




DISEASEDEX™ Emergency Medicine  
Blunt Head Trauma Sample Document

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# BLUNT HEAD TRAUMA

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## Summary

### 0.1 CRITICAL FOCUS

#### A. GENERAL:

1. After ABCs are addressed, management is aimed at controlling and preventing complications of increased intracranial pressure and at rapid neurosurgical decompression of surgically correctable lesions.
2. Protection of cervical spine must be accomplished, as well as priority management of other associated life-threatening injuries.
3. Classic signs and symptoms of head injury can be masked in intoxicated, pediatric, elderly, and multitrauma patients.

B. CERVICAL SPINE IMMOBILIZATION: Any patient with head trauma and altered mental status should be considered to have possible cervical spine injury; immobilize neck until the spine is cleared radiographically.

C. AIRWAY MANAGEMENT: Ensuring a patent airway and initiating effective ventilation is critical.

1. INTUBATION: If airway management is needed, endotracheal intubation is procedure of choice. Rapid-sequence intubation and paralyzing agents are recommended for optimal resuscitation of acutely head-injured patients with suspected increased ICP.
  - a. CERVICAL SPINE CLEARED: Oral intubation may be performed.
  - b. CERVICAL SPINE STATUS UNCERTAIN: Oral intubation in conjunction with in-line stabilization or nasotracheal intubation may be used based on skill and experience of intubationist.

D. IV FLUIDS: In multiply-injured patients, hypotension should be assumed to be secondary to blood loss. Begin NS to maintain BP above 90 mmHg systolic. Avoid routine use of dextrose-containing solutions (potential risk of worsening CNS lactic acidosis and cerebral edema).

E. ANTICEREBRAL EDEMA MEASURES: Treatment modalities may include head elevation, osmotic therapy, diuretics, rapid sequence intubation, barbiturates, hyperventilation, mechanical decompression.

F. NASOGASTRIC TUBE: Indicated to prevent gastric dilatation and to decrease incidence of regurgitation.

G. INTRACRANIAL PRESSURE (ICP) MONITORING: Consider in patients with severe head injury (GCS 8 or less).

H. SEIZURE PROPHYLAXIS: Controversial; decreases incidence of seizures in early post-traumatic period but has no long-term benefit.

I. HEMATOMA EVACUATION: Provides definitive therapy for subdural and epidural hematoma; should be performed as soon as possible.

J. OBSERVATION:

1. Patients with normal mental status and neurologic examination may be observed at home with serial neurologic checks by a responsible adult.
2. Patients with altered mental status require hospital admission for close observation and frequent neurologic checks to detect the development of elevated ICP; consider cranial CT scan.

K. TRIAGE IN MULTIPLE TRAUMA: Decision to proceed to emergent craniotomy vs laparotomy is difficult; ED diagnostic peritoneal lavage or abdominal US, if immediately available, may be helpful in the assessment of BAT.

## 0.2 CLINICAL PRESENTATION

A. MECHANISM OF INJURY: Three mechanisms primarily responsible for brain injury following blunt head trauma include direct tissue deformation, linear/rotary stress, and compression.

1. PRIMARY BRAIN INJURIES: Include concussion, cerebral contusion, brain laceration, shearing injuries, epidural or subdural hematoma, and subarachnoid or intracerebral hemorrhage.
2. SECONDARY BRAIN INJURIES: Pathophysiologic processes leading to further neuronal injury include cerebral edema and hyperemia, increased intracranial pressure, and herniation.

B. EPIDEMIOLOGY: 3% to 4% of persons in the United States sustain head injuries each year; 15% are major. Motor vehicle accidents are common cause; males between 15 and 24 years of age are affected most frequently.

C. CLINICAL FINDINGS: History of head trauma may be obscured in patients with drug or alcohol intoxication. Physical findings may be absent or range from scalp injury only to signs of transtentorial herniation (deep coma, unstable vital signs, fixed and dilated pupils, decerebrate posturing). Associated cervical spine injury and intraabdominal hemorrhage should be suspected, particularly because altered state of consciousness may obscure diagnosis.

1. VITAL SIGNS: Tachycardia is common nonspecific sign. Hypertension with bradycardia (Cushing's reflex) is an ominous sign of elevated ICP. Abnormal respiratory pattern suggests elevated ICP.
2. HEENT: Findings may include retinal hemorrhage, gaze deviation, anisocoria, CSF rhinorrhea or otorrhea, and hearing loss.
3. GI: Nausea and vomiting commonly occur.
4. CNS:
  - a. Glasgow Coma Scale (GCS) should be utilized for serial assessment of head-injured patients. Changes in GCS help predict ultimate prognosis.
  - b. Alteration of mental status may range from mild confusion to deep coma; concussion is commonly associated with amnesia.
  - c. Corneal, oculocephalic, and oculovestibular reflexes may be abnormal, and focal neurologic signs may be present.
  - d. Decerebrate or decorticate posturing may indicate the level of cerebral involvement.

D. COMPLICATIONS: Include postconcussion syndrome (most common sequela), posttraumatic seizures, permanent neurologic deficit (cognitive, motor, sensory), persistent CSF rhinorrhea, hydrocephalus, bacterial meningitis.

## 0.3 DIAGNOSTICS

A. LABORATORY:

1. GENERAL: For isolated blunt head trauma, specific laboratory data, other than ABGs, generally are not of much value.
2. ABGs: Provide invaluable information about adequacy of ventilation and oxygenation in severely head-injured patients.
3. SERUM ALCOHOL LEVEL/TOXICOLOGIC SCREEN: May help differentiate intoxication from head injury or show degree to which these conditions coexist.

## B. RADIOLOGY:

1. CERVICAL SPINE RADIOGRAPHS: Obtain in all cases of significant head injury unless patient is alert, denies neck pain and has no midline tenderness, has no significant distracting injuries, and has a good range of motion of the neck.
2. SKULL RADIOGRAPHS: Little value in management of BHT. Depressed skull fractures and intracranial foreign bodies may be well visualized by skull radiography.
3. HEAD CT SCAN: Definitive diagnostic test because it is both noninvasive and highly accurate in evaluation of intracranial injuries; should be performed in patients with persistent alteration of mental status or focal neurologic deficits.
4. HEAD MRI: No indication in acute traumatic brain injury at this time.

## 0.4 DIFFERENTIAL DIAGNOSIS

A. Includes causes of coma or altered mental status that may have led to traumatic incident, including alcohol and drug intoxication, hypoglycemia, hypoxia, cerebral embolism, infarction, or neoplasm, and seizures.

## 1.0 CLINICAL PRESENTATION

### 1.1 INTRODUCTION

#### 1.1.1 ETIOLOGY

A. MECHANISMS: Three mechanisms are primarily responsible for acute brain injury following BHT.

1. DIRECT TISSUE DEFORMATION: Head is subject to impact injury following acceleration/deceleration and compression injuries. Direct tissue deformation occurs at point of impact and may be associated with injuries to scalp, skull, and brain (Gurdjian, 1978).
2. LINEAR/ROTARY STRESS:
  - a. Brain is less dense than skull and is surrounded by CSF in subarachnoid space. It is therefore free to move at different rate than skull in response to impact (Gurdjian, 1978).
  - b. Motion of brain relative to skull may lead to injuries opposite point of impact (contrecoup lesions), linear and rotary stress on various neuronal and vascular elements, and laceration or contusion of brain as it glides over bony prominences.
3. COMPRESSION: Hematoma formation, vascular dilatation, and edema frequently follow head injury. Because intracranial volume cannot expand, these may cause a shift of brain parenchyma and compression of vital neural elements.

## B. CAUSES:

1. Primary causes are motor vehicle occurrences (including automobiles and trucks, bicycles, motorcycles, pedestrians) (50% of cases), falls (25%), sports and recreational injuries, and direct blows to the head (Jager, 2000; Helling, 1999; Osberg, 1998; Sinson, 1998; CDC, 1997, 1997a; Parkinson, 1985; Gilroy, 1984; Ramos, 1986; Kraus, 1986; Sosin, 1989; Wasserman, 1988; Bachulis, 1988; Sharples, 1990; Simpson, 1992; Ciricillo, 1992; Cohadon, 1991).
2. Air bag deployment in low- or moderate-speed crashes can cause serious head injuries, especially in children or adults of short stature (McKay, 1999).
3. In children younger than 6 years, head injuries are caused by abuse in 20%, while 80% are due to accidental causes, primarily falls (60%) and motor vehicle crashes (25%) (Reece, 2000).
4. BHT accounts for up to 22% of soccer injuries; mainly caused by heading the ball; even minor head trauma may result in organic brain damage (Tysvaer, 1992).
5. Primary injury sustained in horseback-riding accidents; often associated with spinal injuries (Hamilton, 1993).
6. Skateboarding is a significant cause of BHT and is more dangerous than roller or in-line skating. In a series of skating-related injuries, over 1/3 of patients overall had head injuries; skateboarders sustained 50% of head injuries vs 33% of in-line skaters and about 20% of roller skaters. Skateboarding head injuries are considerably more likely to be severe and have more serious consequences (Osberg, 1998).
7. Baby walkers (Partington, 1991; Claydon, 1996; Shane, 1997), misuse of child safety seats (Graham, 1992; Kowal-Vern, 1992), airbag deployment (Perez, 1996; CDC, 1996; Hollands, 1996), and falls (Duhaime, 1992; Lescohier, 1993) are potential causes of significant head injury in young children.
8. Among victims of intentional traumatic brain injury, 37% have blunt mechanisms (Wagner, 2000).
9. Intracranial hemorrhage is present in about 25% of victims of baseball ball assaults who are struck on the head (Groleau, 1993).
10. Among the elderly, falls leading to BHT represent a significant cause of morbidity and mortality. While falls from standing are more common (75%), falls on stairs (20%) or from a height (5%) are more likely to result in an abnormal CT scan (Nagurney, 1998).

11. Head/neck trauma is the major cause of deaths associated with all-terrain vehicles; in majority of fatal cases, rider was not wearing a helmet at the time of the crash (CDC, 1999).
12. Car surfing, in which participants hang on to, or stand on top of, a moving vehicle as though they were surfing, can result in severe or even fatal head injuries (Peterson, 1999).

C. SEVERITY (Stein, 1993a):

1. MINIMAL (All of the following)
  - a. No LOC or amnesia.
  - b. GCS score 15, normal alertness and memory.
  - c. No focal neurologic deficit.
  - d. No palpable depressed fracture.
2. MILD (Any of the following):
  - a. Brief (<5 min) LOC.
  - b. Amnesia for event.
  - c. GCS score <14.
  - d. Impaired alertness or memory.
  - e. Palpable depressed fracture.
3. MODERATE OR POTENTIALLY SEVERE (Any of following):
  - a. Prolonged (>5 min) LOC.
  - b. Focal neurologic deficit.
  - c. Post-traumatic seizure.
  - d. Intracranial lesion on CT scan.

D. PROGNOSIS:

1. Determined by type, location, and severity of brain injury, as well as quality of prehospital, hospital, and posthospital treatment (Gennarelli, 1993). Other factors include patient age, associated injuries, mechanism of injury, neurologic status (GCS score (Dent, 1995)), presence of spontaneous ventilation, duration of posttraumatic amnesia, pupillary responses, CT findings (Quigley, 1997; Ellenberg, 1996; Mamelak, 1996).
2. In severe head trauma, outcome cannot be predicted accurately within 6 h of injury (Waxman, 1992).
3. Morbidity and mortality are strongly related to ICP instability and hypotension during course (Marmarou, 1991; Pigula, 1993; Piek, 1992; Chesnut, 1993). Level of cerebral blood flow also associated with poor neurologic outcome (Robertson, 1992; Chan, 1992).
4. In a study of 1876 patients, all patients with intracranial lesions had at least one of the following: amnesia >5 minutes, vomiting, impaired consciousness, agitation, neurologic signs, or alcohol intoxication (Duus, 1993). Confirmatory, prospective studies are needed.
5. As medical and surgical care of traumatic brain injury continues to improve, and as survivorship increases, attention is increasingly focused on functional outcomes, including quality of life. In a study of 55 head-injured adults admitted to a Level 1 Trauma Center between 1991 and 1994, outcomes were evaluated one year after injury (data were complete for 51 patients) (Hawkins, 1996):
  - a. All patients had initial Abbreviated Injury Scales scores of 3 or more and required transfer to a comprehensive inpatient rehabilitation hospital; 75% of patients had an initial Glasgow Coma Score of 8 or less; mean ISS was 26.
  - b. At one year after injury, 90% of patients were living at home; however, 16% required round-the-clock supervision. Only 57% were rated as completely independent, and just 25% had returned to work full- or part-time; 27% demonstrated inappropriate or disruptive behavior at least once per week.

## 1.1.2 CLASSIFICATION

### A. OVERVIEW

#### 1. TYPES OF BRAIN INJURY (Gennarelli, 1993):

- a. FOCAL: Usually caused by direct blow to the head; causes contusions, brain lacerations, and intracranial hematomas (epidural, subarachnoid, subdural, intracerebral).
- b. DIFFUSE:
  - (1) Occurs with sudden, rapid acceleration or deceleration motion of the head; causes cerebral concussion or more prolonged posttraumatic coma (diffuse axonal injury); 25% mortality.
  - (2) New classification of head injury based on results of initial CT scan has been proposed. Offers possibility of early identification of patients at risk and earlier prediction of severely head-injured patients falling into broad outcome categories, and permits early identification of patients potentially at high risk from intracranial hypertension (Marshall, 1991a):
    - (a) DIFFUSE INJURY I: Includes all diffuse head injuries where there is no visible pathology; mortality is 10%.

- (b) DIFFUSE INJURY II: Includes all diffuse injuries in which the cisterns are present, the midline shift is <5 mm, or there is no high- or mixed-density lesion >25 cc.
- (c) DIFFUSE INJURY III: Includes diffuse injuries with swelling where the cisterns are compressed or absent and the midline shift is 0 to 5 mm with no high- or mixed-density lesion >25 cc.
- (d) DIFFUSE INJURY IV: Includes diffuse injuries with a midline shift >5 mm and with no high- or mixed-density lesion >25 cc; mortality is 50%.

c. MILD HEAD INJURY:

- (1) A widely used term; however, the definition of mild head injury (ie, GCS 13 to 15) may be imprecise.
- (2) A retrospective study of 3370 consecutive patients indicates that significant differences in injury severity exist between patients with GCS 13, 14, and 15 (Culotta, 1996), which may account for the difficulty in predicting outcome in this group.
- (3) A prospective study found significant differences in CT abnormalities, need for neurosurgical intervention, and long-term outcome among patient with a GCS of 13 to 15. Some investigators recommend that only patients with a GCS of 15 be classified as "mild" head injured (Hsiang, 1997).

B. CONTUSION, CEREBRAL

1. DEFINITION: Focal areas of petechial hemorrhage and swelling occurring most commonly in cerebral cortex and less often in cerebellum, brain stem, brain tissues that abut the falx cerebri, tentorium cerebelli, and foramen magnum, and along superior longitudinal sinus (Bullock, 1990a).

2. SITE:

- a. Inferior frontal lobes and temporal tips are areas of cortex most frequently contused (Mendelow, 1983a).
- b. Contusions caused by acceleration impact are most likely to be found at impact site and may be produced by fractured bone abutting cortical surface.
- c. When caused by deceleration impact, contusions are frequently found opposite impact site (contrecoup injuries).

