Traumatic Brain Injury Part I: Introduction and early management

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2026 Update **2026**

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Intended Learning Outcomes

TBI Part I: Introduction and early management

1. Explain the pathophysiological mechanisms of primary and secondary brain injury and apply a physiology-based early neuroprotective strategy.
2. Perform a structured immediate assessment of patients with traumatic brain injury, integrating clinical history, neurological examination, and early monitoring.
3. Apply contemporary airway, ventilation, and oxygenation targets in TBI, including avoidance of routine hyperventilation and prevention of hypoxia.
4. Implement evidence-informed haemodynamic targets to avoid hypotension and optimise cerebral perfusion pressure in the early phase of TBI.
5. Select appropriate fluid resuscitation strategies and hyperosmolar therapies, recognising indications, limitations, and potential adverse effects.
6. Critically appraise the evidence for tranexamic acid use in TBI and apply it appropriately based on injury severity and timing.
7. Recognise and manage neurogenic pulmonary oedema and stress-induced cardiac dysfunction associated with acute brain injury.
8. Describe basic and advanced monitoring modalities in TBI, including neurological assessment and principles of multimodality monitoring.
9. Identify and interpret key laboratory and radiological investigations required in the early management of TBI.
10. Conduct a focused neurological examination and recognise early neurological deterioration requiring urgent intervention or referral.

eModule Information

Relevant Competencies from CoBaTrICE

TBI Part I: Introduction and early management

- **1.1** Adopts a structured and timely approach to the recognition, assessment and stabilisation of the acutely ill patient with disordered physiology
- **1.4** Triages and prioritises patients appropriately, including timely admission to ICU
- **1.5** Assesses and provides initial management of the trauma patient
- **3.6** Recognises and manages the patient with neurological impairment
- **4.3** Administers blood and blood products safely

1. Introduction

Traumatic brain injury (TBI) remains one of the leading causes of death and long-term disability worldwide and represents a major challenge for critical care systems. Contemporary global burden analyses demonstrate that disorders of the nervous system collectively constitute one of the largest contributors to years lived with disability, with TBI accounting for a substantial proportion of this burden. Updated estimates derived from the Global Burden of Disease (GBD) 2021 framework indicate that tens of millions of new TBI cases occur annually across more than 200 countries and territories, with a persistent and significant impact on mortality, disability, and healthcare utilisation.

The epidemiology of TBI has evolved over recent decades. In low- and middle-income countries, road traffic collisions and interpersonal violence continue to account for a large proportion of severe injuries. In contrast, high-income countries have experienced a marked demographic shift, with an increasing incidence of TBI related to falls, particularly among older adults. This change is closely linked to population ageing, frailty, and the widespread use of anticoagulant and antiplatelet therapies. As a result, the median age of patients with moderate-to-severe TBI in high-income settings has increased substantially compared with earlier decades.

Importantly, TBI is a highly heterogeneous condition, encompassing a wide spectrum of primary injury patterns and secondary pathophysiological processes. While the primary injury is not reversible, outcomes are strongly influenced by the prevention and treatment of secondary brain injury, including hypoxia, hypotension, cerebral hypoperfusion, metabolic derangements, and intracranial hypertension. For this reason, early management of TBI requires a structured, physiology-driven approach beginning at the scene of injury and continuing through emergency department resuscitation, imaging, surgery, and intensive care unit (ICU) admission.

Although the majority of patients presenting with TBI have mild injuries, this module focuses on the early management of moderate-to-severe TBI, where timely recognition, meticulous physiological optimisation, and appropriate referral to specialist centres have the greatest potential to improve outcomes. Awareness of mild TBI remains important, particularly in patients with polytrauma or spinal injury, but detailed discussion of concussion and long-term rehabilitation is beyond the scope of this module.

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(Maas et al. 2017; Johnson and Griswold 2017; Coronado et al. 2012; Majdan et al. 2016; Zhong et al. 2025; GBD 2021 Nervous System Disorders. 2024; Maas et al. 2022; Meyfroidt et al. 2022)

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2. Early Management of Head Injury

The primary objective in the early management of traumatic brain injury (TBI) is to prevent or limit secondary brain injury while rapidly identifying and treating life-threatening consequences of the primary insult. Secondary brain injury results from potentially preventable physiological derangements such as hypoxia, hypotension, cerebral hypoperfusion, intracranial hypertension, hypercapnia or hypocapnia, hyperthermia, and metabolic disturbances. These secondary insults are most frequent during the first hours after injury and are strongly associated with worse neurological outcome.

Early management should therefore be structured around a physiology-driven neuroprotective bundle that prioritises airway protection, adequate oxygenation and ventilation, haemodynamic stability, normoglycaemia, normothermia, and early control of intracranial dynamics. While standardised algorithms improve reliability and safety of care, clinicians must remain adaptable and prepared for unexpected deterioration, given the marked heterogeneity of TBI pathophysiology and injury patterns.

