ICP monitoring should be instituted in all patients with severe head injury (Glasgow Coma Score <9) and positive findings on CT, all surveys or studies dealing with the real application of such guidelines show an incidence of ICP monitoring markedly lower than expected.

Randomised clinical trials on the use of ICP monitors are difficult to design, as ICP monitoring is an accepted intervention for more severely injured patients. Nevertheless there is a conceptual agreement that ICP monitoring is not useful in patients who are considered to be unsalvageable. Other patients with less severe injuries, in whom a persistent increase of ICP is not expected, may not need a monitor. Data from observational studies shows that this is often the case in reality. Doubts also exist about the threshold to measure ICP in older patients as their risk of developing intracranial hypertension is lower.

A practical approach to decision-making in relation to whether or not an ICP monitor ought to be placed, is to develop local protocols which would define:

- exclusion criteria
- situations where ICP monitoring is considered mandatory
- situations in which each case should be discussed individually.

Criteria of exclusion would be:

- patients with bilateral unreactive midriasis
- untreatable lesions at CT
- advanced age and comorbidities
- extracranial lesion difficult to manage
- coagulopathy
- high GCS motor score (≥5).

ICP monitoring will be indicated where CT findings indicate a potentially evolving lesion or a diffuse lesion at risk of high ICP (i.e. obliteration of the third ventricle or compressed basal cisterns). ICP monitoring may be indicated in these situations even for patients with less severe neurological score (moderate head injury).

The appropriateness of ICP monitoring is uncertain (and will require individualised assessment and decision-making) where the lesion is less severe e.g. DAI grade 1, isolated tSAH without CT evolution at repeat CT, or isolated focal lesions in atrophic brains.

The flowchart below explains visually a potential method to approach the indication for ICP monitoring.
ICP monitoring – inclusion criteria

Are there specific indications to measure ICP? And no exclusion criteria present?

Yes – strong recommendation
Non-ventilated patients with a higher GCS can still be evaluated clinically. In some patients a second CT may be needed before placement of an ICP monitor.

Yes – strong recommendation
With the exclusion of patients with bilateral unreactive pupils or patients with severe coagulopathy.

Diffuse injury type III or IV

No

Yes – strong recommendation
Suggestive of intracranial hypertension. Risk of post-traumatic cerebral infarction

Anisocoria

No

Yes – strong recommendation
Patients undergoing craniotomy with the intent to remove focal mass in whom the bone flap was not replaced (excludes therapeutic decompression or cases of anisocoria).

Primary decompression

No

DAI type I or II

No

Yes – weak recommendation
Diffuse axonal injury (DAI) with deep lesion, thalamic, mesencephalic and with motor GCS ≤ 4

Multiple injury

No

Yes – weak recommendation
Useful to detect increased ICP levels due to extracranial causes. Contraindicated in patients with traumatic coagulopathy

Other clinical pattern, evaluate case by case

No
ICP monitoring—exclusion criteria

Moderate or severe TBI

Bilateral unreactive mydriasis

YES – ICP monitoring probably not useful
Clinical correlation with GCS 3, after stabilisation of vital functions, without confounding factors

NO

Untreatable CT lesion

YES – ICP monitoring probably not useful
Lesion at the base of the skull, brain-stem lesion, multiple intraparenchymal lesions exerting mass effect, large post-traumatic cerebral infarction

NO

Advanced biological age

YES – ICP monitoring probably not useful
Conservative surgery of the focal lesion and/or medical care may be warranted. Lower risk of high ICP due to natural atrophy

NO

Contusion not evolving & low risk of coning

YES – ICP monitoring probably too aggressive
Evaluation needed after at least 2 CTs. If the patient can be clinically examined, ICP measurement is less vital. In younger patients cranial compliance is less and elevated ICP is more likely. If an increase in peri-lesional oedema is observed, reconsider ICP measurement.

NO

Petechiae without swelling and GCS ≥ 5

YES – ICP monitoring probably too aggressive
MRI recommended to exclude brain-stem and diencephalic lesion. Clinical evaluation will help determine whether or not to measure ICP.

NO

SAH or small lesions

YES – ICP monitoring probably too aggressive
Decision taken after one or more stable CTs in first 24 hours post injury. Favoured by improving GCS levels. This strategy is easier to implement if the patient is not sedated i.e. it is possible to evaluate the clinical status.

Evaluate inclusion criteria to monitor ICP


Find out what proportion of head-injured patients with a GCS <9 in your institution have ICP measured. What is the indication for measuring ICP at your institution?

Read studies which reviewed the decline of mortality in association with introduction of measurement of ICP.


Methods to measure ICP

As previously indicated, ICP can be measured by a variety of methods and in different anatomical sites. The methods include fluid-coupled systems with an external transducer, and fibre optic and piezo-resistive systems. The epidural and intraventricular spaces and the parenchyma are the most commonly used anatomical sites (see figure).
Each has its advantages and disadvantages (see below).

<table>
<thead>
<tr>
<th>Method</th>
<th>Advantages</th>
<th>Problems</th>
<th>Indications/Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular</td>
<td>‘Gold standard’</td>
<td>Infection, Risk of haemorrhage, Occlusion</td>
<td>Absolute contraindication: disturbances of blood coagulation, Relative contraindication: narrow or displaced ventricles</td>
</tr>
<tr>
<td></td>
<td>Recalibration possible</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Reliable and cheap</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>CSF can be withdrawn to lower ICP or to measure compliance</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parenchymal</td>
<td>Reliable</td>
<td>Recalibration not possible, Expensive, Infection (lower than ventricular), Risk of haemorrhage (lower than ventricular)</td>
<td>Absolute contraindication: disturbances of blood coagulation</td>
</tr>
<tr>
<td></td>
<td>Insertion simple</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epidural</td>
<td>Simple</td>
<td>Artefacts, Recalibration, usually not possible, Expensive</td>
<td>Relative contraindication: disturbances of blood coagulation</td>
</tr>
<tr>
<td></td>
<td>Low infection rate</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

You should observe the various techniques used to set-up ICP monitoring systems and observe how the measurement is used to influence individual patient care. What are the implications for your own practice?


**Threshold for ICP treatment**

Conventionally, the ICP threshold for treatment is 20 mmHg. However, after the first days the threshold should be elevated to 25 mmHg.

In patients with temporal lesions or diencephalic lesions, a regional coning may occur even with low ICP values and the threshold of treatment should be lowered to 15 mmHg.
Threshold for CPP treatment

The level of CPP changes according to the type of CT, age and previous arterial hypertension.

Older patients affected by arterial hypertension might need a CPP within the 70–90 mmHg. Conversely children, young male and adult females should probably have a CPP target not below 50 mmHg.

Patient with focal lesions may have an oedematous penumbra in which cerebral vascular resistance is likely to be higher. In such cases high CPP values are suggested utilising regional monitoring or functional imaging in the evaluation of rCBF. In the presence of a standard monitoring in patients with a focal lesion, the target of the clinicians will be to avoid a fall of CPP below predefined values.

Approach to the interpretation of ICP data

Always check the patient as well as the monitor!

1. Check for neurological deterioration

Initially you have to evaluate if the ICP increase was associated with catastrophic brain herniation (new abnormal pupillary dilatation unreactive to light) or a consistent decline of GCS (if the patient is not sedated). If this is the case (though it is relatively infrequent) it is important to apply all efforts to reduce ICP immediately and plan a CT scan if an increase of mass effect is suspected.

2. Test if the increase of ICP is reliable

Excluding the above scenario, you should check the following points before any conclusion with a therapeutic implication is drawn.

- Can you recognise the pulsatile components of the ICP waveform?
- If you compress the external jugular veins (gently and briefly!), can you observe a rise in ICP?
- In the case of ventricular catheters: if you drain CSF, does the ICP decrease?
- Does the ICP value correspond with the clinical situation?

3. Evaluate the level reached by ICP elevation and its duration

ICP level can rapidly rise to very high levels. In such cases, CPP may decrease dramatically and the brain may become hypoperfused. However most of these ICP waves are of short duration and due to hyperaemia waves in patients with abnormal, but not severely reduced, craniocerebral compliance. In such cases analyse rapidly the causes of easily avoidable ICP increases - see below.

4. Short lasting increase of ICP, hyperaemia waves

Causes of increased jugular venous pressure
- Impaired drainage of blood at the neck: check the inclination of the head and that dressings do not compress the jugular veins.
- Increased intrathoracic pressure: avoid patient-ventilator dyssynchrony, exclude pneumothorax, haemothorax, pleural effusion. Decrease PEEP if oxygenation permits.
- Increased intra-abdominal pressure (IAP); measure IAP (bladder pressure) and apply corrective measures if possible when it is elevated.

Causes of increased arterial pressure and/or cerebral vasodilation
- Check ETCO₂, as increased CO₂ may lead to cerebral vasodilatation. Check causes of pain and verify if the infusion pumps with analgesics are appropriately functioning. Use boluses of analgesic to test patient response.
- Verify any sudden increase or reduction of mean arterial pressure. An increase of MAP may be due to pain, ventilator-patient dyssynchrony, fever and shivering. A decline of MAP can be due to a malfunction of catecholamine syringe pumps or at pump change. Verify if the patient is shivering or if the temperature is rising. Treatment takes time and negative reactions to physical cooling must be avoided as well as any hypotension due to bolus doses of antipyretic. In recent years the introduction of low doses continuous infusion of diclofenac has consistently improved the management of fever with very few collateral effects.
- Verify if pupils are reactive and the patient’s motor responses. Suspect crisis if posturing or seizures occur. Check EEG and deepen sedation.
- Check if a sudden reduction of SpO₂ occurred.