3. MILD HEAD INJURY: Patients with mild head injury, particularly those >40 years or who complain of headache, are at increased risk of having an intracranial injury, including cerebral contusion. Absence of loss of consciousness does not exclude intracranial injury (Mikhail, 1992).

4. If large or associated with surrounding edema, mass effect may result in herniation and brain stem compression, producing secondary neurologic deterioration.

C. LACERATION, BRAIN

1. DEFINITION: Tears or rents in normal brain tissue.

2. SITES: Cortical surfaces along fracture lines and floor of anterior and middle fossae.

3. PATHOLOGY: Invoke cellular responses indistinguishable from contusions.

D. HEMATOMA, INTRACRANIAL

1. OVERVIEW

a. Predictors of presence of an intracranial lesion include advanced age, injury caused by a fall, low GCS score, and anisocoria (Gutman, 1992).

b. 12% to 33% of patients with severe head injuries have a lucid period prior to deteriorating into coma ("talk and die" patients) (Lobato, 1991; Jennett, 1977; Marshall, 1983; Rockswold, 1993).

c. Of patients who deteriorate into coma following a seemingly minor or moderate head injury, 75% to 80% have an intracranial lesion potentially requiring surgery. Outcome predictors include GCS score following deterioration into coma, highest ICP during course, degree of midline shift, type of intracranial lesions, and patient age (Lobato, 1991).

d. Patients with mild head injury at increased risk of having an intracranial injury include those >40 years, those injured in falls, and those who complain of headache (Mikhail 1992; Gutman, 1992).

(1) Nonfocal neurologic examination and absence of LOC do not exclude intracranial injury (Mikhail 1992; Ashkenazi, 1990; Schutzman, 1993; Harad, 1992).

(2) Conversely, regardless of age, mechanism of injury, or clinical findings, intracranial lesions cannot be completely ruled out clinically in patients who have LOC or amnesia, even if GSC score is 15; however, likelihood of having lesions requiring neurosurgical intervention is small (0.3% in one study) (Jeret, 1993).

2. HEMATOMA, SUBDURAL

a. DEFINITION: Venous or arterial bleeding into the subdural space; may be acute, subacute, or chronic, depending on time interval from injury to onset of symptoms.

- (1) ACUTE: Up to 3 days from injury to onset of symptoms (Gurdjian, 1978; Wilberger, 1990).
  - (2) SUBACUTE: 3 to 21 days postinjury.
  - (3) CHRONIC: >21 days postinjury; may occur after minor head trauma.
- b. MECHANISM OF INJURY: Usually results from rupture of bridging veins between dura and cerebral cortex or from laceration of brain and cortical arteries.
- (1) DECELERATION: In acute and chronic SDH, the bridging veins may be torn in a deceleration injury of the head, which puts high strain rates on these vessels, causing them to rupture.
  - (2) CONTUSION: Hemorrhage from a large cortical or subarachnoid contusion or laceration is common (Miller, 1982).
  - (3) ARTERIAL RUPTURE: 60% of acute SDHs are arterial in origin (ie, from rupture of small cortical arterials). May be result of head trauma.
  - (4) SKULL FRACTURE: Skull fracture with a tear of the dura or a laceration of the dural sinus is associated with SDH. One study found associated skull fractures in approximately 50% of patients.
- c. CAUSES:
- (1) Primary causes of acute SDH are falls, motor vehicle/motorcycle accidents, and assaults (Wilberger, 1990); may occur after minor head trauma or cervical manipulation.
  - (2) Adults <40 years with acute SDH are most often injured in MVAs, whereas patients >65 years are primarily injured in falls.
  - (3) In infants, may result from violent shaking by the extremities or shoulders, with whiplash-induced intracranial and intraocular bleeding but with no external signs of head or neck trauma (Jayawant, 1998).
- d. EPIDEMIOLOGY:
- (1) INCIDENCE:
    - (a) Acute SDHs account for about 30% of all severe head injuries. The overall incidence of acute SDH in patients who have sustained head trauma is 5% to 12% (Wilberger, 1990).
    - (b) 7% incidence with head trauma in children; the most important complication of head injury in childhood and occurs 10 times more frequently than extradural hematoma.
    - (c) Incidence of SDH in children less than 6.5 years with head injury was 45% in victims of abuse versus 10% in children injured accidentally (Reece, 2000).
  - (2) AGE/GENDER: In the elderly, SDH occurs with equal frequency in men and women; in young persons, incidence is 3 times higher in males (Ellis, 1990; Wilberger, 1990).
- e. CLINICAL PRESENTATION: Suspect in any comatose patient or in any alcoholic with an altered mental status. Symptoms and signs are often nonspecific, nonlocalizing, or absent; patients may be either stable or have a rapidly progressive course.
- (1) Vital sign abnormalities depend on patient's overall condition; Cushing's reflex (hypertension, bradycardia) is indicative of increased ICP and is usually a late finding.
  - (2) Hypotension may reflect shock or, in combination with Cheyne-Stokes respirations, may be a sign of impending herniation.
  - (3) Signs of head trauma often present; pupillary inequality (ipsilateral dilatation), particularly in acute SDH; papilledema (subacute, chronic SDH); visual field defects in some cases.
  - (4) Headache common (especially with chronic SDH); confusion, disorientation, belligerent behavior, particularly with injury to frontal or temporal lobes; depressed consciousness (lethargy, obtundation, coma) occurs in 100% of patients with acute SDH and is common in subacute and chronic SDH.
  - (5) Common focal signs include hemiparesis (may be false localizing sign), Babinski's sign, ankle clonus; decerebrate or decorticate posturing with impending transtentorial herniation; seizures may occur in all 3 types.
  - (6) Abnormalities in thought content (dementia, psychosis), and personality changes common.
  - (7) Patients with mild head injury, particularly those >40 years or who complain of headache, are at increased risk of having an intracranial injury, including SDH. Absence of loss of consciousness does not exclude intracranial injury (Mikhail, 1992).
  - (8) Herniation of temporal lobe with resultant brainstem compression.
- f. MORTALITY:
- (1) ACUTE: 30% to 90%, depending on age and operative timing (Wilberger, 1990; Seelig, 1981; Stone, 1986); 4 times greater in adults <40 years than in those >65 years (74% vs 18%).
  - (2) SUBACUTE: 12% to 25%.
  - (3) CHRONIC: 3% to 12%.
  - (4) MULTIPLE TRAUMA: Concomitant head injury in patients with multiple trauma doubles mortality. Adequate resuscitation probably is most critical factor for optimal survival, particularly in children. Hypotension is main factor contributing to increased mortality (Pigula, 1993; Chesnut, 1993).

### 3. HEMATOMA, EPIDURAL

- a. DEFINITION: Collection of blood between the dura and cranial vault.
- b. INCIDENCE: Present in about 2% of BHT patients (Colohan, 1989); accounts for 7% to 34% of acute mass lesions (Evans, 1990).
- c. The presence of air, not secondary to scalp laceration, in epidural hematomas, suggests a fracture of air-containing structures in the skull base (Ersahin, 1993).
- d. CLINICAL PRESENTATION:
  - (1) Patients may present with some alteration in mental status, ipsilateral pupillary abnormalities, and contralateral hemiparesis; any of these may be absent, even in presence of significant hematoma formation.
  - (2) EDH in posterior cranial fossa should be suspected in patients with BHT involving an occipital blow resulting in a frontal or temporal hematoma (Lui, 1993).
  - (3) Most important CNS sign is alteration in mental status (coma-lucid interval-coma) Focal neurologic signs, including hemiparesis, anisocoria, Babinski sign, and decerebrate posturing, may be present.
  - (4) Patients with mild head injury, particularly those >40 years or who complain of headache, are at increased risk of having an intracranial injury, including EDH. Nonfocal neurologic examination and absence of loss of consciousness do not exclude intracranial injury (Mikhail, 1992; Ashkenazi, 1990; Schutzman, 1993).
  - (5) Rarely, EDH may disappear spontaneously. Onset of generalized brain swelling appears to play a fundamental role in the resolution mechanism.
  - (6) The diagnosis of EDH in children less than 2 years old may be difficult, relying more upon clinical signs and CT scanning rather than symptoms (Beni-Adani, 1999).
- e. PROGNOSIS:
  - (1) CT scanning has improved initial diagnosis and postoperative management and has led to a reduction in mortality (5% to 18% overall; 0% for "pure" and 31% for "complicated" EDH) (Poon, 1991; Pozzati, 1989; Lui, 1993).
  - (2) Mortality is virtually restricted to patients who undergo surgery while in coma (Lobato, 1988; Rivas, 1988).
  - (3) In a study of outcomes in 139 patients with acute epidural hematomas, 46% had a complete or near-complete recovery, 31% had moderate disabilities, 10% were severely disabled, 4% remained vegetative, and 9% died (Heinzelmann, 1996).
    - (a) Key factor in determining outcome was presence of additional intracerebral injuries (hematomas or contusions), which were manifest by an initially low Glasgow coma score.
    - (b) Development of intracranial pressure elevation or other neurosurgical complications also portended a poor outcome
  - (4) In one series, patients operated on within 12 hours of injury has significantly higher mortality than those operated on 12 to 48 hours
  - (5) Concomitant head injury in patients with multiple trauma doubles mortality. Adequate resuscitation probably is most critical factor for optimal survival, particularly in children. Hypotension is main factor contributing to increased mortality (Pigula, 1993; Chesnut, 1993).

### 4. HEMORRHAGE, SUBARACHNOID

- a. Bleeding into subarachnoid space may follow pia-arachnoid tears, brain contusions, or parenchymatous hemorrhages extending into ventricle or subarachnoid space (Olshaker, 1993).
- b. Traumatic SAH may cause delayed cerebral vasospasm; in one study, patients in whom vasospasm developed had a significantly worse outcome (Schmieder, 1996).
- c. In a comprehensive study of 240 patients with traumatic subarachnoid hemorrhage, who were compared with patients with head injuries of similar severity but without SAH, presence of SAH adversely affected outcome. Patients who had traumatic SAH had more intracranial hypertension, had worse Glasgow Coma Scores at discharge, and were less likely to be discharged home (Greene, 1996).
- d. The incidence of SAH in children less than 6.5 years with head injury was 30% in victims of abuse versus 8% in children injured accidentally (Reece, 2000). (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: SUBARACHNOID HEMORRHAGE)

### 5. HEMORRHAGE, INTRACEREBRAL

- a. DEFINITION: Areas of parenchymal hemorrhage >1 cm in diameter resulting from inertial stress propagation through brain substance associated with shearing forces or rapid deceleration injuries (Olshaker, 1993).
- b. INCIDENCE:
  - (1) Occurs in <2% of head injuries. Intraventricular hemorrhage following BHT is uncommon and reflects severity of injury (LeRoux, 1992).
  - (2) May occur more commonly in patients in whom an extradural hematoma has been removed.

c. CLINICAL PRESENTATION:

- (1) May occur more slowly than extradural bleeding and may not be evident on initial CT-scan (Fukamachi, 1985; Soloniuk, 1986; Atluru, 1986; Huneidi, 1992).
- (2) In one study, delayed intracerebral hematoma occurred in 20% of patients with moderate to severe BHT in whom signs of cerebral contusion were absent initially. Indicated by recurrence of increased ICP within 3 to 4 h, despite initial but inconsistent response to osmotic diuretics (Huneidi, 1992).
- (3) Frontal location of intracerebral hematoma is most likely to progress, requiring surgical decompression (Patel, 2000). (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: INTRACEREBRAL HEMORRHAGE)

E. CONCUSSION

1. DEFINITION:

- a. Transient episode of altered mental status following BHT, with a rapid return to an apparently normal neurologic state (Kelly, 1991); also called mild traumatic brain injury syndrome (Rosenthal, 1993; Kelly, 1997; Am Acad Neurol, 1997).
- b. May or may not involve loss of consciousness. Also includes any transient neurologic dysfunction such as confusion, nausea, vomiting, dizziness, headache, slurred speech, disorientation, incoordination, irritability, and amnesia, as well as loss of consciousness, as long as there is obvious improvement over time and resolution of the abnormality.

2. MECHANISM OF INJURY:

- a. Occurs as a result of direct or indirect mechanical force applied to the head (Gurdjian, 1978).
  - (1) DIRECT: Occurs when a moving object strikes the head, or when the moving head strikes a nonmoving object.
  - (2) INDIRECT: Occurs as a result of impact to another part of the body with secondary involvement of the head.
- b. Most commonly results from automobile accidents, falls, home and industrial accidents, and sports injuries (Gurdjian, 1978; Albright, 1985; Kelly, 1991).
- c. In a study of 23,566 sports injuries, there were 1219 (5.5%) mild traumatic brain injuries; football accounted for 63% (Powell, 1999).

3. PATHOPHYSIOLOGY: The degree of traumatic brain injury is minor. Typical lesions include focal cortical contusions and diffuse axonal injury (DAI) (Rosenthal, 1993).

4. COMPLICATIONS:

- a. Postconcussion syndrome (characterized by headache, inability to concentrate, dizziness, and/or visual disturbances; may last months to years); personality changes; posttraumatic seizures; 85% to 90% recover fully within one year (Alexander, 1995),
- b. Patients with mild head injury, particularly those >40 years or who complain of headache, are at increased risk of having an intracranial injury; absence of loss of consciousness does not exclude intracranial injury (Mikhail, 1992).

c. SECOND-IMPACT SYNDROME:

- (1) Increasingly, it is recognized that repeated, seemingly minor blows to the head, especially during football, hockey, boxing, skiing, and other contact sports, can have cumulative and catastrophic effects (Am Acad Neurol, 1997; Kelly, 1997).
- (2) Occur when a second concussion is sustained before an individual has had time to recover from an initial injury. May be sudden and severe; characterized by acute brain edema and rapid neurologic deterioration. Edema, coma, and death appear to result from failure of cerebral autoregulation and vascular congestion (Kelly, 1997).

F. FRACTURE, SKULL

1. GENERAL:

- a. Significance relates to the intracranial sequelae of head trauma. Presence of skull fracture is associated with a higher risk of intracranial injury (Ashkenazi, 1990).
- b. Most patients with skull fracture do not have an associated acute traumatic intracranial lesion and, conversely, many patients with an acute traumatic lesion do not have skull fracture (Ashkenazi, 1990).
- c. The complication rate in basilar skull fracture without intracranial abnormality is low. In a study of 114 patients, meningitis was the most serious complication, developing in only one patient (Kadish, 1995).
- d. CT can be useful in detecting fractures of the anterior cranial fossa (Asano, 1995)

## 2. INCIDENCE:

- a. GENERAL: In one study, one third of head-injured patients had symptoms, neurologic deficits, or evidence of a basal skull fracture on initial evaluation; half of these patients had fractures on x-ray (Miller, 1990).
- b. CHILDREN: The skull is the most frequent fracture site in children <3 years and linear fractures of the parietal bone are the most common type, whether due to abuse or accidental injury (esp falls). Accounts for 60% to 80% of such injuries overall and for >75% of fractures in infants (Kowal-Vern, 1992; Leventhal, 1993; Shane, 1997). Skull fractures were present in 43% of children with severe head injuries (Feickert, 1999).