Intensive care management of TBI is a dynamic process that begins at the scene of injury and continues through emergency department resuscitation, imaging, surgical intervention, and ICU admission. During this phase, patients may transition rapidly between clinical environments, including the emergency department, operating theatre, radiology suite, and intensive care unit. It is therefore essential that intensive care practitioners are familiar with prehospital and early emergency management principles and are able to anticipate ICU-specific challenges before formal admission.

Optimal early care requires frequent reassessment of neurological status and physiological variables, prompt recognition of evolving intracranial or extracranial pathology, and early involvement of neurosurgical and trauma services when indicated. Practising a structured, reproducible approach while maintaining clinical vigilance is key to reducing avoidable secondary brain injury and improving patient outcomes.

2. 1. Clinical history

Accurate and timely history-taking is a critical component of early assessment in traumatic brain injury (TBI) and should be undertaken as soon as this can be achieved without delaying life-saving interventions. Information is often obtained from multiple sources and assembled incrementally, including eyewitnesses, prehospital providers, emergency department staff, and relatives.

Of particular importance are details of the mechanism of injury, estimated force and energy transfer, use of protective devices (such as seatbelts or helmets), and the patient's neurological and physiological status at the scene and during transport. Early documentation of level of consciousness, pupillary responses, blood pressure, oxygenation, and ventilation status provides an invaluable baseline against which subsequent neurological and physiological deterioration can be detected.

A focused clinical history should include:

- Level of consciousness at the scene and during transport, with emphasis on the Glasgow Coma Scale motor score and pupillary reactivity
- Vital parameters recorded prehospital (blood pressure, oxygen saturation, ventilation status)
- Estimated blood loss and evidence of extracranial haemorrhage
- Prehospital and emergency treatments administered, including airway interventions, fluids, vasopressors, and medications
- Mechanism of injury, including speed, height of fall, and use of protective devices
- Relevant past medical history
- Suspected influence of alcohol or recreational drugs
- Possible medical causes precipitating the event (e.g. seizure, syncope, arrhythmia, stroke)
- Use of anticoagulant or antiplatelet therapy, including drug type and timing of last dose

Particular attention should be paid to pre-injury antithrombotic therapy, as anticoagulants and antiplatelet agents are increasingly prevalent and are associated with higher risks of intracranial haemorrhage progression and delayed bleeding. Where uncertainty exists, early discussion with haematology and repeat imaging strategies should be anticipated.

While prehospital airway management and intubation may be appropriate in selected patients, the strongest evidence supports early prevention of hypoxia and hypotension rather than any specific airway strategy. Decisions regarding prehospital or early in-hospital intubation should therefore be individualised, taking into account transport time, available expertise, associated injuries, and the risk of secondary brain injury.

Although essential information may be lost if not actively sought, history-taking must never delay immediate resuscitative measures.

Note

Promoting road safety measures contributes to healthcare.

Task

Test your knowledge:

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?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

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’. The GCS (in particular the motor score) may also be of prognostic benefit (see section on IMPACT Score).

Note

Recording of on-site observations is vital

Task

Test your knowledge

At an accident scene, tracheal intubation may be carried out. How does this benefit traumatic head injury patients?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

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. There is however, clear evidence that preventing hypoxia and hypotension improves outcome.

Note

Contemporary trauma systems emphasise early physiological stabilisation and rapid access to definitive care. The priority is prevention of hypoxia and hypotension rather than adherence to a fixed prehospital strategy.

Early level of consciousness provides both a baseline for monitoring and contributes to prognostic models such as IMPACT and CRASH, although prognostication should remain cautious in the early phase.

In text References

(Attewell, Glase and McFadden. 2001; Stewart et al. 2003; Forbes et al. 2017; Gravesteijn et al. 2021; Meyfroidt et al. 2022; Hawryluk et al. 2019; Hossain, Rostami and Marklund. 2023; Maas et al. 2022)

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2. 2. Immediate assessment and treatment

The initial assessment and management of patients with traumatic brain injury (TBI) follows standard trauma principles and prioritises the rapid identification and correction of life-threatening conditions. Assessment and treatment are closely interlinked and should proceed simultaneously, with frequent reassessment.

The immediate priorities are to:

- Secure or maintain a patent airway
- Optimise oxygenation and ventilation
- Initiate haemodynamic resuscitation
- Identify intracranial and extracranial injuries
- Reassess neurological status repeatedly

Prevention of secondary brain injury during this early phase is critical and requires meticulous attention to oxygenation, ventilation, blood pressure, and cerebral perfusion.

