

Competing Priorities in the Brain Injured Patient: Dealing with the Unexpected

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1. Introduction

Management of the multiply injured trauma patient can be defined by its complex nature and the necessity to reconcile multiple competing clinical priorities. Approach to single anatomic region/organ system traumatic injury tends to be relatively straight forward, although increasing severity of any isolated injury can by itself pose a formidable therapeutic challenge. In fact, any such “isolated” injury can be life threatening if severe enough and/or not managed optimally.

When the effects of simultaneous injuries to different anatomic regions and organ systems are combined, the cumulative complexity of trauma management can increase dramatically.¹ This chapter discusses clinical approaches to patients with traumatic brain injury in the context of multiple simultaneous associated injuries, focusing on addressing competing priorities and triage strategies needed to successfully manage these patients.

2. Team management and leadership

Effective diagnostic and therapeutic approaches to the multiply injured patient require the presence of well-functioning trauma systems and integrated specialty teams.² The optimal approach to the multiply injured patient involves the involvement of trauma-trained surgeons, intensivists, orthopedic specialists, urologists, neurosurgeons, and interventional radiologists.^{3, 4} Highly skilled team management, leadership, and communication skills are of critical importance.⁴ Excellent communication between physicians and teams, including awareness of important clinical pitfalls and constant vigilance on the part of all participating teams (i.e., presence of multiple cross-checks), as well as the need for centralized care planning (including multi-disciplinary patient care conferences) are crucial.⁵

3. Physiologic and outcome considerations

Restoration and maintenance of homeostasis is critical in management of the multiply injured patient. This often formidable task requires the achievement of a delicate balance between satisfying individual organ-system physiologic needs while reconciling the

frequent necessity for micro-management of often competing specific organ- or body system-oriented considerations (i.e., maintenance of relatively lower blood pressure levels in a patient with concomitant aortic and brain injury *versus* maintenance of higher blood pressures to ensure adequate brain perfusion). The critical nature of clinical decision making is exemplified by the finding that morbidity and mortality in head injured patients can as much as triple in the presence of hypotension.⁶ Additionally, when matched for injury severity and age, multiply injured patients with brain trauma have a significantly worse long term outcomes compared to multiply injured patients without brain trauma.^{1,2}

Early predictors of outcome from traumatic brain injury (TBI) include age, Glasgow Coma Scale, pupillary exam, computed tomographic (CT) characteristics, and the presence of hypotension (systolic blood pressure <90 mmHg).⁷ The key consideration is to maintain homeostasis and to triage clinical management in a manner that optimizes recovery of all affected organ systems. This involves the need for intimate knowledge of individual organ system tolerance limits, the knowledge of common clinical management pitfalls, as well as familiarity with temporal evolution of injuries and injury patterns in the context of overlapping priorities and biomedical parameter ranges.

The overarching goal is to prevent the so-called “secondary hits” that have been shown to adversely affect outcomes.² These secondary insults, in contrast to the primary trauma, are amenable to prevention and may be reversible if detected and managed promptly.⁸ As many as 40% of patients with TBI exhibit some form of significant neurologic deterioration during their hospital stay, most often from secondary insults.³ Significant proportion of patients with traumatic brain injury are initially lucid after injury but deteriorate quickly. Therefore, practitioners should always have a high index of suspicion and should avoid potentially dangerous clinical assumptions (i.e., assuming acute intoxication) when approaching patients with possible TBI. One must remember that nearly one-third of all head injured patients who die may belong in this category⁹, and up to 75% of these have an identifiable and avoidable secondary insult.¹⁰ Hypotension accompanies severe head trauma in approximately 35% of cases and entails a near doubling of mortality (from 27% to 50%).¹⁰ Hypotension in the setting of hemorrhagic shock is also associated with increased mortality. A hematocrit of less than 30% may be associated with a reduction in blood oxygen carrying capacity and potential worsening of cerebral ischemia. Acute anemia associated with head injuries has been cited to carry an associated mortality of 52%.¹⁰ Despite that, universal blood transfusion triggers continue to be controversial, and there is evidence to suggest that “dosing” blood by unit(s) as opposed to absolute hematocrit targets may be more prudent in the context of brain tissue oxygenation.¹¹⁻¹³ The age of the transfused blood may also be an important consideration, with more favorable cerebral oxygenation responses seen following transfusions of blood stored for fewer than 19 days.¹¹