## 3. RISK FACTORS:

- a. With bilateral epidural hematomas with both collections present at the same time on emergency CT bleeding is arterial in origin and related to the presence of a skull fracture interrupting the branches of the middle meningeal artery (Arienta, 1986; Barlow, 1985).
- b. Basilar skull fractures are present in 25% of patients with facial fractures. Incidence increases with number of facial fractures and their anatomic location; particularly associated with fractures of orbital wall/rim, maxilla/zygoma, and orbital floor (Slupchynskyj, 1992).

## 4. CLINICAL PRESENTATION:

- a. Factors associated with increased risk of open and depressed skull fractures include:
  - (1) Assault with any object.
  - (2) CSF leak.
  - (3) Clinical signs of basilar fracture:
    - (a) Drainage from the ear
    - (b) CSF drainage from the nose
    - (c) Hemotympanum
    - (d) Battle's sign
    - (e) Raccoon eyes
    - (f) Blindness
    - (g) Hearing loss
    - (h) Cranial nerve deficit
- b. Cranial nerve palsies develop in almost 25% of children with basilar skull fractures and are permanent in 50% of cases (Kitchens, 1991).
- c. Absence of scalp soft tissue injury does not always rule out skull fracture (Banerjee, 1991).
- d. Symmetrical cranial fractures in infants are most likely result of compression of head between two surfaces, rather than result of localized impact (Hiss, 1995).
- e. A head CT should be obtained for patients with any one of the four Miller criteria (nausea, vomiting, headache, signs of depressed skull fracture) present after minor head trauma and a GCS of 14 (Miller, 1996; Holmes, 1997). However, these criteria should not be used as the only selection criteria for head CT scanning in these patients, since, when used alone, do not detect all patients with intracranial injuries who require neurosurgical intervention (Holmes, 1997).
- f. In infants, risk for intracranial injury is increased almost fourfold in presence of skull fracture (Quayle, 1997). Presence of lethargy and temporal or frontal bone fractures is predictive of intracranial injury (Shane, 1997).

### 1.1.3 EPIDEMIOLOGY

#### A. INCIDENCE:

1. Each year, 3% to 4% of persons in the US (7.5 to 10 million) sustain head injuries. Approximately 85% of these injuries are minor (lacerations or contusions of scalp) and about 15% are major (concussion, skull fracture, intracranial hemorrhage). The average annual estimate of new traumatic brain injury treated in the US equals 444/100,000 persons (Jager, 2000).
2. BHT is one of the most common childhood injuries, annually accounting for more 500,000 ED visits and 95,000 hospital admissions (Schutzman, 2001).

#### B. AGE:

1. Incidence of head injuries is highest in children <5 years, then decreases up to 14 yr (Cohadon, 1991; Lescohier, 1993). A second peak occurs in the 15- to 24-year age group, followed by a gradual decline with age (Cohadon, 1991). A third peak in the incidence of traumatic brain injury occurs among those age 65 years and above (CDC, 1997).
2. Highest incidence of head injury is in children less than 5 years of age (1091/100,000), followed by persons over 85 years (Jager, 2000).

3. One study found that vehicle occupants <30 years of age are more likely to sustain diffuse head injury and serious truncal injury than focal intracranial lesions; patients >60 years injured in falls are more likely to have operable intracranial mass lesions without significant injury to the torso (Moulton, 1992).
4. Survival is decreased in elderly blunt head trauma victims compared with that of younger patients (Pennings, 1993; Knudson, 1994).
5. Head injuries in children younger than 6.5 are caused by abuse in 20% while 80% are due to accidental causes (Reece, 2000).

C. GENDER: Injury and subsequent mortality is disproportionately high in males, especially those between 15 and 24 years of age (Sosin, 1989; Cohadon, 1991). Males are 1.6 times more likely than females to sustain head injury until age 65 (Jager, 2000).

#### D. MORTALITY:

1. GENERAL: Overall mortality is 33% to 50% (Cohadon, 1991; Phair, 1991; Marshall, 1991). Has been reported to be as high as 40% for patients with focal brain injuries and 25% for those with diffuse brain injuries (Marshall, 1991). Annual head injury-associated death rate in US is 17/100,000 residents. (Sosin, 1995).
2. CHILDREN:
  - a. BHT accounts for 7000 deaths/year in children (Schutzman, 2001). Mortality in a series of 150 children with severe head injuries was 22% (Feickert, 1999).
  - b. Major cause of death in children aged >1 yr, accounting for 15% of deaths in children aged 1 to 15 years and 25% of those aged 5 to 15 years (Kraus, 1990).
  - c. Mortality from head injuries in children younger than 6.5 years was 13% in victims of abuse vs 2% in those injured accidentally (Reece, 2000).
3. IMPROVING OUTCOME:
  - a. Extracranial injuries are highly influential in determining outcome in severe BHT. Significant improvement in outcome in a sizable proportion of patients could be accomplished by improving ability to prevent or reverse hypotension, as well as pneumonia, sepsis, and coagulopathy (Chesnut, 1993; Piek, 1992).
  - b. In children, largest reduction in mortality results from preventing deterioration and secondary brain injury in patients with mild/moderate intracranial injury and subsequent deterioration while limiting unnecessary imaging procedures (Schutzman, 2001).

### 1.1.4 PATHOPHYSIOLOGY

#### A. OVERVIEW

1. GENERAL: Initial shear-strain brain injury at interface of grey and white is followed by progressive damage involving (White, 1993; Goodman, 1994):
  - a. Progression of subtle focal axonal damage to axonal transection 6 to 12 h after injury.
  - b. Progressive development of tissue microhemorrhages between 12 and 96 h postinjury.
  - c. Development of tissue and CSF lactic acidosis that does not appear to be explained by tissue ischemia.
  - d. Ischemic brain damage and diffuse axonal injury are common findings in patients with fatal head injuries (Bennett, 1995).
2. BIOCHEMICAL EVENTS:
  - a. Following ischemic or shear injury, a complex cascade of biochemical events appears to mediate significant secondary developments, resulting in neuronal degeneration (Bullock, 1993).
  - b. Excessive release of excitatory neurotransmitters (eg, acetylcholine, glutamate, aspartate) leads to loss of calcium homeostasis, increased free radical production, tissue acidosis (Hayes, 1994; Siesjo, 1993), lipid peroxidation (Hall, 1993), and cell death.
  - c. It is widely believed that oxygen free radicals are generated after severe head injury; presumably, these radicals overwhelm the natural scavenger systems, which include superoxide dismutase, catalase and glutathione (Faden, 1996). Unchecked, free radical accumulation then leads to secondary brain tissue damage, via membrane lipid peroxidation, loss of microvascular autoregulation, vasospasm, and edema formation.
3. PRIMARY BRAIN INJURY:
  - a. DEFINITION: Damage from initial insult; occurs at moment of impact and results from transfer of kinetic energy to the brain. May cause spectrum ranging from minor injury to irreparable injury with death, depending on cause and severity of inciting injury (Salluzzo, 1992).
  - b. MECHANISM OF INJURY: Involves neural or vascular elements of the brain. Primary injury in head trauma is mechanical, including hemorrhage, mass effect, contusion, and actual tissue loss (Strauss, 1998).
    - (1) FOCAL IMPACT (eg, direct blow to head): Produces cerebral contusion and lacerations, which primarily affect cerebral cortex and tend to produce localized defects.

(2) DECELERATION INJURY (eg, MVA): May cause shearing injuries of white matter and diffuse axonal injury; patients often have bilateral and symmetric involvement of hemispheres and present in coma.

c. PATHOPHYSIOLOGIC CASCADE (Gennarelli, 1993):

(1) FOCAL BRAIN INJURY: Trauma leads to local mass effects from contusion or hematoma, which can cause brain shifts, herniation, and brainstem compression (latter is presumptive precipitating event of coma). Prolonged brainstem compression results in brainstem hemorrhage.

(2) DIFFUSE BRAIN INJURY: Initial insult results in primary defect in axonal membrane that causes excessive accumulation of calcium ions within intracellular compartment of the axon. This produces ionic shifts within the axon interior and subsequent axonal depolarization, and leads to altered transmission of the neural networks, widespread neurologic dysfunction, and primary coma.

4. SECONDARY BRAIN INJURY (Salluzzo, 1992; Doberstein, 1993; Gennarelli, 1993):

a. INCIDENCE: Up to 40% of all BHT patients show evidence of delayed secondary neurologic deterioration.

b. MECHANISM:

(1) Physiologic events are mediated by complex cascade of biochemical processes occurring as result of complications of intracranial or extracranial injuries sustained at time of initial impact. Hypoxia, ischemia, or both, can result in significant cell loss.

(2) Brain dysfunction may occur within minutes or be delayed for hours to days. May lead to further damage of nervous tissue, prolonging or contributing to neurologic deficits.

(3) Secondary brain injury occurs as the result of physiologic responses to direct injury. including edema, inflammation, tissue necrosis, and thrombosis. These effects may be mediated by active cytokines and vasoactive substances, including nitric oxide and prostaglandins (Strauss, 1998).

c. EXTRACRANIAL (SYSTEMIC) INSULTS:

(1) Hypotension (most significant).

(2) Hypoxia.

(3) Electrolyte imbalance.

(4) Acidosis

(5) Other (eg, anemia, hypercarbia, hyperglycemia, hypoglycemia).

d. INTRACRANIAL INSULTS:

(1) Intracranial hypertension.

(2) Delayed intracerebral hematoma.

(3) Cerebral edema.

(4) Hyperemia.

(5) Carotid artery dissection.

(6) Seizures.

(7) Vasospasm.

e. PROGNOSIS:

(1) Adversely associated with outcome. Must be prevented or promptly corrected before irreversible neurologic damage occurs. Preliminary studies of pharmacologic intervention with neuroprotective drugs appear promising for treatment of many of these secondary posttraumatic events (Bullock, 1993).

(2) Lipid peroxidation can be inhibited by antioxidants. They are capable of inhibiting posttraumatic events, eg, edema, metabolic dysfunction, and ischemia (Hall, 1993).

B. ISCHEMIA, CEREBRAL

1. Probably most important cause of secondary insult to brain and plays important role in majority of fatal cases (Bouma, 1991, 1992). Associated with cascade of events that worsen primary traumatic events. Increases accumulation of calcium ions, which can lead to axonal degeneration (Gennarelli, 1993).

2. Hypotension reduces cerebral perfusion pressure, with resultant cerebral ischemia and infarction and is a primary contributor to increased morbidity and mortality related to severe trauma to extracranial organ system (Chesnut, 1993; Pigula, 1993; Winchell, 1996).

3. Delayed cerebral arterial spasm resulting in ischemia is frequent complication (30% in one series). May occur regardless of GCS score, although it is more common in patients with severe head injury (Martin, 1992).

C. VASOSPASM, CEREBRAL

1. Delayed cerebral arterial spasm resulting in ischemia is frequent complication (30% in one series). May occur regardless of GCS score, although it is more common in patients with severe head injury. Severity may be comparable with that seen in aneurysmal subarachnoid hemorrhage (Martin, 1992).

2. Ischemia in the first 8 hours after brain injury may be caused by compression of the microvasculature rather than by vasospasm of the larger vessels (Schroder, 1998).

#### D. HYPEREMIA, CEREBRAL

1. Increased cerebral blood flow (hyperemia) occurs commonly after traumatic brain injury (Romner, 1996; Kelly, 1996); however, neither the mechanism of hyperemia nor its significance is well understood (Kelly, 1996). Head trauma may directly impair normal cerebrovascular autoregulation (pressure-mediated as well as metabolic), leading to dilatation of the cerebral vascular bed and increased intracranial volume (Newell, 1996).
2. Loss of autoregulation leads to vicious cycle in which increased cerebral blood flow causes elevated intracranial pressure and reflex increase in mean arterial pressure and cerebral blood flow.
3. Although controversial (Miller, 1994), cerebral hyperemia is thought to be more common in children than adults (Bruce, 1981) and may be a delayed cause of rapid neurologic deterioration (Humphreys, 1990).
4. Vasospasm and ischemia, as well as hyperemia, may occur during severe head injury. Vasospasm often occurs late after traumatic brain injury and especially in patients with traumatic subarachnoid hemorrhage (Romner, 1996; Schmieder, 1996).

#### E. INTRACRANIAL PRESSURE, INCREASED

##### 1. GENERAL:

a. CAUSES: May be due to expanding hematoma or development of cerebral hyperemia or edema (Miller, 1985; Doberstein, 1993).

(1) Cranial cavity has fixed volume and limited ability to compensate for increases in fluid compartments, ie, blood and edema fluid, that may follow head injury.

(2) Major adaptive mechanism is increase in rate of resorption of cerebrospinal fluid (Rosman, 1983).

(3) As intracerebral volume increases, compliance of this compartment, or its ability to tolerate volume increments, decreases; therefore, small increase in volume may produce large increase in ICP.

b. EFFECTS OF INCREASED INTRACRANIAL PRESSURE (ICP): Pathophysiologic effects of elevated ICP are impaired cerebral blood flow, ischemia of neural tissue, and herniation.

(1) Maintenance of cerebral blood flow is dependent upon cerebral perfusion pressure (CPP), which is difference between mean arterial pressure (MAP) and ICP ( $CPP = MAP - ICP$ ).

(2) Elevation of ICP above 20 mmHg may compromise cerebral blood flow. When CBF is <45%, gross neuronal tissue changes occur, and when CBF is <20%, pupils may dilate.

(3) In a study of 188 brain-injured children, there were no survivors among patients with CPP less than 40 mmHg (Downard, 2000).

(4) CPP should be maintained above 70 mmHg (Marshall, 2000).

##### 2. EDEMA, CEREBRAL:

###### a. GENERAL:

(1) An increase in brain volume due to an increase in its water content or brain tissue edema often develops in conjunction with contusions, lacerations, and hemorrhage.

(2) Swelling may be general or localized and reflects one of the major effects of trauma. Edema can lead to various neurologic deficits and may be severe enough to cause transtentorial herniation.

(3) Exerts its effect by increasing ICP and altering cerebral blood flow.

(4) Hypoxia and hypotension may play important role in pathogenesis of diffuse brain swelling (Aldrich, 1992).

###### b. TYPES:

###### (1) VASOGENIC CEREBRAL EDEMA:

(a) Is the most common and is associated with an increased permeability of the brain endothelial cells. As a result of this increased permeability, there is a leakage of protein, water, and sodium into the extracellular space. The protein is evidenced by an increase in cerebrospinal fluid (CSF) protein, as well as by abnormalities of brain swelling noted on CT scan.

(b) The etiologies of vasogenic edema include trauma (eg, epidural or subdural hematoma, or lightning or electrical injury), hemorrhage, ischemia (usually focal in nature as in cerebral thrombosis), infections (eg, rabies and meningitis), and tumor. It also may occur as a late (>24 hr) manifestation in patients resuscitated from cardiac arrest.

###### (2) CYTOTOXIC CEREBRAL EDEMA:

(a) Is caused by brain endothelial cell swelling with a resultant decrease in the extracellular space. This type of edema is theorized to be secondary to ATP-dependent sodium pump failure, which causes an increase in the amount of sodium in the interior of the cell, with a concomitant increase in size secondary to osmotic flow of water following the sodium.