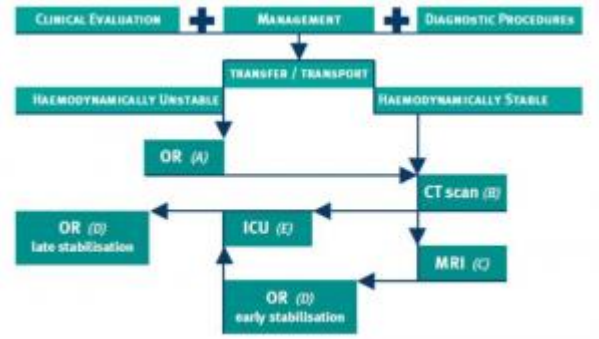

Figure 1: Prioritisation of injuries and initial management

Figure 1:
(A) Extracranial life-threatening haemorrhage. (Extremely rarely, intracranial haemorrhage without prior CT)
(B) Head. Extracranial as clinically indicated
(C) In selected patients only, particularly MRI of the spine in spinal injury patients
(D) Intracranial haematoma. Major extracranial injuries
(E) Indications for admission to ICU: coma with diffuse brain injury; post-neurosurgery; altered consciousness and requiring intubation; multi-injured/cardio-respiratory instability; brain-stem death(?)

Note

Primary resuscitation prioritises management of airway, breathing and circulation.

2. 2. 1. Airway, breathing

Hypoxia is strongly associated with increased mortality and worse neurological outcome following traumatic brain injury and must be avoided at all stages of care. Airway protection and ventilation should therefore be instituted promptly when airway reflexes are impaired, oxygenation or ventilation is inadequate, or when neurological status is deteriorating.

During initial resuscitation, supplemental oxygen should be administered to all patients with suspected or confirmed TBI. Once airway control and adequate oxygenation are achieved, inspired oxygen concentration should be titrated to maintain arterial oxygen saturation above 94–95%, avoiding prolonged exposure to unnecessarily high FiO_2.

Ventilation strategies should aim for **normocapnia**, with a target arterial $PaCO_2$ of approximately **35–38 mmHg**. Routine hyperventilation in the early phase of TBI is not recommended, as it can reduce cerebral blood flow and precipitate cerebral ischaemia. Continuous capnography should be used in all intubated or ventilated patients to ensure tight control of ventilation.

Short-term hyperventilation ($PaCO_2$ as low as 25–30 mmHg) may be used **only as a temporising measure** in patients with signs of imminent cerebral herniation while awaiting definitive interventions such as surgical decompression. When hyperventilation is used, its duration should be minimised, and concurrent optimisation of oxygen delivery and haemodynamics is essential.

Tracheal intubation should be performed using a rapid sequence induction technique by experienced personnel, with strategies to minimise increases in intracranial pressure and avoid peri-intubation hypotension. Induction agents with minimal haemodynamic depression should be selected, and vasopressors should be readily available.

Ketamine is considered safe in patients with TBI and may be particularly useful in haemodynamically unstable patients. Manual in-line stabilisation of the cervical spine should be maintained during airway management until spinal injury has been excluded.

Aspiration should be actively prevented and promptly treated, and lung-protective ventilation strategies should be employed once the patient is stabilised, particularly in those with associated thoracic injury or impaired lung mechanics.

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.
- Adopt strategies to minimise any immediate hypotension or hypoxia 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 SpO_2 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 CO_2 (et CO_2) is mandatory and $PaCO_2$ should be kept between 35–38 mmHg in normotensive patients. Avoid hypercapnia.
- Apply hyperventilation in patients with abnormal papillary light reflexes not due to direct iris effects (eg traumatic mydriasis). If hyperventilation leads to hypotension then discontinue.
- While hyperventilation ($PaCO_2$ as low as 22mmHg or 3kPa) may be lifesaving when waiting for definitive management to reduce severe intracranial hypertension such as surgery it should be carried out for as short a period as possible (30 minutes or less) as it has been shown to cause cerebral ischemia, and for long periods is associate with poorer outcome.

Task

Test your knowledge

If we suspect an potential impairment in the airway of a patient and decision is made to anaesthetise a patient, which specific considerations should we have?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

If the patient requires intubation, especially when consciousness is already impaired, the aim is to minimise the risk of secondary brain damage due to induced raised ICP (intracranial pressure) and decreased perfusion pressure. Rapid sequence induction (RSI) is the recommended technique using a combination of sedative with low cardiopressant effects (e.g. midazolam, ketamine), analgesic (fentanyl) and fast acting muscle relaxant (e.g. succinylcholine, rocuronium) agents.

When intubating where possible (and it is compulsory in some guidelines) capnopgraphy should be used. The Major complications of airway management in the UK (National Audit Project) clearly showed that the use of capnography routinely in intubation could significantly reduce deaths and brain damage, particularly in intensive care units.

> **Warning**
>
> When intubating a patient, think of possible injuries to the cervical spine and minimise the risk of possible further neurological insult. This usually requires an extra person to manually maintain inline stabilisation.

> **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.

