



Severe Traumatic Brain Injury in Sports

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A 28 year-old male is snowmobiling with his father in western Colorado. A deer runs into the path of the two vehicles, causing him to swerve and hit a tree. He hits his head after being thrown from the snowmobile, and lies motionless amidst the trees. Luckily, he was wearing a helmet, and his father has with him a cell phone to call for help.

Within 15 minutes, help is available to transport him to the nearest hospital. During this interval, the patient lost consciousness for up to 10 minutes, and then was only able to speak in short phrases. He complained of a headache, and of being cold. The father thought that there was blood coming from inside of the helmet, but he hesitated to remove the helmet prior to arrival of the rescue crew. During the 30 minute transport to the closest hospital, the patient became more obtunded, responding only to loud verbal stimuli with moaning. He was able to maintain his airway, and appeared to be adequately oxygenating.

Upon his arrival at the Emergency Department, the physical exam revealed the following vital signs: BP 100/60, P 110, RR 12, T 98.8. He was now only responsive to painful stimuli with a withdrawal reflex. Upon removal of the helmet, a large laceration over the right temporal-parietal region was noted, as well as several abrasions and contusions to the face. His pupils were noted to be pinpoint, equal, and reactive. He appeared not to have Doll's eyes. He continued to withdraw to pain during his initial evaluation. His estimated weight was approximately 80 kg.

What are the next steps that should be taken in the Emergency Department?

Key Clinical Questions

What information exists that guides our Emergency Department management of severe TBI?

What are the key therapies in the management of severe TBI in the ED?

What are the indications for operative intervention in severe TBI patients?

What clinical findings predict outcome in severe TBI patients?

Introduction

The Emergency Physician commonly manages patients who present to the Emergency Department with traumatic brain injury (TBI). Although most patients have only minor TBI, there is a significant minority of patients who sustain severe TBI. The role of the Emergency Physician is to stabilize these patients, assess their overall extent of injury, and determine the need for immediate operative intervention. In order to accomplish this task, it is necessary to be familiar with the current guidelines and recommendations that direct these critical tasks.

Epidemiology and Pathophysiology

Each year, there are approximately 1.6 million head injuries in the United States, and 1 million patients are treated and released from an Emergency Department (ED).¹ These injuries result in an additional 230,000 hospital admissions, cause 50,000 deaths, and result in 80,000 patients with permanent neurological disabilities per year. Over 50% of all trauma fatalities are a result of traumatic brain injury (TBI), and TBI is the leading cause of death and disability in the United States. TBI results in a lost productivity costs and annual healthcare costs of \$40 billion.

In TBI patients, brain edema results from vasogenic, hydrostatic, osmotic, and cytotoxic effects. As a result of interstitial edema, brain fluid volume increases and intracranial pressure (ICP) rises. Cerebral perfusion pressure (CPP) is the difference between the patient's mean arterial pressure (MAP) and ICP, as is shown below:

$$\text{CPP} = \text{MAP} - \text{ICP} \quad (\text{Normal example} = 80 \text{ mmHg} = 90 - 10)$$

Both elevated ICP and decreased MAP (as in hemorrhagic shock) can cause CPP to diminish to a critical level that will increase cell death and morbidity following TBI. Cerebral blood flow (CBF) will be disturbed when ICP is above 40 mmHg, and ICP levels above 60 mmHg is uniformly lethal. In most patients, therapy for elevated ICP should begin when ICP is consistently above 20 mmHg. Once cell death begins as a result of TBI, there is secondary auto-destruction, which cause oxygen radical formation, intracellular calcium shifts, glutamate toxicity, and a cycle of ongoing cell death.

Medical Literature and Internet Search Information

The information obtained from the medical literature came from the MEDLINE/PubMed search engine.² The keywords utilized in the search included the terms “TBI, Guidelines, Diagnosis, therapy, and Emergency Department”. These terms provided the 1996 guidelines from the *J Neurotrauma* and the Italian guidelines from the *J Neurosurg Sci*.

The search of the Internet included a search using www.google.com³ and the key words TBI and head trauma. From this search, the American Association of Neurological Surgeons (AANS) website was found and the website of the Brain Trauma Foundation.⁴ Using www.google.com, a search for the Cochrane database was made, finding the website www.update-software.com. On this website, the Cochrane Library can be searched using key words in order to find abstracts of the reviews done on topics such as TBI and mannitol.