(b) Etiologies of cytotoxic cerebral edema include Reye's syndrome, hypercarbia, and hypoxia associated with cardiac arrest; less common than vasogenic edema following head

trauma. Cerebrospinal fluid protein is normal, indicating no increase in capillary permeability (although cytotoxic edema following cardiac arrest usually is not sufficient to cause sustained ICP elevation).

(3) OBSTRUCTIVE CEREBRAL EDEMA: Is best exemplified by obstructive hydrocephalus, which causes a backflow of CSF resulting in cerebral edema.

c. CLINICAL SIGNS:

(1) Clinical manifestations are a product of an increase in ICP. Generally does not develop until 24 to 48 hours after head trauma (Rosman, 1983).

(2) Increased ICP may be manifested clinically by headache, vomiting, papilledema, pupillary asymmetry, and weakness of the abducens nerve.

(3) In severe ICP elevation, cardiovascular changes (eg, Cushing's phenomenon) and respiratory changes (eg, periodic breathing) will be seen.

F. HERNIATION, CEREBRAL

1. ETIOLOGY: Occurs when expanding mass lesion produces local increase in ICP within fixed space of rigid skull. Brain shifts caudally through tentorial opening and foramen magnum.

2. CLINICAL PRESENTATION: Specific syndrome depends on direction of brain tissue displacement.

a. TRANSTENTORIAL:

(1) UNCAL: Rapidly expanding lateral mass displaces temporal lobe, which compresses upper brainstem, 3rd cranial nerve, and posterior cerebral artery.

(a) Headache

(b) Vomiting

(c) Mydriasis (ipsilateral to side of herniation and contralateral to hemiparesis)

(d) Altered mental status (rapid progression to stupor and coma)

(e) Hemiparesis (ipsilateral)

(2) CENTRAL: Slowly expanding, multifocal supratentorial mass lesions causes downward and lateral shift of diencephalon and upper pons (rostral-caudal deterioration).

(a) Headache

(b) Altered mental status (progressive obtundation)

(c) Miosis (small reactive pupils)

(d) Cheyne-Stokes respirations

(e) Extensor plantar responses (bilateral)

(f) Paucity of focal motor signs

(g) Decorticate, then decerebrate posturing

b. TONSILLAR (FORAMEN MAGNUM): Large posterior fossa mass pushes cerebellar tonsils through foramen magnum, compressing medulla.

(1) Headache

(2) Vomiting

(3) Hiccoughs

(4) Meningismus

(5) Altered mental status (rapid progression to stupor and coma)

(6) Skew deviation of eyes

(7) Irregular respirations

(8) Hypertension

**1.1.5 PREDISPOSING FACTORS**

A. ALCOHOL ABUSE

1. Common contributing factor in MVAs, falls, and acts of violence and thereby predisposes to head injury (Bullock, 1990a).

2. Alcohol abuse is a predisposing factor for traumatic brain injury from intentional causes (Wagner, 2000).

3. Ethanol intoxication affects management of patients with acute brain injury by increasing likelihood of intubation, ventilatory support, ICP monitoring, and, possibly, development of pneumonia (Gurney, 1992).

B. DRUG ABUSE

1. Common contributing factor in MVAs, falls, and acts of violence and thereby predisposes to head injury.

2. Drug abuse is a predisposing factor for traumatic brain injury from intentional causes (Wagner, 2000).

## C. CHILD ABUSE

### 1. GENERAL:

- a. Principal cause of serious head injury in children less than 2 years and leading cause of death in alleged child abuse; accounts for up to 2/3 of cases (Reece, 2000; Jayawant, 1998; Lloyd, 1998; Duhaime, 1998; Elner, 1990; Duhaime, 1992; Kowal-Vern, 1992).
- b. Head injuries in children younger than 6 years are caused by abuse in 20% of cases (Reece, 2000). Abuse and neglect may be particularly prevalent causes of blunt head trauma in infants <3 months of age (Stewart, 1993).

### 2. CNS INJURIES: Account for majority of deaths and permanent disabilities in abused children (Duhaime, 1998; Marmon, 1989; Merten, 1990).

- a. INTRACRANIAL HEMORRHAGE: More common in nonaccidental vs accidental trauma (Jayawant, 1998; Duhaime, 1992); accompanied by signs of direct head trauma, eg, subgaleal hemorrhage, skull fracture, cerebral contusion, and external contusions (Elner, 1990). Neurologic outcome is also poorer (Goldstein, 1993).
- b. SDH or SAH:
  - (1) May result from violent shaking by the extremities or shoulders, with whiplash-induced intracranial and intraocular bleeding but with no external signs of head or neck trauma (Jayawant, 1998; Duhaime, 1992; Elder, 1991).
  - (2) In children less than 7 years with head injury, incidence of SDH and SAH was 45% and 30%, respectively, in victims of abuse vs 10% and 8%, respectively, in children injured accidentally (Reece, 2000).
- c. EPIDURAL HEMATOMA: Less likely to result from abuse (Shugerman, 1996).

### 3. OCULAR TRAUMA: Frequently accompanied by ophthalmic manifestations, particularly retinal, vitreous, and subdural optic nerve hemorrhages (Jayawant, 1998; Duhaime, 1998; Elner, 1990; Elder, 1991; Duhaime, 1992).

- a. In one series, ocular injuries were present in 7 of 10 children who died of alleged child abuse, and all 7 died of direct head trauma (Elner, 1990).
- b. In accidental trauma, retinal hemorrhage is a rare finding (Reece, 2000; Johnson, 1993). Retinal hemorrhages were identified in 33% of head-injured victims of child abuse vs 2% of children with accidental head injuries (Reece, 2000).

### 4. FALLS:

- a. Should be suspected with head injuries reported to occur in falls of low height. Infants and young children are relatively resistant to injuries from free falls; falls of <10 feet are unlikely to produce serious or life-threatening injury (Tarantino, 1999; Williams, 1991; Chadwick, 1991; Duhaime, 1992; Root, 1992; Leventhal, 1993).
- b. In a study of 167 infants who fell less than 4 feet, the vast majority sustained minor or no injury; the only two infants who developed intracranial hemorrhage were victims of abuse (Tarantino, 1999).

### 5. MORTALITY: Deaths from head injuries in children younger than 6.5 years was 13% in victims of abuse vs 2% in those injured accidentally (Reece, 2000).

### 6. SUMMARY: Likelihood of head injuries being inflicted based on specific injury is as follows:

- a. HIGHLY LIKELY:
  - (1) Thin subdural brain swelling
  - (2) Chronic subdural hematoma in children >3 mo of age
  - (3) Massive subgaleal hematoma
- b. POSSIBLY:
  - (1) Subarachnoid hemorrhage
  - (2) Cerebral contusion
  - (3) Complex skull fracture
- c. UNLIKELY:
  - (1) Concussion
  - (2) Epidural hematoma
  - (3) Linear parietal skull fracture

## D. INFANT WALKERS

1. Reported infant walker-related injuries include skull fracture, concussion, and intracranial hemorrhage (Chiaviello, 1994; Coats, 1991; Rieder, 1986; DiMario, 1990; Fazen, 1982).

## E. SEIZURES

1. Patients with epilepsy are at increased risk for head injuries due to falls, with such injuries at high risk for traumatic intracranial hematomas. Until a mass lesion has been excluded by CT, any decrease in level of consciousness or focal neurologic deficit should not be attributed to the seizure itself (Zwimpfer, 1997).

## F. FALLS

### 1. GENERAL:

a. Head injuries occur in 1/3 of persons who fell less than 20 feet and account for the majority of deaths in these patients (Helling, 1999).

b. Actual height of a fall has been shown to be a poor predictor of injury severity (Goodacre, 1999).

2. YOUNG CHILDREN: Potential cause of significant head injury in young children (Reece, 2000; Greenes, 1998; Duhaime, 1992; Lescohier, 1993). However, in a study of 167 infants who fell less than 4 feet, 85% had minor or no head injury (Tarantino, 1999).

3. ELDERLY: Falls leading to BHT represent a significant cause of morbidity and mortality in this population. While falls from standing are more common (75%), falls on stairs (20%) or from a height (5%) are more likely to result in an abnormal CT scan. A focal neurologic exam is predictive of need for neurosurgery (Nagurney, 1998).

## G. TRAUMA, ATHLETIC

1. GENERAL: Sports are a primary cause of BHT (Osberg, 1998; CDC, 1997a; Kraus, 1986; Sharples, 1990; Tysvaer, 1992).

2. FOOTBALL: In a study of 23,566 sports injuries, there were 1219 (5.5%) mild traumatic brain injuries; football accounted for 2/3 of these (Powell, 1999).

3. SOCCER: BHT accounts for up to 22% of soccer injuries; mainly caused by heading the ball; even minor head trauma may result in organic brain damage (Tysvaer, 1992).

4. SKATEBOARDING: Significant cause of BHT and is more dangerous than roller or in-line skating. In a series of skating-related injuries, over 1/3 of patients overall had head injuries; skateboarders sustained 50% of head injuries vs 33% of in-line skaters and about 20% of roller skaters. Skateboarding head injuries are considerably more likely to be severe and have more serious consequences (Osberg, 1998).

5. SNOWBOARDING: BHT, including intracerebral hemorrhage and subdural hematoma, has been increasingly reported with snowboarding (Machold, 2000; Ferrera, 1999); occipital impact is frequently involved in BHT due to snowboarding (Nakaguchi, 1999).

## 1.2 ASSOCIATED CONDITIONS

### A. TRAUMA, MULTIPLE

1. Approximately one third of patients with significant head injuries (blunt and penetrating) have other serious injuries; the figure is higher if only blunt trauma is considered (Miller, 1990).

2. Fractures of long bones and injuries to face, neck, chest, and abdomen are common.

3. Patients with both significant head injury and femur fracture are at high risk for sustaining visceral injuries.

a. Delayed stabilization of lower extremity fractures does not seem to worsen the head injury but does not decrease risk of pulmonary complications (Poole, 1992). Early fracture repair in severely brain injured patients had been associated with significantly increased risks of hypotension and decreased cerebral perfusion pressure (Townsend, 1998).

b. Early fracture fixation may be deleterious after head injury, possibly due to secondary brain injury and greater fluid resuscitation requirements (Jaicks, 1997).

c. Others have documented no adverse neurologic effects or increased mortality in patients with head injuries who underwent early fracture fixation (Scalea, 1999).

4. Concomitant head injury in patients with multiple trauma doubles mortality. Adequate resuscitation probably is most critical factor for optimal survival. Hypotension is main factor contributing to increased mortality (McMahon, 1999; Pigula, 1993; Chesnut, 1993).

5. Circulating catecholamine levels, especially norepinephrine, are significantly correlated with severity of injury in patients with BHT as part of multisystem trauma (Woolf, 1992; Levitt, 1995).

### 6. TRIAGE:

a. Management priority of intracranial versus intraabdominal trauma requires individual assessment. The decision to obtain head CT before laparotomy must be made on a case-by-case basis with consideration of the patient's hemodynamic status and the likelihood of intracranial and intraabdominal injury (Gutman, 1991). In general, patients with severe shock and hemoperitoneum should proceed directly to the OR with intracranial monitoring, while patients who are relatively stable should undergo cranial CT first (Hsiang, 1997).

b. In a study of multitrauma, head-injured patients with GCS score <13, head CT scan prior to general surgery appeared to be safe, provided that the patient was normotensive or had hypotension that resolved with administration of IV fluids. The likelihood of detecting a lesion requiring craniotomy was similar to the likelihood of truncal injury requiring general surgery (Winchell, 1995).

c. In a study of 800 multitrauma patients, laparotomy without prior head CT was safe, provided there were no lateralizing neurologic signs (Wisner, 1993).

d. One study of 112 multitrauma patients successfully used US and the finding of large hemoperitoneum to triage those patients who should proceed directly to laparotomy (Huang, 1995).

#### B. TRAUMA, CERVICAL SPINE

1. Although several retrospective studies have found that <5% of head-injured patients had cervical spine injury. such injury must always be suspected in presence of head trauma (Link, 1995; Michael, 1989; Gbaanador, 1986; O'Malley, 1981; Neifeld, 1988; Bayless, 1989; Soicher, 1991; Hills, 1993; Bucholz, 1983; Williams, 1992; Frye, 1994).
2. An altered state of consciousness secondary to head injury and/or intoxication may obscure diagnosis of a cervical spine injury.
3. Alert patients with minor head injury and no neck complaints are unlikely to have a cervical spine fracture.
4. Inclusion of upper cervical spine images with head CT should be considered in evaluation of patients with significant head trauma (intracranial hemorrhage or skull fracture) (Kirshenbaum, 1990).
5. In a series of 188 patients with cervical spine or spinal cord injuries, 35% had associated severe or moderate head injury; severe head injury and skull base fractures were more frequent with upper cervical injuries (Iida, 1999). (FOR FURTHER INFORMATION, SEE CLINICAL REVIEWS: CERVICAL SPINE TRAUMA, NEUROGENIC SHOCK)

#### C. TRAUMA, THORACIC, BLUNT

1. Occurs commonly in association with serious blunt head trauma. Hypoxia secondary to pulmonary dysfunction complicates management of serious head injuries.
2. Concomitant head injury in patients with multiple trauma doubles mortality. Adequate resuscitation probably is most critical factor for optimal survival, particularly in children. Hypotension is main factor contributing to increased mortality (Pigula, 1993; Chesnut, 1993). (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: BLUNT CHEST TRAUMA)

#### D. TRAUMA, ABDOMINAL, BLUNT

1. Occurs commonly in association with serious blunt head trauma. Abdominal examination is unreliable in presence of altered mental status secondary to head injury and/or intoxication.
2. Concomitant head injury in patients with multiple trauma doubles mortality. Adequate resuscitation probably is most critical factor for optimal survival, particularly in children. Hypotension is main factor contributing to increased mortality (Pigula, 1993; Chesnut, 1993). (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: BLUNT ABDOMINAL TRAUMA)

#### E. TRAUMA, FACIAL

1. Patients with facial fractures, especially those injured in motor vehicle accidents, have high incidence of associated significant head injuries (Sinclair, 1988; Davidoff, 1988; Fitzpatrick, 1989; Slupchynskyj, 1992; Haug, 1992).
2. In one large series, BHT occurred in 17.5% of patients with facial fractures; simple LOC was the most frequent type of head injury. BHT was most likely in patients with mandibular fractures and likely in those with isolated maxillary fractures; patients with both maxillary or mandibular and zygoma fractures sustained the most severe intracranial injuries (Haug, 1992).
3. Basilar skull fractures are present in 25% of patients with facial fractures. Incidence increases with number of facial fractures and their anatomic location; particularly associated with fractures of orbital wall/rim, maxilla/zygoma, and orbital floor (Slupchynskyj, 1992). (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: FACIAL FRACTURE)

#### F. FRACTURE, RIB

1. Significant head injuries occur in 33% to 50% of patients with first or second rib fractures and is often the cause of death (Fermanis, 1985).

#### G. FRACTURE, STERNAL

1. Closed head injury occurs in about 20% of patients with sternal fractures (Buckman, 1987; Wojcik, 1988).

### 1.3 VITAL SIGNS

#### A. BLOOD PRESSURE

##### 1. OVERVIEW

- a. Although most commonly unaffected by blunt head injuries, blood pressure may be elevated or decreased.