5. Rapid increase of ICP, high values, plateau waves, Lundberg A waves

Differing from, yet similar to, hyperaemia waves, small changes in intracranial volume, can provoke prolonged elevation of ICP once intracranial compliance is markedly reduced. This type of ICP elevation has been described by Lundberg as type A and has a typical morphology charaterised by a rapid rise of ICP until reaching a plateau (namely plateau waves) which can last several minutes but usually ends rapidly with a return of ICP to previous values.

The physiology of plateau waves is complex and their impact of outcome uncertain.

In the plateau phase a reduced but preserved flow is still present due to vasodilation. This may explain why their impact as secondary insults is uncertain; however if untreated, brain herniation can occur during the plateau if the elevated ICP is long lasting.
All therapies directed at improving cerebral compliance may be effective in abolishing plateau waves and among them secondary decompression seems the more effective. A very prompt approach can be tried in the seconds in which the ICP rapidly rises with therapies able to induce an acute vasosconstriction, directly (hyperventilation, indomethacin), or indirectly through metabolic coupling (barbiturate, propofol) or through autoregulation (boluses of indirect catecholamine, mannitol, hypertonic saline).


6. Progressive increase of ICP due to systemic, potentially reversible causes

Frequently ICP increases progressively over hours. Besides anatomical reasons, which are discussed below, a persistent state of hyperthermia, an absolute or relative hyponatraemia or status epilepticus may be associated with a prolonged increase in ICP.


7. Progressive increase of ICP due to intracranial causes - enlargement of perilesional oedema

In patients with traumatic contusions, after the first few days, it is common to observe an increase of perilesional vasogenic oedema; the evolution of core haematoma is less probable. Management is guided initially towards preserving brain tissue by controlling ICP and increasing medical therapy - especially if the lesion is located in cortical areas associated with clinically important neurological functions (eloquent areas). If the ICP cannot be controlled, external decompression may be considered, with or without de-bulking of the haemorrhagic core of the contusion if the area is less functionally relevant.

8. Progressive increase of ICP due to intracranial causes - worsening of diffuse swelling

An increase of cytotoxic oedema in patients with marked initial secondary insult, hypotension, cardiac arrest and worsened diffuse swelling may occur gradually over days. A further cause could be Post-Traumatic Cerebral Infarction, which may present after a time delay.


The place of advanced monitoring techniques in head injury

The strategy of an escalating response to rising ICP and falling CPP, described in the foregoing section, assumes that all patients with severe brain injury are similar and should be managed in the same way, but growing evidence suggests a range of patterns of disordered pathophysiology. Different patient subgroups may require different treatment strategies. Advanced neuromonitoring techniques which look at cerebral oxygenation, cerebral perfusion, and metabolism can give us deeper insight into the various pathophysiological mechanisms involved and help to target rational therapy.

This concept was clearly expressed by John Douglas Miller some years ago.


Global measurement

ICP: Measurement of ICP remains the cornerstone of monitoring in the patient with traumatic brain injury. When linked to data on MAP it provides a measurement of CPP, which is a rough estimate of CBF. Global CBF seems to be dependent on CPP predominantly in the first hours post injury. Conversely, in patients who survive and stabilise in ICU, CPP and global CBF are not correlated, probably as the result of preserved autoregulation and maintenance of CPP within autoregulatory range.

SjO₂: Newer adjuncts to ICP monitoring include jugular venous saturation monitoring (SjO₂) which allows the clinician to monitor cerebral oxygen extraction for any given CPP. While continuous SjO₂ monitoring has not been shown to be of benefit, episodic SjO₂ can detect, albeit infrequently, clinically occult episodes of cerebral ischaemia
and increased oxygen extraction, thus allowing, for example, the optimisation of hyperventilation in the treatment of intracranial hypertension. Infrequently, a decline of SjO2 is associated with very critical CPP and in such case is an ominous sign.

Critical level of anaemia may be detected in association with low values of SjO2.

The main reason that SjO2 is not more frequently used is that it seems to be more a confirmative monitoring than an informative one. The second reason is that SjO2, after the early few days, reflects more the decline of cerebral metabolic rate for oxygen (CMRO2) than the appropriateness of CBF. High stable SjO2 values are indicative of post-traumatic CMRO2 depression or the result of therapies which induce a decline of neuronal activities (deep sedation, propofol, barbiturate, hypothermia).


Brain tissue oxygen levels (ptiO2) and thermal diffusion regional cerebral blood flow (TD-rCBF) measurements:
A valuable alternative to SjO2 is the measurement of ptiO2 and TD-rCBF. These monitoring devices are extremely focal but once placed in apparently normal brain, they seem to be sensitive to the same variables that affect SjO2 - a recognised global monitoring modality.

While TD-rCBF is pure, looking only at rCBF, ptiO2 is affected by multiple factors, so is a proxy of rCBF, arterial oxygenation and regional cerebral metabolic rate for oxygen (rCMRO2).


Focal measurement

ptiO2 and TD-rCBF: The apparent limitation of ptiO2 and rCBF (their regionality) is conversely the reason that they can be placed in perilesional oedematous regions.
Here they can easily document how the penumbra is sensitive to changes of physiological variables. Unfortunately in clinical practice the intention to place the sensor in the oedematous area has success in only half of attempted cases.


Microdialysis: Microdialysis is a technically feasible method of monitoring biochemical disturbances in the brain if placed in the penumbra. Ideally two catheters are placed with one of them placed in apparently normal tissue for comparison. Although it doesn't measure the rate of molecular production or consumption, it opens a ‘window at the bedside’ on brain metabolism. Conditions of metabolic crisis, with lactate production in excess of pyruvate, abnormal cell firing and cytotoxic damage, associated with glutamate elevation, excessive consumption or defect in the metabolism of glucose can be detected at the bedside only by this method.


Further monitoring

TCD: Transcranial Doppler may be better used in the non-invasive estimation of autoregulation. However it is operator dependent and relatively unstable. It is routinely applied in only a few centres.

Continuous evoked potential monitoring remains a promising monitoring modality. It has the unique capability to detect reduction in neuronal activity in association with declines in CPP. New equipment improves its application at the bedside. One major limitation is the difficulty of forcing wave morphology into analyzable data. Today most studies analyse the relationship with outcome rather than describing patterns guiding therapy and management.

Continuous EEG monitoring may detect non-convulsive status epilepticus (NCSE).
No study has yet shown that so-called ‘targeted’ therapy is superior to conventional therapy, and the complexity and rapid pace of scientific and technological advance makes this a field which is best done in specialised centres. However, a review of survival of patients treated with ‘aggressive’ management suggests that, in patients managed in centres which apply ICP and advanced monitoring, mortality is lower.


**THINK** Is a poor prognosis by itself a justification for the use of complex monitoring and treatment? If not, what other factors should determine the use of complex monitoring techniques? May there be a price to pay and how frequently is the price worth paying? As you relate to care of relevant patients, reflect on these issues.

**Neuro imaging developments:** More recently neuroimaging modalities have been introduced in the diagnosis of specific physiopathological conditions and their monitoring. These include

- **CT perfusion.** A modified angio-CT in which a non-diffusible tracer is able to measure at high resolution regional CBF (rCBF) (quantitative) and cerebral blood volume. The absolute values of CBF should be evaluated with caution.

- **XeCT.** Xenon CT is a method to measure quantitatively regional CBF using Xenon as a contrast diffusible agent. Data results are highly reliable with a high spatial accuracy. It allows the combination of evaluation of rCBF with CT imaging. More recently the technique has been applied at bedside with the introduction of portable CT scanner.


- **PET.** A quantitative measurement of regional cerebral metabolic rate for glucose (rCMRG). In specialised centres with cyclotron, it can measure regional cerebral metabolic rate for oxygen (rCMRO₂), regional CBV, regional oxygen extraction (rOEF), and regional distribution of benzodiazepine receptors (a marker of tissue viability). It is however a technique currently reserved for research centres. PET-CT can produce similar imaging coupled with a traditional CT.

- **MRI diffusion.** Quantifies with a ratio the typology of regional brain oedema, cytotoxic or vasogenic.

- **MRI perfusion.** Produces a non-quantitative mapping of regional perfusion, and allows a description of penumbra, the area in which reduced perfusion is mismatched from the cytotoxic oedematous area.

- **MRI spectrometry.** Produce a semiquantitative mapping of cerebral metabolites suggesting areas of ischaemia and excitotoxicity.


Another way to approach neuromonitoring is according to the aim of care. If your centre is predominantly interested in preserving global function of the brain, you can consider the measurement of ICP alone. SjO₂ may be easily added to such monitoring.

If more accuracy and a desire to incorporate measurement of perfusion is desired, then tissue O₂ partial pressure (ptiO₂) or thermal diffusion cerebral blood flow (TD-CBF) and microdialysis placed, in apparently normal tissue at CT, may be added.

