Head Injuries

14

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Learning Objectives

- Describe the pathophysiology of head trauma and the distinction between primary and secondary brain injuries.
- Explain the impact of decreased cerebral perfusion pressure in contributing to adverse outcomes.
- Identify serum biomarkers for the diagnostic work-up and outcome prediction in traumatic brain injury.
- Understand the risk of hypoxia and hypotension in contributing to secondary brain injury.
- Establish the concepts of appropriate resuscitation for limiting the risk of secondary brain injury.
- Define the optimal timing and modality of long bone fracture fixation in polytrauma patients with associated head injuries.
- Recognize patients who require early neurosurgical consultation, hospital admission, or transfer to a higher level of care.

14.1 Introduction

Traumatic brain injury (TBI) represents the leading cause of death and long-term neurological impairment in mainly young trauma patients worldwide [1–4]. Technological innovation in recent years, with the introduction of neuroproteomics and a new generation of laboratory testing modalities, has improved the diagnostic work-up for TBI patients [5]. However, despite advances in diagnostic imaging, neurointensive care modalities, and the quality of neurosurgical care, the clinical outcome of patients with severe TBI remains poor. Of critical importance, there is currently no specific pharmacological therapy available for the treatment or prevention of secondary cerebral insults [6, 7]. The extent of posttraumatic brain damage is determined by a combination of the *primary* trauma, resulting from mechanical forces applied to skull at the time of impact, and the subsequent evolution of

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H.-C. Pape et al. (eds.), *Textbook of Polytrauma Management*,
https://doi.org/10.1007/978-3-030-95906-7_14

secondary brain injuries [8]. The primary trauma is characterized by either focal or diffuse brain injury patterns, whereas secondary brain injuries evolve over time and are characterized by a complex cascade of molecular and biochemical events leading to neuroinflammation, brain edema, and delayed neuronal death [9]. The immune-pathophysiological sequelae of TBI are highly complex and involve numerous brain-derived proinflammatory mediators, such as cytokines, chemokines, complement anaphylatoxins, excitatory molecules, electrolyte disturbances, and blood-derived leukocytes that migrate across the blood–brain barrier (BBB) [10–12]. These events culminate in the breakdown of the BBB and allow leakage of circulating neurotoxic molecules from the peripheral blood stream into the subarachnoid space of the injured brain, which is otherwise protected from the systemic circulation under physiologic conditions. The resulting proinflammatory environment in the injured brain promotes the development of brain edema and brain tissue destruction by leukocyte-released proteases, lipases, reactive oxygen species, and terminal complement activation proteins (Fig. 14.1). Ultimately, the extent of secondary brain injury, characterized by neuroinflammation, ischemia/reperfusion injuries, cerebral edema, intracranial hemorrhage, and intracranial hypertension, represents one of the key determinants of poor outcomes after severe TBI. Iatrogenic factors, such as permissive hypotension, prophylactic hyperventilation, overzealous volume resuscitation, and the inconsiderate timing and extent of surgical procedures for associated injuries further contribute to preventable secondary cerebral insults [13]. Early hypoxia and hypotension are significant contributors to the evolution of secondary brain injury and must be prevented during the early resuscitation of polytrauma patients with associated head injuries [14]. In light of the complex underlying pathophysiology and the inherent vulnerability of the injured brain to "second hit" insults, it is imperative for the trauma team to closely coordinate the timing of the surgical priorities for the management of associated injuries in head-injured patients.

14.2 The Quest for a Serum Biomarker

At present, there is a lack of reliable serum biomarkers for routine use in the diagnostic work-up and outcome prediction for TBI patients. A mul-

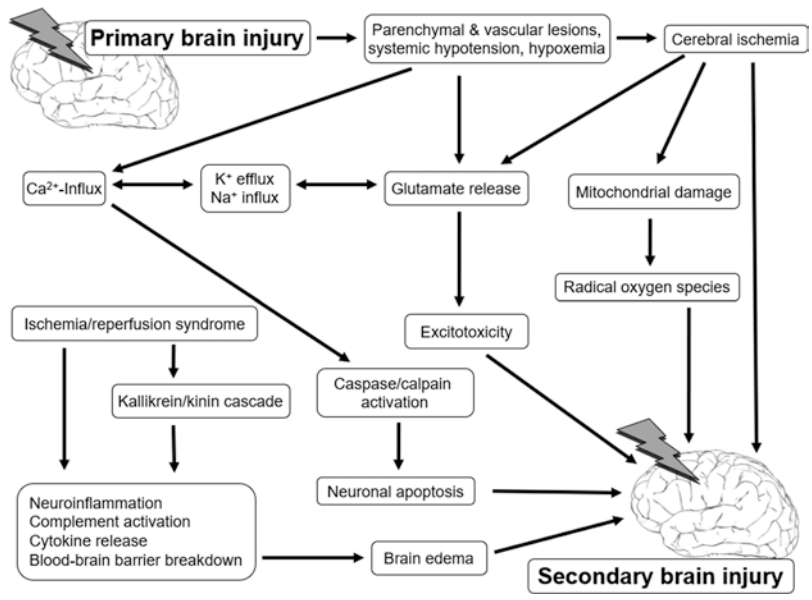


Fig. 14.1 Pathophysiology of primary and secondary brain injury. (See text for details)

tiplicity of potential candidate molecules has been identified in recent years [15]. Early research from the 1980s defined the properties of an "ideal" TBI biomarker as such: (a) must be highly specific for brain tissue; (b) must be released from the brain only after relevant tissue damage occurred; (c) must appear in cerebrospinal fluid (CSF) and serum rapidly after TBI and mirror the time course of injury; (d) must reflect the severity of neurological injury; and (e) must be of clinical relevance [16]. The currently established and novel speculative biomarkers originating from injured neurons, axons, or glial cells are depicted in Fig. 14.2. There is ongoing debate on the potential advantages of CSF versus peripheral blood samples. Some authors argue that biomarkers in CSF are preferred over serum, due to the close proximity of the intrathecal fluid to the injured brain, independent of the BBB integrity. In contrast, serum biomarkers are more practicable for routine sampling due to access to peripheral blood samples. Confounding variables that alter biomarker serum levels include associated extracranial injuries and presence of traumatic-hemorrhagic shock. This aspect is of particular importance considering that most head-injured patients do not undergo routine CSF sampling, and only selected patients with severe TBI are candidates for CSF drainage through indwelling intraventricular catheters. The currently most widely used serum biomarkers for TBI include neuron-specific enolase (NSE), S100 calcium-binding protein B (S100B), and glial fibrillary acidic protein (GFAP) [16]. NSE is a glycolytic enzyme which catalyzes the conversion of 2-phosphoglycerate to phosphoenol pyruvate during glucose metabolism which is present in high concentrations in neurons and neuroendocrine cells. The release of NSE into serum has been considered a sensitive surrogate of the severity of brain damage. A serum NSE level <10 ng/mL is considered within the normal range. Elevated serum NSE levels correlate with intracerebral CT pathology and are predictive of