##### 2. BLOOD PRESSURE, DECREASED

###### a. INCIDENCE:

- (1) Occurs in 1/3 of patients with severe BHT (Chesnut, 1993; Doberstein, 1993).
- (2) In concussion, decreased BP, either absolute (systolic pressure <115 mmHg) or relative (as compared with previous BP determinations), is a consistent finding 2 to 3 days postconcussion.

b. CAUSES:

- (1) Usually reflects extracranial bleeding; exceptions are massive bleeding from scalp lacerations and large intracranial hematomas in children (Rosman, 1983).
- (2) Other causes include MI, cardiac contusion or tamponade, tension pneumothorax (Doberstein, 1993).
- (3) May occur as preterminal event prior to herniation and brain stem compression. Also may play important role in pathogenesis of diffuse brain swelling (Aldrich, 1992).
- (4) When associated with normal pulse rate, hypotension may be due to spinal shock.
- (5) A review of a large trauma database described a small subset of patients that may experience "neurogenic hypotension," but investigators caution that this is a diagnoses of exclusion. More commonly, this finding probably was due to diuretic use in the absence of extracranial trauma (Chesnut 1998).

c. PROGNOSIS:

- (1) Hypotension as secondary brain insult is major determinant of outcome in severe BHT. Reduces cerebral perfusion pressure, with resultant cerebral ischemia and infarction. Increased morbidity and mortality related to severe trauma to extracranial organ system appears primarily attributable to associated hypotension (Chesnut, 1993; Pigula, 1993; Marmarou, 1991; Prough, 1997).
- (2) Associated with 150% increase in mortality (Chesnut, 1993). In children, hypotension with or without hypoxia causes significantly increased mortality compared with adults (Pigula, 1993).

3. BLOOD PRESSURE, INCREASED

- a. Hypertension in association with bradycardia (Cushing reflex) may occur following sudden rise in intracranial pressure and is ominous sign.
- b. Present in 10% of patients with epidural hematoma (Jamieson, 1968).

B. HEART RATE

1. OVERVIEW

- a. A normal pulse rate associated with hypotension may be indicative of spinal shock or secondary to medications patient may be taking, eg, beta-blockers.

2. BRADYCARDIA

- a. Hypertension in association with bradycardia (Cushing reflex) may occur following sudden rise in intracranial pressure and is ominous sign. Transient heart block may occur (Wirth, 1988).

3. TACHYCARDIA

- a. Common, nonspecific sign that may reflect fear, anxiety, drug effect, hypovolemia, or several other factors.

C. RESPIRATIONS

1. RESPIRATIONS, CHEYNE-STOKES

- a. The occurrence of an abnormal respiratory pattern usually suggests elevated intracranial pressure or primary involvement of brain stem respiratory centers.
- b. Cheyne-Stokes respiratory pattern is due to diffuse cortical process and may be sign of impending transtentorial herniation.

2. RESPIRATION, APNEIC

- a. The occurrence of an abnormal respiratory pattern usually suggests elevated intracranial pressure or primary involvement of brain stem respiratory centers.
- b. Apnea is sign of brain stem dysfunction at medullary level and carries poor prognosis (Gurdjian, 1979).
- c. Apneic respiration may also be due to drug effect, aspiration of gastric contents, or upper airway obstruction.

3. RESPIRATIONS, INCREASED

- a. May be due to brain stem involvement (central neurogenic hyperventilation) secondary to either transtentorial herniation or direct injury ischemia at midbrain level.
- b. Tachypnea may also be caused by hypoxia. Causes of hypoxia following head injury include upper airway obstruction, aspiration, hypovolemia, cervical spine injury, and injury to thoracic cage and/or pulmonary parenchyma.
- c. Increased respiratory rate and hypoxia also may be manifestations of neurogenic pulmonary edema, a poorly understood complication of head injury.

D. TEMPERATURE

1. OVERVIEW

- a. Abnormal temperature is uncommon in acute phase of head injury.

## 2. TEMPERATURE, INCREASED

- a. Abnormalities in temperature are uncommon acutely, but increase in temperature may occasionally occur and carries poor prognosis.
- b. Early hyperthermia (temperature  $>38.5$  C within first 24 h of admission) is associated with GCS of 8 or less, presence of cerebral edema or diffuse axonal injury on CT scan, and systolic hypotension, and predicts longer ICU length of stay and worse neurologic outcomes (Natale, 2000).

## E. CUSHING'S PHENOMENON

1. Classic rise in blood pressure and fall in pulse are late findings in the course of increased ICP and are unreliable in diagnosing a rising ICP.

## 1.4 PRESENTATION BY BODY SYSTEM

### 1.4.1 GENERAL DISCUSSION

- A. Typical signs and symptoms of head injury may not be present in intoxicated (Staffeld Cook, 1994) and pediatric (Dietrich, 1993) patients.
- B. Physicians are imperfect in their ability to predict the presence of intracranial injury based on clinical signs and symptoms (Reinus, 1994) or based on decreased Glasgow Coma Scale scores.
- C. Occult intracranial injury (ie, brain injury in absence of neurologic signs or symptoms) is a well-described entity in children less than 2 years old; risk is highest in those less than 1 year old (Gruskin, 1999; Greenes, 1998).
- D. In a prospective study of 608 children less than 2 years old with head trauma, 14 (48%) of 30 with intracranial injury were asymptomatic; infants less than 3 months of age were at highest risk (Greenes, 1999).

### 1.4.3 HEENT PRESENTATION

#### A. OVERVIEW

1. Injuries to scalp, maxillofacial structures, and cervical spine are commonly associated with head injuries (Davidoff, 1988; Michael, 1989); however, major brain injury does not need to be accompanied by external evidence of scalp injury.
2. Physical examination abnormalities directly attributable to head injury are discussed below.

#### B. LACERATION, SCALP

1. In the patient with an abnormal mental status or who is comatose, evidence of scalp lacerations should arouse the suspicion of a subdural hematoma.
2. Absence of scalp laceration does not rule out skull fracture (Banerjee, 1991).
3. Gentle digital palpation of the depths of an anesthetized scalp wound may detect an unsuspected skull fracture (Banerjee, 1991).

#### C. ABRASION, SCALP

1. In the patient with an abnormal mental status or who is comatose, evidence of scalp abrasions should arouse the suspicion of a subdural hematoma.

#### D. EDEMA, SCALP

1. In the patient with an abnormal mental status or who is comatose, evidence of scalp edema should arouse the suspicion of a subdural hematoma.
2. Soft tissue swelling  $\geq 4$  mm is usual concomitant of acute skull fracture (Kleinman, 1992).
3. Swelling secondary to subgaleal hematoma following minor head trauma has been reported (Cooling, 1991).

#### E. HEMATOMA, SCALP

1. Examination of the scalp for hematoma will provide evidence for the presence of head trauma.
2. Hematoma and ecchymosis of the mastoid area (Battle sign) are suggestive of basilar skull fracture but usually do not appear for up to 24 hours post-trauma.
3. Absence of scalp hematoma does not necessarily rule out skull fracture (Banerjee, 1991).
4. EDH in posterior cranial fossa should be suspected in patients with BHT involving an occipital blow resulting in a frontal or temporal hematoma (Lui, 1993).
5. Scalp hematoma is a useful clinical indicator of possible intracranial injury in infants. Of 14 asymptomatic infants with intracranial injury in one series, 13 (93%) had an associated significant scalp hematoma (Greenes, 1999).

#### F. ECCHYMOSIS, SCALP

1. In the patient with an abnormal mental status or who is comatose, evidence of scalp ecchymosis should arouse the suspicions of a subdural hematoma.

#### G. ECCHYMOSIS, PERIORBITAL

1. May follow direct fracture of orbital roof or anterior cranial fossa.

#### H. BATTLE'S SIGN

1. Area of ecchymosis behind pinna (Battle's sign) represents blood dissecting from basilar skull fracture but is usually not seen in first 12 to 24 hours following head injury.
2. If noted immediately following head injury, it may mean that direct mastoid trauma is present.

#### I. OTORRHEA, CEREBROSPINAL FLUID

1. Indicates meningeal disruption at site of basilar skull fracture, usually in petrous bone area.
2. RING SIGN: Helps detect CSF leak and diagnose basilar skull fracture. When CSF fluid mixed with blood is placed on filter paper, CSF will separate from blood and produce one or more concentric rings of clearer fluid. Reliable indicator of presence of CSF in blood but is not exclusive for CSF (Dula, 1993).

#### J. RHINORRHEA, CEREBROSPINAL FLUID

1. Basilar skull fracture of anterior fossa or cribriform plate may produce CSF rhinorrhea if there is meningeal disruption at fracture site (Gurdjian, 1978).
2. RING SIGN: Helps detect CSF leak and diagnose basilar skull fracture. When CSF fluid mixed with blood is placed on filter paper, CSF will separate from blood and produce one or more concentric rings of clearer fluid. Reliable indicator of presence of CSF in blood but is not exclusive for CSF (Dula, 1993).

#### K. HEMOTYMPANUM

1. Due to petrous or temporal bone fracture medial to tympanic membrane; indirect sign of basilar skull fracture (Schaffer, 1985).
2. May be seen in up to 80% of pediatric temporal bone fractures (Lee, 1998).

#### L. VISUAL ACUITY, DECREASED

##### 1. GENERAL:

- a. Complete or incomplete loss of vision may be noted either as result of direct nerve injury (usually from frontal impact and basilar skull fractures affecting optic nerve or chiasm) or from contusion of occipital temporoparietal areas of brain from contrecoup injuries (Elisevich, 1984; Upson, 1984; Kitchens, 1991).
- b. Head trauma, especially with associated facial fractures, is associated with an increased incidence of ocular injuries and mandates a scrupulous ocular exam with consideration of an ophthalmology consult (Pelletier, 1998).

##### 2. CONCUSSION:

- a. Patients frequently complain of blurred vision immediately following the injury; however, it is rare for this to last more than 1 or 2 hours after the amnesia clears.
- b. A blow over an occipital pole may give rise to blindness and possibly hemianopsia; this usually resolves within 1 to 2 days.

#### M. DIPLOPIA

1. Due to strabismus from injury to cranial nerve III or IV (Kitchens, 1991; Kwartz, 1990). Requires formal test of ocular motility (Kwartz, 1990).

#### N. FUNDUSCOPIC CHANGES

##### 1. PAPPILLEDEMA

- a. Funduscopic examination is usually normal immediately following head injury. Signs of papilledema generally do not develop for several hours.
- b. Of 426 patients with head injury, 15 (3.5%) developed papilledema, and only one patient was noted to have papilledema immediately after the event (Selhorst, 1985).

##### 2. HEMORRHAGE, RETINAL

- a. Bilateral retinal hemorrhages suggest subarachnoid hemorrhage.

##### b. CHILDREN:

- (1) Results from direct trauma or shaking of and whiplash injury to a young child. After the perinatal period, this finding in a child <3 years may be indicative of CNS injury resulting from child abuse. May occur in serious accidental head injury but is never seen in trivial head injuries (eg, falls from low heights) (Duhaime, 1992; Elder, 1991).

(2) Funduscopic examination of young children presenting with altered consciousness is mandatory; presence of retinal hemorrhages in absence of a history of witnessed severe trauma indicates nonaccidental trauma (Duhaime, 1992).

(3) Retinal hemorrhages were seen in about 90% of infants <1 year of age who had been shaken but in none who had been injured accidentally. The rarity of retinal hemorrhage in accidental trauma has been confirmed (Johnson, 1993).

(4) In children younger than 6.5 with head injuries, retinal hemorrhages were identified in 33% of victims of abuse versus 2% from accidental causes (Reece, 2000).

(5) Retinal hemorrhages caused by accidental mechanisms are usually ipsilateral to intracranial hemorrhage, often unilateral, and isolated to the posterior retinal pole (Christian, 1999).

#### **1.4.4 NECK PRESENTATION**

##### **A. TENDERNESS, NECK**

1. Careful examination of the cervical spine for tenderness should be performed because of a strong possibility of associated neck trauma. If mental status is abnormal, physical examination is not reliable, and cervical films are indicated. Neck sprains often will not be detected until 24 to 72 hours postinjury.

#### **1.4.7 GASTROINTESTINAL PRESENTATION**

##### **A. NAUSEA**

1. GENERAL: Occurs commonly in association with blunt head trauma, especially in children (McKissock, 1960).

2. CONCUSSION: Frequently accompanies concussion immediately following the accident and may continue through the second or third days postconcussion.

##### **B. VOMITING**

1. Occurs commonly in association with blunt head trauma, especially in children.

2. In patients with mild head injury, postinjury vomiting does not correlate with findings of acute radiographic abnormalities (Hsiang, 1997). Vomiting after mild head injury in children does not relate to injury mechanism or type of head injury (Brown, 2000).

3. A head CT should be obtained for patients with any one of the four Miller criteria (nausea, vomiting, headache, signs of depressed skull fracture) present after minor head trauma and a GCS of 14 (Miller, 1996; Holmes, 1997). However, these criteria should not be used as the only selection criteria for head CT scanning in these patients, since, when used alone, do not detect all patients with intracranial injuries who require neurosurgical intervention (Holmes, 1997).

4. Persistent vomiting may reflect elevation of intracranial pressure.

#### **1.4.10 NEUROLOGIC PRESENTATION**

##### **A. HEADACHE**

###### **1. IMMEDIATE (within 24 hours):**

a. GENERAL: Severe headache is not always common complaint immediately after head injury. Severity of headache often is inversely related to severity of injury (ie, the milder the head injury, the more severe the headache) (Yamaguchi, 1992).

b. SDH: In one series, headache was present in about 80% of patients with chronic subdural hematomas but in only about 10% of patients with acute subdural hematomas. (McKissock, 1960).

c. EDH: Complaint in about 1/3 of patients with epidural hematoma (McKissock, 1960).

###### **d. CONCUSSION:**

(1) Almost always present, although patients who are drowsy or confused may not complain of this symptom. Patients with mild head injury, particularly those >40 years or who complain of headache, are at increased risk of having an intracranial injury (Mikhail, 1992).

(2) A head CT should be obtained for patients with any one of the four Miller criteria (nausea, vomiting, headache, signs of depressed skull fracture) present after minor head trauma and a GCS of 14 (Miller, 1996; Holmes, 1997). However, these criteria should not be used as the only selection criteria for head CT scanning in these patients, since, when used alone, do not detect all patients with intracranial injuries who require neurosurgical intervention (Holmes, 1997).

###### **2. DELAYED (24 to 72 hours):**

a. CONCUSSION: Headache is most common symptom occurring during second or third day postconcussion. However, a few patients never experience any headache.

b. EDH: Nonresolving headache may be associated with a delayed epidural hematoma (Ashkenazi, 1990).

### 3. LATE (POSTCONCUSSION PERIOD):

- a. Common symptom in postconcussion phase. Some special forms of headache may be distinguished:
  - (1) Headache at site of scalp wound or bruise felt mainly when brushing the hair; due to localized traumatic neurosis.
  - (2) Headache due to neck injury, felt mainly in occipital region.
- b. May persist for months; 25% to 35% of patients experience headache 6 weeks postinjury, and 90% still complain of headaches 1 year postconcussion.