Most disability is due to focal lesion involving eloquent regions (frontal and temporal lobes). Such lesions are frequently surrounded by oedema which can be functionally categorised as penumbra. With such a perspective, ptiO₂ or TD-rCBF, and microdialysis might be placed tentatively in perilesional oedematous tissue. Regional CBF may be measured in a tomographic way by means of XeCT or CT perfusion and the type of oedema by means of diffusion MRI.

The place in clinical practice of standard and advanced monitoring techniques in TBI is further developed in the next Task, which deals with various treatment strategies.
The intensive care management of severe head injury consists of the provision of high quality general care and various strategies aimed at preventing or reducing secondary brain damage while the underlying pathology is resolving. Control of the factors involved in the ‘vicious cycle’ previously described is of specific relevance. Possible manipulations of the cellular and molecular disturbances previously outlined are also under scrutiny.

We shall deal firstly with the specific treatment strategies and conclude with comments on the general care of neurotrauma patients.

**Therapeutic intervention level**

Most ICUs apply protocols to the care of increasing levels of intracranial hypertension. Correspondingly, a scale of therapeutic intervention has been developed. While these strategies are accepted worldwide, each centre has its own scale, in which barbiturate, deep hypothermia, hyperventilation and secondary external decompression are considered as last tier therapies. It is helpful to audit the number and heterogeneity of ICP therapies regularly used within your ICU, both to monitor your own management and to improve communication among different specialists within the department and hospital.


**Specific management**

This can helpfully be considered under four headings: surgery for haematoma; swelling of the brain; intracranial infections and seizures.

**Surgery**

Often immediate surgery is the primary treatment e.g. for large intracranial haematomas and contused brain lobes, and for depressed fractures and other penetrating injuries.

**THINK** In your daily practice, think about how decisions are reached about whether and when to operate; become involved in assessing and preparing patients before surgery and in their reassessment and post-operative care; and reflect on the issues surgery raises about effective communication among professionals and with families (see ‘Patient Challenges’ and Task 1).
**Swelling of the brain**

According to North American guidelines and the recommendations of the European Brain Injury Consortium, the critical values for specific intervention are: ICP >20 mmHg, CPP <70 mmHg.

The conventional approach to the treatment of elevated ICP involves the sequential application of various treatment modalities, starting with basic therapies and the least invasive of interventions and progressing, as indicated, through the sequence of interventions outlined below including the monitoring of the ICP and CPP.

**Basic treatment**

Appropriate sedation and analgesia is critical since stress and pain may cause a rise in ICP. Mechanical ventilation is also mandatory, at least during the initial stages. The patient’s head should be elevated at 15-30 degrees. Care should be taken to ensure that wound dressings and collars do not compress the jugular veins.

**THINK** Does analgesia and sedation in head-injured patients require a different approach from other trauma patients?  
See PACT ‘Sedation and analgesia’ module for information.

**Standard interventions**

**Sedation**

In patients with a stable, elevated ICP, sedation and analgesia is central to management. The endpoint of sedation should be to lower baseline ICP by decreasing the reaction of the brain to stimulation. In patients with reduced cranial compliance, the vasodilatation induced by stimulation may increase the cerebral blood volume and may lead to a persistent ICP elevation.

Conversely in patients with less severe injuries, and with enough preserved intracranial compliance, the level of sedation required may be lighter. Indeed in such cases the increases of ICP are self-resolving. Adequate levels of sedation may reduce the incidence of early seizures and non-convulsive status epilepticus. Finally sedation and analgesia can blunt autonomic responses to TBI.

In the few days after injury, in patients in whom there is chance of a prompt recovery, short-term sedative and analgesic regimens are suggested (propofol and remifentanil, or fentanyl). Conversely in patients with severe CT findings, initial high ICP and at high risk of secondary insults, neurological examination seems not to be of use and may be potentially dangerous. In such patients midazolam, and in more severe cases diazepam, can be used as sedative, with fentanyl as an analgesic. The half-life of each of them may be prolonged, especially in severely injured patients, when the duration of ICP instability is prolonged thus requiring these drugs to be infused for a number of days.

Once the ICP levels begin to be successfully controlled, daily sedation interruption may be useful to reduce the total dose.
CSF drainage

If ICP rises above 20 mmHg, draining CSF via a ventricular catheter is a safe and generally reliable method of returning the value to more acceptable levels.

Mannitol

Administration of mannitol is an effective method to decrease ICP. Given intravenously it stays in the vascular compartment and creates a temporary osmotic gradient - osmolarity rises. When serum osmolarity exceeds 320 mOsmol, administration of mannitol is stopped.

The normal single dose is 0.3 g/kg intravenously, given over a period of 15 to 20 minutes.

Q. Sometimes treatment with mannitol is intensified in refractory cases (see the second patient in the ‘Patient Challenges’). What is the main risk of this course of action to neurological and other pathology?

A. Rebound increase in ICP due to worsening of vasogenic oedema (because of the damaged blood brain barrier) and renal dysfunction.

Second-line therapies

It is expected that patients who require these last tiers of therapies cumulatively are not more than 20-25% of the patients in whom ICP is measured. If higher rates pertain in a unit’s audit of its practice, especially the rate of secondary decompressive craniotomy, then consideration is indicated to review whether this may represent failure of first level therapies and/or underuse of early surgery for mass lesions.
Hyperventilation

Hypocapnia causes cerebral vasoconstriction thereby lowering cerebral blood flow and consequently ICP. In severe hypocapnia, however, the degree of cerebral vasoconstriction may be sufficiently extreme as to result in hypoperfusion. However as the reduction in CBF is compensated by an increase of oxygen extraction, a net decline in oxygen consumption has not been yet demonstrated. Hyperventilation to a level of PaCO$_2$ between 30 and 35 mmHg is usually safe.

The monitoring of SjO$_2$ during hyperventilation might disclose low values (e.g. 60%), which should induce clinicians to reduce ventilation. Hyperventilation should be used once a transtentorial herniation is discovered. This measure could be specifically effective in patients with an evolving haematoma, while preparing for transfer to theatre.


Q. If you felt that a greater degree of hyperventilation was desirable, how might this be achieved with less risk of ischaemia?

A. Additional and more sophisticated monitoring of cerebral oxygenation might assist in this aim using techniques such as jugular bulb oximetry and brain tissue PO$_2$; see Task 3.

Barbiturates

Apart from their sedative effect, high doses of barbiturates, such as thiopentone and methohexital, reduce cerebral metabolism and lower ICP. As barbiturates have numerous side effects (e.g. hypotension, especially in volume-depleted patients, hypothermia, immunodepression and infection) they are only indicated as ‘second-line therapy’ when other treatments prove ineffective. Continuous EEG-recording is suggested to monitor their effects. The dose of barbiturate should be targeted to control ICP and not to reduce EEG activity. Conversely, if ICP is unresponsive to barbiturates, further doses, once burst suppression has been already achieved, are inappropriate.

Although not proven, a way to minimise the dose infused is to add barbiturates to previous benzodiazepine-based sedation. The synergistic effect of benzodiazepine helps to reduce the side effects of barbiturate when infused alone.

The long half-life of barbiturates should be considered when infused even for a couple of days. Hypothermia, may lead to a further increase of barbiturate half-life due to depression of the hepatic metabolic rate.
Q. What particular type of infection is associated with the use of barbiturates in head-injured patients?
A. Respiratory infection.

Q. Give two reasons why this might occur with barbiturates.
A. Reduced respiratory ciliary activity and immune function.

Q. What other class of drug which has been used in the management of severe head injury has been reported to have a similar side effect?
A. Steroids.

Q. Does this class of drug have a recognised role in the current management of TBI?
A. Current orthodoxy is that steroids have no part to play in TBI - see reference below. Many units, however, still use them and further trials are underway to explore possible benefit in some subgroups, e.g. patients with intraparenchymal lesions and vasogenic oedema.

Decompressive craniectomy

The present section discusses cranial decompression which is done to control elevated ICP. This is called secondary decompression. Primary decompression is applied after primary surgery for mass effect. Sometimes this is not a choice of the surgeon but more a necessity.

Secondary decompressive craniectomy ‘to give the swelling brain more space’ is an effective method to lower ICP which has been shown to produce a worthwhile outcome in some cases. It serves as ‘a second-line therapy’ and seems to be indicated especially when medical therapies fail to control ICP or the risk of associated complications become excessively high.
In patients with focal intraparenchymal lesions, which have not been primarily evacuated, secondary decompression may be undertaken in association with contusion evacuation. For patients with diffuse injury, the efficacy on long-term outcome of decompressive craniectomy remains controversial. The reason may be that the TBI-associated axonal damage is in part independent from intracranial hypertension and/or that the complications associated with decompression may outweigh any immediate benefit on the intracranial pressure.

Secondary decompression, as with all second tier therapies, should be applied to most severe cases with persistently high ICP values, aiming for an acceptable disability or a good recovery. The extent of decompression should be large enough to avoid cortical ischaemia and venous engorgement in relation to the bone border.

One recent international multicentre study of early decompressive craniectomy vs conventional therapy (see Cooper et al. below) of intracranial hypertension showed disappointing six month outcomes in those patients who underwent decompression, though the methods of the study have been criticised.