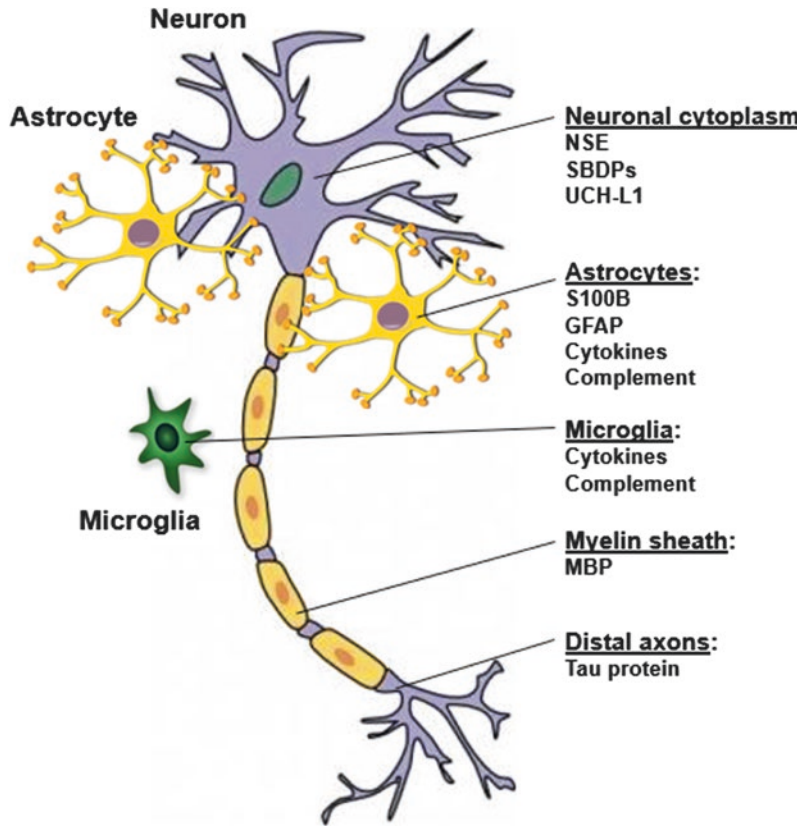


Fig. 14.2 Cellular source of serum biomarkers for traumatic brain injury
Abbreviations: *GFAP* Glial fibrillary acidic protein, *MBP* Myelin basic protein, *NFL* Neurofilament, *NSE* Neuron-specific enolase, *SBDPs* Spectrin breakdown products, *UCH-L1* Ubiquitin carboxy-terminal hydrolase-L1

poor outcomes after TBI. In patients with DAI, a cut-off at NSE levels >50 ng/mL revealed 100% sensitivity and 100% specificity for predicting post-injury mortality [17]. In contrast, NSE has a low sensitivity for differentiating patients with mild TBI from healthy controls. Recent studies detected extracranial sources of NSE in polytrauma patients, including hemorrhagic shock, long bone fractures, and ischemia/reperfusion injuries, which limits the value of this serum biomarker for isolated head injury.

The astroglia-derived S100B is one of the most promising serum markers for TBI, being released rapidly after trauma with a short half-life of less than 60 min. Significantly elevated S100B levels have been described in the serum of head-injured patients and have been shown to correlate with the severity of TBI and predictive of adverse outcomes. In patients with TBI (GCS ≤8), elevated serum S100B levels greater than 1.0 ng/mL are predictive of secondary brain injury and postinjury mortality after TBI. However, in spite of a high sensitivity, the low specificity represents a shortcoming of S100B as the preferred biomarker, since the protein is not just expressed by astroglial cells but has also been detected in adipocytes, chondrocytes, melanoma cells, and hematopoietic cells. Some studies therefore suggested that elevated S100B levels after TBI are reflective of BBB damage, resulting in protein leakage from the periphery into the intrathecal compartment, rather than reflecting the extent of direct neuronal injury. Finally, GFAP represents a "classic" astrocyte-specific cell marker, and its exclusive brain-specific expression and release renders the protein a very intuitive biomarker for TBI above other candidate molecules, such as S100B or NSE. Clinical studies confirmed this notion by demonstrating significantly elevated GFAP serum levels around 7 pg/mL in TBI patients on the day of admission, compared to tenfold lower levels at 0.7 pg/mL in control subjects without head injuries. Future validation studies in large-scale longitudinal multicenter trials will have to confirm the notion of GFAP as a potential "silver bullet" among the available serum biomarkers to monitor the course of treatment and predict outcomes in TBI patients.

14.3 Hypoxia and Hypotension: The "Lethal Duo"

Episodes of hypoxia and hypotension represent the main independent predictive factors for poor outcomes after severe TBI [14]. In a landmark article published in 1993, Chesnut and colleagues analyzed the impact of hypotension, as defined as a systolic blood pressure (SBP) <90 mmHg, either during the resuscitation phase ("early") or in the ICU ("late"), on the outcome of head-injured patients prospectively entered into the Traumatic Coma Data Bank (TCDB) [18]. Early hypotension occurred in 248 of 717 patients (34.6%) and was associated with a doubling of post-injury mortality from 27% to 55%. Late hypotension occurred in 156 of 493 patients (31.6%), of which 39 patients (7.9%) had combined early and late hypotensive episodes. For 117 patients with an exclusive hypotensive episode occurred in the ICU, 66% either died or survived in a vegetative state, as defined by a Glasgow Outcome Scale (GOS) score of 1 or 2 points. The authors furthermore determined that mortality is drastically increased in combination with hypotension (SBP <90 mmHg) and hypoxia (PaO_2 ≤60 mmHg) [18–20]. A different study confirmed the notion that severe secondary insults occur during the neurointensive care period in more than 35% of all head-injured patients, including episodes of hypoxia, hypotension, elevated intracranial pressure (ICP), and decreased cerebral perfusion pressure (CPP) [21].

National guidelines by the *Brain Trauma Foundation* recommend that blood pressure and oxygenation be monitored in all head-injured patients, and advocate to maintain a systolic blood pressure >90 mmHg and a PaO_2 >60 mmHg, respectively [22]. This notion is of particular importance in view of the ongoing debate on the controversial concept of "permissive hypotension" in patients with traumatic hemorrhage from penetrating or blunt torso injuries [23, 24]. The strategy of "permissive hypotension" is mainly based on a landmark article from the 1990's advocating a modified prehospital resuscitation concept for hypotensive patients with penetrating torso injuries, by delay-

ing fluid resuscitation until arrival in the operating room [25]. This proactive concept is certainly intuitive from the perspective that traditional resuscitation with aggressive fluid administration may lead to increased hydrostatic pressure and displacement of blood clots, a dilution of coagulation factors, and an undesirable hypothermia in critically injured patients [26]. However, in light of the vulnerability of the injured brain to secondary insults mediated by hypoxia and hypotension during the early post-injury period, the concept of hypotensive resuscitation, which has seen an unjustified expansion from penetrating to blunt trauma, in absence of high-level evidence, appears contraindicated for patients with traumatic brain injuries [14].