## B. ALTERED MENTAL STATUS

### 1. OVERVIEW

#### a. GENERAL:

- (1) Most important sign to follow may range from mild confusion to deep coma for assessment of head-injured patient.
- (2) Isolated transient loss of consciousness is frequently an indicator of significant injuries. In one series, predicted need for an emergency operation in 4% to 19% of patients; most of the procedures were nonneurosurgical (Owings, 1998).
- (3) Causes include diffuse cerebral event, eg, cortical swelling or large or bilateral intracranial hematomas; direct injury to brain stem; secondary compression of brain stem following transtentorial herniation; or a metabolic process affecting cortex or brain stem, eg, hypoxia, hypovolemia, or a drug effect.
- (4) Eighty percent of patients who deteriorate into coma after sustaining an apparently nonsevere head injury have a mass lesion potentially requiring surgery (Lobato, 1991).
- (5) MENTAL STATUS EXAMINATION: GCS score does not provide information regarding mental status.
  - (a) Should be performed on all patients with normal GCS scores and should include determination of orientation to time, person, and place and evaluation of short-term memory (Salluzzo, 1992).
  - (b) Less significant in patients with abnormal GCS scores because they already are in higher risk group for intracranial injury (Salluzzo, 1992).
  - (c) Motor component of GCS score may be equivalent to overall score for mortality prediction and when deciding on triage to trauma center (Ross, 1998).
- (6) CT SCAN: Should be obtained in the ED for patients with LOC or amnesia, regardless of GCS score; scans are abnormal in almost 20% of patients with GCS scores >13 (Harad, 1992).

#### b. CONCUSSION:

- (1) Patients often experience a loss of consciousness (LOC), which may be the result of temporary interruption of the reticular activating system responsible for maintaining a state of alertness.
- (2) By the time patients are seen in the ED, they usually have recovered consciousness.
- (3) LOC is not necessary or reliable to make the diagnosis of concussion.

#### c. EPIDURAL HEMATOMA:

- (1) The classic description is that of loss of consciousness, return to a "lucid" state of consciousness, followed by lapse into unconsciousness.
- (2) In a review of patients with posterior fossa EDH, 70% to 80% had continuous or transient consciousness before surgery (Pozzatti, 1989). Another study reported no LOC or amnesia in the majority of cases (Miller, 1990).
- (3) The presence of coma predicts a higher mortality in patients with epidural hematoma (Lobato, 1988).
- (4) A study of serial changes in acute EDH size and associated changes in LOC and ICP found that the hematoma enlarged in 65% of patients. Eight of 13 patients unchanged level, 2 deteriorated, and 3 improved, indicating relative stability in the state of consciousness despite the marked changes in hematoma size (Sakai, 1988).
- (5) Data suggest that a period of stability in LOC is a period during which compensatory mechanisms can maintain the stability of the intracranial condition during progressive enlargement of the hematoma (Sakai, 1988).
- (6) EDH can develop within hours and be associated with a sudden neurologic change despite normal blood pressure and no clinical evidence of elevated ICP to explain the delayed appearance. Slower hemorrhage is thought to be due to a venous source, usually a torn dural sinus (Pozzatti, 1989).
- (7) Symptoms, including altered mental status, cannot be relied upon in infants. The diagnosis of EDH depends upon clinical signs and CT scanning in children less than 2 years old (Beni-Adani, 1999).

d. SUBDURAL HEMATOMA:

(1) CHANGES IN AROUSAL:

(a) Any change in alertness from lethargy to obtundation and coma may represent increasing intracranial pressure with structural or metabolic changes in brainstem ascending reticular activating system (found in midbrain and upper pons).

(b) Also occurs with diffuse bilateral cerebral hemispheric dysfunction.

(2) CHANGES IN THOUGHT CONTENT: Any change in orientation, or evidence of confusion, personality change, or delirium, may represent injury to the frontal or parietotemporal lobes secondary to contusion of intracerebral hemorrhage.

(3) A lucid interval has been reported in 12% to 37% of patients with acute SDH (Jamieson, 1968).

2. GLASGOW COMA SCALE

a. Permits descriptive and reproducible assessment of patient's state of consciousness; best scale for predicting outcome and is simplest and least time-consuming system. Correlates highly with severity of brain injury (Rocca, 1989; Salluzzo, 1992).

b. Use of GCS score should replace terms such as lethargy or stupor, which are nonspecific and vary in meaning from one observer to another.

c. Should be utilized for repeated observation of head-injured patients to help predict prognosis. The GCS score was derived as a prognosticator, not as a treating device. To make an accurate assessment for treatment, other parameters need to be evaluated, eg, vital signs, mechanism of injury, and associated injuries (Kohi, 1984; Winkler, 1984).

BEST MOTOR RESPONSE:	Obeys	6
	Localizes pain	5
	Withdraws	4
	Flexion to pain	3
	Extension to pain	2
	None	1
BEST VERBAL RESPONSE:	Oriented	5
	Confused conversation	4
	Inappropriate words	3
	Incomprehensible	2
	None	1
EYE OPENING:	Spontaneously	4
	To speech	3
	To pain	2
	None	1

d. INJURY SEVERITY: Rate of change of the GCS may be as important as the absolute value (Schaffer, 1985; Cohadon, 1991):

(1) Mild: GCS between 13 and 15 (High-risk mild head injury = GCS score of 13 or 14, or GCS score of 15 with acute radiographic abnormalities) (Hsiang, 1997).

(2) Moderate: GCS between 9 and 12

(3) Severe: GCS between 3 and 8

e. INTOXICATION: May not be accurate in presence of alcohol or drugs. One study of 257 head-injured patients revealed that alcohol-intoxicated patients had significant depression of their GCS score related to blood alcohol level (Jagger, 1984).

f. MENTAL STATUS EXAMINATION: GCS score does not provide information regarding mental status.

(1) Should be performed on all patients with normal GCS scores and should include determination of orientation to time, person, and place and evaluation of short-term memory (Salluzzo, 1992).

(2) Less significant in patients with abnormal GCS scores because they already are in higher risk group for intracranial injury (Salluzzo, 1992).

(3) Motor component of GCS score may be equivalent to overall score for mortality prediction and when deciding on triage to trauma center (Ross, 1998).

g. CT SCAN: Should be obtained in the ED for patients with LOC or amnesia, regardless of GCS score; abnormal in almost 20% of patients with GCS scores >13 (Harad, 1992).

## C. AMNESIA

### 1. GENERAL:

- a. Most reliable sign in establishing the diagnosis of concussion: concussion is present following trauma to the head whenever there is complete amnesia, even if for only a short period of time.
- b. A period with unclear but not totally absent memories constitutes a borderline case and probably should be considered a concussion if typical symptoms are present in the days and weeks postinjury.

### 2. TYPES:

- a. May have two components - retrograde and posttraumatic (or anterograde). Posttraumatic amnesia is present in virtually every case; a retrograde amnesia of 5 to 10 seconds is common.
- b. Unusual for the patient to remember any painful sensation at the moment of impact, although at times the amnesia does not commence until 2 to 3 seconds after the accident.
- c. In a small number of cases, there may be period of retrograde amnesia lasting 1 to 3 hours.

### 3. DURATION:

- a. Best general guide to the severity of the concussion; always should be estimated with care and recorded.
- b. In measuring its duration, the correct end-point is where the memories become clear and continuous. There frequently are "islands" of more or less clear memory earlier than the true end-point of the amnesia.
- c. Duration of posttraumatic amnesia may predict long-term outcome from mild and moderate head injuries (VanderNaalt, 1999).

4. CT SCAN: Should be obtained in the ED for patients with LOC or amnesia, regardless of GCS score; scans are abnormal in almost 20% of patients with GCS scores >13 (Harad, 1992).

## D. NEUROLOGIC SIGNS, FOCAL

### 1. HEMIPARESIS

- a. Most common motor sign following blunt head trauma and suggests impending transtentorial herniation. In unconscious patients, decreased motor tone suggests hemiparesis; monoparesis is uncommon.
- b. As a hematoma expands and exerts pressure across the tentorium, compression of the pyramidal tract produces contralateral muscle weakness or paralysis.
- c. Occurs in 33% to 70% of patients with an epidural hematoma; most common with lateral hematomas and rare with basal and posterior fossa hematomas (Jamieson, 1968; Gallagher, 1968).
- d. Motor abnormality generally occurs contralateral to the lesion.
  - (1) In series of 125 patients with epidural hematoma, limb paresis was present in 85 patients and was contralateral to the lesion in 95% of cases (McKissock, 1960).
  - (2) False lateralization of motor paresis occurs with compression of contralateral cerebral peduncle against free edge of the tentorium, which is referred to as cruciate syndrome, or Kernohan's notch.
- e. Some authors feel that hemiparesis is less valuable than pupillary dilatation as localizing sign.
- f. Hemiparesis or other focal territorial sign with normal CT scan mandates consideration of a carotid vascular injury.

### 2. APHASIA

- a. Expressive or receptive aphasia may follow head injury and indicates involvement of dominant hemisphere (McKissock, 1960).
- b. Cause of this deficit is unclear; it may be due to anterior cerebral artery spasm or brainstem injury (Ferrara, 1985).

### 3. BABINSKI SIGN

- a. Indicates pyramidal tract abnormality and head injury; may develop as a result of compression of the peduncles; may be one of earliest signs of compression (Gallagher, 1968).
- b. In one report of children with head trauma, presence of isolated Babinski sign was not indicative of significant injury or poor outcome (Parish, 1985).

## E. REFLEX, CORNEAL, DECREASED

1. This reflex requires intact fifth and seventh nerves, both of which originate in the pons. Therefore, an absent corneal reflex may indicate pontine dysfunction or injury to cranial nerves V or VII.

## F. REFLEX, OCULOCEPHALIC

1. Oculocephalic reflex ("doll's eye" maneuver) consists of conjugate eye deviation contralateral to direction of head rotation.
  - a. Response is dependent upon intact connections between vestibular apparatus, gaze centers in pons, and brain stem nuclei of third and sixth nerves.
  - b. In comatose patient, normal oculocephalic reflex indicates brain stem is intact.
2. Testing for this reflex should never be done in head-injured patient until cervical spine injury has been ruled out.

## G. REFLEX, OCULOVESTIBULAR

1. Tests same neural reflex arc as the oculocephalic reflex but with a stronger stimulus, namely cold water squirted against tympanic membrane.
  - a. Normal response (ie, indicating intact brain stem) in comatose patient is conjugate, tonic deviation of eyes in direction of stimulus.
  - b. Normal response in conscious patient is fast component of nystagmus away from irrigated ear.

## H. PALSY, CRANIAL NERVE

1. MECHANISM OF INJURY: Direct trauma to the nerve; stretching injury; vascular compromise (Kitchens, 1991; Keane, 1992).
2. INCIDENCE:
  - a. In one series, occurred in almost 25% of children with basilar skull fractures (Kitchens, 1991).
  - b. Olfactory, facial, and audiovestibular nerves injured most often, followed by optic and ocular motor nerves; trigeminal and lower cranial nerves rarely involved (Keane, 1992).
3. PRESENTATION: Difficult to diagnosis in severe BHT; coma may obscure all but III, VI, VII nerve damage (Keane, 1992).
  - a. OLFACTORY NERVE (CN I):
    - (1) Most frequently injured nerve in BHT (up to 8% of cases overall and to 30% with severe BHT or anterior fossa fractures); more common with occipital than frontal blows and can result from trauma to any part of head; anosmia often permanent (Keane, 1992; Kitchens, 1991).
    - (2) Most patients with complaints of olfactory dysfunction have anosmia and rarely regain normal olfactory function; parosmia prevalence decreases over time (Doty, 1998).
  - b. OPTIC NERVE (CN II): Often results from blow to ipsilateral outer eyebrow and occasionally from temporal-parietal blow; usually associated with severe head injury with LOC; typically results in total blindness or field defect (Keane, 1992; Kitchens, 1991). Also occurs with fractures at the skull base (Poon, 1999).
  - c. OCULOMOTOR NERVE (CN III):
    - (1) Causes diplopia due to external strabismus; signs of injury include dilated pupil and turned-out eye; nerve damage must be distinguished from diffuse orbital muscle limitation (Keane, 1992; Kitchens, 1991).
    - (2) Third nerve palsy with intact sensorium is not due to acute brainstem compression/herniation.
  - d. TROCHLEAR NERVE (CN IV): Rarely diagnosed acutely; presents with internal strabismus; diagnosis requires high level of patient cooperation; usually resolves completely (Kitchens, 1991; Kwartz, 1990; Keane, 1992).
  - e. TRIGEMINAL NERVE (CN V): Usually associated with facial lacerations and crush injuries to lateral skull; gasserian ganglion and trigeminal trunk rarely injured in BHT (Keane, 1992; Kitchens, 1991).
  - f. ABDUCENS NERVE (CN VI): Patient cooperation necessary for diagnosis; usually resolves completely (Kitchens, 1991).
  - g. FACIAL NERVE (CN VII):
    - (1) One of most frequently injured CNs in BHT (up to 5%); associated with petrous bone fracture; resolves completely within 3 mo in 60% to 75% of cases; deficits beyond this time are permanent (Kitchens, 1991; Keane, 1992; Nageris, 1995; Glarner, 1994; Simo, 1996).
    - (2) Important to distinguish between central and peripheral involvement of facial nerve. The former, characterized by paralysis of lower half of face with sparing of forehead, suggests pressure effect at cortical level and is more likely to be associated with further neurologic deterioration.
  - h. VESTIBULOCOCHLEAR NERVE: May occur with temporal bone fractures or labyrinthine concussion; most commonly caused by conductive defects associated with hemotympanum; may result in permanent minor hearing loss (Kitchens, 1991; Keane, 1992).
  - i. LOWER CRANIAL NERVES: Cranial palsies to nerves IX, X, XI, and XII have been described with associated basal skull fractures, especially those involving occipital condyle fractures (Legros, 2000).
4. PROGNOSIS: Varies with nerve involved; chances of recovery greatest for facial nerve (40%), intermediate for ocular motor system, and least likely for olfactory, optic, and audiovestibular nerves (Keane, 1992).

## I. POSTURING, DECORTICATE

1. Indicates hemispheric level of dysfunction and may precede transtentorial herniation.

## J. POSTURING, DECEREBRATE

1. As a hematoma expands and intracerebral pressure increases, pressure on the brainstem may produce a decerebrate response to stimuli.
2. Poor prognostic sign indicating midbrain level of CNS function.

- a. In one series, this sign was seen in 50% of patients with impending transtentorial herniation and correlated well with poor functional recovery (Hoff, 1978).
- b. However, in another study of 62 patients with bilateral decerebration, 20 patients survived and 11 had good recovery (Mahapatra, 1985).

## K. PUPILLARY CHANGES

### 1. OVERVIEW

- a. May warn of impending cerebral herniation, may provide clues regarding presence of concomitant toxic or metabolic disorder, and may give information about integrity of optic nerve and retina (Salluzzo, 1992).

### 2. GAZE DEVIATION

- a. May be caused by injuries involving cortex, brain stem, cranial nerves III, IV or VI, eye muscles, or orbit.
  - (1) CORTEX: Conjugate deviation of both eyes toward side of head injury or away from paretic or paralyzed side suggests cortical lesion.
  - (2) BRAIN STEM: Skew deviation of eyes occurs in brain stem injuries. Also seen are uncoordinated movements of eyes without fixation.
  - (3) CRANIAL NERVES: Cranial nerves innervating eye muscles most commonly affected by blunt head trauma are the oculomotor (III) and abducens (VI) nerves.
    - (a) Injury to a oculomotor nerve will cause external deviation of eye with slight downward deviation. Pupillary dilatation usually precedes disturbances of extraocular motor function when caused by third nerve injury (Byrnes, 1979).
    - (b) Inward deviation of eye will follow injury to sixth cranial nerve.