Hypothermia

Moderate hypothermia reduces cerebral metabolism and cerebral blood volume and is effective in reducing ICP, but like barbiturate infusion, prophylactic use has been associated with more adverse effects than benefits.

Management of hypothermia requires a knowledge of the three phases of the therapy: the induction phase, where the aim is to get down to the target temperature of 34 °C as quickly as possible; the maintenance phase, where the aim is to tightly control core temperature, with minor or no fluctuations (maximum, 0.2-0.5 °C); the rewarming phase, with slow and controlled warming. Target rates which have been used for the rewarming phase are 0.2-0.5 °C/hr (utilised in post-cardiac arrest patients) and 0.1-0.2 °C/hr for others (see below).

Several methods are available to rapidly cool the patient but the most simple can be achieved by combining a rapid infusion of 1500-3000 mL of cold (4 °C) fluids with surface cooling device. Intensive care should include careful monitoring of fluid balance, to compensate for cold diuresis, with prevention of hypovolaemia or hypotension, tight control of glucose and electrolyte concentrations, prevention of infective complications and the loss of skin integrity, adjustment of doses of various drugs (including sedatives and opiates) and the prevention of shivering.
During the maintenance phase, try to tightly control core temperature, with only very minor fluctuations tolerated (maximum 0.2-0.5 °C). Clinical efforts should be directed to focus on prevention of long-term side effects, such as pneumonia, wound infections and pressure ulceration of the skin.

Slow and controlled rewarming (slower than 0.2-0.5 °C/hr) is suggested. Numerous animal studies have shown that rapid rewarming can adversely affect outcome and that slow rewarming preserves the benefits of cooling. In clinical studies, rapid rewarming have been associated with an increased the risk of hyperkalaemia and there is the risk of transient regional or general imbalances between cerebral blood flow and oxygen consumption—i.e., increased oxygen consumption relative to perfusion.


**THINK Can you think why this form of treatment might not work?**

Other pharmacological agents

Numerous pharmacological agents are being developed and tested specifically for the treatment of patients with head injury, e.g. blockade of glutamate receptors, scavenging of free radicals, and blockade of calcium channels.

Several novel pharmaceutical compounds have yielded impressive results in tissue culture or animal experimentation. Most clinical trials have so far been disappointing, however. Several reasons can explain failures. More recent research has moved toward the test of drugs, hormones, and other endogenous substances whose mechanism of potentially protective action may entail multiple neuroprotective mechanisms.

A second parallel line of research involves the combination of agents with complementary targets and effects rather than focusing on a single target with multiple agents.

**Intracranial infections**

Intracranial infections such as meningitis, brain abscess, and subdural empyema are complications of injuries which penetrate the dura mater and create a portal of entry for pathogenic bacteria such as depressed skull vault fractures, skull base fractures, and the injuries caused by stabbing, shooting or by a blast. It is most common when primary management of the wound has been deficient either because of limited healthcare resources or because the mechanism of injury has not been recognised.

Most intracranial infections in TBI are avoidable. Septicaemia due to intracranial infections following TBI is extremely rare.

Q. Are prophylactic antibiotics of proven value in skull fracture? Explain your answer

A. No rigorous clinical trial has shown benefit after skull base fracture but in practice, many neurosurgeons still use penicillin to prevent infection by endogenous pneumococci.

Q. Outline possible collateral effects and considerations of prophylaxis in these circumstances - in relation to the patient and to the environment.

A. Any approach with short-term prophylaxis should consider the impact on microbial ecology of the ICU and re-evaluate the cost-effectiveness of this approach. A further collateral effect of prophylaxis is to reduce the chance to detect early causes of patient infection in other site as the clinical picture will be obscured and because the induced bacteriostasis limits the growth of micro-organisms in laboratory culture.

The treatment of established intracranial infection may also involve the drainage of pus and the prolonged use of antibiotics as guided by cultures of pus, blood, CSF and clinical response.

Q. Which supplementary investigation would you use to monitor the efficacy of treatment and the need for further surgery?

A. Serial CT scanning would be the main method.

Ask your radiological colleague to show you some of the past series and relate these to the clinical findings.

Seizures

Seizures reflect disordered electrical discharges in the damaged brain. They are common after TBI (5-15% in various series), especially after operative haematoma evacuation, penetrating injury (including depressed skull fracture with dural penetration) and when there has been focal neurological signs or intracranial sepsis. Seizures can be classified as early or late (before or after seven days from the injury), and as generalised (grand mal) or focal. Repeated or prolonged seizures constitute status epilepticus. Late seizures imply a permanent risk of epilepsy and the need to consider long-term prophylaxis.

Prevention of seizures is the goal. Some drugs commonly used in the ICU should be avoided or used with caution.
Prepare a list of the drugs in common use in your ICU and determine which may provoke seizures. You may be surprised at how many such drugs there are.

**NOTE** Usually in the clinical setting you cannot tell whether a certain drug is responsible for a seizure or whether it is ‘just the TBI by itself’. If a patient develops seizures following TBI - especially if they are pharmacoresistant - it is useful to remain aware of the possible predisposing influence of current medications and to consider discontinuing them where suspicion arises.

Anticonvulsants such as phenytoin and carbamazepine reduce the liability to early seizures (but not to late seizures), especially in high risk subgroups. Preventing intracranial infections is important.

In the conscious patient a single brief seizure may only require supportive treatment, but in the comatose patient every seizure can threaten life or function and should be treated. Seizures enormously increase the cerebral metabolic demand for oxygen, and raise ICP and lower CPP to critical levels by increasing CBF and by increasing CO₂ retention in spontaneously breathing patients.

| A seizure in the comatose patient is a life-threatening emergency |


Q. A head-injured patient, several hours after evacuation of a large extradural haematoma, has his sedation reduced to allow neurological assessment prior to extubation. He is observed to start having a generalised seizure. Presuming ABCs are addressed, outline your pharmacological approach.

A. An intravenous bolus dose (10 mg or more) of diazepam will probably abort the seizure. An intravenous infusion of phenytoin should then be commenced.

Q. Outline your ongoing management of the phenytoin therapy?

A. Phenytoin should be given no faster than 25 mg/min (as it is cardiotoxic) until the loading dose (usually 1000 mg in an adult) is given. Then start a maintenance adult dose of 250-300 mg per day as guided by clinical response and the plasma level of the drug.
Q. If the patient’s convulsions do not stop within 30 minutes, outline further pharmacological steps.

A. Consider thiopentone (thiopental) (a 200 mg bolus followed by an infusion of 5-15 mg/min). Clonazepam can be used as an alternative to phenytoin, especially if the seizures are mainly focal.
It is impossible to care for the brain without caring for the whole organism. Optimum support for the injured brain sometimes seems injurious to the body as a whole (e.g. hypothermia, barbiturate coma). High quality intensive care is needed to complement complex interventions and to prevent avoidable morbidity. Many PACT modules deal with aspects of the overall critical care of the patient and extensive links to these modules are used below. However, certain physiological systems are exposed specifically to TBI-related conditions and complications and these are summarised below.

**Central nervous system**

*Avoid oversedation*

The negative effect of oversedation has been elucidated in patients with extracranial disease and includes cardiovascular depression with increased need for inotropes and slow weaning from ventilation. However, sedation in severe TBI is a standard treatment to reduce sudden increases in ICP due to noxious or painful stimulation. The depth or duration of sedation may thus be greater than initially planned, often due to accumulation of sedatives and analgesics.

In those patients in whom ICP levels have been controlled for some days and CT findings are stable or satisfactory, sedation should be stopped or reduced substantially. If required, sedation can be restarted at a level which maintains calmness in the patient and stable ICP. Finding the balance may be difficult and take many hours. Daily assessment of the need for sedation and stopping sedation unless indicated is part of standard ventilator care.

See PACT modules on Sedation and analgesia and Mechanical ventilation.


**Agitation after withdrawal of sedation**

Prolonged sedation and analgesia may be associated with tolerance and with features of withdrawal when drugs are stopped. These signs and symptoms include central nervous system activation, gastrointestinal disturbances and sympathetic hyperactivity.
The central nervous system manifestations include hyperactive deep tendon reflexes, clonus, frequent yawning, sneezing, and hypertonicity. A reversible instability and elevation of ICP can be observed. Withdrawal syndrome is more severe, appears earlier in younger patients, and is a particular problem in paediatric ICU.

Symptoms due to withdrawal can be confused and overlap with those due to paroxysmal sympathetic hyperactivity (also termed paroxysmal autonomic instability syndrome with dystonia, PAID). This is a syndrome complicating severe TBI and shares similar clinical symptoms with the withdrawal syndrome. Elevated sympathetic nervous system activity occurs and is characterised by increased heart rate, respiratory rate, and blood pressure; also redeployment of blood to skeletal muscle and the central nervous system, diaphoresis, and hyperthermia. In the case of TBI, elevated sympathetic activity may almost be considered normal and is blunted in the acute phase by sedation and analgesia applied to control elevated ICP levels. However, during weaning from sedation, this paroxysmal sympathetic hyperactivity may appear and last for weeks thus representing a potentially treatable cause of increased secondary morbidity.