14.4 Classification of Head Injuries

Traumatic brain injuries are classified by *severity*, using the Glasgow Coma Scale (GCS), or by *morphology*, using non-contrast computerized tomography (CT) imaging [27].

14.4.1 Severity of Injury (GCS)

The patient's level of consciousness is rapidly evaluated by the GCS score (Table 14.1), which grades the severity of TBI as mild (GCS 13–15), moderate (GCS 9–12), and severe (GCS 3–8). Of the three underlying criteria (eye opening, verbal response, motor response), the *best* motor response represents the most reliable predictor of outcomes. Of note, the GCS must be reevaluated and documented in regular time intervals to detect a deterioration in GCS over time ("patients who talk and die"). Importantly, the post-resuscitation GCS score is needed to grade the severity of TBI due to the confounding influence of cerebral hypoperfusion in patients with traumatic-hemorrhagic shock on the level of consciousness. The "classic" GCS grading scale from the 1970s was recently modified (a) to be more easily applicable; (b) to avoid inflicting pain for testing; and (c) to account for non-testable parameters ("NT," see revised GCS scale in Table 14.1) [27].

Table 14.1 Glasgow coma scale

Original scale	Revised scale	GCS score
Eye opening (E)[a]		
Spontaneous	Spontaneous	4
To speech	To sound	3
To pain	To pressure	2
None	None	1
	Non-testable	NT
Verbal response (V)[a]		
Oriented	Oriented	5
Confused conversation	Confused	4
Inappropriate words	Words	3
Incomprehensible sounds	Sounds	2
None	None	1
	Non-testable	NT
Best motor response (M)[a]		
Obeys commands	Obeys commands	6
Localizes pain	Localizing	5
Flexion withdrawal to pain	Normal flexion	4
Abnormal flexion (decorticate)	Abnormal flexion	3
Extension (decerebrate)	Extension	2
None (flaccid)	None	1
	Non-testable	NT

Best possible score: 15. Worst possible score: 3
[a]The GCS score is calculated as E + V + M

14.4.2 Morphology of Injury (CT)

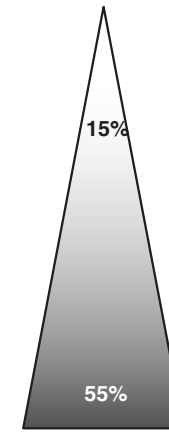
The morphological classification of TBI is determined by a non-contrast CT scan of the head. This includes the descriptive assessment for presence of skull fractures, intracranial hematomas, diffuse axonal injury, cerebral contusions, as well the Marshall classification of axial CT scans which stratifies intracranial lesions as either focal, evacuated vs. non-evacuated hematomas, or as grade I-IV diffuse injuries (Table 14.2) [28].

14.4.2.1 Skull Fractures

Skull fractures in younger patients are generally a sign of high-energy trauma. Fractures occur either in the cranial vault or in the base of the skull. The presence of a linear vault fracture alone is associated with a 400-fold increased risk

Table 14.2 Marshall classification of head CT scan

CT classification	Definition	Mortality
Diffuse Injury (DI) I	Normal CT scan (clinical diagnosis)	
Diffuse Injury (DI) II	Open basal cisterns, midline shift 0-5 mm, high- or mixed-density lesions < 25 cc.	
Diffuse Injury (DI) III	Compressed or absent basal cisterns, midline shift 0-5 mm, high- or mixed- < 25 cc	
Diffuse Injury (DI) IV	Absent basal cisterns, midline shift > 5 mm, high- or mixed-density lesions < 25 cc	
Evacuated mass lesion (EML)	Any surgically evacuated intracranial lesions	
Non-evacuated mass lesion (NEML)	High- or mixed-density lesion > 25 cc, not surgically evacuated	



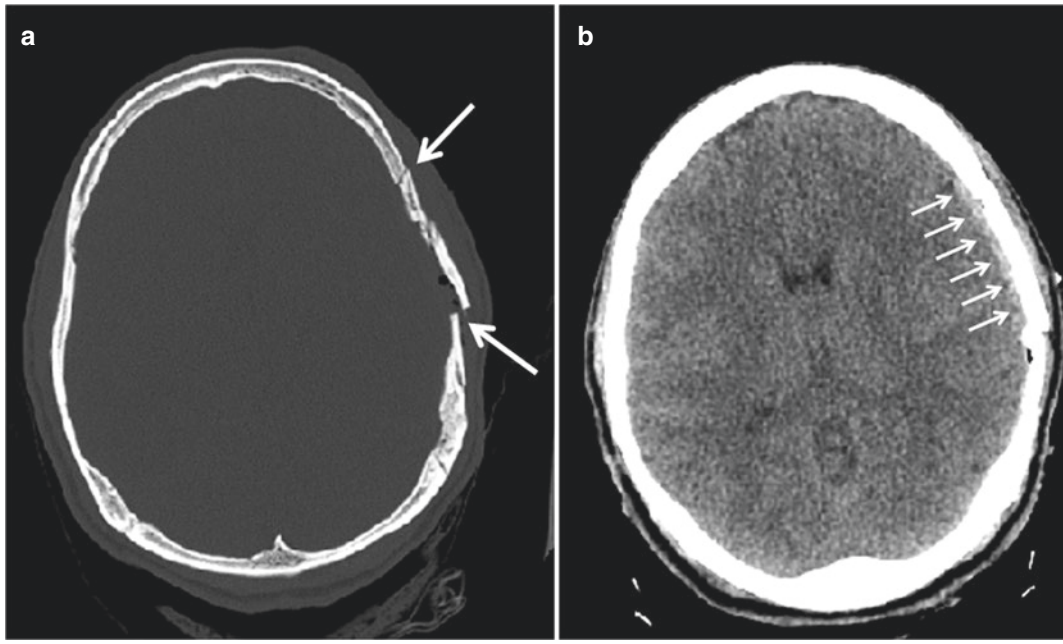


Fig. 14.3 Skull fracture with associated subdural hematoma

Case example of a 40-year-old male patient who was assaulted with a baseball bat and sustained a segmental comminuted fracture of the left temporal and parietal bone (arrows in panel **a**). The small air bubbles under the skull fracture indicate presence of an open fracture (panel **a**). The non-contrast axial CT scan image demonstrates an underlying subdural hematoma (arrows in panel **b**)

of an intracranial hematoma (Fig. 14.3). Clinical signs of basilar skull fractures include leakage of cerebrospinal fluid (CSF) from the ear (otorrhea) or nose (rhinorrhea), periorbital ecchymosis ("racoon eyes"), and retroauriocular ecchymosis (Battle's sign). Rarely, skull fractures may be associated with neurological dysfunction of the cranial nerves VII (facial paralysis) and VIII (hearing loss), or with a carotid artery dissection. Open skull fractures expose the cerebral surface

and are associated with a high risk of infections (meningitis, meningoencephalitis).

