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Acute head trauma presents a number of diagnostic and therapeutic challenges for the emergency physician (EP). In the past, attention has been paid to preventing and managing the initial (primary) brain injury. Recent studies have recognized the importance of secondary brain injury caused by post-traumatic factors contributing to cerebral ischemia (hypotension, hypoxia, etc.). This understanding has given the EP a principle role in preventing ongoing cerebral injury by reducing the severity of secondary insults. Timely and appropriate interventions can prevent further cerebral compromise and significantly improve patients outcome. To provide optimal care, the EP must recognize which patients are most at risk for having intracranial injury and, therefore, require more extensive evaluation and treatment.

The purpose of this article is to review the epidemiology, pathophysiology, clinical presentation, and management of blunt and penetrating head trauma in the adult and pediatric populations. The principles presented should assist the EP to individually tailor the management of head-injured patients. While the scope of head trauma also includes prevention and injury to the eyes, facial bones, and neck, these issues will not be discussed.

The Editor

Traumatic Brain Injury: State-of-the-Art Protocols for Evaluation, Management, and Resuscitation

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Epidemiology

Acute head trauma/traumatic brain injury (TBI) is common, with approximately 2 million incidents per year in the United States, resulting in 52,000 deaths, 220,000 hospital admissions, and 100,000 people sustaining temporary or permanent disabilities.[1-4] Advances in primary prevention (airbags, restraint-belt compliance, helmet laws) have contributed to a decline in death due to TBI by 22% over the past two decades. However, almost 34,000 people die annually prior to arrival at the ED.[2,3] Of those who survive to reach the ED, 80% have minor head trauma, with a post-resuscitation Glasgow Coma Scale (GCS) score of 13-15; 10% have moderate head trauma (GCS 9-12); and 10% have severe head trauma (GCS less than or equal to 8). *(See Table 1.)*[1,5] Mortality correlates inversely with GCS and is generally worse among children younger than 5 and adults older than 60 years of age.[6,7] Death attributable to TBI is 3.4 times more common in males and peaks in two age groups: 15-24, and 65 and older.[2] For patients younger than 25 years of age, TBI remains the most common cause of traumatic death.[8]

While blunt TBI is much more common than penetrating TBI, in 1990, firearms surpassed motor vehicle crashes (MVCs, both vehicle vs vehicle and pedestrian vs vehicle) as the most common cause of head-injury related death.[2]

Associated Injuries. Up to 60% of patients with blunt TBI have an associated systemic injury, and the mortality of those with an associated systemic injury (22%) is double the mortality of those with an isolated head injury (11%).[9,10] Cervical spine (c-

spine) injuries occur in 1.2-15.0% of head-injury cases, most commonly in those with a lower GCS.[11-14] Extracranial injuries increase mortality as a result of hypotension and hypoxia. This increase is most dramatic in patients with a post-resuscitation GCS of 7-12, because associated injuries add little to the already high mortality of patients with more severe head injury.[9,15,16] For this reason, it is critical to be vigilant about the diagnosis and treatment of associated injuries in patients with head trauma that is moderate or at the high end of severe in order to prevent unnecessary hypotension and hypoxia.

The EP also must be aware of systemic complications associated with injury to the brain. Disseminated intravascular coagulation (DIC), which is more common with penetrating TBI but is present in up to 90% of cases of severe blunt TBI, can occur from activation of the extrinsic coagulation cascade by exposure of brain tissue thrombplastin to circulating blood.[17,18] DIC may precipitate delayed intracranial hemorrhage; accordingly, an initially stable patient with DIC who deteriorates must have a head CT to rule out this complication, even if he/she previously had a negative head CT.[19,20]

Neurogenic pulmonary edema leading to adult respiratory distress syndrome can develop either quickly or on a delayed basis (minutes to days) after the initial brain injury and can be reversed by lowering the elevated intracranial pressure (ICP) that causes this condition.[21] ECG changes may occur in up to 50% of patients with intracranial hemorrhage, resulting from an increase in left ventricular pressure following increased intracranial pressure and from elevated levels of circulating catecholamines. The most common ECG changes are supraventricular tachycardia, prolonged QT interval, diffuse large upright or deeply inverted T waves, ST segment depression, and U waves.[21-23] The above conditions should be managed to ensure adequate cerebral perfusion and oxygenation.

Mechanisms of Brain Injury

Head trauma causes injury to the brain via two mechanisms direct and indirect both of which can contribute to primary and secondary brain injury.[24] Direct injuries occur from the initial impact of an object with the skull. Damage can occur either directly beneath the involved area causing a scalp laceration, skull fracture, epidural hematoma, or brain contusion from coup injury or, damage can be removed from the impact site as propagation of impact energy through the brain disrupts distant structures. Indirect injury occurs when cranial contents are set in motion within the skull; the brain remains attached to the cranial vault by its meningeal and vascular attachments and is buffered by CSF. During such acceleration-deceleration injuries, vessels bridging the cranium to the brain are torn and axonal integrity is disrupted by shear and strain forces, causing subdural hematomas, diffuse axonal injury (DAI), and concussions.[24] A contra-coup injury combines elements of both direct and indirect injuries: the brain sustains damage as it moves inside the cranium and hits the inner surface of the cranium opposite the initial site of impact.

Both direct and indirect mechanisms can produce primary and secondary brain injury. Primary injury is the initial irreversible mechanical injury from impact or shearing. This includes brain lacerations, DAI, intracerebral hemorrhages (not hematomas), contusions, and brain tissue avulsions.[24] Areas of the brain suffering irreversible primary injury are surrounded by a penumbra of tissue that is injured but potentially salvageable.[25] Expert and efficient care by the EP can have a significant impact on the patient s quality of life by sustaining viability of these tissues.

After initial direct and/or indirect mechanisms cause primary brain injury, subsequent secondary insults may occur resulting in ischemic brain injury. This is the leading cause of brain death from TBI.[26,27] The importance of secondary injury is suggested by reports of patients who talk and deteriorate, implying the primary injury is not the sole determinant of the patient s neurologic outcome.[28-30]

Secondary Injuries. Secondary injuries represent, for the most part, ischemic injuries that result from diverse physical or metabolic insults to the brain. These include decreased cerebral perfusion pressure (CPP), hypotension, hypoxia, anemia, and seizures, all of which impair oxygen delivery to the brain and thereby contribute to cerebral ischemia. CPP is a function of intracranial pressure and mean arterial pressure (CPP = MAP-ICP) and should be maintained at greater than 70 mmHg.[31] This figure is derived from observations of increased mortality of head-injured patients with

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Table 1. The Glasgow Coma Scale