**Management and prevention**

Oversedation increases the risk of tolerance and consequently of withdrawal syndrome. Unfortunately, more severely injured patients may have higher ICP levels which need more sedation; the same patients are more at risk of paroxysmal sympathetic hyperactivity.

Withdrawal may be less problematic if use of short-acting benzodiazepines (e.g. midazolam) is avoided. Alpha-2 agonists (clonidine, dexmedetomidine) can be used to reduce withdrawal from opioids.

Concerning paroxysmal sympathetic hyperactivity, first-line oral medications include methadone, gabapentin, benzodiazepines and centrally acting alpha-2 agonists. Bromocriptine is a second-line medication often used in combination with other medications.

See the PACT module on Sedation and analgesia.


Respiratory system

Patient positioning

Appropriate positioning may reduce the frequency of pulmonary complications and silent (micro-) aspiration. The patient should be inclined head-up at 30°, and should be nursed if possible alternatively on their back, right and left sides. Nursing head-up is also part of the ventilator care bundle (see above under sedation). While such positioning is important, its effect of increasing ICP due to altered venous drainage needs to be minimised.

Kollef M. SMART approaches for reducing nosocomial infections in the ICU. Chest 2008; 134: 447-456. PMID 18682466

Early and ultra-early tracheostomy

Early tracheostomy (4-6 days post injury), or ultra-early tracheostomy (1-3 days post injury) have been claimed as advantageous in reducing the use and duration of sedation, duration of mechanical ventilation and in preventing pulmonary and laryngeal complications. It may also accelerate safe discharge from ICU to a lower level of care and so enhance early physical rehabilitation. However, this is a controversial area and most studies have failed to show any effect on outcome. It would seem appropriate to adopt an early tracheostomy approach in specific patients in whom a tracheostomy seems inevitable.

See the PACT module on Airway management.


Gastrointestinal system - nutrition and stress ulceration

Feeding is important to reduce catabolic effects and maintain immunological competence. There is evidence that the catabolic response in head-injured patients may be more profound and/or prolonged than in multiple trauma patients without head injury. Nutrition via the enteral route is more physiological, less expensive and is associated with fewer complications than parenteral nutrition. It may also be associated with accelerated normalisation of nutritional status. There is a minor increased risk of reflux and potential for aspiration. However, gastric pH is higher (more neutralised) by enteral nutrition and the airway has a significant degree of
protection against aspiration due to the cuffed tube. Enteral nutrition should be started shortly after admission with the intention of reaching full nutritional intake by day 3 in ICU.

Some years ago the incidence of stress ulcers in patients with head injuries was thought to be approximately 10%, twice the rate in other patient groups. While the incidence of stress ulcers has decreased generally, severe TBI is an accepted risk factor for stress ulceration. Early enteral feeding and adequate use of analgesics and sedation are perhaps the most effective prophylaxis. H2 antagonists, which reduce acid production, is an established therapy and proton pump inhibitors are also used. However, with such agents there may be an increased risk of healthcare associated infections such as pneumonia and clostridium difficile colitis.

See the PACT module on Nutrition.

Curtis L. Early, high quality enteral nutrition significantly improves outcome in head trauma patients. J Neurotrauma 2011; 28(10): 2197-2198. PMID 21846247

**Metabolic function**

**Disturbances of sodium balance**

Disturbances of serum electrolytes are observed in approximately 60% of all patients with severe TBI. Changes of serum sodium concentration are the most important to manage appropriately. Rapid recognition and management of such disturbances is dependent on serial clinical examination and careful monitoring: hourly urine output, blood pressure and central venous pressure measurements as well as frequent measurement of serum electrolytes and osmolarity and urine specific gravity and osmolality.

**Hypernatraemia**

The most common causes of hypernatraemia (serum sodium >150 mmol/L) in patients with TBI are: central or neurogenic diabetes insipidus (DI) and osmotic diuresis (mannitol infusion). Central or neurogenic DI occurs when the brain does not secrete adequate levels of antidiuretic hormone. The diagnosis is probable once these signs are present:

- High urine output (>3 mL/kg BW/h)
- Low urine specific gravity (1001-1005)
- Low urinary osmolality <150 mOsmol/kg and a
- Urinary sodium below 50 mmol/L.

Increases of serum sodium completes the diabetes insipidus picture. Treatment include parenteral desmopressin acetate or argipressin administered as soon as the diagnosis has been confirmed. Afterwards any depletion of fluid and potassium should be corrected. Administration of additional water via the enteral route may be necessary to replace the calculated free water deficit. In a patient with hypernatraemia, the first priority is to correct the cause and the correction of the
serum sodium per se should be done carefully. The transmembrane water transfer works more on sodium gradients than on absolute values. Chronic sodium derangements are less dangerous than acute ones. In severely affected patients, the main aim should be to avoid a further sodium increase.

Hyponatraemia

In general medicine, severe hyponatraemia is defined as a serum sodium level <120 mmol/L but in the TBI patient, different thresholds exist.

A sodium level of 140 mmol/L is often considered borderline hyponatraemia, while levels <135 mmol/L define hyponatraemia. Syndromic reasons for hyponatraemia are cerebral salt wasting syndrome (CWS) and syndromes of inappropriate ADH secretion (SIADH-syndrome, Schwartz-Bartter-Syndrome).

However the commonest causes are

- Low sodium intake
- Iatrogenic hyponatraemia (use of hypotonic solution, excessive correction of hypernatraemia)
- Increased sodium losses (e.g. via the gastric tube)

In young patients managed with arterial pressure levels higher than their physiological values with the aim to preserve CPP, a natriuresis can occur due to a compensatory mechanism, called ‘aldosterone escape’. In such patients consider targeting CPP according to their physiology and reduce arterial values to more appropriate values.

A definitive diagnosis of the underlying cause of hyponatraemia is sometimes difficult to obtain. While in both syndromes (SIADH and CWS) urinary sodium is elevated, conversely the volume of urinary output is completely different, low in SIADH and high in CWS. In patients with sodium depletion due to poor intake, the urinary sodium is low. Treatment depends on the cause. In the case of SIADH, this includes fluid restriction and the use of loop diuretics while in cases of CWS treatment involves the administration of fludrocortisone acetate and hypertonic intravenous sodium solutions.

See the PACT module on Electrolytes and Homeostasis.

Fraser JF, Stieg PE. Hyponatremia in the neurosurgical patient: epidemiology, pathophysiology, diagnosis, and management. Neurosurgery 2006; 59(2): 222-229; discussion 222-229. PMID 16883162


Control of blood sugar levels

Extreme hyperglycaemia is dangerous for the brain. However, active control of glycaemia, as a routine, has not been shown to improve patient outcome, probably because of the dramatic consequences of the increased incidence of hypoglycaemia associated with such protocols. Today tight control of glycaemia is not recommended.

In the specific field of neurotrauma there are further concerns that a strict control of hyperglycaemia could further impair glucose diffusion in lesioned area. Lesioned areas e.g. the contusional perilesional area, are affected by critical values of rCBF, and frequently, by an accelerated glucose metabolism (hyperglycolysis) and a reduction of serum glucose could further impair glucose availability. If a centre wishes to obtain the theoretical benefit from a tight glucose control, complex cerebral monitoring such as cerebral microdialysis, should be used. Today in TBI patients, it is suggested to maintain glycaemia in the 150 mg/dL to 200 mg/dL (8.3-11 mmol/L) range.

See the PACT module on Electrolytes and Homeostasis.


Haematology/coagulation - venous thromboembolic disease

Patients with TBI are certainly at high risk of deep venous thrombosis (DVT), given that patients are older and affected by more comorbidities. The prophylaxis of DVT with heparin conflicts with the risk of inducing new intracerebral bleeding or of increasing the volume of the core in lesioned areas. The risks of DVT in trauma patients are well defined, while in patients with TBI it is now clear that CT evolution for haemorrhagic core intraparenchymal lesion is unlikely after the first 3 days post injury. Consequently prophylaxis with low molecular weight heparin should be considered after the first 3 days post injury in combination with the application of mechanical compression devices to the leg.

See the PACT module on Bleeding and thrombosis.
Infections - prevention and management

Patients in coma are at high risk of pneumonia. Indeed, some may have pulmonary aspiration before leaving the scene of trauma or reaching hospital. There is a general agreement that pulmonary infection severity in such patients is minimised by early intubation and ventilation. Pulmonary complications can be expected 3-7 days post injury, usually starting with a moderate increase of temperature, a deterioration in the purulence and volume of tracheal secretions and a decline of oxygenation. X-ray changes can vary from reduced ventilation at the lung bases, to infrequently, frank pneumonic change. Various strategies of antimicrobial prophylaxis and therapy have been recommended. Wide spectrum prophylaxis is not acceptable due to increasing rate of multiresistant bacteria. It is reasonable to utilise agreed, hospital, VAP protocols to guide diagnosis and initial therapy but specific microbiological/Infectious Diseases advice may be required for refractory infections where the patient may be a carrier of resistant organisms. Fever leading to an increase of ICP should be treated with antipyretics however continuous use of antipyretic drugs can mask infection. Specific treatments may increase the risk of severe pneumonia, including hypothermia, and barbiturate use.