14.4.2.2 Intracranial Lesions

Intracranial lesions are stratified as diffuse or focal injuries. Diffuse brain injuries frequently present normal on the initial CT scan. Mild diffuse brain injury is a simple concussion, whereas *diffuse axonal injury* (DAI) represents a neuronal "shearing injury" from high-energy acceleration-deceleration mechanisms and is associated with a dismal prognosis (Fig. 14.4). Focal lesions include epidural, subdural, and intracerebral hematomas, as well as cerebral contusions. *Epidural hematomas* are uncommon but dangerous due to being underestimated (so-called lucid interval) with the potential for rapid deterioration and adverse patient outcomes. These hematomas present in biconvex or lenticular shape on axial CT scan and typically result from a laceration to the middle meningeal artery (Fig. 14.5). In contrast, subdural hematomas are typically of venous origin due to shearing injuries to bridging veins of the cerebral cortex. *Subdural hematomas* are more common and present in about 30% of all patients with severe head injuries. In contrast to the lenticular shape of epidural bleeding, the shape of subdural hematomas conforms to the contour of the brain (Fig. 14.6). Subdural hematomas are surrogate markers of severe underlying parenchymal brain injury. *Cerebral contusions, subarachnoid hemorrhage, and intracerebral hematomas* are very common injury patterns in patients with severe TBI. Most contusions are found in the frontal and temporal lobes (Fig. 14.7). Contusions can evolve over time and form intracerebral hematomas with perifocal neuroinflammation and cerebral edema which may occasionally lead to a mass effect requiring surgical decompression.

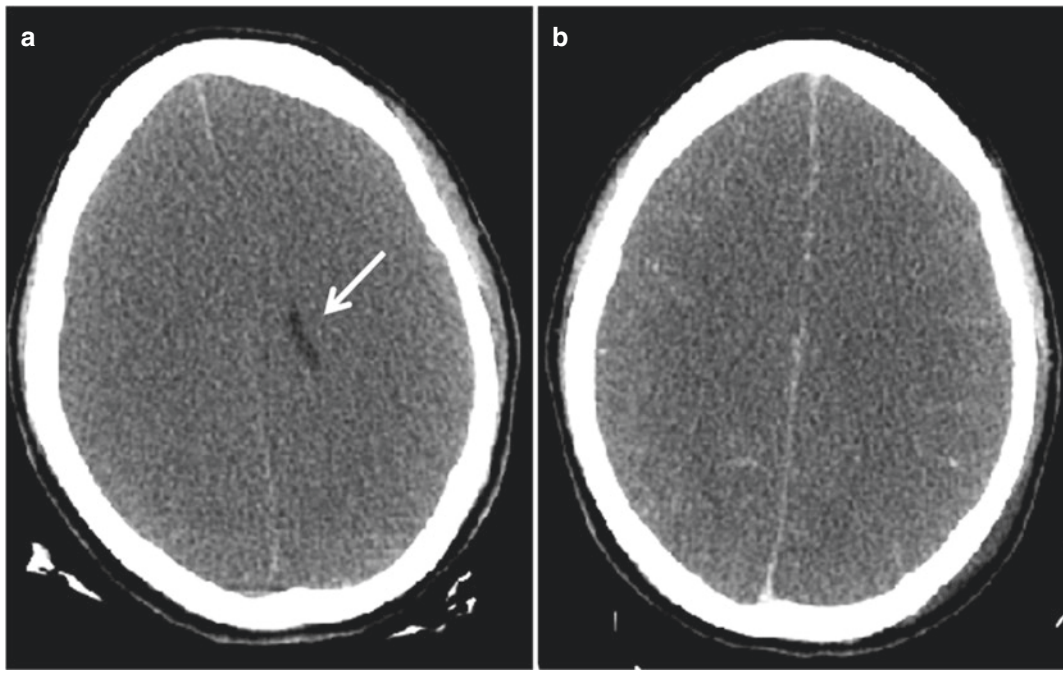


Fig. 14.4 Diffuse brain injury

Case example of a 43-year-old male patient who sustained a high-speed multivehicle deceleration injury. He was comatose with a GCS of 3 and intubated at the scene. The initial non-contrast CT scan demonstrates diffuse brain swelling with effacement of the intracranial subarachnoid spaces (panel **a**). The arrow in panel A demonstrates the residual lateral ventricle in the left hemisphere. A repeat non-contrast CT scan obtained on day 3 reveals small punctuate subarachnoid hemorrhage (panel **b**). In spite of maximal intensive care therapy, this unfortunate patient died from uncontrolled cerebral edema with tonsillar herniation

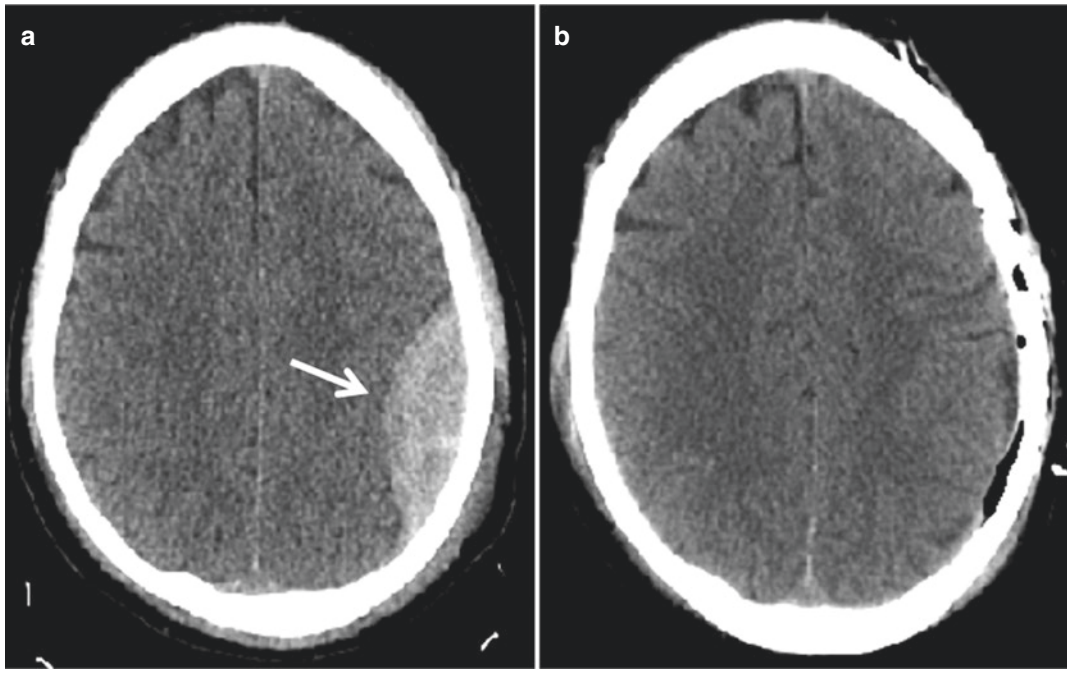


Fig. 14.5 Epidural hematoma

Case example of a 33-year-old male who sustained a fall on his head while intoxicated. He was brought to the ED with a GCS of 14. The non-contrast CT scan of the head demonstrates a large lenticular shape epidural hematoma along the left frontoparietal convexity, measuring 20 mm in thickness (panel **a**). The patient was taken to the operating room for a craniotomy with hematoma evacuation. The postoperative CT scan demonstrates absence of the hematoma and post-craniotomy intracranial air (panel **b**)