EYE OPENING	SCORE	SIGNIFICANCE
Spontaneously	4	Reticular activating system is intact; patient may not be aware
To verbal command	3	Opens eyes when told to do so
To pain	2	Opens eyes in response to pain
None	1	Does not open eyes to any stimuli
VERBAL RESPONSE	**SCORE**	**SIGNIFICANCE**
Oriented-converses	5	Relatively intact CNS; aware of self and environment
Disoriented-converses	4	Well articulated, organized, but patient is disoriented
Inappropriate words	3	Random, exclamatory words
Incomprehensible	2	Moaning, no recognizable words
No response	1	No response or intubated
MOTOR RESPONSE	**SCORE**	**SIGNIFICANCE**
Obeys verbal commands	6	Readily moves limbs when told to
Localizes painful stimuli	5	Moves limb in an effort to remove painful stimuli
Flexion withdrawal	4	Pulls away from pain in flexion
Abnormal flexion	3	Decorticate rigidity
Abnormal extension	2	Decerebrate rigidity
No response	1	Hypotonia, flaccid; suggests loss of medullary function or concomitant spinal cord injury

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systolic blood pressure lower than 90 mmHg or intracranial pressure greater than 20 mmHg. Head injury may cause an increase in ICP through several mechanisms: 1) hyperemia, which may direct more blood toward the injured cerebrum; 2) cerebral edema, which is exacerbated by impairment of the blood brain barrier; 3) intracerebral hemorrhage; and 4) extra-axial hematomas. Each of these mechanisms adds more volume to the fixed-space cranium, which results in higher intracranial pressure.

Cerebral oxygen delivery is also threatened immediately post-trauma by the loss of normal cerebral vascular autoregulation, an incompletely-understood process by which cerebral blood flow (CBF) is maintained essentially constant over a range of MAP (from 50-150 mm Hg) and CPP. In brains suffering from even minor traumatic injury there may be a loss of normal autoregulation.[32] The autoregulatory curve is shifted to the right, which requires that the MAP or CPP be above normal to attain normal CBF.[33-35] CBF is lowest for the first 24 hours post-injury and increases over the next three days in patients who do not die from refractory elevated ICP.[31]

The importance of preventing secondary injury is supported by the substantial contribution that hypotension, hypoxia, and anemia make to mortality. In a landmark study, Chesnut investigated patients with a TBI post-resuscitation GCS score of 8 or less from the time of injury through resuscitation and found that hypotension (SBP < 90) occurred in 35% (11% isolated, without concurrent hypoxia) and was associated with a 150% increase in mortality. Hypoxia (PaO_2 < 60) occurred in 45% of patients (22% isolated) and also significantly increased mortality, although not to the extent that hypotension did. Combined hypotension and hypoxia occurred in 23% of cases and was more detrimental than either alone.[15] Because significant trauma can cause both significant cerebral and systemic injuries, those with lower initial GCS scores are more likely to have hypotensive events.[16] The EP must appreciate the greater likelihood of associated injuries that could cause hypotension in patients with lower GCS scores.

Clinical Presentation

History and Physical Examination. Evaluation and treatment of head trauma adheres to the same fundamental principles used in managing all patients with trauma: consideration for the airway, breathing, and circulation (ABCs) with special attention to c-spine immobilization. The previous section highlighted the need for complete and thorough resuscitation to prevent hypotension and hypoxia. While even one episode of hypotension or hypoxia may be associated with a significant increase in mortality, the risk rises with prolonged duration and increasing frequency of these events.[16] Consequently, it is of paramount importance to prevent further occurrences. All patients with a history of head trauma should have their cervical spine immobilized, preferably in the prehospital setting, but certainly upon reaching the ED or until their c-spine can be cleared clinically or radiographically.

Upon the patient s arrival in the ED, important historical points must be elicited from the patient, emergency medical service personnel, family members, and/or witnesses. *(See Table 2.)* This information is important for two reasons: 1) risk-stratifying the patient for severity of injury to determine need for immediate management and more focused physical exam and ancillary studies; and 2) prognostic purposes, (e.g., predicting the increased mortality with in-the-field hypotension). For example, there is added risk of brain injury in MVCs associated with intrusion into the vehicle compartments, required extrication from the vehicle, and side-impact collisions (presumably due to the proximity of the head to the side window compared to the windshield).[9] These historical points should raise the suspicion of more severe head injury in a patient with seemingly minor trauma. Standard laboratory tests to be obtained include a CBC, electrolytes, liver function tests, BUN, creatinine, urinalysis, type and screen/cross, and DIC profile (PT/PTT/fibrinogen/D-dimers). Other tests to consider are a blood alcohol level and urine drug screen.

Table 2. Important Historical Information

A. Mechanism of injury:

1) Falls: a) height of fall, b) position landed in, c) duration of loss of consciousness or amnesia, if any
2) Motor Vehicle Crashes: a) vehicle vs. vehicle or pedestrian vs. vehicle, b) driver or passenger, c) type of vehicle (car, van, bus), d) restrained or unrestrained, e) airbag deployed, f) speed of vehicle(s), g) frontal or side impact, h) how much compartment intrusion, i) was patient thrown from the vehicle and how far, j) roll-over accident, k) extrication time, l) head impact, m) duration of loss of consciousness or amnesia, if any
3) Blunt Object/Assault: a) type of object (bat, fist, etc.), b) number of blows, c) duration of loss of consciousness or amnesia, if any
4) Penetrating Injury: a) type of object, b) number of gunshots heard/felt, c) distance from weapon at time of assault, d) duration of loss of consciousness or amnesia, if any

B. Vital signs at scene and in transport

C. Initial GCS

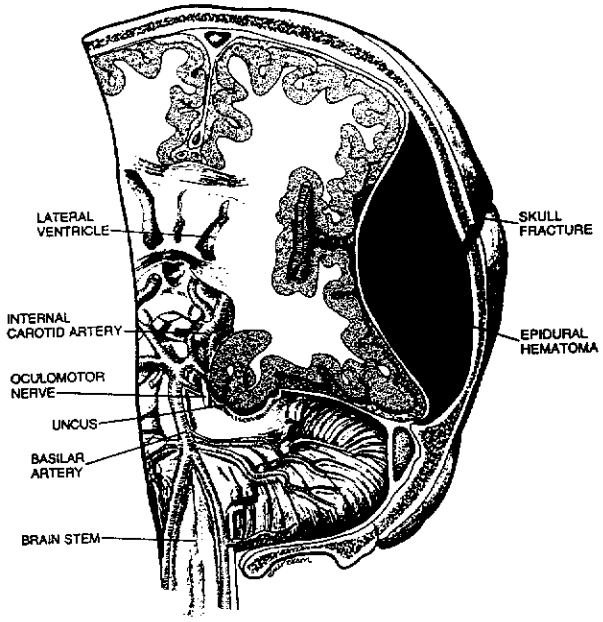
D. AMPLE history: Allergies, Medications, Past medical history, Last meal, ETOH/Other drugs

E. Post-traumatic events: cardiac arrest, apnea, seizures, vomiting

F. Patient's current condition, especially compared to baseline

G. Patient's focal complaints; what the patient remembers before and after the accident

Figure 1. Illustration of Skull Fracture and Epidural Hematoma



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Specific historical findings encountered in general and neurologic examinations should alert the physician to the presence of rising ICP and the need for emergent diagnostic and therapeutic intervention. For example, persistent vomiting, severe headache, or papilledema suggest an early increase in ICP. Altered mentation (GCS Eye < 4 or GCS Verbal < 5) may be due to many causes, including DAI, unilateral lesions with mass effect on the contralateral cerebral hemisphere, and injury to or pressure on the reticular activating system in the brainstem. Systemic causes of altered mentation include hypotension, hypoxia, hypoglycemia, acid/base/electrolyte disturbances, and hypothermia.