Brain Trauma Foundation and Cochrane Recommendations

The Brain Trauma Foundation (BTF) has both pre-hospital and in-hospital guidelines available at its website, www.braintrauma.org.⁴ The pre-hospital guidelines can be printed from the website, but the in-hospital guidelines can only be reviewed from the website.⁴ The in-hospital guidelines, called the [Management and Prognosis of Severe Traumatic Brain Injury](#), were developed in 2000.⁵ They are an update of the guidelines that were published in 1996.⁶ The in-hospital guidelines have been accepted by the American Association of Neurological Surgeons (AANS), the first such protocols ever accepted by the Association. These guidelines are also endorsed by the World Health Organization's (WHO) Committee on Neurotraumatology.

The Department of Transportation National Highway Traffic Safety Administration (NHTSA) awarded the Brain Trauma Foundation a grant to develop Guidelines for emergency medical service providers and their medical directors on the prehospital assessment and treatment of traumatic brain injury. The [Guidelines for Prehospital Management of Traumatic Brain Injury](#), were developed with the assistance of a national group of EMS experts, and are available to print from the Braintrauma.org website.⁷

The Cochrane Library has guidelines for several aspects of TBI management, including anti-epileptic drugs, barbiturates, calcium channel blockers, hyperventilation, and mannitol, dating from 1997 to 2001.⁸⁻¹² (Each review is updated when new information warrants a new review.)

Acute Management of Traumatic Brain Injury (TBI)

The following headings provide the areas of TBI management that are addressed by the BTF or Cochrane guidelines, or are relevant to the management of TBI in the Emergency Department. The BTF guidelines utilize three classes of evidence (I-III) and three recommendation levels (standards, guidelines, and options). The Cochrane Reviews simply state a reviewer's conclusion based on the available data from the medical literature.

Initial Resuscitation

The BTF guidelines have no firm standards or guidelines stated, instead, they offer only options that may be useful in the acute setting. These include rapid physiologic resuscitation and the use of sedation and short acting neuromuscular blockade as needed. These guidelines state that intracranial hypertension treatment should be delayed unless herniation and/or rapid neurologic deterioration are suspected clinically.

Blood Pressure and Cerebral Perfusion Pressure

Although there are no standards regarding blood pressure management, the BTF recommends that SBP should be maintained above 90 mmHg and that if possible, MAP should be maintained above 90 mmHg and CPP above 70 mmHg. These values should be achieved using judicious fluid infusion as needed.

Hypoxia

Regarding the management of hypoxia, the BTF again states no standards, but suggests that the patient's PaO₂ should be maintained above 60 mmHg. A recommended option states that endotracheal intubation should occur when the GCS is < 9, when there is persistent hypoxia, or if the patient is unable to maintain their airway.

Hyperventilation

The BTF guidelines do provide a clear standard for hyperventilation, stating that in the face of a presumed or measured normal ICP, the pCO₂ should not be maintained below 25 mmHg even in severe TBI patients. Their guidelines also state that early prophylactic hyperventilation, with pCO₂ levels below 35 mmHg, should also be avoided. Several options are provided, including the brief use of hyperventilation in the face of acute neurologic deterioration or persistent intracranial HTN that fails other medical therapies. The option to test for cerebral ischemia using jugular venous O₂ saturation monitoring is suggested if it is necessary to maintain the pCO₂ below 30 mmHg.

The Cochrane Review of this issue states that there is only one randomized controlled trial (RCT) regarding hyperventilation, and that there is still considerable uncertainty regarding its use in TBI. The reviewer concluded that although there is a possible beneficial effect on mortality with the use of hyperventilation in TBI, it is not clear that its use improves neurologic outcome.

Mannitol

The BTF guidelines state that mannitol does control increased ICP, and that it could be used in severe TBI in doses up to 1 gr per Kg body weight, although this is not a standard of care in TBI. As with hyperventilation, the option is to use it in the face of a rapid neurologic decline and presumed herniation. The physician is guided to avoid hypovolemia with its use, and to keep the serum osmolarity above 320 mOsm. It is suggested that intermittent boluses are preferred over constant mannitol infusions.

The Cochrane Review of mannitol in TBI points out that there are few RCTs and, as such, there is uncertainty regarding its use. It may be useful, however, in the setting of measured (not presumed) increased ICP, and may be superior to pentobarbital in the setting of increased ICP.

