

# Neurosurgical and Intensive Care Management of Traumatic Brain Injury

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## Abstract

Optimal management of patients with traumatic brain injury (TBI) remains a challenge, despite significant improvements in pathophysiologic understanding and treatment strategies in recent decades. Because primary brain injury sustained at the time of trauma is irreversible, the TBI management mainly aims for early detection and treatment of secondary brain injury such as space-occupying intracerebral hematomas and brain edema. Prevention of secondary brain injury requires a high standard of care and understanding of both medical and surgical treatment modalities. This review focuses on practical recommendations for neurosurgical and intensive care management in patients with severe TBI.

## Keywords

- ▶ traumatic brain injury
- ▶ neurosurgery
- ▶ neurocritical care

Traumatic brain injury (TBI) is the leading cause of death and disability in young adults.<sup>1</sup> Population-based studies have shown mortality rates up to 45% and significant neurologic disability in almost half of the surviving patients.<sup>2,3</sup> Furthermore, the long-term effects of neuropsychological dysfunction may create significant difficulties in social reintegration, independence, and employment.<sup>4</sup>

Facial fractures have long been suggested to provide a cushion zone to the cranium and therefore may limit the extent of severe brain injury.<sup>5</sup> However, more recent studies showed that the impact forces transmitted to the head are often associated with brain injury.<sup>6,7</sup> The incidence of TBI in patients with maxillofacial fractures is approximately 25%.<sup>8</sup> Therefore, facial bone fractures may be considered as highly suspicious for associated brain injury.

TBI may result in various injuries, including skull fractures, diffuse axonal injury, and cerebrovascular injuries. Primary brain injury sustained at the time of trauma is irreversible and remains the most important determinant of neurologic outcome. Therefore, the TBI management focuses on preventing secondary brain injury in the hours and days following trauma, which may have intracranial (e.g., hematoma, brain edema) as well as extracranial causes (e.g., hypoxia, hypotension).

The severity of brain injury correlates closely with neurologic outcome and is classified by the initial postresuscitation Glasgow Coma Scale (GCS, ▶ **Table 1**) and further by findings on computed tomographic (CT) imaging.<sup>9,10</sup> Severe TBI is defined as GCS 8 or less and associated with poor, but in most cases not hopeless, prognosis. The overall severity of patients with multiple injuries can be assessed using the Injury Severity Score (ISS) according to different anatomical body regions, where an ISS greater than 14 is generally accepted as a definition of a patient with major trauma.<sup>11</sup>

Noncontrast CT scan is the diagnostic method of choice in the acute phase after TBI. The presence and extend of mass lesions and their consequences, leading to high intracranial pressure (ICP), should be considered. However, a direct correlation between CT imaging and high ICP is lacking.<sup>12</sup> Therefore, ICP monitoring using minimally invasive brain tissue probes should always be considered in unconscious and/or sedated patients with TBI.

The need for surgical intervention is based on both the patient's neurologic condition and findings on CT imaging.<sup>13–15</sup> The most important concern in patients with TBI is the development of high ICP due to a pathophysiologic cascade of intra- and extracellular derangements leading to secondary brain injury.<sup>16</sup> Recently, it has been shown that

**Table 1** Classification of severity of traumatic brain injury

Severity	Glasgow Coma Scale
Mild	13–15
Moderate	9–12
Severe	3–8

Note: Severity of traumatic brain injury is classified into mild, moderate, and severe according to the Glasgow Coma Scale.

multimodal neuromonitoring using brain tissue oxygen tension and microdialysis monitoring is more effective in detecting secondary brain injury than ICP monitoring alone.<sup>17</sup> Multimodal neuromonitoring may further allow monitoring the effects of medical and/or surgical interventions aiming at ICP control.

## Surgical Issues and Considerations

### Depressed Skull Fracture

Depressed skull fractures are classified as either “closed” or “open,” depending on the presence or absence of an overlying scalp laceration, respectively. An open fracture depressed greater than the thickness of the skull requires surgical repair and prophylactic antibiotics to prevent bacterial infection (→ Fig. 1).<sup>18</sup> Surgical objectives include (1) debridement of the contaminated bone fragments and soft tissue, (2) evacuation of intracranial hematoma and repair of a dural laceration if present, and (3) reconstruction of the skull. Observation can be considered for closed fractures depressed less than 1 cm without evidence of dural penetration and cerebrospinal fluid leak.