Anisocoria (asymmetric pupils) suggests early ipsilateral uncal herniation due to an expanding extra-axial hematoma that causes compression of the third cranial nerve.[24] *(See Figure 1.)* Uncal herniation is the most common herniation syndrome and progresses from anisocoria and sluggish pupils to fixed, dilated, and nonreactive pupils. Motor findings are normal early in this syndrome, but typically evolve from compression of the ipsilateral cerebral peduncle, which produces a contralateral Babinski reflex, hemiparesis, and decerebrate posturing. However, Kernohan's notch syndrome occurs in up to 25% of cases; the contralateral peduncle is compressed leading to a Babinski reflex, hemiparesis, and decerebrate posturing ipsilateral to the mass lesion and blown pupil.

Pinpoint pupils (< 2 mm) and signs of upper motor neuron damage (bilateral weakness, increased muscle tone, and Babinski reflexes) are the initial manifestations of the central transtentorial herniation syndrome caused by an expanding lesion at the top of the brain or at the frontal or occipital poles. These signs progress to mid-fixed, nonreactive pupils, sustained hyperventilation, decorticate then decerebrate posturing, and eventually slow and shallow respirations. Pinpoint pupils and flaccid quadriplegia with sudden cardiopulmonary failure characterize the cerebellotonsillar syndrome in which the cerebellar tonsils herniate through the foramen magnum and rapidly displace the brainstem. If left untreated, each of these syndromes will progress to death from cardiopulmonary arrest. Another manifestation of life-threatening increase in ICP is the triad of hypertension, bradycardia, and irregular respirations (the Cushing reflex), which can occur from increased ICP due to any cause but is only present one-third of the time.[11]

Severe Head Trauma

General Principles. Approximately 10% of head trauma patients who survive to the ED have severe head trauma (post-resuscitation GCS ≤ 8 within 48 hours of trauma).[1,36] Severe head trauma has an overall mortality of nearly 40%, and the risk of

dying from this injury correlates inversely with GCS.[1,6] The mortality will be modified in any individual patient by considering other poor prognostic indicators such as age older than 60, pupillary nonreactivity, low GCS Motor score in particular, swelling or mass effect seen on head CT, anemia, hypotension, and hypoxia.[6,15,30,37,38] A preresuscitation GCS (e.g., one done in the field) is not a marker for the severity of head trauma and is not prognostic of neurologic recovery because of interference with assessment of neurologic status caused by hypoxia, hypotension, hypoglycemia, alcohol or other drug intoxication, and the possibility of a surgically evacuable mass lesion.

Although the EP usually has only a preresuscitation GCS to help determine whether to intubate, only a post-resuscitation GCS can be used to judge the severity of injury and prognosis.[39] Other factors that interfere with an adequate assessment of GCS include: ocular trauma and periorbital edema (eye); intubation, pre-verbal children, and non-English speaking patients (verbal); paralytic drugs, spinal cord injuries, and extremity fractures (motor).[24] It is important to recognize that one can have a significant intracranial process and a normal GCS.

Patients with severe head trauma will either be in coma or unconscious but arousable. These patients usually are incoherent and unable to follow commands. They may have focal neurologic deficits as a result of direct or indirect injury. Signs of elevated ICP as described may be present. As with all trauma, emphasis must be placed first on managing the ABCs.

Intubation. In managing the ABCs, all patients with severe head trauma should be intubated. This is performed after examining and clearing the airway of debris, loose teeth, and regurgitated food. Intubation is necessary because of the likelihood of impending loss of consciousness (LOC) and subsequent inability to maintain a patent airway and adequate respirations. Intubation and paralysis have the additional benefit of reducing cerebral and whole-body oxygen requirements. Rapid-sequence intubation (RSI) should be performed with an oral endotracheal tube, while holding in-line c-spine immobilization, provided there are no contraindications to this technique.[40] *(See Table 3.)* The patient should be preoxygenated with 100% O_2 through a bag-valve mask while an assistant maintains pressure on the cricoid cartilage (Sellick maneuver) to decrease the amount of air entering the stomach; the patient should be maintained on 100% O_2. Pretreatment with fentanyl blunts the elevation in heart rate and MAP that occurs with endotracheal intubation, and lidocaine attenuates the rise in intracranial pressure. Because succinylcholine, which may be given later, causes a transient elevation of intracranial pressure, a defasciculating dose of vecuronium should be given during pretreatment. While waiting for the vecuronium to take effect, oxygenation should be continued and the EP should perform a rapid neurologic assessment prior to intubation and paralysis, including pupillary size and reactivity, movement of extremities, and the presence or absence of corneal reflexes. Documentation and description of these findings is useful to neurosurgical consultants and transfer facilities, and may also help detect spinal cord injury and paralysis that may otherwise go unnoticed. This brief examination essentially comprises the disability portion of the primary survey.