Therapy directed at correcting hypovolemic shock includes prompt volume expansion with crystalloid solutions, followed by administration of blood products as per established trauma guidelines. It is hypothesized that, following traumatic brain injury, cerebrovascular dysfunction results in loss of brain compliance, resulting in increased sensitivity to elevated venous pressures. Increased central venous pressure (CVP) occurring with vigorous crystalloid resuscitation may therefore contribute to the loss of brain compliance and the development of intracranial hypertension.¹⁴ Cerebral perfusion pressure (CPP), defined as the difference between mean arterial pressure (MAP) and intracranial pressure (ICP) is an important factor in determining the adequacy of cerebral blood flow. Cerebrovascular autoregulation, present in the uninjured brain, is lost when the CPP falls below 50 mmHg.¹⁵

Xenon 133 scanning to measure cerebral blood flow in brain injured patients found that cerebrovascular dysfunction and global cerebral ischemia was seen in 13% of patients with a GCS of 8 or less, and of these patients 63% were in a persistent vegetative state or died.¹⁶ In some cases, patients with spinal cord injury may exhibit hypotension secondary to dysfunction of the sympathetic nervous system and loss of peripheral vascular tone, an important consideration due to the association between the relatively frequent co-occurrence of TBI and spinal injury.^{17, 18}

4. Overview of injury mechanisms and related considerations

Blunt trauma is associated with some form of brain injury in as many as 40-50% of patients.^{19, 20} Among those with TBI, the incidence of associated injuries can exceed 60%.¹⁹ Moreover, the very presence of TBI and an associated injury approximately doubles the mortality (from ~10% to ~20%) when compared to TBI alone.¹⁹ In patients with a GCS of less than 8, mortality is as high as 45%. In terms of the components of the GCS scale, a motor score of 2 or less was associated with the lowest survival.⁵ Certain associated injuries, when combined with TBI carry an especially high mortality, including great vessel (50% mortality), liver (39%), bowel (37%), spleen (34%), lung (34%), spine (26-32%), and various skeletal injuries (18-29%).²¹ Of note, many of these "high mortality" concurrent injuries tend to be associated with either blood loss and hemorrhage (i.e., femur fracture, splenic/hepatic laceration) or hypoxia (i.e., pulmonary injury).

5. Concurrent spinal injury

Spinal injuries and TBI frequently occur together. Combination of TBI and spinal injury without neurologic deficit carries an approximate mortality of 25%, which increases to about 33% when neurologic deficit is present.²¹ In addition to a defined set of priorities associated with the management of TBI, the trauma practitioner must be aware of important considerations unique to the setting of spinal injury. The overarching consideration is the avoidance of secondary injury by preventing both hypotension and hypoxia.⁸ This spans an entire spectrum of preventive measures, including adequate spinal immobilization with spinal precautions, avoidance of excessive manipulation during patient transfers and procedures (i.e., endotracheal intubation), and provision of adequate cardio-respiratory support. Patients with spinal cord injuries (SCI) may be at increased risk of respiratory failure due to diaphragmatic and/or intercostal muscle dysfunction, depending on the level of SCI.²² The most common causes of spinal cord injuries are motor vehicle crashes (48%), falls (21%), assaults (15%) and sports-related accidents (14%).⁴ It is imperative that the practitioner be aware of the possibility of spinal cord injury in the multiply injured patient. Of all trauma patients that die within the first 30 minutes after injury, 20-25% have a cervical SCI.⁶