### 3. ANISOCORIA

- a. CAUSES: Unilateral pupillary dilatation with poor response to light may be due to direct or indirect injury to oculomotor nerve, compression of nerve or brain stem during transtentorial herniation, ocular trauma, instillation of mydriatic, or prior illness or injury involving eye or optic nerve.
  - (1) In setting of head trauma, it should be assumed that patient with dilated, poorly responsive pupil and altered mental status has expanding intracranial mass lesion (Gutman, 1992). The presence and side of pupillary dilation does not reliably predict the presence and side of an intracranial lesion (Chesnut, 1994).
  - (2) In study of patients who, after sustaining head injury, were found to develop intracranial hemorrhage, 24/36 were noted to have normal pupils on arrival to hospital (Edna, 1983).
  - (3) Anisocoria in the setting of epidural hematoma is associated with a three times higher mortality; outcome is improved with rapid surgical evacuation after the development of anisocoria (Cohen, 1996).
- b. DIRECT INJURY: Oculomotor nerve may be directly injured at tentorial opening, in area of posterior clinoid process, and as it exits cranial cavity along superior orbital fissure.
  - (1) In one series, 31% of third nerve injuries were classified as direct.
  - (2) Should be suspected when pupil dilatation occurs immediately following injury, if level of consciousness improves despite pupillary dilatation, and if vital signs are stable (Byrnes, 1979).
- c. INDIRECT INJURY: In patients with dilated pupil and altered mental status, one should generally assume that the nerve is indirectly affected.
  - (1) Mechanisms of injury include compression by expanding or shifting intracranial structures, midbrain ischemia involving third nerve nucleus, or trapping of nerve against bony, ligamentous, or vascular intracranial structures.
  - (2) Third nerve palsy is on same side as expanding lesion in 80% to 85% of cases.
  - (3) Dilated pupil is late sign of elevated intracranial pressure, but generally precedes extraocular function abnormalities when caused by third nerve compression (Byrnes, 1979).

## L. DIZZINESS

- 1. CONCUSSION: Common feature; may occur immediately following trauma and last for several days; frequently aggravated by a change in head position.

## M. RESTLESSNESS

- 1. May be a sign of increased ICP and impending deterioration in children with mild to moderate head injury. Deterioration may result from delayed cerebral hyperemic state (Humphreys, 1990).

## 1.5 COMPLICATIONS

### A. SEIZURES, POSTTRAUMATIC

- 1. GENERAL: Serious complication; can cause secondary brain damage through increased metabolic requirements, increased ICP, cerebral hypoxia, and/or excessive release of neurotransmitters (Chiaretti, 2000).

2. **INCIDENCE:** In two studies of patients with severe head injury, incidence of seizures was 7% to 20% at one year and 10% to 20% at two years postinjury (Temkin, 1990).
3. **RISK FACTORS:**
  - a. Occur more commonly after intradural than extradural hematoma; additional intradural injury increases the risk of epilepsy (Jamjoom, 1991).
  - b. In a large cohort study of 4541 patients with traumatic brain injury, significant risk factors for late seizures were brain contusion with SDH, loss of consciousness or amnesia for >1 day, and age >65 y. Probability in first year ranged from 1.5% to 7% based on severity of injury; after first 5 years, only those patients with moderate or severe injury had increased incidence of unprovoked seizures (Annegers, 1998).
  - c. In children, a GCS score of 3 to 8 appears to be predictive of posttraumatic seizures (Lewis, 1993).
4. **CONCUSSION:** A study of over 4200 patients with mild closed head injury (defined as a blow to the head, loss of consciousness, or posttraumatic amnesia <30 minutes), 2% developed seizures within the 1st week and 1% within the 1st 24 hours.
  - a. Majority had tonic clonic seizures. Half with seizures had normal CT scans. Most commonly seen on abnormal CT scans were intracerebral parenchymal damage, followed by subdural hematoma, then epidural and subarachnoid hemorrhages.
  - b. Parenchymal damage was most common in the frontal and temporal lobes; incidence was higher in patients with seizures between the 2nd and 7th day postinjury.
  - c. More than 90% of these patients had no neurologic deficits 6 months postinjury.
5. **CT SCAN:** Should be performed on all patients developing seizures within the first week postinjury, even though incidence of correctable intracranial hemorrhage is low.
6. **PROPHYLACTIC ANTICONVULSANTS:** Lessens risk of seizures in immediate posttraumatic period (within 7 days) (Wohns, 1979; Temkin, 1990; Hauser, 1990; Lewis, 1993), but no long-term benefit has been shown to seizure prophylaxis (Scheirhout, 1998).

## B. POSTCONCUSSION SYNDROME

1. **DEFINITION:** A relatively well-defined group of disturbances that usually follow mild head injury, often last months or years, and frequently prevent the return to preaccident activities or employment.
2. **CHARACTERISTICS:**
  - a. Headache, vertigo, anxiety, fatigue, irritability, visual disturbances, memory difficulty, cognitive difficulties (loss of ability to process more than one kind of information simultaneously), reduced tolerance for alcohol, loss of insight, and personality changes. Someone in an intellectually demanding role may notice these symptoms for a year postinjury (Mittenberg, 1991, 1992; Evans, 1994; Packard, 1994a).
  - b. **REACTION TIME:** Used to document objectively the presence of neurologic impairment. In one report, reaction times of patients with concussion were significantly slower than controls; the reaction times showed improvement over 6 months (MacFlynn, 1984).
  - c. Postconcussive symptoms probably under recognized and may even occur in multiple trauma patients who were not initially thought to have had a head injury. In an uncontrolled study of patients who had been evaluated and discharged from a Level 1 trauma center after blunt trauma, none had a history or had signs or symptoms of traumatic head injury. In a 1-month telephone follow-up, 1/3 reported symptoms consistent with a postconcussive syndrome (mostly insomnia, headaches, irritability or fatigue). No noninjured "control" patients were studied (Chambers, 1996).
3. **CONTRIBUTING FACTORS:** May include brain injury (focal cortical contusions and diffuse axonal injury); head and neck injury; posttraumatic emotional stress; iatrogenic contributions (eg, poor physician follow-up or discharge instructions); and secondary stress (McMillan, 1987).
4. **PROGNOSIS:**
  - a. Neurologic status immediately following the injury is not predictive of the severity of neuropsychologic deficits (Leininger, 1990).
  - b. One study found that half of concussion patients had one symptom 6 weeks postinjury; 15% still had symptoms at the end of one year (Rutherford, 1977, 1979).
5. **SECOND-IMPACT SYNDROME:**
  - a. Increasingly recognized that repeated, seemingly minor blows to the head, especially during football, hockey, boxing, skiing, and other contact sports, can have cumulative and catastrophic effects (Am Acad Neurol, 1997; Kelly, 1997).
  - b. Occurs when a second concussion is sustained before an individual has had time to recover from an initial injury. May be sudden and severe; characterized by acute brain edema and rapid neurologic deterioration. Edema, coma, and death appear to result from failure of cerebral autoregulation and vascular congestion (Kelly, 1997).
  - c. A review of published reports casts doubts on the frequency and existence of second-impact syndrome, recommending more accurate reporting to help verify its existence (McCroory, 1998).

#### C. RHINORRHEA, CEREBROSPINAL FLUID, PERSISTENT

1. CSF leaks occasionally follow basilar skull fracture, sometimes years after initial injury. Surgical repair is generally required (Chandler, 1983; Nageris, 1995; Glarner, 1994).

#### D. HEMATOMA, SUBGALEAL

1. Has been reported following minor head trauma (Adelola, 1975; Kuban, 1983; Cooling, 1991). Does not per se indicate presence of serious underlying head injury; however, there may be significant hemodynamic compromise (Cooling, 1991).
2. Infants can lose up to 50% of their total blood volume into the subgaleal space (Anton, 1999).
3. Key clinical features to assess are patient's hemodynamic status, need for blood transfusion, and correction of any coagulopathy (Cooling, 1991).
4. Aspiration generally is not recommended unless diagnosis is uncertain, complications exist (eg, dissection into retro-orbital space), or patient is markedly symptomatic (Cooling, 1991).

#### E. HYDROCEPHALUS

1. Subarachnoid hemorrhage occurring at time of head injury may lead to impaired resorption of CSF and development of communicating hydrocephalus.
2. In one report of 17 patients with posttraumatic hydrocephalus, onset of symptoms was within one year in all but one case. Main symptoms were dementia, impaired level of responsiveness, and ataxia (Cardoso, 1985).
3. CT criteria for making this diagnosis are as follows:
  - a. Dilatation of unobstructed ventricular system;
  - b. Obliteration of cerebral sulci;
  - c. Areas of periventricular low density;
  - d. Rounding of frontal horns of lateral ventricles.

#### F. ANOSMIA

1. Avulsion of the first cranial nerve (olfactory nerve) may occur secondary to fracture of the cribriform plate and generally results in permanent loss of smell (Kitchens, 1991).
2. Most patients with complaints of olfactory dysfunction have anosmia and rarely regain normal olfactory function; parosmia prevalence decreases over time (Doty, 1998).

#### G. BLINDNESS

1. Some cranial nerve deficits observed after blunt head injury may be permanent. Injury to optic nerve manifests as sudden rather than gradual loss of vision. In one series, 80% of children with CN II injury had total blindness without improvement (Kitchens, 1991).
2. Cortical blindness, which is most often transient, may occur (Woodward, 1990).

#### H. HEARING LOSS

1. Acoustic nerve is a frequently injured cranial nerve. Hearing loss may occur secondary to fracture of petrous ridge or direct injury of tympanic membrane or structures of middle ear (Glarner, 1994).
2. Conductive hearing loss follows longitudinal fractures of the temporal bone and varying degrees of sensory losses with transverse fractures. Mild high-frequency sensorineural hearing loss also may occur from BHT not necessarily involving temporal bone fracture (Zimmerman, 1993). Significantly more common than sensorineural or mixed hearing loss (Lee, 1998).
3. Conductive hearing loss also can occur after mild head injury as a result of blood in the middle ear or disruption of the ossicular chain (Evans, 1992).
4. No correlation between cause of injury, LOC, or GCS scores and presence, type, or degree of hearing loss (Zimmerman, 1993).

#### I. MENINGITIS, BACTERIAL

1. Violation of meninges, clinically detected by presence of CSF rhinorrhea or otorrhea, may lead to meningitis (Glarner, 1994). Most common organisms are *Streptococcus pneumoniae* and *Haemophilus influenzae* (Rosman, 1983).

#### J. THROMBOSIS, CEREBRAL SIGMOID SINUS

1. Should be suspected in patients with persistent or delayed gait ataxia, headache, and protracted vomiting following trauma to the back of the head. May occur after mild trauma and in absence of skull fracture. Course usually is benign, with symptoms resolving within 6 weeks of injury (Taha, 1993).

#### K. THROMBOSIS, CEREBRAL, SAGITTAL SINUS

1. Superior sagittal sinus thrombosis is rare complication. Altered mental status, seizures, headache, and hemiparesis are most common presenting symptoms (Ferrera, 1998; Hesselbrock, 1985).
2. Diagnosis is made by findings on CT scanning, cerebral angiography, or MRI (Ferrera, 1998).
3. Outcome is variable, with both persistent neurologic deficits (Ferrera, 1998) and full recovery (Bousser, 1985) reported.

#### L. ANEURYSM, TRAUMATIC

1. Fracture of skull (clivus, sella turcica) in proximity to vascular structures may lead to complications such as aneurysm or stenosis (Meguro, 1985; Liu, 1985; Holmes, 1993).
2. One report of three patients with fracture of the sella turcica revealed vascular complications in all cases (Kojima, 1985).

#### M. SYNDROME OF INAPPROPRIATE ANTIDIURETIC HORMONE

1. SIADH may be a complication of blunt head trauma (Lieblich, 1985; Born, 1985a) and may be diagnosed utilizing the following criteria:
  - a. Hyponatremia and serum hypoosmolality;
  - b. Urine osmolality greater than serum osmolality;
  - c. Continued renal loss of sodium;
  - d. Normal renal, adrenal, and thyroid function;
  - e. Patient not dehydrated.

#### N. EDEMA, PULMONARY, NEUROGENIC

1. Poorly understood complication of severe blunt head injury. A review of fatal and nonfatal head injuries suggested that neurogenic pulmonary edema may occur in as many as 50% of patients dying of isolated head injuries within 96 h of the injury (Rogers, 1995).
2. Mechanism of neurogenic pulmonary edema that occurs as consequence of head injury in absence of pulmonary aspiration, multiple organ failure or direct trauma to the lungs, abdominal contents, diaphragm or thoracic cage is unknown (Rogers, 1995).
  - a. However, numerous pathogenic factors may be involved, including pulmonary hypertension, increases in pulmonary blood volume, inflammatory responses, humoral vasoactive mediators and capillary permeability defects.
  - b. Characterized by congestion, hemorrhage and edema within the lungs, in absence of similar changes in other visceral organs ("selective edema").

#### O. PNEUMONIA

1. Prolonged hospitalization in head-injured patients is associated with 50% incidence of infectious complications, most commonly pneumonia.
2. Tends to occur early in course (within first week postinjury), which is consistent with increased risk of initial aspiration of oropharyngeal or gastric contents because of associated LOC. Intubation and mechanical ventilation may be relatively low risk in absence of other injuries (Hsieh, 1992).

#### P. COGNITIVE IMPAIRMENT

1. Subtle cognitive disorders may develop, especially after moderate to severe head injury (Gensemer, 1989; Williams, 1990; Levin, 1990). Also occurs in 20% of patients following mild head injury (Evans, 1992; Fisher, 1994; Levitt, 1994).
2. Formal neuropsychological testing is necessary to document subtle cognitive impairments, which have significant ramifications on both treatment and prognosis after mild traumatic brain injury (Collins, 1999).

#### Q. PERSONALITY CHANGE

1. May occur after mild (Fisher, 1994) as well as moderate to severe (Gensemer, 1989; Williams, 1990; Levin, 1990) head injury.

#### R. SECOND IMPACT SYNDROME

1. Increasingly recognized that repeated, seemingly minor blows to the head, especially during football, hockey, boxing, skiing, and other contact sports, can have cumulative and catastrophic effects (Am Acad Neurol, 1997; Kelly, 1997).
2. Occurs when a second concussion is sustained before an individual has had time to recover from an initial injury. May be sudden and severe; characterized by acute brain edema and rapid neurologic deterioration. Edema, coma, and death appear to result from failure of cerebral autoregulation and vascular congestion (Kelly, 1997).

3. A review of published reports casts doubts on the frequency and existence of second-impact syndrome, recommending more accurate reporting to help verify its existence (McCrory, 1998).

#### S. HYPOPITUITARISM

1. Thought to occur in <1% of patients with BHT; however, autopsy studies of patients who died as result of head injury revealed 8% to 22% incidence of necrosis of anterior pituitary.
2. May present as early as two hours postinjury.