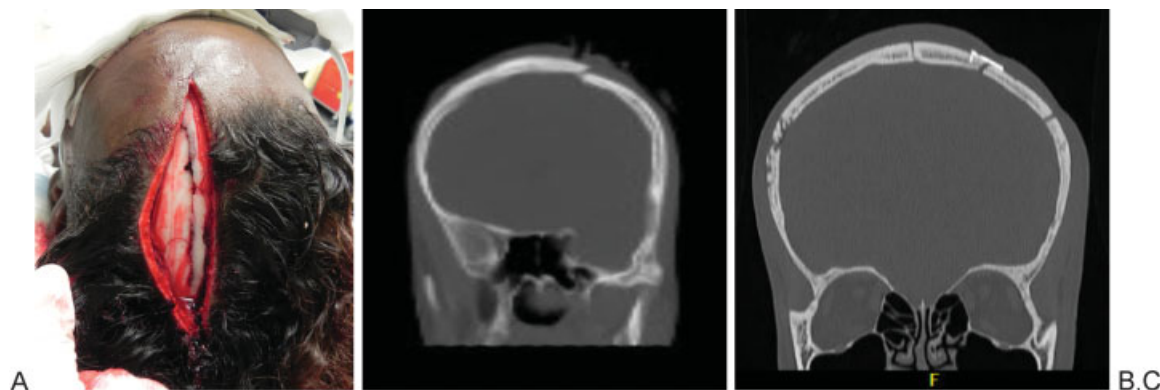
### Frontal Sinus Fracture

Fractures of the frontal sinus involve the (1) anterior table, (2) posterior table, or (3) both. Management strategies include prevention of short- and long-term complications, correction of aesthetic deformity, and reestablishment of normal sinus function.<sup>19</sup> In particular, posterior table fractures are associated with a high risk of cerebrospinal fluid (CSF) leak,

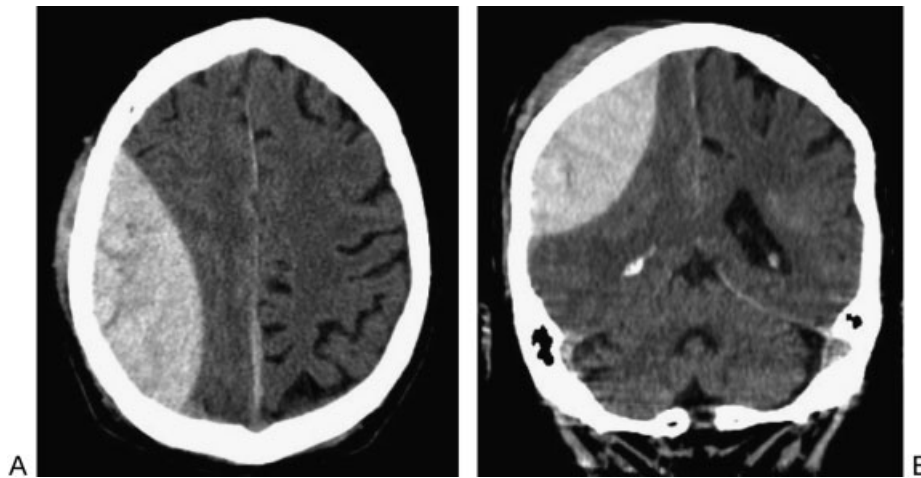
meningitis, and mucocele formation. Therefore, surgical repair (frontal sinus obliteration or cranialization) can be considered for significant disruption of the posterior table (displacement >1 table width), CSF leak, and nasofrontal tract obstruction.<sup>19,20</sup> Frontal sinus obliteration is performed in the presence of significant posterior table displacement and nasofrontal tract obstruction. Frontal sinus cranialization should be considered with CSF leak and/or severe comminution of the posterior table. The timing of surgery mainly depends on the patients' condition, often requiring a staged procedure with wound debridement in the acute phase and surgery for frontal sinus fracture in a second step. There is no definitive evidence regarding prophylactic antibiotic use. Observation can be considered for anterior table fractures as well as posterior table fractures displaced less than 1 table width and no evidence of CSF leak. In these cases, clinical and radiologic follow-up should be performed to rule out long-term complications (mucocele formation, meningitis, brain abscess).