A distinct set of complicating factors can be brought about by hemodynamic derangements associated with spinal cord injury. Included are factors such as hypotension requiring vasopressor administration, bradycardia requiring pharmacologic and/or procedural intervention, patient positioning restrictions, as well as inability to perform a reliable injury assessment below the level of neurologic injury associated with spinal cord disruption. Cervical SCI may cause profound changes in heart rate and rhythm, blood pressure, and cardiac output. Patients may exhibit hypotension secondary to dysfunction of the

sympathetic nervous system and loss of peripheral vascular tone.¹⁷ Patients with high cervical SCI (levels C1-C5) have significantly higher requirements for cardiovascular intervention (i.e., need for of vasoactive agents or assistive device use) than patients with lower injuries (levels C6-C7).¹⁷ Shortly after spinal cord injury (seconds to minutes) there may be a systemic pressor response characterized by widened pulse pressure that results from short-term outflow of sympathetic activity and adrenal hormones.²³ This pressor response is then quickly replaced by neurogenic shock characterized by bradycardia and hypotension.²⁴ In order to maintain proper systemic tissue perfusion, affected patients commonly require fluid resuscitation, supplemented by administration of vasoactive agents (if patient remains hypotensive despite adequate fluid resuscitation). Because patients with high spinal cord injuries are more susceptible to developing pulmonary edema, it is important to limit the amount of fluid resuscitation while maintaining a systolic blood pressure of 100-110 mmHg.⁸

Ventilatory management strategies for patients with diaphragm dysfunction differ from those used in cases without muscular functional deficits. With injuries involving spinal segments of T1 or higher, the intercostal muscles, important in expanding the anterior-posterior dimension of the thoracic cavity, are flaccid. With higher injuries of the cervical spine, the diaphragm itself may become paralyzed. This results in a paradoxical inward movement of the abdominal wall during inspiration.²⁵ Pulmonary capacity is further reduced while the patient is supine. Because of the weakened diaphragm, the abdominal contents push cephalad.²⁶ Initial respiratory management is aimed at providing adequate ventilatory support while reducing ventilator associated complications.

6. Concurrent chest injury

The simultaneous presence of TBI and chest injury involves a distinct set of clinical circumstances and considerations. Life-threatening pulmonary injuries may require aggressive ventilatory approaches, and when considered in the context of TBI, may predispose the patient to both systemic and brain hypoxia. Pericardial tamponade and tension pneumo- and/or hemothorax also represent a life threat by causing hypotension and brain hypoperfusion. In addition, traumatic aortic injury may impose a unique set of hemodynamic restrictions with regards to maintenance of narrow blood pressure and heart rate ranges.²⁷ For a given mean arterial pressure, any rise in ICP results in a decrease in CPP. In order to maintain adequate cerebral perfusion, CPP should be maintained around 60-70 mmHg.

One special consideration specific to the patient with TBI is the entity of acute lung injury (ALI) associated with isolated brain trauma.²⁸ This clinico-pathologic entity may not be associated with traumatic pulmonary injury *per se*. Instead, it may be more closely reflective of the global increase in TBI severity (i.e., the presence of a large mass lesion or midline shift on imaging is associated with 5-10 fold increase in risk of ALI).²⁸

In addition to ALI, patients with severe brain injury can develop neurogenic pulmonary edema (NPE), which is defined as increased interstitial or alveolar lung water occurring in the absence of cardiac or pulmonary disorders or hypervolemia.²⁹ The disease process is characterized by alveolar hemorrhage, pulmonary vascular congestion, and the presence of protein rich exudate.³⁰ Comparatively, the incidence of NPE following severe head injury (20%) is similar to the incidence of NPE in subarachnoid hemorrhage (23%).⁹ NPE can present within minutes to hours of the insult and usually resolves by 72 hours. Significant

pulmonary edema past this time point suggests another diagnosis. During the injury there is a sympathetic discharge that causes increases in arterial and venous pressures and subsequent vascular damage. This damage is thought to result in vascular extravasation and the development of NPE. In animal models NPE is most reproducible with insults to the nucleus tractus solitarius or the noradrenergic A1 cell group.³¹ Interestingly, the exudate seen in NPE has a much higher protein content than that seen in cardiogenic pulmonary edema, supporting a distinct physiologic process.³²