Barbiturates

There are no standards regarding high dose barbiturates, but the BTF suggests that its use can control increased ICP when all other therapies, both medical and surgical, fail to decrease ICP. It is suggested that this therapy only be used in patients who are hemodynamically stable and those for whom death is not certain.

The Cochrane Review of this subject states that barbiturates work through lowering cerebral metabolism, but because there are few RCTs, that there is no evidence of improved outcome. The studies to date have shown hypotension in 25% of patient treated with this modality, and the reviewer suggests that this adverse effect might offset any of the benefit of this TBI treatment.

Steroids

The BTF guidelines state that there is no role for steroids in TBI, given that they have not been shown to decrease ICP or improve patient outcome in any studies to date.

Calcium Channel Blockers

The Cochrane Library includes a review of calcium channel blockers in severe TBI, pointing out that these drugs may prevent vasospasm and maintain cerebral blood flow. Despite the fact that there are four RCTs, there still is considerable uncertainty, the reviewer points out. Pooled data from two RCTs of traumatic SAH patients has shown that the use of nimodipine decreases mortality by 40% and decreases death or disability by 33%.

Seizure Prophylaxis

The BTF guidelines point out that there is no role for anti-epileptic drugs (AEDs) in TBI patients in order to prevent the occurrence of late post-traumatic seizures. There are guidelines that suggest that although they will not change long-term outcome, the use of phenytoin or carbamazepine or phenytoin may reduce the risk of early seizures in high-risk patients and possibly reduce the risk of ICP spikes in association with these early post-traumatic seizures.

The Cochrane Review of AEDs in TBI suggests that these drugs might be helpful in reducing the cytotoxic metabolism that causes glutamate to accumulate following seizures. In six RCTS, their use of AEDs reduces the risk of early seizures by 66%. For every 100 patients who are prophylaxed with an AED post-trauma, 10 would remain seizure-free for the first week. But, as was stated in the BTF guidelines, this early AED use has not been shown to reduce the occurrence of late seizures or alter long-term neurologic outcome.

Antibiotic Prophylaxis

Neither the BTF guidelines nor the Cochrane Library include any mention of prophylactic antibiotics in TBI. The ePocrates database (www.ePocrates.com) and the Sanford guide also have no specific recommendations regarding antibiotic use in penetrating TBI. Tintinalli's Emergency Medicine Comprehensive Study Guide suggests that antibiotics only be given with neurosurgical consultation, and that in patients who present with a fever late following a skull fracture, that antibiotics should be given.¹² Within 72 hours of injury, pneumococcus should be treated, and after this time interval, Staph aureus and gram negatives should be treated using vancomycin and a third generation cephalosporin such as ceftazadime.

Intracranial Pressure (ICP) Monitoring

ICP monitoring is suggested by the BTF when the TBI patient's GCS score is < 9, or when the CT shows either space-occupying lesions or edema that compresses the basal cisterns. It is also suggested in patients with a normal CT if two of these three findings are present: age > 40 years, persistent SBP < 90 mmHG, or the presence of motor posturing. ICP monitoring is felt not to be useful in TBI patients with GCS scores > 8, unless there is a space-occupying lesion seen on CT.

Elevated ICP Management

The BTF recommends that ICP be managed using an ICP monitor, and that CPP be maintained above 70 mmHg. Ventricular drainage is encouraged, as is the use of repeat CT scans when indicated. First-line therapies include the use of hyperventilation to a pCO₂ of 30-35 mmHg, or the use of mannitol in doses up to 1 gr/kg. Second tier agents include the use of barbiturates and hyperventilation to a PCO₂ < 30 mmHg.

Emergent Cranial Decompression

Used as far back as in the days of Hippocrates, emergent cranial decompression, or placing a Burr hole in the skull, is used to evacuate extradural hematomas in the setting of presumed tentorial herniation. When rapid, progressive neurologic deterioration occurs, with coma, a fixed and dilated pupil, hemiplegia, and a presumed skull fracture on the side of the blown pupil, a likely intracranial hematoma is present on the same side. In this situation, a temporal Burr hole is placed in proximity to the middle meningeal artery.¹⁴ When bilateral fixed pupils are present, this procedure can be repeated on the contra-lateral side. Although no mention is made of this procedure in the BTF guidelines, indications for this procedure are discussed in the EM Reports TBI discussion, part II.¹⁵