In text References
(Coles et al. 2007; Davis et al. 2006; Protheroe and Gwinnutt. 2011; Hossain, Rostami and Marklund. 2023; Carney et al. 2017; Zeiler et al. 2014)

2. 2. 2. Circulation

Hypotension is a major contributor to secondary brain injury and is independently associated with increased mortality and poor neurological outcome following traumatic brain injury (TBI). Even a single episode of hypotension can significantly worsen prognosis. Contemporary evidence indicates that cerebral injury may occur at blood pressure thresholds previously considered acceptable, particularly in older patients.

During the early management of TBI, systemic hypotension must be actively avoided. In adult patients, a systolic blood pressure (SBP) of at least **110 mmHg** should be targeted, while higher thresholds (120–130 mmHg) may be appropriate in elderly patients or those with known chronic hypertension. Until intracranial pressure (ICP) is measured, maintaining a mean arterial pressure (MAP) of approximately **80–85 mmHg** is a pragmatic strategy to support cerebral perfusion.

Haemodynamic resuscitation should begin immediately and continue throughout transport, imaging, and procedural interventions. Fluid therapy should be judicious, with early use of vasopressors where necessary to achieve blood pressure targets. **Norepinephrine** is the preferred first-line vasopressor in patients with TBI, as it reliably increases MAP with minimal impact on heart rate and intracranial pressure.

In patients with combined traumatic brain injury and extracranial haemorrhage, priorities must be carefully balanced. Life-threatening haemorrhage should be controlled rapidly; however, prolonged permissive hypotension is contraindicated once immediate bleeding control has been achieved, due to the high risk of cerebral hypoperfusion.
Once ICP monitoring is established, cerebral perfusion pressure (CPP) targets should be individualised, typically aiming for **CPP ≥ 60–70 mmHg**, while avoiding excessive vasopressor use that may exacerbate cerebral oedema or systemic complications. Continuous reassessment of neurological status and haemodynamic variables is essential.

- Insert at least two large bore peripheral intravenous cannulae.
- If hypotension occurs: first check for extracranial injuries.
- A systolic blood pressure of < 110mmHg doubles mortality after TBI. However, this is no a clear threshold at which low blood pressure increases mortality and morbidity. A recent observational study found for each 20 point increase in systolic pressure there was a decrease in the adjusted odds of death of 18.8% (Spaite et al). This suggests that clinically meaningful hypotension may not be as low as current guidelines suggest and so hypotension should be avoided where possible.
- 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, **Norepinephrine** is the preferred first-line vasopressor, as it reliably increases MAP with minimal impact on heart rate and intracranial pressure.
- 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.
- 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 or a MAP target of 90mmHg while ICP is unknown.
- The concept of permissive hypotension is controversial and no mortality benefit using this strategy has been shown. However, in actively bleeding patients it makes intuitive sense to not drive the bleeding through hypertension. This is commonly taken as a systolic pressure of 90 mmHg but the target may need to be individualised and be higher.
- Isolated intracranial injury in the adult does not cause hypotension, except in extremely severe patients, usually with bilaterally dilated unreactive pupils

In text References
(Feinstein et al. 2005; Earle et al. 2007; Spaite et al. 2017; Wiles 2013; Fuller et al. 2014; Brenner et al. 2012; Carney et al. 2017; Spaite et al. 2017)

2. 2. 3. Fluid resuscitation and hyperosmolar therapy

Adequate intravascular volume is essential to maintain cerebral perfusion and prevent secondary brain injury following traumatic brain injury (TBI). Fluid resuscitation should aim to restore and maintain euvolaemia while avoiding hypotonic solutions, which may exacerbate cerebral oedema.

In the early phase of TBI, **0.9% sodium chloride** remains the preferred crystalloid solution. Hypotonic fluids, including dextrose-containing solutions, should be avoided. The use of balanced crystalloid solutions remains controversial in TBI; while they may be appropriate in later phases or in selected patients, their lower chloride and sodium content may increase the risk of cerebral oedema in the acute setting.

Fluid administration should be guided by frequent reassessment of haemodynamic status and response to therapy. Excessive fluid loading should be avoided, particularly once blood pressure targets are achieved, and early vasopressor support should be used where necessary.

Fluid resuscitation:

- Use intravenous isotonic solutions (e.g. NaCl 0.9%, Ringer's-Solution (Hartmann's solution), for volume resuscitation. A large meta-analysis is consistent with the possibility of harm with balanced crystalloids after TBI and many consider it prefereable to use NaCL 0.9% after TBI (Hammond et al. 2019).
- 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.
- Use of colloids, glucose-containing hypotonic soutions and other hypotonic solutions or albumin as maintenance fluids is not recommended by the last ESICM consensus.
- Use of synthetic colloids, albumin, hypertonic solutions, glucose-containing and other hypotonic soluctions must be avoided in the TBI patient during fluid resuscitation.
- The aim is normovolaemia during resuscitation of TBI patients.
- Use of central venous pressure alone as an enpoint to guide fluid therapy must be avoided.
- Use of blood pressure and fluid balance and dynamic hemodynamic variables such pulse pressure variation or stroke volume variation in guiding fluid therapy must be integrated to other variables such as cardiac output, SvO2 and blood lactate to optimize fluid therapy.
- It is strongly recommended monitoring blood electrolytes monitoring as a safety endpoint in fluid therapy.