Induction is best achieved by thiopental, which decreases ICP, blunts the ICP response to intubation, and decreases the cerebral metabolic rate, thereby matching cerebral oxygen demand and supply. Because of its propensity to cause hypotension, if the patient is not hemodynamically stable, a smaller dose or alternative agent should be used. In the hypotensive patient, etomidate (0.15-0.30 mg/kg IV) should be considered because of its minimal cardiovascular effects. If neither thiopental nor etomidate is available, midazolam (0.1-0.3 mg/kg) may be used. Ketamine is contraindicated because it increases cerebral cortical activity and oxygen requirements. Paralysis is achieved with succinylcholine. Patients without known elevated ICP and without signs of elevated ICP should be ventilated to achieve a $PC0_2$ of 35-45 mmHg (eucapneic), especially in the early post-trauma phase.[41]

Hemodynamic Resuscitation. Once airway and breathing are stabilized, attention must be paid to the patient s hemodynamic status. In adults, isolated head injury alone does not cause hypovolemic shock; therefore, the presence of hypotension in the head trauma patient mandates a vigorous search for internal and external bleeding (e.g., open fracture). SBP should be maintained at least 90 mmHg and, preferably, between 120-140 mmHg.[31] Normal saline (NS) or lactated Ringer s solution (LR) are appropriate resuscitation fluids. However, evidence is accumulating favoring the use of 250 mL of 7.5% hypertonic saline or 7.5% hypertonic saline/6% Dextran 70 (HSD) as the initial resuscitation fluid in hypotensive patients with a GCS score lower than 8.[42] In this regard, a cohort analysis of several studies comparing 250 mL of HSD to 250 mL of NS or LR found that patients resuscitated with HSD had decreased fluid requirements and higher rates of survival to discharge.[43] HSD may improve survival by decreasing ICP.[44] Persistent hypotension following the initial bolus of HSD should

Table 3. Rapid Sequence Intubation (RSI)

1. **Preoxygenation**
   1. Administer 100% oxygen by bag-valve mask for 5 minutes or four vital capacity breaths
   2. Hold cricoid cartilage pressure (Sellick maneuver)
2. **Pretreatment**
   1. Fentanyl (3-5 mcg/kg IV)
   2. Lidocaine (1.5 mg/kg IV)
   3. Vecuronium (0.01 mg/kg IV)
3. **Wait 2-3 minutes (if possible)**
   1. Continue preoxygenation
   2. Perform rapid neurologic exam
4. **Sedation/Induction**
   1. Thiopental (3-5 mg/kg IV; 0.5-1 mg/kg if hypotensive)
   or
   Etomidate (0.15-0.3 mg/kg if hypotensive)
   or
   Versed (0.1-0.3 mg/kg)
5. **Paralysis**
   1. Succinylcholine (1.5 mg/kg IV)
6. **Intubation with c-spine immobilization**
7. **Immediately after Intubation:** Release Sellick maneuver, institute positive pressure ventilation, and consider longer-term sedation and neuromuscular blockade; maintain on 100% O_2

Adapted from: Walls RM. Rapid-sequence Intubation in head trauma. *Ann Emerg Med* 1993;22:1008-1013.

Table 4. The Acute Neurologic Examination in the Severely Head-Injured Patient

- Primary Survey
  - Airway, breathing, circulation
- Level of consciousness
- Mental status
- Glasgow Coma Scale score
- Pupillary size and responsiveness
- Motor examination
  - Strength, symmetry, abnormal movements
- Brain stem function
  - Respiratory rate and pattern
  - Eye movements
  - Oculocephalic response (doll s eyes), if C-spine cleared
  - Oculovestibular response (caloric testing)
- Cranial Nerves
  - Pupillary responses
  - Gag reflex
  - Corneal reflexes
  - Facial symmetry
- Tendon and pathologic reflexes
  - Symmetry
  - Babinski response
- Head and neck
  - External signs of trauma
  - Signs of basilar skull fracture
- Cervical and thoracolumbar spine
  - Deformity, step-offs

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be treated with NS or LR.[45] Considering its benefits, HSD is underused in the prehospital and ED resuscitation of head-injured patients.[41] Blood should be given for refractory hypotension or severe anemia with the goal of achieving a hematocrit of 30-33%, which is optimal for rheological considerations.[41] The patient should be on maintenance IV fluids (NS or LR) after hemodynamic stability is achieved. Mannitol is not an appropriate resuscitation fluid because it causes osmotic diuresis.[45]

Once respiratory and hemodynamic stability have been achieved, the physician can perform the secondary survey, which includes a more thorough acute neurologic exam.[24] *(See Table 4.)* The head of the patient s bed should be elevated to 30 to decrease ICP by encouraging cerebral venous return.

Elevated Intracranial Pressure. If a patient has elevated ICP (> 20 mmHg) that may be confirmed from an ICP monitor or is assumed from the clinical findings of intracranial hypertension, immediate intervention is required to correct the problem and maintain normal ICP. Recently, many therapeutic options have been analyzed and categorized as Standards, Guidelines, and Options by the Brain Trauma Foundation report *Guidelines For The Management Of Severe Head Trauma*.[31] If the patient already has a ventriculostomy, acute reduction should be attempted first by ventricular drainage. Hyperventilation to an initial $PC0_2$ =30-35 mmHg immediately lowers ICP, as decreased $PC0_2$ and consequent alkalosis cause cerebral vasoconstriction and decreased CBF, thereby limiting the amount of intracranial space occupied by blood vessels and reducing the rate of edema formation. Hypocapnia also restores the normal cerebral autoregulatory curve.[46] However, use of hyperventilation to lower ICP must be done with caution because reduced CBF can reach ischemic thresholds, and will do so more quickly in the presence of subdural hematomas, diffuse cerebral injuries, and hypotension. Hyperventilation should never be performed in the absence of confirmed or highly-suspected elevated ICP, as severely head-injured patients who receive prolonged prophylactic hyperventilation have worse outcomes at three and six months after injury than those who do not receive this treatment.[47]

If an initial post-traumatic head CT was negative for a mass lesion, it should be repeated in the presence of signs and symptoms of elevated ICP; an abnormality may have developed in the interim. Any identified surgically amenable lesion should be evacuated. If elevated ICP persists and the patient is not hypotensive, mannitol boluses (0.25-1 g/kg) should be given. Mannitol boluses raise CBF by: 1) elevating MAP as an initial volume expander; 2) reducing ICP through osmotic cerebral dehydration lasting 90 minutes to six hours; and 3) reducing blood viscosity through hemodilution, which encourages microvascular flow.[41,48,49] Smaller doses and boluses are preferable to continuous infusion because mannitol opens the blood brain barrier, allowing it and other small osmotically active solutes to pass into the brain; their accumulation there eventually causes a reverse osmotic shift, drawing fluid into the brain and potentially exacerbating cerebral edema. This effect is most pronounced when mannitol is in circulation for prolonged periods, such as with continuous infusions. Mannitol should not be given if the serum osmolality is greater than 320 mOsm because renal toxicity increases at these levels.[41]

If hyperventilation and mannitol fail to reduce ICP, second-line therapy may be initiated. One option is the administration of high dose pentobarbital therapy (barbituate coma) in hemodynamically stable patients with a loading dose of 10 mg/kg over 30 minutes followed by 5 mg/kg/h × three hours and then a maintenance dose of 1 mg/kg/h. Hyperventilation to a PCO_2 less than 30 mmHg may be attempted but only with monitoring of cerebral oxygen extraction, jugular venous oxygen saturation, or cerebral blood flow to tailor the therapy to avoid cerebral ischemia. There is no indication for prophylactic barbituate use or glucocorticoid (steroid) use at any time in the treatment of TBI, because neither has been shown to improve patient outcome.[50-52]