14.5 Initial Assessment and Management

The standardized approach to the initial assessment of multiply injured patients with associated TBI is performed by the ATLS® protocol, as described elsewhere in this textbook (see Chap. 6) [27]. The main goal in the early management of head-injured patients is the prevention and restoration of variables which contribute to the evolution of secondary brain injury, including hypoxemia, hypotension, hypercarbia, and hypoglycemia. Patients with severe TBI (GCS ≤8) require immediate endotracheal intubation for airway protection and adequate ventilation and oxygenation. The fluid replacement therapy of choice in TBI patients consists of isotonic electrolyte solutions, such as Ringer's lactate. Urinary output helps guide the patients' response to resuscitation, aimed at >0.5 mL/kg/h in adults and 1–2 mL/kg/h in pediatric patients. A pitfall in TBI patients with increased urinary output may related to a postinjury complication termed "syndrome of inappropriate ADH secretion" (SIADH). Neurologic evaluation of TBI patients occurs after the stabilization of vital systems, per ATLS® guidelines. A head CT should be obtained under the following circumstances: (1) altered level of consciousness with GCS <14; (2) abnormal neurological status; (3) differences in pupil size or reactivity; (4) suspected skull fracture; (5) intoxicated patients. Furthermore, the head CT must be repeated whenever a patient's neurologic status deteriorates. Significant associated injuries, such as blunt chest trauma, intraabdominal hemorrhage, pelvic ring disruptions, and long bone fractures must receive adequate attention due to the potential of contributing to the development of secondary brain damage. Due to the interrelation between cerebral perfusion pressure (CPP) with intracranial pressure (ICP) and mean arterial pressure (MAP), an increased systemic blood pressure should not be therapeutically low-

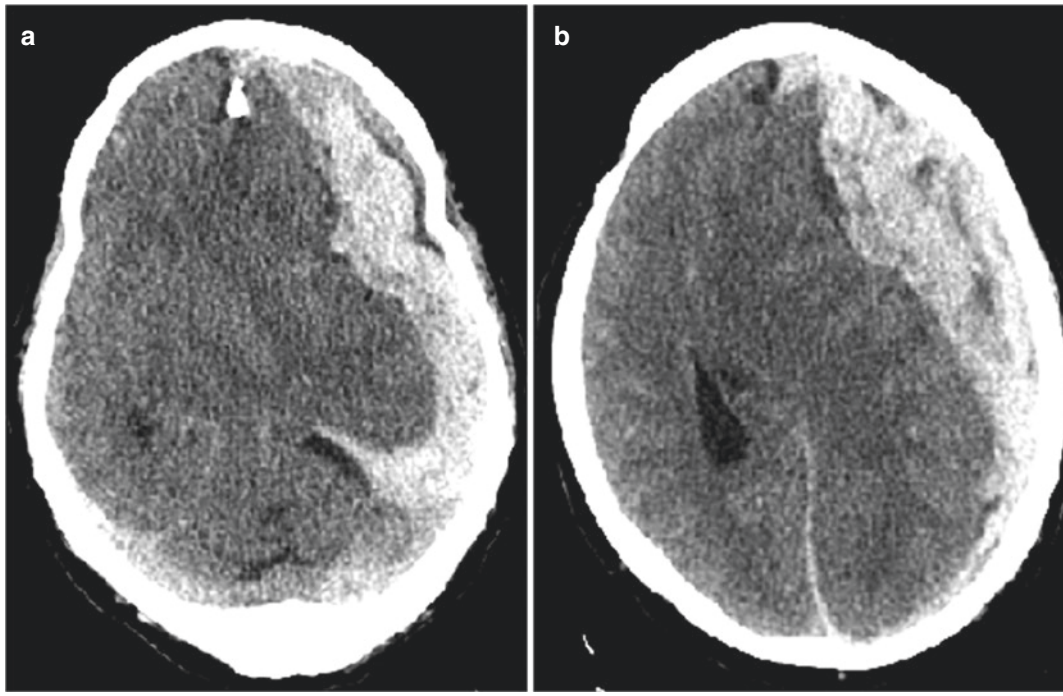


Fig. 14.6 Subdural hematoma

Case example of a 65-year-old male patient on long-term anticoagulation for atrial fibrillation who sustained a low-energy fall on his head. The patient was brought to the ED with a GCS of 12. The non-contrast CT scan demonstrated a large subdural hematoma of 40 mm diameter over the left cerebral hemisphere (panel **a**). There is a significant intracranial mass effect with rightward midline shift, effacement of the left lateral ventricle (panel **b**), and subfalcine herniation. The patient was taken to the operating room emergently for a decompressive craniectomy (not shown)

ered in head-injured patients, as this may be reflective of "demand hypertension" to retain adequate cerebral blood flow (CPP = MAP-ICP). Similarly, permissive hypotension should not be advocated in polytrauma patients with associated TBI due to the potential detrimental impact on preventable secondary cerebral insults. According to the "Monro-Kellie doctrine," the total intracranial volume remains constant, implying that expanding mass lesions will result in a reduced CPP. Therefore, CPP maintenance above 70–80 mmHg may have to be therapeutically achieved by surgical evacuation of intracranial mass lesions and by attenuation of brain swelling by the use of osmotic drugs (e.g., mannitol) and therapeutic CSF drainage through intraventricular catheters (Fig. 14.8). Since elevated ICP values >20 mmHg are associated adverse outcomes, the indwelling ICP monitoring is generally recommended under the following conditions:

1. S*evere* TBI (GCS ≤8) with abnormal admission CT scan;
2. S*evere* TBI (GCS ≤8) with normal CT scan, but prolonged coma >6 h;
3. Postoperative monitoring after surgical hematoma evacuation;
4. Neurological deterioration (GCS ≤8) in patients with initially mild or moderate extent of TBI;
5. Head-injured patients requiring prolonged mechanical ventilation, e.g., for management of associated extracranial injuries, unless the initial CT scan is normal.