Non-operative and Operative Intervention Recommendations

A series of guidelines was developed by Italian neurosurgeons and neurointensivists, and are published in three parts. These three documents cover the initial assessment and management of TBI patients, and the criteria for medical and surgical management for these patients.¹⁶⁻¹⁸

Initial Assessment and Management

The initial evaluation of the TBI patient using the motor component of the GCS score is discussed in patients who are comatose (eye score = 1, verbal score = 1,2). In this situation, the motor component takes on great prognostic significance, and it should be scored using the best motor response from either side of the body. The indications for cranial CT are stated, including the loss of two points on GCS, ICP above 25 mmHg, or a decrease in CPP below 70 mmHg or O₂ saturation below 50% for over 15 minutes. These guidelines recommend that intubation be achieved using rapid sequence induction (RSI) with use of the sedatives thiopental, midazolam, or ketamine, and use of the paralytics succinylcholine or vecuronium.

Criteria for Medical Therapy

One unique concept from these guidelines relates to the use of inotropes in the TBI patient who is hypotensive. The recommendation is made that inotropes only be used once the blood volume is restored, and is indicated to maintain MAP above 90 mmHg and to achieve a CPP above 70 mmHg when the ICP is elevated. These guidelines state that the use of these agents should not be in lieu of those therapies that provide a reduction in elevated ICP.

Criteria for Surgical Therapy

These guidelines state absolute and relative criteria for surgical intervention. Knowledge of these indications will help the Emergency Physician to plan for the actions for the neurosurgeon when consultation is made for the TBI patient. Absolute surgical criteria include the presence of a focal lesion that causes a midline shift > 5 mm, and a space-occupying lesion > 25 cc in volume. Relative criteria for surgical intervention include ICP > 20 mmHg or a CPP < 70 mmHg despite optimal medical therapies.

Brain Trauma Foundation Outcome Prediction Guidelines

In its 2000 TBI guidelines, the BTF also developed standards for outcome prediction in TBI. The presence of clinical findings was correlated with mortality using available class I evidence, looking for a 70% positive predictive value (PPV) as its cutoff for being clinically useful. The clinical findings that are related to mortality, all of which can be detected in the Emergency Department, are reviewed below.

Glasgow Coma Scale (GCS) Score

As the GCS score declines, mortality increases in a step-wise manner. It is a standardized bedside test that preferably should be recorded after pulmonary and hemodynamic stabilization, and without the presence of sedatives or paralytics. The GCS score is viewed to be useful because many health care personnel, with good inter-rater reliability, can do it easily.

Age

As is seen with the GCS score, there is a step-wise increase in mortality as age increases. This is true in TBI as it is in other types of trauma patients.

Pupil Exam

The pupil exam is important to note in the acute setting, since the bilateral absence of a light reflex suggests a higher mortality than in patients who do not have this finding. Asymmetry is defined as > a 1 mm diameter difference, a dilated pupil is one that is > 4 mm in size, and a fixed pupil is one that has < a 1 mm response to light. The pupil exam should be recorded over time, and should whether the pupils are fixed, dilated, and are asymmetric at rest or to light. As with the GCS score, it is best to record the pupil exam after adequate pulmonary and hemodynamic resuscitation has taken place.

Hypotension and Hypoxia

A persistent SBP < 90 mmHg has a 67% PPV for mortality, and when seen with hypoxia, there is a 79% PPV for a bad outcome. Because these parameters are so important, it is suggested that they be recorded frequently during the resuscitation of TBI patients in the acute setting.

Cranial CT Findings

Four categories of CT findings are viewed in the BTF guidelines to have prognostic value:

Basal Cisterns and Elevated ICP

The presence of compressed or absent basal cisterns suggests a three-fold increased risk of increased ICP and mortality. This finding may be associated with papillary findings, focal lesions on CT, GCS scores, and the insults that result from hypoxia and hypotension.

Subarachnoid Hemorrhage

Subarachnoid hemorrhage (SAH) occurs in up to 56% of severe TBI, and is most commonly seen over the convexity of the brain. For whatever severity of injury noted to the brain, mortality doubles in the presence of traumatic SAH. If there is blood in the basal cisterns, there is a 70% PPV of a bad outcome. The volume and extent of traumatic SAH is related to outcome, independent of the other injuries noted in TBI patients.