7. Concurrent abdominal injury

Traumatic abdominal injuries are among the most lethal overall, with intra-abdominal and pelvic hemorrhage continuing to be associated with significant morbidity and mortality. This section will discuss diagnostic and therapeutic approaches needed to effectively manage concurrent abdominal and traumatic brain injuries. Included in the discussion is the management of the abdominal compartment syndrome and the damage control approach to severe abdominal trauma. At times, increasing ICP may be noted in patients with TBI and significant abdominal injury. In highly select cases, the correct diagnosis and surgical decompression of the abdominal compartment syndrome (ACS) may improve intracranial hypertension that is otherwise unresponsive to traditional medical therapy.³³

It is not uncommon for the brain injured patient to have simultaneous abdominal injury, especially in the setting of blunt polytrauma. In this case, the presence of intra-abdominal hypertension (IAH) can exacerbate elevations in ICP. Thus, the presence of IAH is an independent risk factor for secondary brain injury.³⁴ The increase in intraabdominal pressure is directly reflected in intrathoracic pressure and central venous pressure. Elevations in central venous and jugular venous pressures result in increased resistance to cerebral outflow, which causes an increase in ICP and decrease in CPP.³⁵ Animal experiments have demonstrated that IAH of >20 mmHg causes significant increases in ICP and decreases in CPP. Additionally, elevations in CSF lactate and interleukin-6 were also seen, suggesting the associated presence of cerebral ischemia.³⁶

Treatment for refractory ICP elevations in the setting of IAH involves several modalities including neuromuscular blockade, vasopressor use to preserve CPP, and abdominal compartment release.³⁷ In the setting of new onset end-organ dysfunction (i.e., renal failure, worsening pulmonary dysfunction) many physicians would advocate abdominal fascial release (a.k.a., abdominal damage control).³⁸ Several authors advocate more liberal use of decompressive laparotomy, extending this paradigm to patients with refractory elevations in ICP without intra-abdominal hypertension. In a group of 17 poly-trauma patients with refractory increases in ICP, decompressive laparotomy resulted in significant ICP reductions from 30.0 ± 4.0 to 17.5 ± 3.2 mmHg.³⁹

Occasionally, emergent laparotomy and abdominal damage control may be coupled with damage control neurosurgery (DCNS) in the acute setting. Initial neurosurgical interventions include arrest of intracranial bleeding followed by evacuation of hematoma/mass lesion. Therapeutic craniectomy appears beneficial in children with diffuse brain edema.⁴⁰ However, DCNS can not be fully recommended in adults until the ICP becomes uncontrolled despite optimal medical therapy.^{41, 42}

Stopping non-cranial hemorrhage is critical in the overall management of the multiply injured patient with brain trauma. Continued emphasis on team work and close

collaboration between clinical teams is crucial.⁴³ In the multiply injured patient, an ICP monitor (mostly under local anesthesia or during craniotomy) can be inserted in the emergency room or in the intensive care unit while the patient is being stabilized. At times, ICP monitoring is initiated in tandem with emergency laparotomy, thoracotomy or any other life-saving procedures.⁴³ Occasionally, a craniotomy concurrent to other operative procedures may be required if the patient has a significant intracranial mass lesion and evidence of critical ICP increases on clinical exam. General surgeons may have to occasionally perform neurosurgery in remote locations for patients with TBI as statistics have shown that early simple interventions have resulted in increased rates of survival between 10-50%.^{44, 45} Simpson *et al* have recommended evacuation of an extradural hematoma by the general surgeon in a remote location if the trauma center is >1-2 hours away.⁴⁴ Rinker *et al* advise emergency craniotomy if the GCS is <8, there are lateralizing signs such as a dilated pupil, hemiparesis or development of sustained bradycardia and hypertension.⁴⁵ Immediate availability of neurosurgeons may not be essential if a properly trained and credentialed trauma surgeon can appropriately monitor patients for neurologic deterioration and facilitate early transfer to a center capable of full-time operative and postoperative neurosurgical care.⁴⁶ In certain extreme situations, the performance of an emergency burr hole may be life saving.⁴⁷

8. Concurrent skeletal injuries

Skeletal injuries are associated with a number of unique therapeutic challenges, especially when associated with significant blood loss and need for emergent skeletal fixation. This section discusses best approaches to deal with brain injured patients who also present with fractures, dislocations, and other musculo-skeletal emergencies. Included in the discussion is the topic of extremity compartment syndrome.