The ICU management of polytrauma patients with associated head injuries primarily focuses on preventing secondary brain injury by maintaining adequate oxygen delivery and hemodynamic stability. This includes the prevention of hypoxemia, hypercarbia, and hypotension, with a

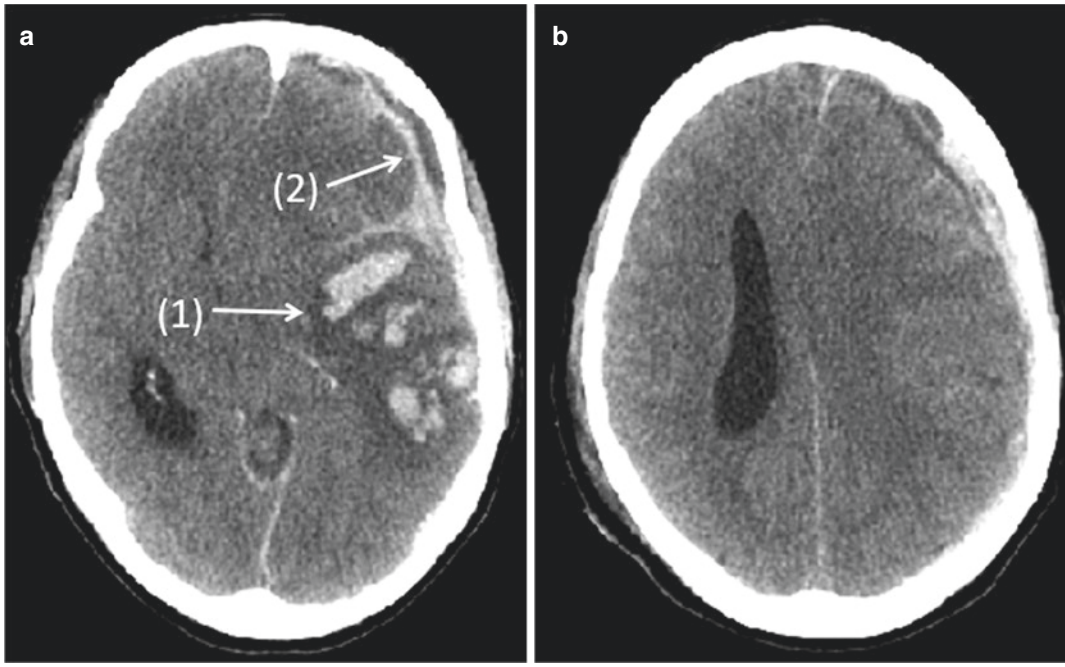


Fig. 14.7 Intracerebral hemorrhage
Case example of a 54-year-old patient who sustained a fall from a height while rock climbing. The patient sustained multifocal intracranial injuries, including a large intraparenchymal contusion hemorrhage in the left temporal lobe (1), an acute left frontoparietal subdural hematoma (2), and multiple scattered areas of punctuate subarachnoid hemorrhage (panel **a**). The intracerebral contusion hemorrhage led to perifocal brain swelling with left hemispheric edema, complete effacement of the left lateral ventricle, and a significant rightward midline shift (panel **b**)

goal of PaO_2 >13 kPa, $PaCO_2$ between 3.3 and 4.5 kPa, and MAP ≥80 mmHg. The historic concept of "blind" prophylactic hyperventilation has been largely abandoned due to inherent the risk of inducing focal ischemic insults. Further aspects of the baseline intensive care for TBI patients include the prevention of hyperthermia, hyperglycemia, and hyponatremia.

For further guidance, the most recent fourth edition of the "Guidelines for the Management of Severe Traumatic Brain Injury" by the Brain Trauma Foundation was published in September 2016, and represents the current evidence-based standard of care [22].

14.6 Pharmacological Therapy

Mannitol is used to reduce elevated ICP levels and is typically administered as a 20% bolus (20 g mannitol in 100 mL). Mannitol augments the intravascular volume, increases cerebral blood flow, and diminishes intracranial volume. Classic indications for the application of mannitol include clinical signs of transtentorial herniation (i.e., loss of consciousness, decerebrate rigidity, ipsilateral pupil dilatation, contralateral hemiparesis), or progressive neurological deterioration with decreasing GCS scores. When administering mannitol, serum osmolarity should be kept <315 mOsm/L and hypovolemia must be avoided by adequate fluid replacement. Systemic hypotension is considered a contraindication for mannitol due to the risk of increasing cerebral ischemia. Hypertonic saline in concentrations of 3% or higher represents another therapeutic modality of reducing elevated ICP, and may be a preferrable option in hypotensive trauma patients [29]. High-dose glucocorticoids have been widely used as an empirical treatment of brain edema in TBI patients from the 1960s until 1990s. However, the large-scale prospective randomized, placebo-controlled