Midline Shift

In patients over age 45 and > a 5 mm midline shift, there is 78% PPV of a bad outcome. In any patient, if there is > a 15 mm midline shift, there is a 70% likelihood of an unfavorable outcome. Although midline shift is associated with increased ICP, the presence of other findings on CT, such as a space-occupying lesion, are more important than the shift itself. The presence or absence of a midline shift should be assessed regularly after surgical therapy is provided.

Intracranial Lesions

In all cases of coma, an intracranial lesion should be suspected. In the presence of any traumatic mass lesion, there is a 78% likelihood of a poor outcome. In a patient with a mass lesion who is over 45 years old, there is a 79% chance of death or a vegetative state. Subdural hematomas are associated with a higher mortality than are extradural hematomas, and the hematoma volume is related to outcome. The worst outcomes are seen in subdural hematomas, diffuse axonal injury (DAI) and epidural hematomas, respectively.

Conclusions: Severe TBI Patients and the Emergency Physician

There are a number of sources available for the Emergency Physician to utilize in order to learn how to optimize the acute management of sports injury patients with severe TBI.

Key Learning Points

1. Index Medicus and other Internet sources have guidelines and other information that allows the Emergency Physician to learn about TBI with a home computer.
2. The Brain Trauma Foundation, the Cochrane recommendations, and published guidelines identified using guidelines.gov all are useful sources of information regarding the management of severe TBI patients.
3. The diagnosis of severe TBI in comatose patients involves the use of the GCS score, especially the motor component. Liberal cranial CT use is also a key strategy in the diagnosis of severe TBI.
4. The acute management of severe TBI involves maintaining SBP above 90 mmHg, CPP above 70 mmHg, and the PaO₂ above 60 mmHg.
5. Airway management requires RSI that establishes the use of both sedative agents and paralytics. This strategy allows the airway to be secured without causing precipitous rises in ICP or aspiration.
6. Judicious hyperventilation (pCO₂ 30-35 mmHg) and bolus infusions of mannitol are only indicated when ICP is suspected or proven to be elevated, based on clinical or CT findings, or ICP monitoring. ICP monitoring is suggested when the GCS score is < 9, or when CT or clinical findings confirm a space-occupying lesion or cerebral edema.
7. Although seizure prophylaxis may be of benefit acutely, it has not been established to prevent the occurrence of seizures long-term following severe TBI.
8. The use of barbiturates, steroids, and calcium channel blockers has not been shown to be effective in improving outcome in severe TBI patients.
9. Emergent cranial decompression is indicated when an extradural hematoma is suspected based on the presence of a fixed and dilated pupil or hemiplegia on the same side as a likely skull fracture.
10. Operative intervention is indicated in the presence of a focal lesion that causes a midline shift > 5 mm, or a space-occupying lesion of > 25 cc volume.
11. Low GCS scale scores, increased age, absent or asymmetric pupil light reflex, hypotension, and hypoxia all suggest a worse outcome in the setting of severe TBI. On CT, compressed basal cisterns, subarchnoid hemorrhage, midline shift, and intracranial lesions all suggest a worse clinical outcome.

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Patient Outcome

Upon arrival to the ED, the Emergency Physician managed the airway using RSI (succinylcholine and midazolam) and endotracheal intubation. Despite having vital signs suggestive of traumatic hemorrhage, the patient's vital signs stabilized after an infusion of two liters of crystalloid in the ED. The peritoneal lavage was equivocal. Abdominal CT findings suggested a small liver hematoma and minimal intraperitoneal blood. The cranial CT demonstrated non-depressed linear skull fracture and an epidural hematoma with a 5mm midline shift. Following the infusion of 200 cc of mannitol (20 gr, or 0.25 mg/kg), the patient was transported by helicopter to the closest trauma center. Upon arrival at the trauma center, the patient was taken directly to the OR, where the epidural hematoma was evacuated. Following surgery, the patient was admitted to the ICU and remained on a ventilator for 10 days.

Twenty days following the accident, the patient was discharged to a rehabilitation facility. By six months, the patient was able to drive and function adequately at home, but had some limitations at work. The patient also complained of persistent headaches and amnesia.

Annotated Bibliography

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[http://www.braintrauma.org/pdflibrary.nsf/Main/Guidelines/\\$File/Management+and+Prognosis+of+Severe+Traumatic+Brain+Injury+Preview.pdf](http://www.braintrauma.org/pdflibrary.nsf/Main/Guidelines/$File/Management+and+Prognosis+of+Severe+Traumatic+Brain+Injury+Preview.pdf) . 2000.