Maintaining hypothermia is not discussed in the *Guidelines For The Management Of Severe Head Trauma* among the treatment options for elevated ICP. Two studies show hypothermia (T= 32-33 C) may be the safest and most effective means of managing elevated ICP because it reduces cerebral oxygen requirements.[53,54] Combining hypothermia with paralysis prevents the shivering response that raises temperature and systemic oxygen requirements. However, hypothermia has poor prognostic implications in trauma and has the potential to cause deleterious systemic effects (e.g., bradycardia, PEA, coagulation defects).[41]

Radiographic Studies. Each patient with severe head trauma

Table 5. Who to Scan

1. All GCS < or = 13
2. GCS = 14-15 if High Risk
3. Consider head CT for any LOC

HIGH RISK	LOW RISK
Focal neurologic findings	No focal findings
Asymmetric pupils	Normal pupils
Skull fracture	No other injuries
External signs of trauma	Trivial mechanism
Multiple trauma	Accurate history
Serious painful distracting injury	Injury > 24 hours ago
Unreliable/unknown history of injury	Initial GCS = 14-15
Initial GCS = 13	No change in consciousness
LOC	Intact orientation/memory
Post-traumatic amnesia	Currently asymptomatic
Progressively worsening headache	Reliable home observers
Vomiting	
Post-traumatic seizure	
History of bleeding disorder/anticoagulants	
Recent ingestion of intoxicants	
Suspected child abuse	
Age > 60, < 2 years old	

Adapted from: Biros, MH. Head trauma. In: Rosen P, et. al. eds. *Emergency Medicine: Concepts and Clinical Practice.* 4th ed. St. Louis: Mosby; 1998:416-447.

should undergo cranial CT, including bone windows, because of the high incidence of associated intracranial lesions. *(See Table 5.)* Up to 25% of patients with a GCS score of 8 or lower will have a lesion warranting neurosurgical intervention.[24] The most common findings are subdural hematomas (SDH), which occur in up to 30% of severe head trauma patients.[36,55,56] Epidural hematomas occur in 0.5% of all head-injured patients and in approximately 1% of those who present in coma; in 40% of cases, they are associated with other intracranial lesions, most commonly SDH, and 80% occur at fracture sites over the middle meningeal artery or a dural sinus.[36,57,58] Contusions, traumatic subarachnoid hemorrhage (SAH, 33% of all severe head trauma), intracerebral hematomas (at least 12% of all severe head trauma), and subdural hygromas (10% of all severe head trauma) are other common findings on initial head CT.[24,59,60]

The head CT should extend to visualize C_1 and C_2, inasmuch as approximately 14% of patients with blunt head trauma and a GCS less than 6 have fractures of either C_1 or C_2; 40% of these are missed by plain radiographic c-spine series.[61] Skull fractures occur with increasing frequency as the severity of head injury increases; however, because patients with severe head trauma will undergo head CT, including bone windows, a plain skull radiograph is unnecessary.[62,63]

Neurosurgical consultation should be obtained for all patients with severe head trauma. Because almost 60% of patients with severe head trauma have associated systemic injuries, the ED physician frequently must prioritize between the need for diagnostic procedures, such as diagnostic peritoneal lavage or abdominal CT, and the need for head CT or burr holes. Chesnut has presented a model to resolve these dilemmas that considers both cerebral and systemic priorities.[41] Patients in category A have ongoing evidence of intracranial hypertension and require an emergent head CT or cranial decompression to prevent herniation and death. If the patient is hemodynamically unstable, these patients may be served best by resuscitation in the operating room while surgeons decompress the cranium. Patients in category B have no evidence of ongoing herniation. Primary survey, resuscitation, secondary survey, and diagnostic procedures may be performed prior to head CT so long as they are accomplished within 15-30 minutes. Patients in category C have head injury which is not of immediate threat to the patient. They may wait for a head CT, but careful and frequent neurologic assessment is mandatory to detect deterioration from an expanding mass lesion.[28-30] Transfer for diagnostic imaging is often accompanied by periods of physiologic instability for the patient. *(See the figure on the rapid reference card inserted with this issue for a protocol outlining procedures for safe transport of these patients.)*[41]

Severe head trauma is one of several reasons to administer prophylactic anticonvulsants to head-injured patients. A complete list of indications is presented in Table 6.

Moderate Head Trauma

Almost 10% of head-injured patients who present to the ED have moderate head trauma (post-resuscitation GCS = 9-12) and nearly 20% die as a consequence of their injury.[64] These patients have the most to gain by expeditious ABC management; it is therefore important to be vigilant in the prevention of secondary brain injury.[9,16] Standard (and optional) laboratory tests as required in severe head trauma should be obtained.

Confusion, lethargy or somnolence, seizures, focal deficits, amnesia, headaches, and vomiting mark the varied clinical presentation of moderate head trauma.[24] Patients with moderate (and minor) head trauma should be intubated if the clinical context warrants such intervention; intubation is required for an expanding neck hematoma, hemodynamic instability, or inability to maintain a patent airway because of loss of gag reflex. After managing the ABCs, the clinician should obtain a head CT since nearly 40% of patients with a GCS as high as 13 (on the border between moderate and mild trauma) will have an abnormal CT.[65]

Patients with moderate head trauma should be admitted for a period of observation even if they have a normal head CT. The typical course (90%) for these patients is neurologic improvement over the next 48 hours. A repeat head CT should be performed on patients who deteriorate or fail to improve during that time.[36]

During the three immediate post-traumatic months, patients frequently experience prolonged periods of disability including inability to work (up to 70%) secondary to chronic headaches (up to 90%) as well as difficulties with memory and concentration (up to

Table 6. Indications for Acute Seizure Prophylaxis in Severe Head Trauma

Severe head injury (GCS < or =10)
Paralyzed and intubated patient
Seizure within 24 hours after injury
Depressed skull fracture
Penetrating head wound
Cortical contusion
Acute subdural hematoma
Acute epidural hematoma
Acute intracranial hemorrhage
Prior history of seizures
In children add: Diffuse cerebral edema

Adapted from: Biros MH. Head trauma. In: Rosen P, et al. eds. *Emergency Medicine: Concepts and Clinical Practice.* 4th ed. St. Louis: Mosby; 1998:416-447. Bullock MR, Chesnut RM, Clifton G, et al. Guidelines for the management of severe head injury. *J Neurotrauma* 1996;13:639-734.

Table 7. Head Injury Precautions

The patient should return immediately to the emergency department under any of the following circumstances:

1. Persistent vomiting or headache
2. Unable to arouse from sleep
3. Development of focal neurologic deficit(s)
4. Lethargy
5. Confusion
6. Amnesia
7. Patient is not acting like himself/herself

90%).[36] Patients with a history and persistent symptoms should be referred for an MRI to detect changes that may not be present immediately after injury.