### Epidural Hematoma

Epidural hematomas (EDHs) are mainly caused by an arterial bleeding of dural vessels, typically affecting the middle meningeal artery, and are associated with temporoparietal skull fractures. The classic clinical presentation includes a brief loss of consciousness followed by a lucid interval for several hours before neurologic deterioration occurs. Large EDHs may cause contralateral hemiparesis, impaired consciousness, and ipsilateral pupillary dilatation. The CT appearance of EDHs is a high-density biconvex shape adjacent to the skull (→ Fig. 2). Surgical intervention is recommended in cases of a hematoma volume greater than 30 cm<sup>3</sup>, independent of the patients' neurologic condition.<sup>13</sup> A frontotemporoparietal craniotomy is performed to gain sufficient access to middle meningeal vessels. The surgical objectives include (1) removal of the space-occupying hematoma and (2) control of the bleeding source. Rebleeding is prevented by placing tack-up sutures along the dura margin and central dura before replacing the bone flap. Evacuation of EDHs within a few hours after trauma is associated with a mortality rate of approximately 10%. Although a burr-hole approach can be a



**Fig. 1** (A) Depressed skull fracture with overlying scalp laceration after a bicycle accident (initial Glasgow Coma Scale [GCS] 14). (B and C) Coronal computed tomographic (CT) scan (bone window) before and after reconstruction of the skull.



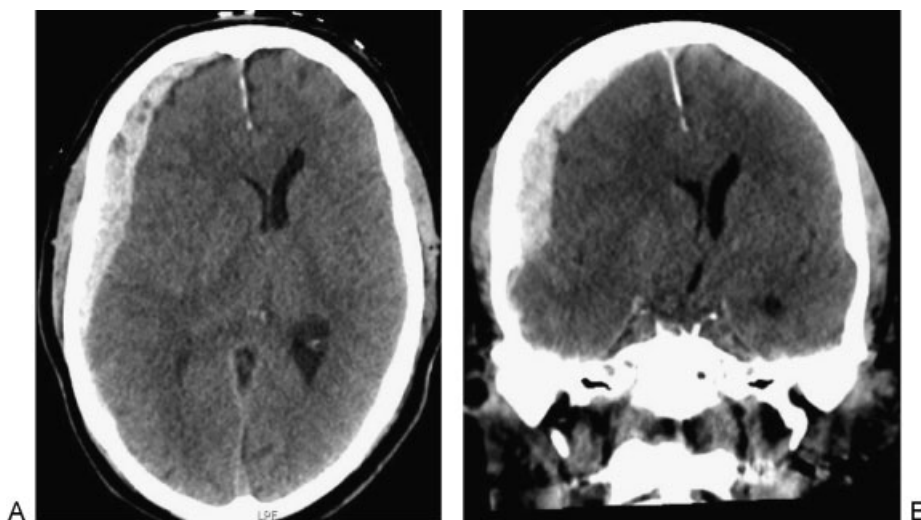
**Fig. 2** Typical frontotemporoparietal epidural hematoma after a fall causing impaired consciousness (Glasgow Coma Scale [GCS] 12) and left-sided hemiparesis (axial [A] and coronal [B] computed tomographic [CT] scan; soft tissue window).

lifesaving measure, it may be insufficient to control the bleeding source.<sup>21</sup> Observation can be considered in patients without significant neurologic deterioration (GCS >8, absent neurologic deficit) and CT findings (hematoma volume <30 cm<sup>3</sup>, maximum thickness <15 mm, and midline shift <5 mm). Conservative management should always include close neurologic monitoring of the patient and follow-up CT scan within 6 to 8 hours.