In the presence of a functioning gastrointestinal tract, parenteral nutrition should be avoided since it increases the risk of (IV) catheter related infection/bacteraemia. However, in almost all TBI patients, central lines/catheters are required for infusion of catecholamines and sedation. As soon as possible, often when catecholamines are no longer needed, central IV catheters should be removed, with sedatives being administered either via peripheral lines or by the gastrointestinal route. Such interventions can reduce the duration a central venous line is in place and reduce the risk of catheter associated infections.

Urinary sepsis is also a significant risk due to the need for long-term indwelling urinary catheters in TBI patients. Diagnosis should be made on the blood leukocyte count and systemic signs together with colony count on urine culture and, although diagnostic criteria are imprecise in the presence of indwelling urinary catheterisation, on urine leukocyte count. Appropriate antibiotics should be administered intravenously unless absorption from the GI tract is assured. Use of a urinary catheter care bundle may reduce the risk of urinary sepsis in TBI patients.

See PACT modules on Severe infection, Pyrexia (for diagnosis of catheter related infection; CRI) and Infection prevention and control.


Management of other injuries

TBI patients may have several extracranial priorities. Patients with any type of cord lesion should be stabilised surgically as soon as possible to allow patient mobilisation and reduce pulmonary complications. Patients with cervical spine lesions may have had anterior stabilisation and consequently tracheostomy should be delayed to allow the surgical wound to repair. Patients with pulmonary contusion and severe gas impairment may need an approach which recognises the conflict between strategies to improve gas exchange and those required for ICP control. Abdominal surgery, haemoperitoneum, post-traumatic or post-surgical paralytic ileus may all increase intra-abdominal pressure (IAP) and contribute to ICP elevation.

Patients with pelvis and long bone fractures of lower limbs have a higher risk of DVT. The same fractures may cause pain and limitation of mobilisation. They are best stabilised as soon as possible, in the first days post injury, preferably with external devices, to reduce any further haemodynamic impairment and to allow early mobilisation.
Late complications of traumatic brain injury

Post-traumatic hydrocephalus

Incidence and pathophysiology: Approximately two thirds of all patients suffering from severe brain injury will develop post-traumatic enlargement of the ventricles. True post-traumatic communicating hydrocephalus, may occur in up to 1-5% of patients.

THINK Can you remember the clinical and radiological definitions of severe brain injury? Refer to previous text for details.

In those patients who go on to develop post-TBI hydrocephalus, it occurs in up to 25% within two weeks of injury and in 90% within six weeks.

Q. List the two main risk factors for post-TBI hydrocephalus?

A. Primary intraventricular haemorrhage and post-traumatic meningitis are the two biggest risk factors.

Meningitis causes impaired absorption of CSF in the arachnoid villi as it flows over the convexities. Primary and/or secondary decompression are now also considered a cause of hydrocephalus, probably due to a reduction of vault surface area for CSF reabsorption.

Diagnosis: Post-traumatic hydrocephalus is normally slowly progressive. Its clinical presentation is variable, making diagnosis a challenge especially in severely impaired TBI patients. The main clinical markers are prolonged recovery from coma and delay in rehabilitation. CT scans will reveal progressive ventricular enlargement and compression of the cerebral sulci (see below). Typically there is a plateau in patient recovery from coma or sometimes a decline after initial progress. Additional investigations may include lumbar puncture, CT cisternography and radionuclide absorption tests.

Often however, the differential diagnosis between atrophy and hydrocephalus remains unclear, and it may be uncertain in patients who are more disabled or in a vegetative state whether the treatment of hydrocephalus will improve patient function. In such cases, a CSF withdrawal test is indicated: liquor is continuously drained by means of an external spinal catheter and improvements of neurologic performance can be recorded to guide the decision to place a ventricular shunt.

Treatment consists of CSF shunts, either ventriculo-peritoneal or ventriculo-atrial. Even after decompressive craniectomy, there is a high incidence of hydrocephalus which may persist after cranioplasty or bone-flap replacement. In some of these cases, depending on the evidence from serial clinical and CT follow-up, definitive shunting may be required.
Sunken skin flap syndrome

The ‘sunken skin flap syndrome’ (SSFS) is defined as a secondary neurological deterioration in the presence of a sinking skin flap in patients with large craniectomies. The syndrome occurs several weeks to months after decompressive craniectomy and is frequently related to being in the upright position and to daytime-dependent, fluctuating headaches. Concurrent with the sinking of the skin flap and the underlying brain tissue, a deterioration of neurological function with new symptoms and signs is observed. These include focal signs, disturbances of consciousness, epileptic seizures and neuroendocrine abnormalities.

These patients are at risk of paradoxical herniation if they undergo diagnostic lumbar puncture or therapeutic lumbar CSF drainage. Although several changes in CSF hydrodynamics, cerebral blood flow and brain metabolism have been described as partial aspects of the pathophysiology, full understanding of the underlying condition is still lacking. The incidence of this phenomenon is rare and the associated syndrome is sometimes not appreciated. Cranioplasty leads to clinical improvement in several cases and may avoid secondary deterioration.

Cranioplasty is therefore a therapeutic as well as a cosmetic procedure. The intensivist should be aware of SSFS and of other delayed complications of external decompression when considering whether secondary decompressive craniotomy is indicated as a therapy for unresponsive ICP.


**Post-traumatic meningitis**

**Incidence:** The reported incidence of this complication is 5-15%. In good centres, the incidence of meningitis, which is associated with ICP measurement catheters, is less than 1%.

Q. How does this incidence compare with that of ‘clean’ and ‘clean-contaminated’ cases?

A. <0.5% and <1% respectively.

**Pathophysiology:** Risk factors include open, contaminated wounds, CSF leaks (rhinorrhea or otorrhea) through basal skull fractures, and ventricular catheters for ICP measurement. The causative organisms tend to be those normally colonising the nasopharynx such as *Streptococcus pneumoniae*, *Hemophilus influenzae*, *Neisseria meningitidis*, and Staphylococci species.

**Diagnosis:** In all TBI patients with fever of unknown origin, post-traumatic meningitis must be in the differential diagnosis especially if intracranial air or rhinorrhea/otorrhea has suggested an open skull fracture or if a ventricular drain is in place. Other common clinical and laboratory findings include neck stiffness, leukocytosis and increased C-reactive protein. The diagnosis is confirmed by isolation of the causative organism from CSF cultures. The diagnosis is supported by decreased glucose (<50 mg/dL; 2.8 mmol/L) and elevated white blood cell count (>10,000/mm³) with a high proportion of leukocytosis (>75%), and a high CSF lactate. Additional common CSF findings include elevated protein (>100-500 mg/dL).

**NOTE** Do not necessarily consider a CSF specimen, which is culture positive, as diagnostic of infection if no signs of inflammation are detected in the CSF specimen or in the patient. Consider the possibility that CSF has been contaminated during sampling or that the catheter is colonised.

**Microbiological aetiology:** Is often evaluated by reviewing CSF cultures. Ventricular catheter associated ventriculitis is commonly due to *Pseudomonas aeruginosa*, *Staphylococcus aureus*, *Staphylococcus epidermidis* and sometimes, in epidemic forms, *Acinetobacter baumannii*.

**Prophylaxis:** Antibiotic prophylactic in patients with basilar skull fractures has not been proved to be effective in preventing meningitis.

Persistent post-traumatic cerebrospinal fluid leakage should be treated surgically. In the case of external drainage, routine replacement of the catheter to prevent infection does not seem effective in preventing infection.
**Treatment:** Meningitis has to be treated immediately as the mortality of untreated bacterial meningitis is high.

As soon as CSF has been sent for gram-stain and culture, high-dose antibiotics are commenced on a best guess basis if warranted clinically.

**NOTE** If the diagnosis is suspected, discuss it with the patient’s consultant neurosurgeon urgently. Discuss whether it is safe to perform a lumbar puncture in any comatose patient in view of the risk of brain herniation when increased ICP may be present. In addition to blood culture, try to obtain a specimen of CSF before starting antibiotics.

**Q.** If the gram-stain is positive and suggestive of *Streptococcus pneumoniae*, what antibiotic would be appropriate?

**A.** Intravenous penicillin (20–30 M/day) is usually indicated.

**Q.** If it was suggestive of staphylococcal infection, what might be the appropriate anti-staphylococcal antibiotics?

**A.** Flucloxacillin, oxacillin, vancomycin (if MRSA suspected), and perhaps supplementation with rifampicin. The choice would be made on the basis of local surveillance reports and individual history of antibiotic exposure of the patient.

**Q.** In the case of Gram-negative organisms what might be an appropriate antibiotic?

**A.** A third generation cephalosporin (e.g. high-dose ceftriaxone, cefotaxime which penetrate well into CSF) or a carbapenem antibiotic.

Consider the effect of such antibiotic in the selection of multiresistant organisms and ensure adequate therapy once bacterial aetiology is determined. Treatment will be modified as indicated when culture and sensitivity results become available. In the case of shunt related infections, infected ventricular drains should be removed and replaced since acute hydrocephalus can complicate meningitis. In selected cases, give consideration to intrathecal administration of antibiotics which is feasible and efficacious in *Staphylococcus aureus*, *Staphylococcus epidermidis* (vancomycin and gentamycin) and mutiresistant Acinetobacter infection (consider colistin).