The management of skeletal fractures in brain injured patients continues to be a controversial topic. A comparison of early (<24 hours) versus late fracture fixation demonstrated that early fracture stabilization does not result in increased central nervous system complications.⁴⁸ Same study showed that patients undergoing delayed fixation experienced significantly higher pulmonary morbidity.⁴⁸ Another study showed that lower extremity fracture fixation within 24 hours did not entail greater risk for adverse outcomes in patients with TBI.^{49, 50} The authors did emphasize, however, that avoidance of any undue hypoxia and hypotension is critical.^{49, 50} Orthopedic “damage control” strategies have evolved in order to assist in early management of skeletal trauma in multiply injured patients with TBI who may be unable to tolerate traditional operative approaches or may not even be stable enough to leave their intensive care bed.^{34, 51}

The clinical syndrome of fat embolism can influence the clinical course in the multiply injured patient, especially following long bone extremity fracture fixation.⁵² Although early fracture fixation is thought to minimize the risk of this occurrence, some experimental studies show that intramedullary nailing of femoral fractures and subsequent liberation of bone marrow contents may have a negative influence on the central nervous system function.⁵²

An important aspect of orthopedic care in the multiply injured patient with brain trauma is the lack of reliable physical examination. Due to this limitation, extremity compartment syndrome may evade timely diagnosis. The reliance on the traditional early clinical signs

and symptoms of compartment syndrome – pain on passive motion that is out of proportion to clinical findings and the presence of paresthesias – has to be substituted with heightened index of clinical suspicion and extremity compartment pressure measurements.⁵³ Fasciotomies should be performed in a timely fashion when evidence of elevated compartment pressures is present.^{54, 55}

9. Concurrent vascular injury

Vascular injuries present a special challenge in the context of simultaneous brain trauma. Specifically, direct management priority conflicts can be seen with regards to the need for therapeutic anticoagulation and the risk of secondary intracranial hemorrhage. Likewise, the maintenance of cerebral perfusion pressure can pose an increased risk in patients with concurrent traumatic pseudoaneurysms and other injuries that may necessitate strict blood pressure and heart rate control. In addition, high dose vasopressor use to maintain adequate cerebral perfusion may lead to distal extremity ischemic complications up to and including the need for amputation.

With ever improving quality of modern imaging modalities, cerebrovascular injuries are being detected more frequently. Neurologic assessment following blunt cerebrovascular injury can be difficult and distinguishing cerebral ischemia from cerebral infarction is often complex, especially in the setting of altered mental status.⁵⁶ The main challenge associated with the diagnosis of blunt carotid or vertebral injury (BCVI) is the relative rarity of BCVI and the need for constant vigilance and high index of suspicion. One of the most important clinical findings associated with BCVI is the presence of an unexplained or new neurologic deficit in the setting of otherwise normal (or unchanged) brain imaging. Trauma practitioners should be familiar with major risk factors for BCVI, both from the injury mechanism standpoint (i.e., cervical seat belt sign, blunt assault to craniofacial area with LeFort III fracture pattern, cervical spine fracture) and from the clinical presentation standpoint (i.e., high-speed motor vehicle crash, flexion-extension neck injury).⁵⁷

10. Missed injuries and diagnostic delays

Important in the context of multiple trauma patient with concomitant brain injury are the concepts of missed injury and delayed diagnosis. Delay in diagnosis occurs when an injury is identified after the usually accepted initial phases of trauma evaluation (i.e., primary, secondary, or tertiary surveys) but before the injury manifests as an overt clinical problem.⁵⁸ Missed injury can be defined as a delay in diagnosis that is associated with clinical symptoms and/or is not identified until after discharge from hospital.⁵⁸ Although the incidence goal for missed injuries and diagnostic delays should be “zero”, this target remains elusive. Major series cite missed injury rates between 0.5% and 65%, with anywhere between 1 and 2.3 missed injuries per patient, depending on population under study, type of study (prospective versus retrospective), and diagnostic definitions (missed injury versus delayed diagnosis).⁵⁸ Among missed injuries, over 10% are clinically significant and, of those, 14% to 50% can be associated directly with patient mortality.^{1, 58, 59}