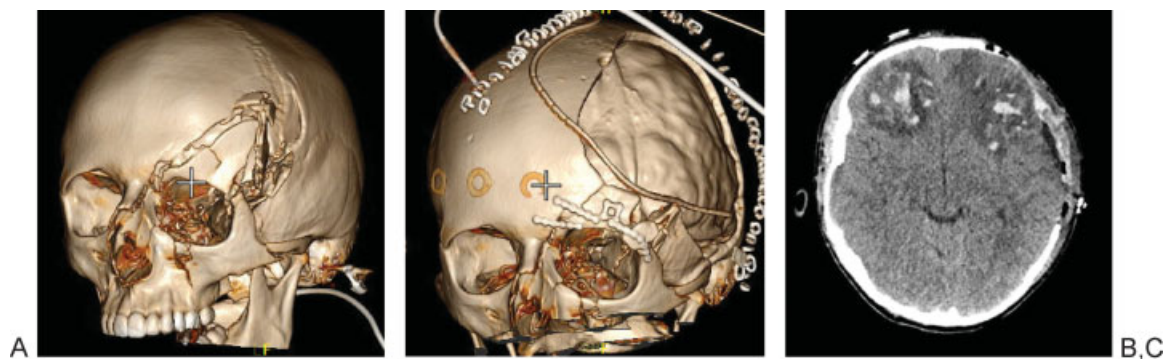
#### Acute Subdural Hematoma

Acute subdural hematomas (aSDHs) commonly arise from blood accumulation around parenchymal lesions as well as disruption of surface or bridging veins. In contrast to EDHs, the impact of damage is usually much higher, associated with further underlying brain injury (e.g., intracerebral hematoma, contusion) and mortality rates up to 60%.<sup>22,23</sup> Symptoms such as impaired consciousness and focal neurologic deficits occur

due to the space-occupying effect with midline shift and possibly brain edema. On CT imaging, aSDHs present as a crescent mass of high density often associated with a midline shift and brain edema (→**Fig. 3**). Surgical intervention is recommended in aSAH with (1) thickness greater than 10 mm or midline shift greater than 5 mm independent of the patient's GCS, and (2) pupillary dilation and/or ICP greater than 20 mm Hg even if the thickness is less than 10 mm and midline shift less than 5 mm.<sup>14</sup> Surgery should be performed in these cases as soon as possible. The subdural space is usually accessed through a large frontotemporoparietal craniotomy (so-called trauma flap) and wide dural incision. The surgical objectives include (1) removal of the hematoma, (2) control of bleeding points on the cortical surface, and (3) watertight closure of the dura. In case of significant brain edema, releasing dural incisions and duraplasty without replacing the bone flap may be performed (decompressive



**Fig. 3** Acute subdural hematoma after a fall (Glasgow Coma Scale [GCS] 8) causing a significant midline shift (axial [A] and coronal [B] computed tomographic [CT] scan; soft tissue window).



**Fig. 4** (A) Combined skull and facial fractures after a high-velocity motor vehicle accident (Glasgow Coma Scale [GCS] 6). (B and C) Postoperative computed tomographic (CT) scan with reconstruction of the supraorbital rim and decompressive craniectomy due to severe bilateral frontobasal contusions causing intracranial hypertension.

hemicraniectomy). Conservative treatment can be considered in patients not meeting the aforementioned criteria for surgical intervention. It is important to note that ICP monitoring should always be performed in comatose and/or sedated patients with aSDH for early detection of secondary neurologic deterioration.

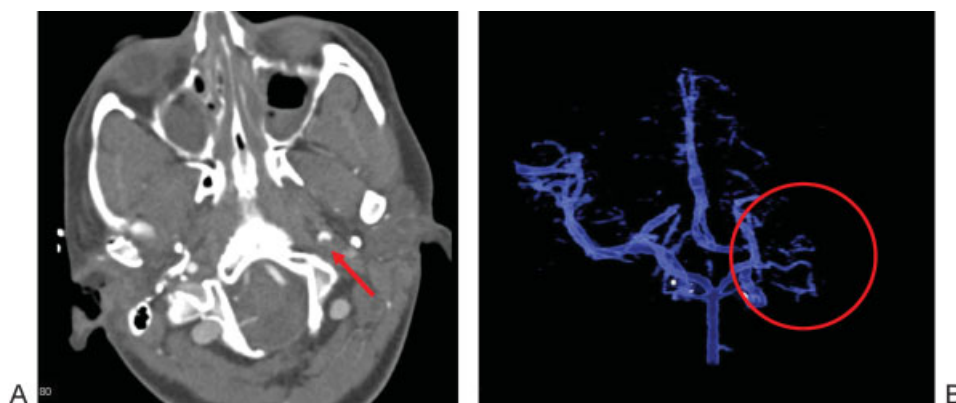
#### Traumatic Parenchymal Lesions

Traumatic parenchymal lesions are classified as focal (intracerebral hematoma, contusions) or diffuse injury (brain edema, diffuse axonal injury). In particular, high-energy trauma is often associated with a combination of both, which often leads to intracranial hypertension (→ Fig. 4). Therefore, ICP monitoring should be considered in all comatose and/or sedated patients with traumatic parenchymal lesions. Surgical intervention should be considered in patients with secondary neurologic deterioration and/or ICP greater than 20 mm Hg.<sup>15</sup> Hematomas and contusions may be evacuated following craniotomy. It has to be noted that only the central bulk of the hematoma is removed to spare potentially vital brain tissue. On the other hand, surgical treatment of diffuse brain injury with intracranial hypertension depends on large bony decompression of the swelling brain. Frontotemporo-parietal or bifrontal decompressive craniectomy can be per-

formed to effectively reduce elevated ICP.<sup>24</sup> The effect of craniectomy on functional outcome in patients with therapy-refractory intracranial hypertension is currently being evaluated in a randomized clinical trial (RESCUEicp trial).