2. 2. 3. 1. Hyperosmolar therapy

Hyperosmolar therapy is a cornerstone of acute management for intracranial hypertension. Both mannitol and hypertonic saline are effective in reducing intracranial pressure (ICP) and improving cerebral perfusion, and either may be used depending on the clinical context.

Hypertonic saline offers several potential advantages, including expansion of intravascular volume and maintenance of systemic blood pressure, and may be preferred in haemodynamically unstable patients. Mannitol may be considered in haemodynamically stable patients with preserved renal function but should be avoided in hypovolaemia, hypotension, or established renal failure.

Treatment of intracranial hypertension should not rely solely on a single ICP threshold. Instead, therapy should be guided by ICP trends, clinical examination, radiological findings, and overall physiological context. As a pragmatic reference, sustained ICP values above **20–22 mmHg** often prompt escalation of therapy in conjunction with other clinical indicators.

Hypertonic saline may be administered as intermittent boluses or continuous infusion. In cases of refractory intracranial hypertension or impending herniation, **23.4% sodium chloride** administered as a small-volume bolus (e.g. 30 mL) may be used as a rescue therapy in experienced centres.

Serum sodium and osmolality should be closely monitored during hyperosmolar therapy. Target serum sodium concentrations commonly range between **145 and 155 mmol/L**, and excessive hypernatraemia or hyperosmolality should be avoided due to the risk of renal injury, electrolyte disturbances, and rebound intracranial hypertension.

- ICP value 20-22mmHg should be used as trigger threshold independent to other variables to start osmotherapy in order to reduce ICP (especially if other Tier 1 neuroprotective measures have not been effective).
- 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.
- In patients with clinical signs of transtentorial herniation high-dose mannitol (1–2 gr/kg/dose) or hypertonic saline (HTS) are potentially useful to reverse herniation. HTS is commonly available in 3%, 5% or 23.4% concentrations.
- There is no clear evidence for the use of Mannitol or HTS and choice is dependent on local protocols. However, Mannitol may lead to a large diuresis and so in patients where hypovolemia or hypotension is a concern the use of HTS may be preferable.
- It is recommened to monitor serum osmolarty and electrolytes during osmotherapy in order to limit side effects.

Think

about the practice in your own region in relation to the choice of fluids for resuscitation in TBI.

In text References

(Finfer et al. 2004; Boone et al. 2015; Gantner, Moore and Cooper. 2014; Mangat and Härtl. 2015; Perel, Roberts and Ker. 2013; van der Jagt 2016; Chacon-Lozsan and Pacheco. 2018; Carney et al. 2017; Myburgh et al. 2007; Gharizadeh et al. 2022; Kochanek et al. 2015)

2. 2. 4. Tranexamic Acid

Tranexamic acid (TXA) is an antifibrinolytic agent widely used in the management of traumatic haemorrhage. Its role in traumatic brain injury has been evaluated in large randomised controlled trials, most notably the CRASH-3 trial.

Current evidence indicates that TXA may reduce mortality in patients with **mild to moderate traumatic brain injury**, particularly when administered **within 3 hours of injury** and in the presence of intracranial haemorrhage. In contrast, no clear mortality benefit has been demonstrated in patients with **severe TBI**, defined by very low Glasgow Coma Scale scores or extensive intracranial injury.

The benefit of TXA appears to be strongly time-dependent, with diminishing effect when administration is delayed beyond the early post-injury period. Importantly, available data do not demonstrate an increased risk of thromboembolic complications associated with TXA use in TBI.

Based on current evidence, TXA should not be considered a routine therapy for isolated severe traumatic brain injury in the intensive care unit. Its use should be limited to selected patients in the early phase after injury, typically as part of a broader trauma or major haemorrhage protocol. For patients admitted to the ICU after the early injury window, TXA is generally not indicated unless there are additional haemorrhagic indications.

As with all interventions in TBI, TXA administration should be individualised, taking into account injury severity, timing, associated extracranial bleeding, and overall risk–benefit assessment.

Key points

- TXA is most effective in **mild–moderate TBI**
- Administer **within 3 hours** of injury
- **No proven benefit** in severe isolated TBI
- No convincing increase in thrombotic events
- Not a routine ICU therapy

In text References

(CRASH-3 trial 2019; Cap. 2019; CRASH-2 trial et al. 2010; CRASH-2 et al. 2011; Dewan et al. 2012; Roberts, Prieto-Merino and Manno. 2014; Maas, Steyerberg and Citerio. 2021; Lawati et al. 2021; Zehtabchi et al. 2014)

2. 2. 5. Neurogenic pulmonary oedema and cardiac regional wall abnormalities in TBI

Acute pulmonary oedema and myocardial dysfunction may occur following severe traumatic brain injury as a consequence of intense sympathetic activation and catecholamine release. This phenomenon, often referred to as neurogenic pulmonary oedema, can develop rapidly after the initial injury and may coexist with stress-induced myocardial dysfunction.