Carotico-cavernous (C-C) fistulae

Pathophysiology: C-C fistulae result from injuries to the internal carotid artery as it passes through the sinus cavernosus. They are more common in blunt trauma than in open injuries, often associated with a basal skull fracture. The tear between the carotid artery and the cavernous sinus causes a high-flow fistula into the sinus resulting in venous hypertension of the sinus and the superior ophthalmic vein, the petrosal sinus and the pterygoid venous plexus. Venous hypertension may cause impairment of cranial nerves II, III, IV, and VI. More serious complications include ipsilateral loss of vision, subarachnoid and intraparenchymal haemorrhage, or ischaemia due to steal of blood from the territory of the major arteries.

Diagnosis: The classical clinical picture includes pulsating exophthalmus and chemosis. In some cases, the sound of the fistula can be heard by a stethoscope pressed onto the temporal region. Transcranial Doppler can also confirm the diagnosis and cerebral angiography usually shows the fistula.

Treatment: The usual treatment for a C-C fistula is closure of the leak by endovascular treatment. If endovascular procedures fail, open surgery is indicated.

NOTE If you suspect a C-C fistula, call your local neurologist/neurosurgeon to perform transcranial Doppler.

Outcome from severe head injury

An integral part of ICU activity should be a registry where the long-term outcome of patients is recorded. This may be organised locally, regionally or nationally and helps to evaluate the clinical value and cost-effectiveness of intensive care medicine. In the specific field of TBI, outcome is more complex as disability rather than survival is a more sensitive outcome.

Intensive care physicians are familiar with the acute care of TBI but ICU treatment of the patient with severe head injury is only the beginning of a lengthy process of recovery which continues with early rehabilitation and ends with the patient returning, as nearly as possible, to his/her former life.

Gaining experience of recovery and rehabilitation after TBI is valuable linking of the ICU phase of care with outcome. This experience may help in decision-making in the ICU and may help to plan rehabilitation needs.

The broad outcomes that can be expected after severe head injury in adults are as follows; one third of patients die, one third recover, one third remain disabled. These data are drawn from ten large series of head-injured patients in industrialised countries.

Q. What factors are most likely to affect outcome?

A.
- the nature and extent of intracranial and extracranial damage
- the depth and duration of post-traumatic coma
- the patient’s age
- general medical health and previous state of function
- the quality of available clinical care.

Prediction of outcome

Much effort has gone into devising ways to predict outcome from TBI as:
- the condition is serious
- it consumes expensive resources
- recovery is prolonged
- relatives understandably wish to be given accurate information about prognosis.

Prediction of outcome is basic to decisions concerning diagnosis, prognosis and therapy. Unfortunately even the most sophisticated prediction models have an individual accuracy of only 80%, and can therefore only supplement and not supplant clinical judgment. Families often find it hard to live with such uncertainty for so long and require much support. You should consider your role in this regard in the early stages of managing a patient with TBI.
The factors of most use in assessing outcome after severe head injury during the early days and weeks are indicated below.

Recovery is most rapid in the early weeks. Unfortunately, this can lead to over-enthusiastic prediction of long-term outcome (especially of cognitive function) by those with limited clinical experience of the later stages of rehabilitation. Over 90% of patients have reached their final category in terms of the relatively crude Glasgow Outcome Scale (see below) at six months, and most international trials on head injury still focus on this measure of outcome. With optimum rehabilitation, further gains can be made over a much longer period, up to two years and sometimes longer in the case of younger adults.

**Measurement of outcome**

Outcome after TBI is measured according the Glasgow Outcome Scale (GOS), originally published in 1975 by Jennett. This scale is in 5 points – dead, vegetative state, severely disabled, moderately disabled, and good recovery. For analysis, it is common to dichotomize outcome as favourable (good recovery and moderate disability) or unfavourable (severe disability, persistent vegetative state). More recently (1998) each of the top three categories, good recovery, moderate recovery, severe disability have been split into a further two levels (upper and lower) to improve discrimination.

The score should be obtained during a personal visit and interview with the patient. However, for practical reasons, a postal and/or a telephonic interview are frequently used. Both scales apply a standardised questionnaire and have been validated.

Interpretation of outcome results for each department should be standardised to patients’ severity. Observed outcomes should be compared with the expected outcomes. Standardisation can be done, using a model based on logistic regression (IMPACT) and comparison of GOS scale values through the sliding dichotomy concept.


PACT module on Clinical outcome.

Audit

You should regularly review the outcome of your patients and compare the treatment results of your ICU with the international standards outlined above. Do not use ‘outcome at discharge’ but try to follow up your patients for six months at least. This may require meeting them and their families to discuss what gains have been made and what problems have emerged during the rehabilitation process. Rehabilitation facilities vary considerably throughout Europe, and you should try to visit those relevant to your institution and understand how they form another part of the clinical chain of care.

Many patients with severe head injury die. It is good practice for those who were involved in the care to hold a mortality conference to review the patient’s injuries and the management in an open and non-judgmental way. This process of audit or quality improvement is becoming increasingly fundamental to clinical practice as it encourages efforts to improve the quality of patient service.

Audit on database review

Doctors and ICUs need to know the outcome of their patients weighted on the basis of illness/injury severity. Using a few variables, usually not more than ten, you can build a database and apply the formula used by established prognostic models (IMPACT). If possible, try to obtain mature data from organisations who build the prognostic model to compare its performance with your data, thus creating a calibration model. In such way you can build an observed/predicted ratio concerning mortality or severe disability. Monitoring your data will allow early recognition of higher than expected rates of death or severe disability.
As there is no effective treatment of primary brain damage, the key issues for the intensive care practitioner, in the early management of patients with traumatic brain injury, are to prevent and/or minimise the risk (and effects) of secondary brain injury. Ascertaining the severity of the primary brain injury at the earliest possible opportunity is important as this will assist you in
  1. detecting secondary brain injury
  2. determining the effects of treatment and
  3. predicting outcome.

Intensive care management of patients with severe TBI comprises high quality general care and various strategies aimed at preventing or reducing secondary brain damage while the underlying pathology is resolving. The primary focus is to maintain, within an acceptable range, the increase of ICP and to maintain adequate cerebral perfusion with oxygenated blood. The management should be individualised on the basis of physiological knowledge.

It is important to maintain prognostic balance in every step of the care to avoid both undertreatment and overtreatment. In ICU, control of the factors involved in maintaining the cerebral perfusion pressure - such as arterial and venous pressures, intracranial pressure and pCO₂ is mandatory. Avoidance of metabolic derangements such as hyperglycaemia and hyponatraemia is also important and meticulous attention to all aspects of Critical Care management is a key factor in improving outcome after head injury.
A 16-year-old boy was hit by a car when crossing the street. You are called to see the patient on admission and find he had a GCS of $E2+M5+V2 = 9$ points before being intubated by the paramedics at the scene of the accident. There are no pupillary abnormalities or focal neurological signs. His vital signs indicate an arterial blood pressure of 110/50 mmHg, a pulse rate of 76 beats/min, and a respiratory rate of 16 breaths/min.

In the emergency room, blood is withdrawn for routine tests, and a peripheral intravenous cannula inserted. On examination a large left parietal scalp wound is identified in the base of which a linear fracture is detected.

Q. What is your assessment of the severity of the head injury in this patient with a GCS of 9 (comprising $E2+M5+V2$)?

A. You conclude that a GCS of 9 indicates a moderately severe head injury.

GCS is an internationally accepted clinical classification used in the assessment of head injury.

See the PACT module on Coma and altered consciousness.

During the time that you are attending to this patient, you note that his neurological and general monitoring is stable and you are arranging to transfer the patient to intensive care.

Q. In diagnostic terms, what would you do next?

A. You should recommend a computed tomography (CT) scan.
The patient’s condition, however, does not remain stable and before CT, which you ordered, can be performed the boy’s condition deteriorates. While accepting that Voice analysis is impaired during intubation, his GCS has nonetheless undoubtedly fallen to 6 (comprising E1+M4+V1) and his left pupil has become dilated. A bolus of mannitol is administered and an immediate CT scan taken. The scan indicates a large extradural haematoma beneath the fracture which is successfully evacuated at emergency operation.

Learning Issues
Pathophysiology of moderate head injury

Appropriate, timely intervention (investigation/treatment)
Immediate medical management of a likely acute rise in intracranial pressure (ICP) - Task 1

Surgical intervention

Q. A trainee colleague from another department who assists in the care of this patient recalls that in his previous institution there were neither CT facilities nor a neurosurgeon. He asks what steps you would have taken in these circumstances if a similar scenario had presented?

A. You indicate that in such acute life-threatening circumstances there are two options available. Either a general surgeon with appropriate training carries out burr-hole drainage of the extradural haematoma and subsequently arranges transfer of the patient to a neurosurgical unit or you give a bolus of mannitol and arrange for immediate, safe transport.

Q. He also wants to know what would have been the priorities if you had decided to transport the patient to another hospital.

A. The main priorities in transporting such a patient are optimisation of cerebral oxygen delivery and minimising the risk of secondary brain damage. This will primarily involve taking temporising, acute measures to reduce ICP (with high-dose mannitol and/or hypertonic saline and/or short-term, pre-evacuation hyperventilation) together with haemodynamic measures to maintain cerebral perfusion pressure (CPP).