The importance of this topic to the brain injured trauma population becomes obvious when one considers the most common contributing factors to missed injury: (a) altered mental

status; (b) presence of distracting injury; (c) administration of analgesia and sedation; and (d) overwhelming or multiple simultaneous injuries.⁵⁸ Whenever the patient's sensorium is diminished, it becomes more difficult to identify injuries as the patient loses the ability to effectively express complaints related to pain and discomfort. Alterations in pain processing may occur with traumatic brain injury, spinal cord injury, hypoxia, shock, intoxication/substance abuse, and administration of sedation for various reasons (i.e., combative patient). The pain response can be altered after a major injury and the patient may not be able to process pain from all injuries equally. For example, a non-displaced ankle fracture may not be readily evident with a concurrent presence of an open femur fracture. Often direct palpation over a specific injury site will elicit a pain response. Therefore, comprehensive repetitive physical examinations may be required in order to effectively identify the complete injury list in the presence of distracting pain. However, even the most detailed physical examination may fail to detect traumatic injuries in the multiply injured patient with concomitant moderate to severe brain trauma. Moreover, concurrent administration of analgesia and sedation may additionally affect the practitioner's ability to reliably detect various types of injuries, from minor to life threatening.⁵⁸ It is important to note that cranio-facial injuries constitute as many as 5%-30% of missed injuries, depending on study cited.^{1, 59, 60}

11. Pitfalls and controversies

This section highlights important pitfalls and controversies associated with management of the multiply injured patients with concurrent brain trauma. We emphasize the need for continuous reassessment of competing priorities and need for centralized team coordination. Because many of the topics mentioned are beyond the scope of this chapter, the reader is referred to other sources as referenced herein. Practitioners should always be aware of potential complications related to massive fluid resuscitation, up to and including the abdominal compartment syndrome.⁶¹ On the opposite end of the hemodynamic spectrum, one should always be cognizant of complications related to use of escalating doses of vasoactive agents, including phenomena such as tachyphylaxis⁶² and the possibility of skin/limb ischemia due to high-dose vasopressor use.⁶³ Although the authors encourage the use of advanced hemodynamic monitoring (both invasive and non-invasive), there are many potential complications associated with both errors in hemodynamic data interpretation and iatrogenic injury related to invasive line placement.⁶⁴

Intra-hospital patient transfers (i.e., transport to operating room or imaging suite) carry its own set of complications, with serious adverse outcomes attributed to such transfers in over 30% of critically ill patients.⁶⁵ Use of any therapies or diagnostic tests that could potentially contribute to additional complications should always be considered in the context of risk-benefit ratio.⁶⁶ For example, although still controversial, evidence suggests that early use of prophylactic anticoagulation is more beneficial than withholding this therapy in the TBI population.^{67, 68}

Additional considerations include the effect of sedative agents on both hemodynamic and metabolic aspects of patient management. For example, the use of propofol for sedation may be associated with complications such as hypotension⁶⁹, pancreatitis⁷⁰, and propofol infusion syndrome.⁷¹ In addition, adjunctive approaches such as therapeutic hypothermia and chemically induced coma are mentioned and referenced for the reader.^{72, 73} These

Provider- and Team-related considerations

- Skilled team management, leadership, and effective communication are of critical importance.
- Coordinated care planning, including multi-disciplinary conferences and open dialogue between various clinical specialties, is important to optimizing patient care.
- Trauma teams should work efficiently, utilizing protocolized care as well as well-functioning clinical management / surveillance systems.
- The overall goal of the trauma team is to reconcile conflicting priorities with the overarching goal of maximizing the outcome from the perspective of the “whole patient”.