Skull Fracture

While it is true that having a skull fracture significantly increases the likelihood of having severe intracranial pathology, plain skull radiographs are not a useful screening tool in deciding who should have a head CT, because patients with a normal x-ray can have severe lesions.[65] The decision to perform a head CT should be based on the history, physical exam, and risk factors listed in Table 6.

Not all skull fractures are significant in their own right.[24] However, those found on CT or clinical exam which are significant include: 1) those which overlie the middle meningeal artery or a major dural sinus and, therefore, predispose to EDH; 2) those which are depressed below the level of the inner table of the skull; 3) basilar skull fractures; and 4) those that are associated with intracranial air, pass through an air-filled sinus, or underlie a scalp laceration (open fracture).[66]

Linear fractures are significant only if they predispose to EDH. Depressed skull fractures may cause underlying brain injury, infections, and seizures. They should be palpated carefully to avoid pushing bone further under the skull table.[24] Basilar skull fractures are linear fractures at the base of the skull, usually through the temporal bone, and are difficult to detect by either plain film or CT. Their presence is suggested by such signs and symptoms as periorbital (raccoon eyes) or retroauricular ecchymosis (Battle s sign); CSF otorrhea or rhinorrhea; cranial nerve deficits due to entrapment; hearing loss from disruption of the otic bones; and a positive ring test in which bloody nasal discharge is placed on filter paper; and CSF (because of its decreased density) migrates further to form a ring around the blood.

The fracture generally heals within one week of injury, and, therefore, prophylactic antibiotics are not given. Patients without other reasons for admission do not require admission for basilar skull fractures alone because the complications (meningitis and osteomyelitis) are delayed.[24,62] Open skull fractures underlie a scalp laceration or disrupt a sinus or the middle ear. These require irrigation and debridement without blind probing.[24]

Scalp Lacerations and Concussion

Scalp lacerations can cause significant bleeding. Methods of hemostasis include direct compression of the bleeding vessel against the skull, injection of wound edges with lidocaine with epinephrine, and clamping of observed bleeding vessels.[24] Irrigation and debridement should be done carefully to avoid depression of underlying fractures. Hair in the wound, but not surrounding the wound, should be removed. A disrupted galea must be repaired, and the skin, dermis, and galea can generally be taken with one needle bite of 3-0 nylon or polypropylene in an interrupted or vertical mattress suture.[67] Because of the rich vascular supply of the scalp, properly managed wounds do not require antibiotics. However, prophylactic antibiotics are appropriate in cases of open skull fracture and penetrating head injury and may be considered for complicated scalp lacerations.[68]

Common in both contact sports and accidents/assaults, concussions are a form of minor head trauma resulting in diffuse axonal injury. Transient confusion and amnesia characterize concussion Grades I and II; a loss of consciousness is not necessary to make the diagnosis of concussion. Any LOC is automatically Grade III.

In patients with isolated head trauma and a GCS of 14-15 the mortality rate approaches zero. Those with a GCS of 13 have a low, but measurable, mortality rate.

Low-risk patients with minor head trauma and a normal examination can be discharged from the ED without a head CT after a 4-6 hour observation period. Patients and their home observers should be instructed in head injury precautions outlined in Table 7. Appropriate follow-up should be arranged. Should deterioration occur during observation, patients should have an emergent head CT because a focal mass lesion may have developed during a lucid interval; both EDH and SDH may present in this manner.[24]

Patients with a GCS of 14-15 with a negative scan and who have no other illnesses or injuries necessitating admission can be safely discharged home, provided they have reliable observers who can bring the patient back to the ED if there is deterioration. One study found that among 395 such patients, none had deterioration after discharge; another found that only 0.03% needed any intervention and no patients required craniotomy.[69,70] Because the elderly have a smaller volume of brain tissue, there is more movement of the brain in head trauma. This leads to more indi-

Table 8. Modified Coma Score for Infants and Children

ACTIVITY	SCORE	INFANT S BEST RESPONSE	CHILDREN (<4 YR)
Eyes opening	4	Spontaneous	Spontaneous
	3	To speech	To speech
	2	To pain	To pain
	1	No response	No response
Verbal response	5	Coos, babbles	Oriented-social, smiles follows objects, converses, interacts with environment
	4	Irritable cry	Confused, disoriented, aware of environment, uncooperative interactions, consolable cries
	3	Cries to pain	Inappropriate words, persistent cries, inconsistent awareness of environment, inconsolable
	2	Moans to pain	Incomprehensible sounds, agitated, restless, inconsolable, unaware of environment
	1	No response	No response
Motor response	6	Normal spontaneous movements	Normal spontaneous movements
	5	Withdraws to touch	Localizes pain
	4	Withdraws to pain	Withdraws to pain
	3	Abnormal flexion	Abnormal flexion
	2	Abnormal extension	Abnormal extension
	1	No response	No response

Adapted from: Biros MH. Head trauma. In: Rosen P, et. al. *Emergency Medicine: Concepts and Clinical Practice.* 4th ed. St. Louis: Mosby;1998:416-447; modified from James HE, Trauner DA. The Glasgow Coma Scale. In: James HE, Anas N, Perkin RM, eds. *Brain Insults in Infants and Children.* Orlando: Grune and Stratton;1985 and Rubenstein JS, Hageman JR. Monitoring critically ill infants and children. *Crit Care Clin* 1988;4:631.)

rect injuries and delayed subdural hematomas, which places them at risk for delayed deterioration. Admission for a short period of observation should be considered for elderly patients (age > 60) with a negative initial head CT.[71,72]

Patients with mild head trauma are at risk for many of the same long-term complications as those with moderate head trauma. Post-concussive syndrome includes poor concentration and memory, sleep-wake disturbances, headache, and anxiety and mood disorders which may affect work and relationships. By three months, 30-50% of patients are still limited by symptoms; this improves to only 10-15% by the end of one year.[73]

Patients Who Talk and Deteriorate

The EP usually sees the patient within the first several hours after injury. He or she does not have the best viewpoint of the severity of the patient s head trauma, because severe TBI is defined as a post-resuscitation GCS less than or equal to 8 within 48 hours after injury. Thus, patients may appear to have minor head trauma in the ED yet actually have slow-developing intracranial lesions that, within the next 48 hours, will move them into the moderate or severe categories. This is illustrated by the fact that nearly 3% of minor head trauma patients and 10-25% of severe head trauma patients will deteriorate unexpectedly; this is known as the talk and deteriorate syndrome.[29,30,36,74] These patients have an initial verbal GCS between 3 and 5, but within 48 hours, develop worsening neurologic signs, including altered mental status, focal neurologic findings, and signs of herniation.[75,76] Of those who deteriorate, 80% will have a focal mass lesion, requiring neurosurgical intervention in the majority of cases; about 20% of cases are due to non-focal causes such as diffuse cerebral edema.[30,75] Between 32-45% of patients who deteriorate go on to die from their head injuries.