Guidelines for the Prehospital Management of Traumatic Brain Injury.

<http://www.braintrauma.org/guideems.nsf> . 2002.

These internet information sources are detailed guidelines for the management of severe TBI in the pre-hospital and hospital settings. They are a must read, in that they describe individual therapies and rank them using evidence-based medicine techniques. Although the pre-hospital guidelines are able to be printed from the internet, the hospital guidelines can only be viewed from the website.

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Langham J, Goldfrad C, Teasdale G, Shaw D, Rowan K: Calcium channel blockers for acute traumatic brain injury. *Cochrane.Database.Syst.Rev.2000.;*(2.):CD000565.

These Cochrane reports are excellent sources of information regarding specific therapies that can be used by the Emergency Physician. These reports should be accessed whenever considering the utility of any acute, including those that are potentially useful in the management of sports-related severe traumatic brain injury.

3. Barron DN, Levitt M, Clements R: Head Trauma and Subdural Hematoma: Part II: Emergency Management of Severe, Moderate, and Minor Head Trauma. *Emergency Medicine Reports* 1 A.D.;22:299-314.

This EM Reports addresses practical issues in the management of TBI by the Emergency Physician. Many of the items discussed in this document are based on the guidelines described above.

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These guidelines, which are based on the clinical management of severe TBI in Italy, are especially useful when considering criteria for surgical treatment and prognostic factors that can be evaluated by the Emergency Physician in the ED. They complement well the information provided in the BTF guidelines and the information provided in the Cochrane reports.

Questions

1. All of the following are true about the initial resuscitation of severe TBI patients **except**:
 - a. No firm BTF guidelines exist as to exact initial resuscitation requirements.
 - b. RSI using sedatives and paralytics is recommended.
 - c. Intracranial hypertension should be assumed and treated in all patients.
 - d. Resuscitation should initially correct hypotension and hypoxia.
 - e. Target SBP should be > 90 mmHg, and target pO₂ should be > 60 mmHg

2. All of the following are true about hyperventilation in severe TBI patients **except**:
 - a. When ICP is likely normal, never achieve a pCO₂ below 25 mmHg.
 - b. Prophylactic hyperventilation to a pCO₂ < 35 mm Hg is recommended.
 - c. Brief episodes of hyperventilation are never recommended.
 - d. Hyperventilation should be considered with acute neurological deterioration.
 - e. It is not clear that hyperventilation improves outcome in severe TBI.

3. All of the following are true about ICP management in severe TBI patients **except**:
 - a. Barbiturates are a standard of care in increased ICP management.
 - b. Steroids are not indicated in the treatment of increased ICP.
 - c. Calcium channel blockers are not a standard for increased ICP patients.
 - d. Mannitol can be given as boluses up to 1 gram per kg body weight.
 - e. ICP monitoring is suggested when the GCS score is < 9 .

4. All of the following are indications for operative intervention in TBI **except**:
 - a. Focal intracranial lesion with > 5 mm midline shift.
 - b. Space-occupying intracranial lesion with a volume > 25 cc.
 - c. ICP > 20 mmHg or CPP < 70 mmHg following medical therapy.
 - d. GCS scale score < 9 with cerebral swelling.
 - e. Sudden neurological decompensation with likely uncal herniation.

5. All of the following suggest a worse outcome in TBI **except**:
 - a. Advanced age and low GCS score.
 - b. Abnormal pupil exam.
 - c. Hypotension and hypoxia.
 - d. Compressed basal cisterns, SAH, midline shift, and ICH on CT
 - e. Concomitant substance abuse in the setting of severe TBI.

Answers

- 1. Answer C** The BTF guidelines state that intracranial hypertension treatment should be delayed unless herniation and/or rapid neurological deterioration are suspected clinically.
- 2. Answer C** Brief episodes of hyperventilation are recommended in the setting of acute neurological deterioration when other medical therapies have failed..
- 3. Answer A** There are no standards regarding high dose barbiturates in patients with increased ICP. Barbiturates can be used when other therapies fail, but significant hypotension may offset any beneficial effects of this therapy in improving outcome.
- 4. Answer D** There is no specific indication for operative intervention based only on the GCS score.
- 5. Answer E** There is no definitive information that suggests that concomitant substance abuse significantly impacts outcome in severe TBI.