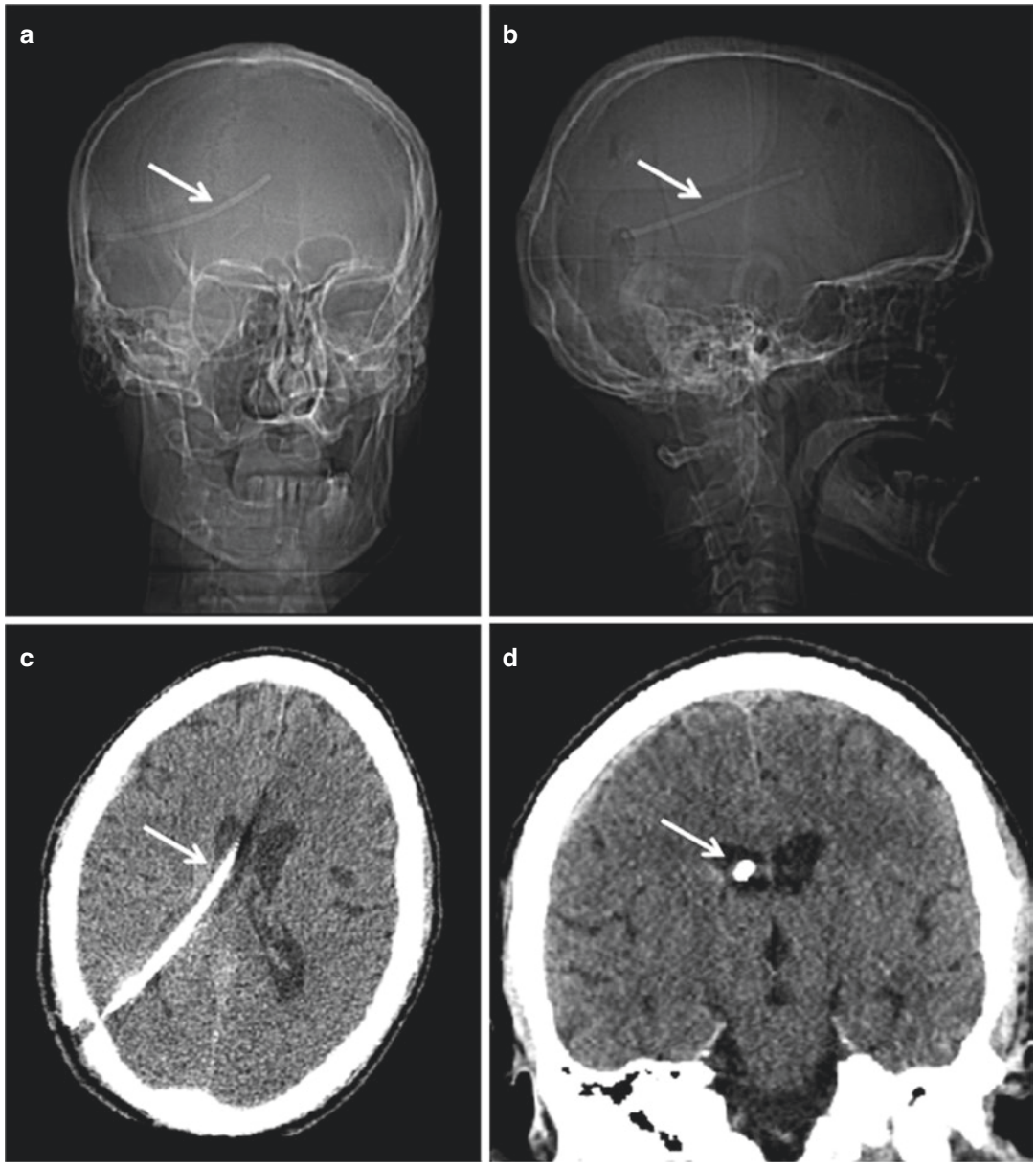


Fig. 14.8 Intraventricular drain

Case example of a 47-year-old head-injured patient with placement of an intraventricular drain which allows intracranial pressure (ICP) monitoring and therapeutic cerebrospinal fluid drainage for managing increased ICP levels. The arrows demonstrate the intraventricular drain on the CT scout images (panels **a** and **b**) and on the axial (panel **c**) and coronal (panel **d**) non-contrast CT images

multicenter "CRASH" trial (**C**orticosteroid **ra**n-domization **a**fter **s**ignificant **h**ead injury) on 10,008 TBI patients revealed a significantly increased mortality in the steroid group compared to the placebo control group during the first 14 days after trauma (21.1% vs. 17.9%, $P < 0.001$) [30, 31]. Thus, corticosteroids are currently considered obsolete and contraindicated in the pharmacological management of TBI patients. Barbiturates are effective in reducing ICP, however, their use is restricted for intensive care therapy with continuous EEG monitoring [27].

14.7 Surgical Management

Surgical indications in patients with TBI include scalp wounds, depressed skull fractures, intracranial mass lesions, and penetrating brain injuries. While scalp wounds can be addressed by a general trauma surgeon, any intracranial lesion is managed by the consulting neurosurgeon.

14.7.1 Scalp Wounds

Scalp wounds represent an underestimated source of significant blood loss that can lead to traumatic-hemorrhagic shock. Early control of scalp hemorrhage is therefore imperative by applying direct pressure and bandages. Smaller size scalp wounds can be closed with sutures at the bedside in the emergency room, whereas larger bleeding wounds are best managed in the operating room, which may include ligating or cauterizing larger vessels, if needed. The wounds must be inspected for foreign bodies or depressed skull fractures prior to closure. Presence of CSF implies an open brain injury with a dural tear.

14.7.2 Depressed Skull Fractures

Depressed skull fractures should be managed surgically by elevation if the depth of depression is larger than the adjacent skull on CT scan, or if the associated intracranial hematoma requires surgical evacuation (see below).

14.7.3 Intracranial Mass Lesions

The specific types of intracranial hematomas associated with severe TBI are depicted in Figs. 14.3, 14.5, 14.6, 14.7. Around one-third of patients with *severe* TBI require emergent surgery for evacuation of mass lesions, most commonly acute subdural hematomas. Of note, even minor lesions in the temporal or posterior fossa may cause compression to the brainstem and obstruction of CSF flow, which place an indication for early surgical intervention. In patients with *mild* or *moderate* brain injuries, a craniotomy is performed for "stable" hematomas on a less urgent basis.

The timing of surgery depends on the clinical condition of the patient, based on the GCS, neurological exam, and CT findings. Surgical hematoma evacuation is typically performed by a craniotomy, whereby the bone flap is replaced by conclusion of surgery. If there is a significant mass effect or swelling of the injured cerebral hemisphere, a decompressive craniectomy is performed, with the bone flap being left off. This helps alleviate elevated intracranial pressure and prevent recurrence of intracranial hematomas. An example of a decompressive craniectomy is shown in Fig. 14.9.

Indications for surgical hematoma evacuation include the following conditions:

- Any type of expanding intracranial hematoma associated with clinical neurological deterioration (GCS) or with >5 mm midline shift (CT) should be evacuated as soon as possible.
- Acute subdural hematoma (SDH) >10 mm thickness ("rule of thumb": larger than the adjacent skull on axial CT scan).
- Acute subdural hematoma (SDH) <10 mm thickness in comatose patients (GCS ≤8) with severe parenchymal injuries and mass effect.
- Acute epidural hematoma (EDH) represents a classic indication for surgical evacuation. Rare exceptions for non-surgical management include patients who are fully awake and alert (GCS 15) with small EDH on initial CT scan. In these selected cases, close clinical monitoring and documentation of clinical findings, in conjunction with short-interval repeat CT scanning, are mandatory to detect worsening hematomas and clinical deterioration, which is associated with dismal patient outcomes.
- Intracranial mass lesions with mass effect and midline shift on CT scan, as well as open or penetrating brain injuries are typically indicated for surgical management by craniotomy/craniectomy with surgical decompression and debridement.

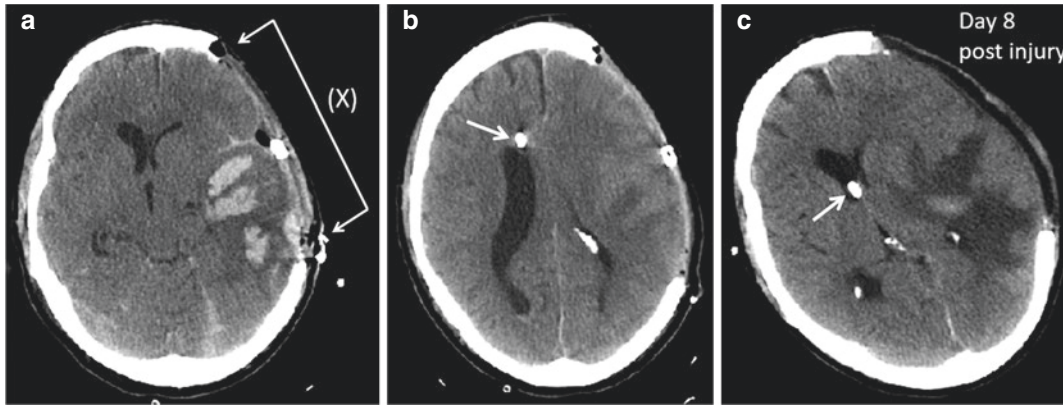


Fig. 14.9 Decompressive craniectomy
The identical patient as depicted in Fig. 14.8 was taken to the operating room for an emergent left-side decompressive craniectomy (X in panel **a**) and placement of an intraventricular catheter (arrow in panel **b** and **c**). The follow-up CT scan on day 8 post-injury reveals an interval resolution of the intraparenchymal hematoma with residual perifocal edema (panel **c**)