Injury-related considerations

- Knowledge of injury patterns is useful in determining the likelihood of any potential associated non-TBI injuries, especially in the setting of concurrent neurological impairment.
- The very presence of associated injuries in the setting of concurrent brain trauma is associated with significantly increased mortality. Among such associated injuries, those that carry highest mortality usually involve risks of hypotension, hemorrhage and/or hypoxia.
- Familiarity with acceptable physiologic parameter ranges inherent to the management of each injured anatomic area or organ system is important to patient care optimization and reconciliation of potentially conflicting therapeutic priorities.
- Non-conventional measures, including various “damage control” approaches permit the most critical injuries to be given higher priority while adequately temporizing other, less critical injuries.

Patient care-related considerations

- The overarching goal is to prevent secondary physiologic insults that have been shown to adversely affect outcomes.
- Maintenance of adequate cerebral perfusion pressure while minimizing hypotensive and hypoxic events is crucial. It is important to note that while these priorities do not change over time, the nature of inciting events may differ (i.e., hemorrhage causing early hypotension versus sepsis causing late hypotension).
- Intra-hospital patient transfers (i.e., for procedural interventions or imaging studies) carry a significant risk, with nearly one-third of such transfers associated with some sort of adverse event (i.e., hypotension, hypoxia, etc). Therefore, such transfers should be undertaken only if absolutely indicated.
- Lack of reliable physical examination in multiply injured patients with TBI predisposes this group to missed injuries and diagnostic delays. Although modern technological advances enable practitioners to partially “compensate” for the lack of adequate bedside assessment, there are no true substitutes for an experienced practitioner with an adequate level of clinical suspicion.

Miscellaneous considerations

- Use of any therapies or diagnostic tests that carry a defined potential for complications should always be considered in the context of careful risk-benefit determination.

Providers should be familiar with potential complications associated with each and every therapeutic agent and procedure. Early recognition of such complications can be life-saving.

Table 1. Important points in management of multiply injured patients with concurrent traumatic brain injury

therapies are still controversial and further research is needed to better define their safety profiles and risk-benefit characteristics, especially in the setting of multiple trauma and competing clinical priorities.

12. The multiply injured patient with TBI – Putting it all together

Management of the multiply injured patient with TBI involves close collaboration of multiple specialties, including critical care, neurosurgery, orthopedic, and trauma experts. Practitioners must always be aware of all competing priorities, including cross-specialty considerations for specific injury patterns and associated pitfalls and complications. Life-threatening injuries should be approached according to the magnitude of the most immediate mortality risk. At times, simultaneous management of multiple injuries may require the initiation of various “damage control” techniques. Complications related to the primary injuries as well as any secondary insults must be recognized and addressed promptly. Early rehabilitation is crucial in order to optimize long-term outcomes in this population. The achievement of these goals requires that trauma teams work efficiently, utilizing protocolized care and well-functioning clinical surveillance systems.^{74, 75} The overall goal of the trauma healthcare team is to reconcile any conflicting priorities with the goal of maximizing the outcome from the perspective of the “whole patient”.

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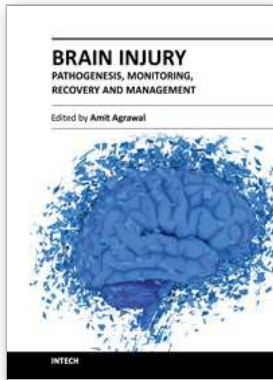
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The present two volume book "Brain Injury" is distinctive in its presentation and includes a wealth of updated information on many aspects in the field of brain injury. The Book is devoted to the pathogenesis of brain injury, concepts in cerebral blood flow and metabolism, investigative approaches and monitoring of brain injured, different protective mechanisms and recovery and management approach to these individuals, functional and endocrine aspects of brain injuries, approaches to rehabilitation of brain injured and preventive aspects of traumatic brain injuries. The collective contribution from experts in brain injury research area would be successfully conveyed to the readers and readers will find this book to be a valuable guide to further develop their understanding about brain injury.

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