Because it is difficult to predict which patients with an initial verbal GCS of 3-5 will deteriorate, it is important that the EP employs appropriate CT scanning, ensures close neurologic observation in the ED and at home, and manages deterioration immediately in order to improve the outcome of these patients.

Pediatric Head Trauma

Epidemiology. Each year, almost five million children in the United States sustain head trauma; the incidence peaks in the 5-7 year age group, with an overall mortality rate of 6%.[77-79] Because cranial sutures are still open, the young child s skull is more elastic than adults, allowing a proportionately (but not absolute)

greater accumulation of edema and hemorrhage before elevated ICP causes brain damage. However, this also allows for more direct damage to the brain since there are large areas unprotected by bone.[79] Overall, children with head trauma have lower mortality rates, and those who survive have better outcomes (because of greater neuroplasticity) than adults with similar injuries (same GCS).[7,79,80] However, young children (age < 5) have a higher mortality from head trauma than adults; this is most likely due to delayed presentation of child abuse and the difficulty obtaining an accurate neurologic exam.[24] Children younger than 2 with severe head injury have an especially poor prognosis because of an immature cerebral autoregulatory mechanism and incomplete myelination. As in adults, mortality correlates with GCS.[81]

Because up to 85% of all head injuries due to child abuse occur in children under age 2, child abuse must be suspected in any child in this age group.[24]

Clinical Presentation. As in adult patients with TBI, historical information is important for evaluation of pediatric head trauma. However, the EP should have a high suspicion for child abuse when there is divergence between the history given by parents and the clinical and radiographic findings or when there are inconsistent histories from purported witnesses.[82] Certain historical features are particularly important because they raise suspicion of more severe injury: for example, children who fall from a height less than 3 m are at little risk for morbidity and mortality, while those who fall more than 5 m are most at risk.[83,84] Other significant risk factors include assault with an object at high velocity that imparts a large amount of energy over a small area of skull (e.g., stone, golf club) and LOC greater than five minutes.[85] Children with minor trauma may have more prominent symptoms than comparably injured adults, including dizziness, lethargy, headache, and vomiting.[24] Patients with severe or persistent symptoms should undergo head CT in the ED.

Children with head trauma frequently have other associated injuries, and the same principles used for evaluating and managing adult trauma patients also apply to children. Attention must be paid to immobilizing the c-spine and evaluating and managing the ABCs. Head trauma can cause hypovolemic shock in children from: 1) scalp lacerations, 2) the accumulation of blood from hemorrhage in the subgaleal or subperiosteal spaces; 3) from an epidural hematoma in the elastic cranium; and 4) the drainage of hemorrhage through a functioning shunt in children with previously treated hydrocephalus.[24]

Following resuscitation, a neurologic exam identical to that employed in adults should be performed. This may be difficult in the young child, and the modified GCS presented in Table 8 can help provide a standard by which to gauge the patient s initial neurologic status and its clinical course.[24] It should be stressed that infants and toddlers may have no detectable deficit other than irritability.[79]

Children with severe head trauma are at great risk for having elevated ICP (up to 80%) because they have a steeper intracranial pressure-volume curve than adults; an equal amount of volume added to the cranium produces a larger rise in ICP.[86,87] Clinical findings suggestive of elevated ICP are more subtle in children; infants may have bulging fontanelles, bradycardia, papilledema, decreasing consciousness, and seizures.[24] Therefore, insertion of an ICP monitor is indicated for a GCS of 5 or less or a GCS of 8 or less with CT evidence of mass lesions or brain injury.[87] In particular, children with a functioning shunt can accumulate a significant amount of intracranial bleeding without manifesting signs of elevated ICP.[88]

Measures must be taken to reduce ICP if there is evidence of elevated ICP by clinical exam or ICP monitoring.[87] If the patient has a ventriculostomy shunt, CSF may be drained to relieve ICP; because of the steep pressure-volume curve, the removal of even a small amount of fluid can markedly reduce ICP. Hyperventilation is also effective for reducing ICP. While it was previously thought that profound hyperventilation of children to a PCO_2 as low as 17 mmHg could lower ICP without compromising CBF, recent literature suggests that, as in adults, cerebral ischemia occurs frequently at $PC0_2$ < 35 mmHg.[89,90] Hyperventilation should therefore be used cautiously and with close monitoring of cerebral oxygen extraction.

Mannitol (0.5-1 g/kg) may be given for persistently elevated ICP. Refractory ICP should be treated with pentobarbital with an initial dose of 3-5 mg/kg and continuous infusion at 1-2 mg/kg/hour.[87] When giving pentobarbital, close attention should be paid to hemodynamic monitoring with arterial and central venous or pulmonary artery catheters to follow the patient s course. Should hypotension develop, therapy should be discontinued and aggressive volume expansion initiated.

As with adults, children with severe and moderate head trauma should undergo head CT, as should patients with minor head trauma who fall into a high-risk category outlined in Table 5. Skull films are not useful in children as a screening tool to determine the need for head CT because, as in adults, patients with a negative x-ray can have severe lesions and those with a positive x-ray require a head CT anyway. No plain skull radiographs are needed in patients who have a head CT.

Children are more likely than adults to have diffuse cerebral edema as opposed to focal, surgically correctable, mass lesions.[91] In children, as in adults, EDH carries a better prognosis than SDH. The prognosis is poor when a focal mass lesion and diffuse injury occur in the same patient, though diffuse cerebral injury alone is generally manageable and is associated with a satisfactory outcome.[92,93]

Pediatric patients who have a normal head CT in the ED may be discharged home safely, as approximately only 1% have delayed symptoms requiring admission for neurologic diagnoses within one month from ED discharge.[94]

Skull fractures often occur in infants with a minor head injury accompanied by a scalp hematoma or deep scalp laceration.[95,96] Skull films are indicated in suspected child abuse because of the high incidence of fractures in these cases. Unlike in adults, linear skull fractures in children may be significant because the meninges may extrude through the skull and form a leptomeningeal cyst.[79] Skull films in older children are rarely useful as screening tools and should be performed only to confirm a diagnosis that is clinically suspected, such as a basilar or depressed skull fracture or penetrating injury.[24,97]

Children with low-risk minor head trauma (i.e., no LOC, hemodynamically stable, normal neurologic exam, asymptomatic) may be discharged home without a head CT to reliable parents who have access to a telephone and to whom the warning signs of delayed head injury complications are explained.[98] For children who are suspected to have been abused, the proper local authorities should be notified while the patient is still in the hospital.