#### Dissection of Cerebral Arteries

Traumatic dissection of the internal carotid artery and/or vertebral artery may lead to cerebral infarction and consequently poor outcome especially in young patients.<sup>25</sup> The most common causes are motor vehicle accidents with severe neck hyperextension and direct blows to the back of the head. Therefore, dissection of cerebral arteries should always be considered in patients with corresponding craniocervical injury. Early CT angiography should be performed to verify or rule out an intramural hematoma caused by transintimal extravasation of blood into the vessel wall.<sup>26</sup> Symptoms may result from occlusion of the true vessel lumen and/or thromboembolism (→ Fig. 5).<sup>27</sup> The therapeutic goal is to prevent delayed ischemic events by starting medical therapy as soon as possible, with heparin acutely, followed by antiplatelet or anticoagulant drugs for 3 months.<sup>28</sup> Arterial cerebral dissection presenting with subarachnoid hemorrhage or refractory to medical therapy may require neurosurgical or endovascular treatment.



**Fig. 5** Computed tomographic (CT) angiography showing intramural hematoma of the left extracranial internal carotid artery (arrow) after a motor vehicle accident (A) leading to thromboembolic occlusion of the middle cerebral artery and consecutive stroke (B).

## Prevention of Secondary Brain Injury

TBI patients should be managed according to a protocol including the *Advanced Trauma Life Support* and *Brain Trauma Foundation Guidelines*.<sup>29,30</sup> The expected benefits include shorter length of hospital stay and improved neurologic outcome.<sup>31,32</sup> After initial resuscitation, diagnostic procedures, and damage control surgery, patients are transferred to an intensive care unit. The main goal of intensive care management is prevention of secondary brain injury by optimizing cerebral perfusion, oxygenation, nutritional supply, and homeostasis.

Intracranial hypertension is a common problem after severe TBI and occurs when the limits of cerebral volume regulation are reached. The Monroe-Kellie doctrine states that the sum of intracranial volumes (brain parenchyma, cerebral blood volume [CBV], cerebrospinal fluid) is constant within the rigid skull, and that an increase in volume of one compartment must be offset by a decrease in volume of another.<sup>33</sup> However, continuing volume expansion due to brain edema or increasing intracerebral hematoma may result in elevated ICP (>20 mm Hg), which should be treated aggressively.

### Multimodal Neuromonitoring

In recent years, multimodal neuromonitoring has gained an important role to provide best medical care tailored to the patient's condition. Monitoring methods include imaging studies (e.g., perfusion CT) as well as minimal-invasive brain tissue probes for bedside monitoring of cerebral hemodynamics and oxygenation (e.g., thermal diffusion probe, brain tissue oxygen tension probe, and microdialysis probe). Bedside neuromonitoring allows for early detection of secondary neurologic deterioration and to guide medical and surgical treatment modalities. Numerous studies have shown that TBI patients may benefit from multimodal neuromonitoring if managed according to standardized treatment protocols.<sup>34–36</sup> However, a thorough understanding of the underlying pathophysiology is mandatory to individualize neurointensive care management.

### Sedation and Analgesia

Sedation is one of the mainstays in early TBI management. The main effect is a decrease in both cerebral metabolic rate and cerebral oxygen consumption. Because of moderate cerebral vasoconstriction, lowering of cerebral blood volume and consequently ICP is achieved.<sup>37,38</sup> On the other hand, sedation bears the risk of affecting hemodynamics, mainly hypotension, and complicates the clinical assessment of the patient. In deep sedation continuous electroencephalographic (EEG) monitoring should be used to assess the level of sedation. Neuromuscular blockade may be used to facilitate mechanical ventilation or minimizing coughing.<sup>39</sup> Most commonly used are benzodiazepines, propofol, and barbiturates. Analgesia is provided by opioids and peripheral acting analgesics.