The underlying mechanism is thought to involve a sudden catecholamine surge leading to increased pulmonary capillary hydrostatic pressure, endothelial injury, and transient myocardial stunning. Stress-induced cardiomyopathy, including Takotsubo syndrome and its variants, has been increasingly recognised in patients with acute brain injury and may contribute to haemodynamic instability and hypoxaemia.

Clinically, patients may present with acute hypoxaemia, bilateral pulmonary infiltrates, and signs of cardiac dysfunction in the absence of primary cardiac disease. Early bedside echocardiography is essential to assess ventricular function, exclude primary cardiogenic shock, and guide haemodynamic management.

Management is primarily supportive and focuses on optimisation of oxygenation, careful fluid balance, and haemodynamic stabilisation. Excessive fluid administration should be avoided. Vasopressor support, most commonly with norepinephrine, may be required to maintain cerebral perfusion pressure, while inotropic support should be reserved for patients with significant myocardial dysfunction confirmed on echocardiography.

With appropriate supportive care, neurogenic pulmonary oedema and stress-induced cardiac dysfunction are often reversible over days. However, their presence is associated with increased morbidity and necessitates close monitoring and multidisciplinary management.

In text References

(Davison, Terek and Chawla. 2012; McLean, Slama and Chew. 2018; Wittstein et al. 2005; Meyfroidt et al. 2022; Rajagopal, Ganesh and Vetrivel. 2017)

2. 2. 6. 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.

2. 2. 6. 1. Basic monitoring

> **Note**
>
> Monitoring warns of impending problems and indicates effectiveness of treatment.

Basic monitoring of patients with isolated head injuries should be initiated, where possible, at the site of the accident and include:

Task

Test your knowledge

What is the basic monitoring required for patients with significant TBI?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

- 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 $PaCO_2$ and Pet CO_2 in the following reference.
- Capnography is strongly suggested in the extrahospital setting where hypoventilation or inadvertent therapeutic hyperventilation is common.

Task

Test your knowledge

How frequently should such monitoring be performed?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

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.

In text References

(Helm et al. 2003)

2. 2. 6. 2. 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, Brain Tissue Oxygenation (or increasingly less commonly jugular bulb catheterisation)).

Note

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

2. 2. 6. 3. Further monitoring

After initial resuscitation, involving airway, breathing, and circulation, additional practical procedures include:

- central venous catheter (moderate/severe injuries; multiple trauma)
- arterial catheter (moderate/severe injuries; multiple trauma)
- nasogastric tube (or orogastric tube in presence of bad facial injuries or suspicion of a baso fo skull fracture)

Warning

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

Note

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

- Cardiac output monitoring may be required in patients who have high vasopressor and inotropic requirements to maintain their cerebral perfusion pressure. Options include Pulmonary Artery Flotation Catheter (in centers used to using it) and many alternatives like non-invasive cardiac monitors, cardiac ultrasound or minimal invasive as PiCCO and LiDCO systems.
- Hemodynamic fluid ressponssivenes variables should be monitored in TBI patients especially during fluid resuscitation.

Note

The variability in both arterial and venous elements of the cerebral circulation means that in order to accurately reflect blood pressure in the Circle of Willis and determine CPP the arterial line transducer should be levelled at the Tragus. Consideration of this should be done in line with local practices.

Task

Test you knowledge

Name complications derived from missplacement or complication of inserting a central venous catheter in head injury patients.

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

Neck bleeding can cause obstruction in the venous return (secondary to application of pressure to stop the bleeding and/or haematoma formation); venous thrombosis or pneumothorax leading to hypotension, hypoxia or both.

In text References

(De Backer et al. 2018; Oddo et al. 2018; Bentzer et al. 2016; Thomas, Czosnyka and Hutchinson 2015)

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2. 3. Patient investigations

2. 3. 1. 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).
- In the context of polytrauma creatinine kinase, troponin and amylase/lipase should also be considered.

Note

The level of consciousness may be altered by electrolyte or metabolic disturbances.

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.

2. 3. 2. Focus on Coagulation

Coagulopathy at presentation is a powerful predictor of outcome and prognosis after TBI resulting in a 9 times higher risk of mortality and 30 times higher risk of unfavourable outcome than in TBI patients without coagulopathy. It is a complex process where a patient may become hypercoagulable or hypocoagulable – it is the latter that is usually most apparent.

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. While the ITACTIC trial found not overall benefit for using TEG in trauma patients with haemorrhagic shock there was a signcal for potential utility for use in patients with TBI with an increased survival at 24 hours in those who had TEG guided therapy. Early haemostatic monitoring including TEG or ROTEM, if accessible, should be performed as soon as possible after trauma especially in the first 72 hours and pateints who have sustained a TBI.