14.8 The "Polytrauma Conundrum": TBI with Associated Femur Fracture

Head-injured patients with associated long bone fractures, as classically exemplified by femur shaft fractures, represent a highly vulnerable population due to the risk of sustaining preventable "second hit" insults [32, 33]. The conundrum related to defining the "optimal" timing and modality of long bone fracture fixation in patients with associated head injuries remains a topic of ongoing debate [34]. Early definitive femur shaft fracture stabilization has been advocated by Larry Bone in his historic landmark article from 1989 [35]. However, polytrauma patients with TBI may be highly vulnerable by early definitive fracture fixation due to hemodynamic instability, refractory hypoxemia, or intracranial hypertension. For example, experimental animal studies demonstrated that femoral reaming and nailing lead to increased ICP levels above 15 mmHg in models of hemorrhagic shock/resuscitation with or without associated traumatic brain injury [36, 37]. These data are corroborated by clinical studies that analyzed changes in ICP and CPP in patients with severe TBI who underwent reamed intramedullary nail fixation of associated femur fractures. In this study, the CPP dropped more than 20 mmHg intraoperatively, which was attributed to intraoperative episodes of systemic hypotension during the reaming and nailing procedure [38]. Multiple additional studies have demonstrated that the early definitive femur fracture fixation within 24 h is associated with early episodes of hypoxia and hypotension and adverse neurological outcomes in TBI patients. In order to avoid the adverse effects or "early total care" (ETC) on the vulnerable brain, the concept of "damage control orthopaedics" (DCO) has been advocated by applying a staged approach of initial temporizing external fixation and delayed definitive fracture fixation of femur shaft fractures, as a preferred "neuroprotective" modality for polytrauma patients with TBI (Fig. 14.10) [32, 33].

When compared to ETC, the "damage control" approach with delayed conversion to definitive care has been shown to decrease the initial operative time and intraoperative blood loss without increasing the risk of procedure related complications, such as infection and non-union. The subsequent procedure after DCO related intramedullary reaming and nailing of femur shaft fractures should be performed outside of

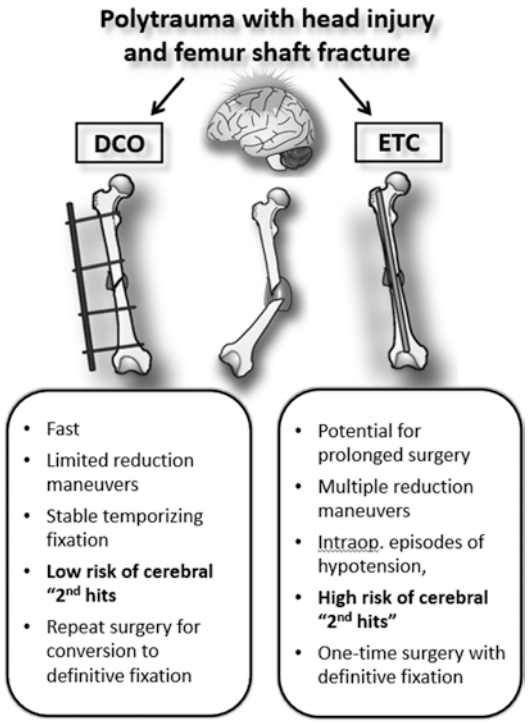


Fig. 14.10 Management strategies for femur shaft fractures in polytrauma patients with TBI
(See text for details)
Abbreviations: *DCO* Damage control orthopedics, *ETC* Early total care

the neuroinflammatory "priming" window, once the post-injury hyperinflammatory response has subsided, ideally within less than 2 weeks of the initial procedure [39]. The delayed conversion procedure is considered safe once the ICP has normalized and patients are awake, oriented, and fully resuscitated.

14.9 Conclusion

Polytrauma patients with head injuries are at risk of sustaining "second hit" cerebral insults which contribute to secondary brain injury and adverse outcomes. The standardized ATLS® approach to the initial assessment and management assures the stabilization of vital functions with the aim of preventing episodes of hypoxemia and hypotension. The presence of intracranial lesions or a deteriorating neurological exam mandates early neurosurgical consultation to assure the optimized coordination of care with the aim of improving long-term outcomes in a highly vulnerable patient cohort.

Key Concepts

The following decision-making strategy represents a "key concept" in terms of providing a pragmatic and safe approach for stratifying polytrauma patients with TBI and associated femur shaft fractures "at risk" for adverse outcomes [32, 33].

- "Damage control orthopaedics" by spanning external fixation in all patients with *severe* TBI (GCS ≤8, intracranial pathology on CT scan, including cerebral edema, midline shift, sub-/epidural bleeding, or open head injuries).
- Optional "damage control orthopaedics" in all patients with *moderate* TBI (GCS 9–13), or patients with GCS of 14/15 with "minor" intracranial pathology on CT scan (e.g., traumatic subarachnoid hemorrhage that warrants observation only). Concomitant neurosurgical procedures may be performed at the same time as DCO, e.g., an emergency craniotomy.
- No additional operations ("second hit!) in patients with refractory intracranial hypertension or unexplained deterioration in neurologic exam.
- Conversion from external to internal fixation in TBI patients who recovered from a comatose state and are awake and alert (GCS 13–15), or comatose patients with a stable ICP (<20 mmHg) and CPP in a normal range (>80 mmHg) for more than 48 h.
- "Early total care" for long bone fractures all patients with *mild* TBI (GCS 14/15) and normal initial craniocerebral CT scan.
- Temporary skeletal traction as a valid adjunct for patients "in extremis," i.e., in severe protracted traumatic-hemorrhagic shock and coagulopathy, who are unsafe to be taken to the operating room until adequately resuscitated.

Take Home Messages

- Polytrauma patients with TBI represent a highly vulnerable patient cohort due to the imminent risk of inflicting a "second hit" insult to the brain related to episodes of hypotension and hypoxemia.
- The primary goal of managing polytrauma patients with TBI is to prevent secondary brain injury. The most important aspect of preventing secondary brain damage is to ensure adequate oxygenation and blood pressure maintenance by managing the trauma patient per ATLS® protocol.
- Secondary brain injury evolves over time and is characterized by neuroinflammation, cerebral edema, breakdown of the blood–brain barrier, evolving intracranial hematomas, increased intracranial pressure and decreased cerebral perfusion pressure, and ultimately delayed (preventable) secondary neuronal cell death.
- A traumatic epidural hematoma represents a neurosurgical emergency which is frequently underestimated due to a lucid interval in awake and oriented patients with the imminent risk of rapid subsequent deterioration ("patients who talk and die").
- Obtaining a head CT scan in TBI patients should never delay the transfer to an appropriate trauma center capable of managing severe TBI by immediate neurosurgical intervention.

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