### Management of Intracranial Pressure and Cerebral Perfusion Pressure

Intracranial pressure (ICP) and cerebral perfusion pressure (CPP) management according to a protocol is mandatory in all

comatose and/or sedated patients with TBI. CPP is calculated by the difference between mean arterial pressure (MAP) and ICP ( $CPP = MAP - ICP$ ). As the volume inside the rigid skull is fixed, space-occupying lesions may lead to raised ICP due to the Monro-Kellie doctrine as mentioned earlier. ICP greater than 20 mm Hg is an independent risk factor of poor outcome and should be treated aggressively.<sup>40,41</sup> Cerebral hypoperfusion is the most important cause of secondary brain injury; therefore, providing adequate CPP is crucial. According to Brain Trauma Foundation Guidelines, a CPP of 50 to 70 mm Hg is recommended.<sup>42</sup> Several treatment strategies exist to control ICP and CPP, including reduction in cerebral metabolism using sedation, induced hyperventilation, hyperosmolar therapy, hypothermia, and surgical approaches. External ventricular drainage is the easiest surgical procedure to reduce intracranial volume. A lumbar puncture is contraindicated due to the risk of brainstem herniation. Decompressive hemicraniectomy as mentioned earlier may serve as an ultima ratio therapy in patients with therapy-refractory high ICP.

### Hyperventilation

Hyperventilation causes cerebral vasoconstriction reducing cerebral blood volume and ICP, but reduction in cerebral blood flow could be detrimental because brain tissue is at risk for ischemia.<sup>43</sup> If hyperventilation is applied, extended neuromonitoring should be installed to measure cerebral hemodynamics and oxygenation<sup>44</sup>.

### Hyperosmolar Therapy

Hyperosmolar therapy with mannitol or hypertonic saline is an important medical treatment option to reduce ICP. Mannitol establishes an osmotic gradient between plasma and brain cells, reducing cerebral edema by drawing water into the vascular system. Hypertonic saline produces a reduction in cerebral edema by moving water out of the brain and thus resulting in decreased ICP.<sup>45</sup>

### Mild Hypothermia

Mild hypothermia (33–35°C) has shown a reduction in ICP and improved outcome in patients with TBI.<sup>46</sup> There is evidence that proactive management of the side effects of hypothermia (e.g., coagulopathy, electrolyte disorders, sepsis) leads to improved outcome.<sup>47</sup> Hypothermia is part of many algorithms targeting patients with uncontrollable intracranial hypertension, although it is still considered experimental. An ongoing study may answer the remaining questions on hypothermia in TBI (Eurotherm3235 Trial).

### Respiratory Care

Patients with severe head injury require a secure airway and mechanical ventilation to maintain arterial oxygen tension greater than 13 kPa and arterial carbon dioxide tension between 4.5 and 5.0 kPa. Hypocapnia should be avoided due to cerebral vasoconstriction and its detrimental effect on CBF. Hypercapnia may raise ICP due to cerebral vasodilation<sup>37</sup>.

### Hemodynamics

Intravascular volume should be maintained using isotonic crystalloids and colloids targeting euolemia and hemoglobin levels between 70 and 100 g/L. Vasoactive drugs are frequently needed to maintain adequate MAP and CPP. Hypotension causes a reduction in cerebral blood flow and may result in cerebral hypoperfusion and ischemia.<sup>48</sup> Hypertension may exacerbate vasogenic edema with increased ICP.<sup>49</sup>

### Nutritional Support and Glycemic Control

Early nutritional support is recommended reducing gastric stress ulcers, enhancing immunologic function, and minimizing the risk of hypoglycemia.<sup>50</sup> Laxatives and prokinetics should be used for stool regulation and reducing intraabdominal pressure. Maintaining glucose levels between 5 and 10 mmol/L is adequate. Hyperglycemia is associated with worse neurologic outcome, whereas hypoglycemia may have even worse effects.<sup>51</sup>

### Coagulation, Deep Venous Thrombosis Prophylaxis

Aggressive management of coagulation disorders is mandatory in the initial resuscitation phase, aiming for normalization of coagulation parameters. Thrombosis prophylaxis with unfractionated heparin or low-molecular-weight heparin may be started in the following 48 to 72 hours after trauma.<sup>52</sup> Up to this point, mechanical devices should be installed such as compression stockings, sequential compression devices, or vena cava filters due to other diseases.

### Conclusion

Optimal treatment of patients with severe TBI requires integrated neurosurgical and intensive care management. Standardized treatment protocols including clear indications for surgical intervention and multimodal neuromonitoring may help manage the individual patient and improve outcome. However, a thorough knowledge of the pathophysiology underlying brain injury is required, in particular because of increasing complexity of management strategies.

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