2. 3. 2. 1. Transfusion thresholds

In a randomised trial, neither the administration of erythropoietin nor maintaining haemoglobin concentrations above 10 g/dL resulted in an improved neurological outcome at 6 months in patients with TBI; however, the transfusion threshold of 10 g/dL versus 7 g/dL was associated with a higher incidence of adverse events including PHI. Furthermore, a mean 7-day haemoglobin concentration of less than 9 g/dL has been associated with increased hospital mortality in patients with severe TBI. The pathophysiology of such complications is complex and even when red-blood-cell transfusion produces an improvement in cerebral oxygenation, this finding has not always been associated with changes in brain metabolism (Maegele et al. 2017).

2. 3. 2. 2. Oral Anticoagulants

The demographic change in TBI is accompanied by an increasing number of patients presenting on oral anticoagulants or antiplatelet drugs. Pre-injury use of antithrombotic agents was associated with greater expansion of extra-axial lesions, higher rates of significant hemorrhagic progression, and higher risk of delayed traumatic intracerebral haemorrhage. A metanalysis has shown that being on warfarin at the time of presentation doubles the risk of a poor outcome. However, a similar analysis of antiplatelet agents did not show the same association.

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 (PCC) should be based on weight and guided by initial PT/INR values. In multiply injured, haemodynamically unstable patients plasma transfusion (FFP) may be more appropriate since it adds both volume and coagulation factors.

Direct Oral Anticoagulants (DOACs) present a particular challenge in TBI, and trauma as a whole as antidotes or specific reversal agents for some of the DOACs are lacking, and usual coagulation tests cannot be used to determine the degree of anti-coagulation. Only one DOAC, Dabigatrin, a direct thrombin inhibitor has a specific reversal agent available (the monoclonal antibody Idarucizumab). Oral direct factor Xa inhibitors include rivaroxaban, apixaban, edoxaban and betrixaban. As yet there is no specific antidote available. Despite their difficulty in reversal they have not yet been associated with worse outcome after trauma.

When assessing the anticoagulation status of a patient it is important to try to ascertain the time since last dose as in general anticoagulation with these agents will have resolved fully after 5 half-lives since the last dose. For example, Dabigatran has a half-life of 12 to 17 hours, and so five half lives are approximately 2.5 to 3.5 days after the last dose. Rivaroxiban has a half-life of 5 to 9 hours and five half-lives will have elapsed by day 1 to 2 after the last dose. The half lives are dependent on renal function (and to a lesser extent hepatic) function and so derangements in renal or liver function may cause a greater degree and/or duration of anticoagulation. Where a reversal agent is not available PCC is often given (and may be given in conjunction

with fresh frozen plasma concentrate to supply factor VII if a 3-factor PCC is used instead of a 4-factor aPCC). Antifibrinolytic agents like TXA may also be given. The complexity of not being able to monitor or often fully reverse these agents means that specialist haematological advice should usually be sought when a patient with severe TBI presents on a DOAC.

2. 3. 2. 3. Anti-platelet agents

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. Platelet-concentrate transfusion in patients with preinjury intake of anti-platelet agents may be considered, although the current data on effects on platelet function and outcome is inconclusive. While there is growing interest in the use of point of care platelet function tests, such as the Platelet Function Analyzer (PFA-100) though more research is required before they can be used in the clinical setting.

In text References
(Barletta et al. 2017; Batchelor and Grayson. 2012; Batchelor and Grayson. 2013; Maegele et al. 2017; Wood et al. 2017; Rossaint et al. 2016; Baksaas-Aasen et al. 2021; Böhm et al. 2021; Mathieu et al. 2020)

2. 3. 3. Radiological investigations

In the emergency room Initial investigations may include X-rays of the chest, pelvis and lateral cervical spine and abdominal focused assessment with sonography for trauma (FAST) scanning. However, plain x-rays may miss important injuries, and it should be emphasised that a FAST scan cannot rule out the presence of bleeding or a significant injury. With this in mind many centers prefer perform a ‘trauma CT’ as soon as possible as the first radiologic examination and only perform plain x-rays or FAST in select circumstances. 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.

The “Trauma CT” consists of CT head, CT cervical spine, and angiography of the thorax, abdomen and pelvis which may include arterial, venous and delayed phases. The CT is reconstructed to allow the thoracic and lumbar spine to be assessed.

To reduce the radiation burden select CT imaging may be considered. However this should be done with caution and serial clinical assessment of patients to ensure occult injuries are not missed. In patients who appear to have an isolated TBI a CT head and cervical spine (C1 to T1) may be performed. However, targeted scanning is best performed in those with more mild injuries (GCS > 13) where the mechanism does not raise suspicion of other injuries and a thorough clinical examination can be performed. There should be a lower threshold for CT neck in the eldery. In patients with a low GCS it can be difficult to exclude other injuries and a trauma series CT is usually indicated.