Early post-traumatic seizures and status epilepticus occur more frequently in children than in adults, but do not predict future epilepsy. Prophylactic anticonvulsants should be administered in the same high-risk population as in adults (see Table 6) and also in patients with diffuse cerebral edema.[99,100]

Penetrating Head Trauma

Epidemiology. Penetrating head trauma may be caused by either missiles or impalement, and is most commonly due to firearms.[24] The majority of patients are male, and assault is the most common cause. In contrast, many women sustain firearm injuries through attempted suicide.[101] Firearms surpassed MVCs as the most common cause of death due to TBI in 1990, and by 1992, 44% of deaths attributable to TBI were due to gunshot wounds (GSW).[2] Firearms cause an estimated 33,000 deaths per year, and, although nearly 75% of patients with a GSW to the head die at the scene, those who survive to the ED have a 60% survival rate.[39,101-103] Injuries due to suicide attempts have a higher mortality (nearly 95%) than those due to assault.[104] Survival is also related to the GCS and pupillary response; mortality is nearly 100% if the GCS is less than 5, but survival approaches 75% if the GCS is greater than 8 and the pupils are reactive.[102] SDH, EDH, and intracerebral tract hematomas are common in penetrating injury. Survival increases when bullets do not penetrate or cross the ventricles or midline (are unilateral), and when there is minimal midline structural shift (< 10 mm).[101] Injuries due to high-velocity, large, or fragmenting bullets are frequently fatal.[24] Survivors of GSWs to the head generally do well, with up to 67% being in good condition two years after injury and up to 60% returning to their former employment.[101,104]

Pathophysiology. Missile injuries to the head can cause several types of damage.[24] Tangential wounds occur from impact at an oblique angle when the bullet travels under the scalp but never penetrates the cranial vault; cortical contusions may occur beneath the site of impact. Perforating injuries occur when the missile loses significant energy passing through the skull and does not penetrate substantially into the brain; local tissue and vascular damage may result. Penetrating injuries are caused by moderate or high-velocity missiles that may enter the skull and then either exit, lodge within brain tissue, or ricochet off the inner surface of the opposite side of the skull. The bullet passing through brain matter causes a tissue cavity that may be three to four times the diameter of the missile. A shock wave propagates which can cause distant damage, including immediate cardiovascular and pulmonary collapse if the brainstem is affected.[105] Similar pathologic changes occur as in blunt TBI, including breakdown of the blood brain barrier, loss of normal cerebral autoregulation, and alteration of cerebral blood flow.[24]

Clinical Presentation. Similar historical information should be obtained as in blunt TBI. *(See Table 2.)* Determining the distance between the patient and the weapon at the time of injury may be difficult; gunpowder on the clothes or skin of the patient indicates close-range injury. Self-inflicted GSWs are suggested by powder burns and injuries on the hand-dominant side.[106]

Patients with a GSW to the head should be managed in the same manner as other head trauma patients, with initial attention paid to the c-spine and ABCs. Patients with a GCS of 8 or less should be intubated immediately.[24] A thorough secondary survey should be performed because patients with a GSW to the head are likely to have a GSW to other parts of the body as well, putting them at risk for hemodynamic instability.

A head CT is mandatory to identify damaged anatomy and potentially evacuable lesions. If the bullet of a non-intubated patient is seen within the cranial vault, the patient should be intubated immediately rather than await the development of coma.[24] Intravenous antibiotics and anticonvulsants should be given as soon as possible. Early neurosurgical consultation is necessary.

Summary

Acute blunt and penetrating head trauma present as a spectrum of injury and result from a variety of causes. Early recognition of the severity of the injury is necessary for prompt initiation of appropriate diagnostic and therapeutic interventions. The emergency physician should manage first the patient s airway, breathing, and circulation. Successful resuscitation will substantially reduce the severity and frequency of secondary insults.

Reduction of suspected or confirmed elevated ICP is a stepwise process that begins with the methods tailored to avoid cerebral hypoperfusion. Refractory elevated ICP should be managed with more intense and closely-monitored first-line therapy or second-line therapy.

Another challenge for the EP is risk-stratification of those patients with mild head trauma in order to determine which require a head CT. This is not necessary in patients with a GCS 13 or less because all of these patients should have a head CT. Identification and evacuation of mass lesions will further improve patient outcome.

Pediatric patients with head injuries should be approached in the same manner as adult patients, with recognition of the prevalence of child abuse and respect for the steeper intracranial pressure-volume relationship.

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Physician CME Questions

73. The largest single cause of death due to traumatic brain injury (TBI) is:
    A. falls.
    B. automobile vs. automobile collisions.
    C. gunshot wounds to the head.
    D. assault with a blunt object.
    E. pedestrian vs. automobile injuries.

74. Which insult to the brain is a preventable cause of secondary brain injury?
    A. Diffuse axonal injury
    B. Intracerebral hemorrhages
    C. Hypotension
    D. Cerebral contusions
    E. Penetrating brain tissue destruction

75. Typical uncal herniation is indicated by:
    A. anisocoria progressing to fixed dilated and non-reactive pupils and contralateral Babinski reflex, hemiparesis, and decerebrate posturing.
    B. pinpoint pupils and flaccid quadriplegia.
    C. Kernohan s notch syndrome.
    D. hypertension, bradycardia, and irregular respirations.

76. In the absence of evidence of elevated intracranial pressure, initial ventilation of severely head-injured patients should aim for a PCO_2 of:
    A. 20-25 mmHg.
    B. 25-30 mmHg.
    C. 30-35 mmHg.
    D. 35-45 mmHg.

77. Which of the following features makes a patient with minor head trauma at low risk for cerebral damage?
    A. unknown history of injury
    B. initial GCS = 14-15
    C. age = 65
    D. worsening symptoms
    E. initial GCS = 13

78. A patient with moderate head trauma and a negative head CT should be:
    A. sent home with head injury precautions.
    B. re-scanned in 6-12 hours regardless of clinical course.
    C. given prophylactic steroids.
    D. admitted for observation.

79. Child abuse as a cause of head trauma must be suspected particularly in children:
    A. under age 2.
    B. who are beginning to ambulate.
    C. whose parents give a history consistent with laboratory and radiographic findings.
    D. ages 2-5.

80. Patients with gunshot wounds to the head who have the bullet still within the cranium should be:
    A. assumed to have no other penetrating injury because the wound is likely self-inflicted.
    B. provided oxygen by face mask and watched closely for deterioration necessitating intubation.
    C. not intubated for fear of masking developing neurologic signs.
    D. intubated prior to the development of coma.

In Future Issues

Meningitis