You will need to be familiar with the local/national/international guidelines for the management/transportation of head-injury patients.
See the PACT module on Patient transportation.

Learning Issues
Role of high-dose mannitol, hypertonic saline and hyperventilation before emergency surgical evacuation of haematoma - Task 2
As you are in the midst of this discussion the driver of the car involved in the accident is admitted to the emergency room and again you are called. The story is that in attempting to avoid the 16-year-old he apparently veered off the road and his vehicle crashed into a stone wall. At the scene of the accident the paramedics noted his GCS as E1+M4+V1 = 6 points. He showed no pupillary abnormalities and no focal neurological signs. His vital signs were normal. Pre-hospital treatment included sedation, tracheal intubation and placement of a peripheral iv line.

**Learning Issues**

Pathophysiology of severe brain injury

Indications for tracheal intubation

Sedation/analgesia

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While the principles are agreed, on site care practice varies from region to region

On examination, his neurological status appears unchanged. His arterial blood pressure is 140/75 mmHg and pulse rate 76 beats/min. Inspection shows some bruises to the scalp but no further injuries. A standard diagnostic evaluation is performed including plain X-rays of the chest, the head, the whole spine, abdominal ultrasound and routine blood tests. No additional injuries are detected. Thereafter, you assist by inserting central venous and arterial catheters; a nasogastric tube is passed and the bladder catheterised.

The initial CT scan indicates small bilateral frontal and temporal contusions, but no space-occupying intra- or extra-axial mass lesion. There is also partial obliteration of the basal cisterns as a sign of raised intracranial pressure.

**Learning Issues**

Extracranial injuries (in multiple trauma)

CT classification of head injury
Interpretation of CT brain
Intra and extra-axial mass lesions

Prioritisation of injuries for operative intervention

Q. This patient’s GCS was noted to be 6 at the scene of the accident. What is the probable significance (in terms of severity of the injury) of this observation?

A. The observation of a GCS of 6 at the scene of the accident is consistent with severe primary brain damage.

Q. When you first saw this patient, his GCS appeared unchanged from that reported by the paramedics. What factors would cause you to be cautious about over-interpreting this observation?

A. The interpretation of the patient’s GCS after transfer to hospital is complicated by prior sedation and tracheal intubation.

Q. Do any of the investigations confirm the initial assessment re the severity of the injury?

A. Yes, the CT findings are unambiguous and consistent with raised ICP.

Concurrent, pressing extracranial injuries (e.g. tension pneumothorax, cardiac tamponade, intracavity bleeding etc.) were not observed in this case. Some extracranial injuries might prompt urgent investigation/treatment other than CT scan (for head injury).

Q. Would the presence of extracranial injuries, which required urgent corrective intervention, affect the prioritisation/sequencing of investigation and treatment of the head injury?

A. Yes, this would have affected the treatment sequence in that life-saving operative interventions would have been required consistent with the principles of advanced trauma life-support (ATLS) i.e. restoring circulation prior to addressing neurological deficit.

Q. Given the neurological and CT findings in this clinically stable patient, it is agreed that the next priority is to facilitate neuro-critical care of his likely raised ICP by measuring the pressure. What would you organise next?

A. The next priority is to take the patient to the operating room and to insert a parenchymal transducer to measure intracranial pressure via a right frontal burr-hole.

The techniques utilised to measure ICP have intrinsic advantages and disadvantages. This patient was taken to the OR for insertion of an ICP monitoring system in a specialist neuro-surgical/critical care centre.
Q. Is there any situation where this might not be appropriate?
A. ICP monitoring outside the specialist neurosurgical environment is controversial.

A parenchymal transducer is inserted and the initial ICP recorded is 24 mmHg. The patient is sedated (midazolam and fentanyl) and ventilated. During the next hours his ICP remains stable at around 15-18 mmHg. A repeat CT on the next day shows an identical picture to the initial CT. Over the next four days the patient’s ICP starts to rise (25-30 mmHg) prompting more aggressive therapy with mannitol, controlled hyperventilation (PaCO$_2$ 32-35 mmHg/4.3-4.6 kPa), and trishydroxymethylaminomethane (THAM).

Another CT on day 4 indicates further enlargement of the bitemporal contusions and total obliteration of the third ventricle. Treatment with mannitol was intensified.

**Learning Issues**

- Intracranial pressure monitoring
- Indications for mechanical ventilation and hyperventilation
- Conservative management of raised ICP
- Benefits/risks of mannitol

Q. This patient was sedated with midazolam and fentanyl. Does the type of sedative agent used matter in the control of raised ICP? Explain your answer.

A. Yes. Sedative agents vary in their effect on cerebral blood flow.
Q. Why was the arterial carbon dioxide level not allowed to fall below 32 mmHg/4.3kPa?
A. Cerebral vasoconstriction is a potential risk in head-injured patients.


In spite of continuous, careful control of ICP using appropriate sedative agents and avoiding the risk of inducing cerebral hypoxia by excessive hyperventilation, on day 6 the patient’s condition deteriorates. The frequency of plateau waves on his ICP recording increases (to a mean ICP of 35-42 mmHg) and both pupils enlarge slightly from 2 mm to 4 mm, and begin to be unreactive to light. A repeat CT now shows further demarcation of the contusions and partial obliteration of the basal cisterns.

Learning Issues
Sedation/Analgesia

Hypocarbia and the risk of cerebral vasoconstriction
Interpretation of ICP patterns
Indications for surgery in severe brain injury

Q. Conservative measures have now clearly failed to control raised ICP. What would you suggest?
A. Surgery is now an option for consideration.
The patient is taken to the OR for bilateral decompressive craniectomy (see scan). Postoperatively his ICP is 15 mmHg. Medical treatment for raised ICP is gradually withdrawn under ICP control over the next four days and the ICP transducer ultimately removed. After 23 days the patient is transferred to a rehabilitation unit. At this time he opens his eyes spontaneously and obeys simple commands. There is no focal neurological deficit. Re-implantation of the bone flaps (removed at decompressive craniectomy) is performed six weeks later.

**Learning Issues**

Weaning from mechanical ventilation

Long-term rehabilitation of the brain-injured patient

Multiprofessional collaboration

Both patients improve after their head injury. In the case of the first patient, recovery is prompt but the second patient takes a full eighteen months before he is well enough to resume his university course in computer sciences. A number of issues arise relating to the post-injury outcome.

**Learning Issues**

Communication with patient/relatives/colleagues

Outcome from TBI

Q. Given the low GCS and evidence of widespread bilateral structural damage in the second patient, is such a good outcome to be expected?

A. Not often. Cognitive problems would be a common sequel following this severity of TBI.
Q. Do you see yourself as influencing the longer-term and continuing management of severely head-injured patients?

A. Yes, Critical Care management is a key step, particularly in the early phase, of the continuum of care of the TBI patient. The primary obvious influence on long-term care will be whether that patient survives ICU or not.

However, the influence of Critical Care will be much broader than simply the issue of survival. The On Reflection piece below addresses some of the totality of this picture including some of the effects of Critical Care on the subsequent success of a rehabilitation programme.

On Reflection:

The primary focus of Critical Care management of TBI patients will be on the acute phase of the condition where immediate interventions are necessary. Ongoing management requires frequent clinical re-evaluation and interpretation of all available data. Clinical signs can be elucidated even in sedated patients, and pupillary reaction to light remains a vital clinical sign and changes in reactivity may be ominous requiring immediate further evaluation, investigation and often, new intervention. The clinical course of these patients may be prolonged and refractory intracranial hypertension, where there is not a surgical option of therapy, may have an insidious onset. All efforts should be made to control ICP medically as, in the absence of a surgically remediable haematoma or lesion, decompressive craniectomy has not been proven to improve outcome though it will improve ICP values. Multidisciplinary communication is paramount to the coordination of effective and efficient critical care and, importantly, to the satisfaction of the patient’s relatives.

See the PACT module on Communication.

Taking a medium and long-term view of what quality Critical Care can add to clinical management, it is important that patients should be well nourished and have optimum management of withdrawal and dysautonomic syndromes. Sedative or antiepileptic drugs require re-evaluation and, if their usefulness is in doubt, are gradually removed to reduce their negative effect on neuroplasticity. Mobilisation, physiotherapy and good skin care during critical illness will be very important to the capacity of the patient to benefit from rehabilitation. Patients with flaccid tetraplegia, not explained by initial injury, should be evaluated for Critical Illness Myopathy and/or Polyneuropathy.

See the PACT modules on Nutrition, Sedation and analgesia, and Neuromuscular conditions.

Evaluation of airway reflexes, of swallowing and of the level of consciousness is undertaken toward the end of ICU stay with the aim of facilitating early (speaking valve) speech and tracheostomy decannulation.

PACT module on Airway management.

Quality and judicious use of antibiotics during Critical Care and the practice of good antibiotic stewardship and infection control principles will hopefully have reduced the
likelihood of the complication of multi-resistant organism carriage and/or infection. The critical care team will have removed any unnecessary central venous catheter before leaving ICU thus reducing the risk of catheter related infection (CRI); similarly the urinary catheter should be removed as soon as this is clinically feasible.

See the PACT module on Pyrexia (for diagnosis of CRI).