The performance of a ‘Trauma CT’ may lead to the identification of previously unknown pathologies. It is very important that such incidental findings are followed and/or treated appropriately.

In patients on warfarin and DOACS with a mild TBI if performed early the initial CT head may be normal or only show a small lesion eg small contusion. It is prudent in such patients, especially those with symptoms, to continue to observe and repeat their CT head within 12 to 24 hours (or earlier if deterioration) to ensure no significant bleeding or lesion progression.

In text References

(Huber-Wagner et al. 2009; Sierink et al. 2016; Treskes et al. 2017; Nishijima et al. 2012; Mason et al. 2017; Nishijima et al. 2012)

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2. 4. 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 clinical 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 in accordance with privacy laws.)
- The relatives should preferably be seen by a senior member of staff but it can be useful to all if junior medical staff and where possible the bedside nurse 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 ACE course on Communication.

2. 5. 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 Figure 2 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.

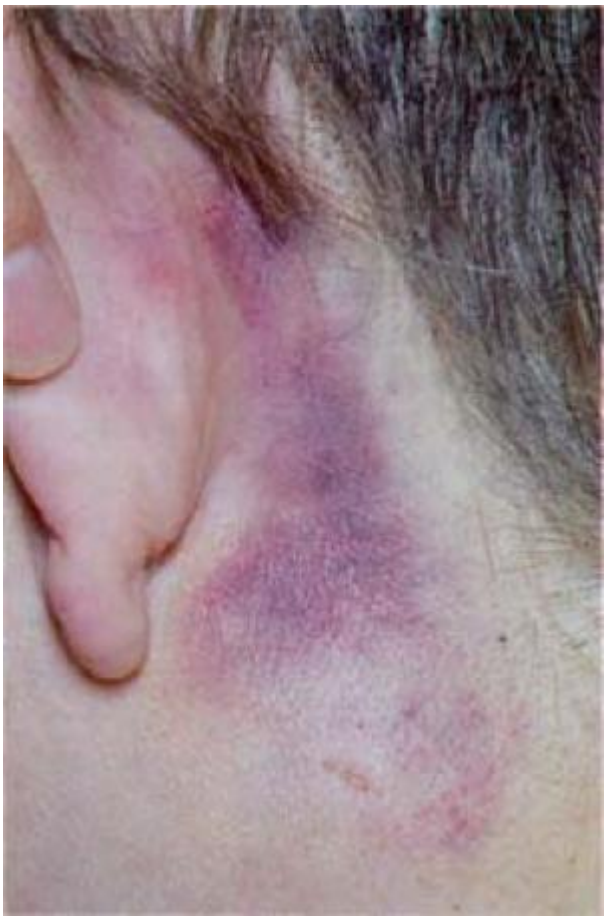

Figure 2:Battle's sign

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.

2. 5. 1. 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.
- Assess for focal neurology with a full neurological examination whenever appropriate.

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.

Note

There is no substitute for clinical examination... appropriately repeated

- 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.

Task

Test your knowledge

A neurological examination of a patient with severe TBI and neurological worsening 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?

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

This indicates transtentorial herniation due to the developing haematoma, probably associated with elevated intracranial pressure.

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

COMPLETE TASK THEN CLICK TO REVEAL THE ANSWER

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 short-term hyperventilation (with an FiO2 1.0) 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 supraorbital pressure and trapezius pinch are the 2 methods of producing a painful stimulus currently recommended, Other methods like pinching the ear lobe or cheek, or pressing firmly on the fingernail bed are not recommended. Painful stimuli in the cranial nerve distribution will avoid spinally mediated responses. 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.

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 re-evaluated 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.

Challenge

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 ACE course 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.

In text References

(Juul et al. 2000; Young, Ropper and Bolton. 1998)

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2. 6. 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 brain 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. The RAIN study suggested that transfer to specialist trauma centres was cost-effective, even when neurosurgical intervention was not indicated. 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.

In text References

(Grieve et al. 2016; Harrison et al. 2013; Patel et al. 2005)

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2. 7. Concept of tiered management

The above managment and neuroprotective measures are commonly considered in a Tiered fashion where treatments are grouped together from those which are most simplest to give with least side effects (eg head up 30 degrees, sedation) through to life saving measures with siginiciant risk (eg decompressive craniectomy).

Examples are from The Brain Trauma Foundation and the Seattle International Severe Brain Injury Consensus Conference (SIBICC).

Task

Review the guidelines below.

- A management algorithm for patients with intracranial pressure monitoring: the Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC)
- A management algorithm for adult patients with both brain oxygen and intracranial pressure monitoring: the Seattle International Severe Traumatic Brain Injury Consensus Conference (SIBICC)
- Guidelines for the Management of Severe Traumatic Brain Injury, Fourth Edition