#### T. COAGULOPATHY

1. Coagulopathy present on admission is important marker to predict occurrence of secondary brain injury. Is one of mechanisms by which initial destruction of cerebral tissue causes delayed insults. Associated with poor neurologic outcome (Stein, 1993).
2. Patients with severe head injuries may develop a diffuse coagulopathy, due to fibrinogen depletion and disseminated intravascular coagulation. Presumably, the coagulopathy develops as the result of release of tissue thromboplastin from the injured brain. A marked depletion of coagulation factors may occur within four hours of injury, leading to a higher likelihood of death (Hulka, 1996).
3. Some studies indicate that the risk of developing coagulopathy is higher in children (May, 1997) and that degree of coagulopathy correlates with severity of injury.
4. Patients with isolated severe head injury develop procoagulant factors within 6 h of injury, causing regional and systemic hypercoagulability; may set off DIC in this patient subset (Scherer, 1998).

#### U. TRAUMA, ARTERIAL, CAROTID

1. Traumatic carotid-cavernous sinus fistula is a rare complication of head injury and can result in steal phenomena, massive epistaxis, and intracerebral hematoma (Iida, 1995).
2. Dissection, thrombosis, and pseudoaneurysm also have been reported (Goodwin, 1994; Klufas, 1996).

#### V. MENINGIOMA

1. An international, multicenter, case-control study of 1178 glioma patients and 330 meningioma patients suggests that previous head trauma may cause a mild increase in the risk of developing meningioma. The authors of the study warn that their results should be interpreted cautiously (Preston-Martin, 1998).

## 2.0 LABORATORY DATA

### 2.2 HEMATOLOGIC

#### A. WHITE BLOOD CELLS

1. OVERVIEW
  - a. The WBC count is usually normal.
2. WHITE BLOOD CELLS, INCREASED
  - a. May be reflection of an acute stress reaction.

#### B. HEMATOCRIT

1. HEMATOCRIT, DECREASED
  - a. Hematocrit value may be decreased if there is significant blood loss from scalp or associated extracranial injuries, or in children with cephalohematoma.
  - b. Anemia (Hct <30) can lead to secondary brain injury by potentiating cerebral ischemia; often present in conjunction with arterial hypotension (Doberstein, 1993).

#### C. COAGULATION TESTS

1. OVERVIEW
  - a. Coagulopathy present on admission is important marker to predict occurrence of secondary brain injury; associated with poor neurologic outcome (Stein, 1993).
  - b. Fibrinolytic parameters (plasma levels of plasmin inhibitor-plasma complex (PIC) and D-dimer) on admission are reliable prognostic markers of head injury in patients who talk and deteriorate (Takahashi, 1997).
2. FIBRIN DEGRADATION PRODUCTS
  - a. FIBRIN DEGRADATION PRODUCTS, INCREASED
    - (1) Elevated fibrin degradation products and elevated partial thromboplastin time are associated with poor prognosis (Olson, 1989; Crone, 1987).

### 3. PARTIAL THROMBOPLASTIN TIME

#### a. PARTIAL THROMBOPLASTIN TIME, PROLONGED

(1) Elevated fibrin degradation products and elevated partial thromboplastin time are associated with poor prognosis (Olson, 1989; Crone, 1987).

### 4. PROTHROMBIN TIME

#### a. PROTHROMBIN TIME, PROLONGED

(1) In patients with increased INR due to chronic anticoagulation with warfarin, the incidence of clinically significant intracranial injury is extremely low after minor blunt head trauma without LOC or neurologic signs (Garra, 1999).

## 2.3 ELECTROLYTES

### A. SODIUM

#### 1. SODIUM, DECREASED

a. Commonly noted several days after head injury secondary to inappropriate secretion of antidiuretic hormone but is not seen acutely.

#### 2. SODIUM, INCREASED

a. Occasionally, hyponatremia and diabetes insipidus may be present secondary to hypothalamic injury.

### B. POTASSIUM

#### 1. POTASSIUM, DECREASED

a. Patients with severe head injuries are at increased risk of hypokalemia (Polderman, 2000).

## 2.4 CHEMICAL SURVEY

### A. GLUCOSE

#### 1. GLUCOSE, INCREASED

a. Nonketotic hyperglycemia may occur following significant head trauma, most likely due to secretion of insulin antagonists, eg, cortisol and growth hormone. Increases in glucose level may correlate with the severity of head injury (Yang, 1995; Margulies, 1994).

b. Some data suggest that increased glucose levels may indicate worse neurologic outcome (Chiaretti, 1998; Lam, 1991; Michaud, 1991). However, one study found that peak glucose did not add to the power of admission GCS score and Simplified Acute Physiology Score to predict neurologic outcome (Margulies, 1994).

### B. CREATINE PHOSPHOKINASE

#### 1. CREATINE PHOSPHOKINASE, INCREASED

a. BB FRACTION: Reported to be elevated in as many as 50% of patients sustaining serious head injury.

(1) High elevations may indicate more serious brain injury, but, in general, there is not good correlation between these two variables (Karpman, 1981; Kaste, 1981; Rabow, 1986).

(2) It has been suggested that an "extrapolated" CSF CK-BB level, which theoretically occurs immediately after injury, may be accurate predictor of brain injury (Hans, 1987).

b. MB FRACTION: Reported to occur in as many as 93% of patients sustaining serious head trauma. Nonspecific EKG changes and autopsy evidence of myocardial necrosis seem to be more common in patients with elevation of MB isoenzyme level, but this correlation has not been tested in large clinical study (Kaste, 1981; Hackenberry, 1982).

### C. PHOSPHATE

#### 1. PHOSPHATE, DECREASED

a. Marked hypophosphatemia has been reported in five patients within 24 hours after severe head injury. Decrease in phosphate was transient and corrected spontaneously after correction of ventilation-induced acid-base disturbances (Gadisseux, 1985).

b. Others have documented hypophosphatemia in patients with severe head injury that required phosphate administration (Polderman, 2000).

### D. AMYLASE

#### 1. AMYLASE, INCREASED

a. Elevated levels of pancreatic amylase, without associated pancreatitis, have been reported in patients with intracranial bleeding (Justice, 1994).

## E. LIPASE

### 1. LIPASE, INCREASED

- a. Elevated levels of lipase, without associated pancreatitis, have been reported in patients with intracranial bleeding (Justice, 1994).

## F. MAGNESIUM

### 1. MAGNESIUM, DECREASED

- a. Preliminary data suggest that decreases in ionized magnesium may correlate with increases in the degree of head trauma (Polderman, 2000; Memon, 1995).

## G. CATECHOLAMINES, PLASMA

1. Although not clinically useful, increases in plasma catecholamines (norepinephrine, epinephrine, and dopamine) have been shown to correlate with the severity of head injury (Yang, 1995; Goldstein, 1996).

## 2.6 ARTERIAL BLOOD GASES

### A. OVERVIEW

1. INDICATIONS: Useful in following oxygenation and ventilation parameters in intubated patients. Decision to intubate should be guided by clinical factors.

### B. HYPOXIA

1. Commonly present initially; due to prolonged apneic period in the field secondary to brain stem involvement. Other causes may include upper airway obstruction, pneumothorax/hemothorax, ARDS, aspiration pneumonia, pulmonary contusion (Doberstein, 1993).
2. May significantly worsen cerebral insult caused by the head injury. However, hypoxia appears to have a less deleterious effect on outcome than hypotension, particularly in children (Chesnut, 1993; Pigula, 1993).

### C. ACIDOSIS, RESPIRATORY

1. Hypercarbia and respiratory acidosis occur commonly and may be due to upper airway obstruction, aspiration, drug effect, CNS depression, or associated injuries.
2. One study has shown a significantly higher incidence of respiratory acidosis in patients with Glasgow Coma Scale scores of 4 or less.
3. Hypercarbia causes cerebral vasodilatation and may cause an increased intracranial pressure, which adds to insult caused by acute head injury.

### D. ALKALOSIS, RESPIRATORY

1. Hypocarbia and respiratory alkalosis occur uncommonly in victims of blunt head trauma.
2. Hyperventilation occurring in patients with significant head injury suggests midbrain involvement.

## 2.9 MISCELLANEOUS

### A. INTERLEUKIN-6

1. Serum interleukin-6 levels are elevated in children with head injury. Although levels are higher with increasing injury severity, there is no correlation with mortality or neurologic outcome (Kalabalikis, 1999).

### B. INTERLEUKIN-8, CEREBROSPINAL FLUID

1. Levels of interleukin-8 in the CSF of children are elevated in the first several days after traumatic brain injury; degree of IL-8 elevation correlates with mortality (Whalen, 2000).

### C. PROTEIN, C-REACTIVE

1. Serum C-reactive protein levels are elevated in children with head injury. Although levels are higher with increasing injury severity, there was no correlation with mortality or neurologic outcome (Kalabalikis, 1999).

## 3.0 RADIOLOGIC DATA

### 3.1 GENERAL DISCUSSION

#### A. MANAGEMENT STRATEGY:

##### 1. LOW-RISK GROUP:

- a. CRITERIA (Snyder, 1990):
  - (1) Asymptomatic
  - (2) Headache

- (3) Dizziness
  - (4) Scalp hematoma
  - (5) Scalp laceration
  - (6) Scalp contusion or abrasion
  - (7) Absence of moderate-risk or high-risk criteria
  - b. RECOMMENDATION:
    - (1) GENERAL: Observation alone; discharge patients home with head-injury information sheet (listing subdural precautions) and a second person to observe them (Snyder, 1990).
    - (2) CHILDREN (2-20 yr): The AAP/AAFP practice parameter states that children with minor closed head injury (GCS=15) and no LOC who have a normal neurologic examination can be discharged from the emergency department to the care of a responsible caregiver without further radiologic evaluation (Am Acad Pediatr, 1999; Preboth, 1999).
2. MODERATE-RISK GROUP:
- a. CRITERIA (Snyder, 1990):
    - (1) Change of consciousness at time of injury or subsequently
    - (2) History of progressive headache
    - (3) Alcohol or drug intoxication
    - (4) Unreliable or inadequate history of injury
    - (5) Age <2 years or >60 years (unless injury trivial) (Borczuk, 1995)
    - (6) Posttraumatic seizure
    - (7) Multiple trauma
    - (8) Serious facial injury
    - (9) Signs of basilar fracture
    - (10) Possible skull penetration or depressed fracture (scalp injuries included)
    - (11) Suspected physical child abuse
  - b. RECOMMENDATION:
    - (1) GENERAL: Extended close observation (watch for signs of high risk group); consider CT examination and neurosurgical consultation; skull series may (rarely) be helpful if abnormal but do not exclude intracranial injury if normal (Snyder, 1990).
    - (2) CHILDREN (2-20 yr): The AAFP and AAP practice parameter states that children with minor closed head injury (GCS=15) and brief LOC who have a normal neurologic examination should be observed by a competent caregiver for at least 24 hours. Although immediate neuroimaging is not mandatory, cranial CT scan is the procedure of choice for those imaged (Am Acad Pediatr, 1999; Preboth, 1999).
3. HIGH-RISK GROUP:
- a. CRITERIA (Snyder, 1990):
    - (1) Depressed level of consciousness not clearly due to alcohol, drugs, or another cause (eg, metabolic and seizure disorders)
    - (2) Focal neurologic signs
    - (3) Decreasing level of consciousness
    - (4) Penetrating skull injury or palpable depressed fracture
    - (5) Infants (less than 2 years old) with significant scalp hematoma.
  - b. RECOMMENDATION: Neurosurgical consultation, emergency CT examination, or both (Snyder, 1990).

### 3.2 PLAIN FILMS

#### A. RADIOGRAPHY, SPINE, CERVICAL

- 1. GENERAL: Cervical spine films are most important plain films in evaluation of patients with head injuries. Cervical spine injury is easily overlooked, especially in patient with altered mental status.
- 2. INDICATIONS: Cervical spine series (cross-table lateral, AP, and open-mouth views) should be obtained on all patients with significant head trauma, unless the following conditions are present (Fischer, 1984; Neifeld, 1988; Bayless, 1989).
  - a. Patient is fully awake and alert (with no altered mental status or loss of consciousness).
  - b. Patient denies neck pain and has no significant distracting pain or injuries.
  - c. Patient has no cervical (posterior or anterior) tenderness.
  - d. Patient has normal active range of motion of neck.
  - e. Mechanism of injury not suspicious for cervical spine injury.
- 3. FINDINGS:
  - a. Angulation or subluxation of vertebral bodies.
  - b. Widening of predental space (>3 mm in adults, >4 mm in children).
  - c. Widening of retropharyngeal soft tissue space (>1/2 width of vertebral body at C-3 or C-4).

- d. Changes in bony density.
- e. Malalignment of lateral masses of C-1.
- f. Narrowing or widening of intervertebral disc spaces anteriorly or of posterior elements and dorsal spines posteriorly (suggests ligament injury).

## B. RADIOGRAPHY, SKULL

1. INDICATIONS: Although detailed recommendations have been proposed and validated (Masters, 1987; Richless, 1993), skull x-rays rarely alter management. CT is the procedure of choice. Difference in cost between skull x-rays and CT is small in view of the inability of skull x-rays to detect intracranial lesions (Stein, 1991; Yealy, 1991). A two-film skull series is as accurate as a three-view series (McGlinchey, 1998).
  - a. PATIENTS UNDERGOING CT: Not necessary for routine patient evaluation; may be useful in equivocal cases of bony injury not detected by CT or in selected moderate-risk patients (especially children <2 years) (Salluzzo, 1992; Hackney, 1991; Yealy, 1991).
  - b. PATIENTS NOT UNDERGOING CT: Useful in identifying depressed fractures and occult skull penetration. Should be obtained if skull cannot be palpated adequately through the hematoma to rule out depressed fracture or if integrity of skull cannot be verified by visual or digital examination in assault with sharp or unknown object (Salluzzo, 1992).
  - c. CHILDREN:
    - (1) May be indicated in children <7 years, as well as in skeletal survey as part of some child abuse evaluations (Salluzzo, 1992). Studies have confirmed that skull x-rays cannot be used to screen for intracranial injuries in children. Significant brain injuries may be present in absence of skull fracture. Clinical neurologic symptoms and signs should be used to determine need for imaging, and if imaging is required, CT scan, not plain skull ray, is procedure of choice (Frush, 1998; Lloyd, 1997).
    - (2) Skull x-rays may be recommended in infants and children under age 2 and whenever depressed skull fracture, a penetrating injury, or nonaccidental trauma (child abuse) is suspected (Lloyd, 1997).
    - (3) In one series, skull fractures were present in 43% of children with severe head injuries (Feickert, 1999).

## C. RADIOGRAPHY, CHEST

1. INDICATIONS: Obtain in most cases of serious head injury to rule out associated injury to pulmonary system and to search for conditions that may lead to impaired ventilation and aggravate insult to injured brain.

## 3.5 CT SCANS

### A. COMPUTED TOMOGRAPHY, HEAD

#### 1. INDICATIONS

##### a. OVERVIEW:

- (1) Noncontrast CT is imaging study of choice in acute blunt head trauma and is superior to MRI and skull radiography (Murray, 1996).
- (2) May be indicated in children less than 2 years old, especially in those under 1 year old, in view of the risk of occult intracranial injury (ie, brain injury in absence of neurologic signs or symptoms) (Beni-Adani, 1999; Gruskin, 1999; Greenes, 1998).
- (3) Indicated for all symptomatic infants with head injury and all asymptomatic infants with significant scalp hematomas (Greenes, 1999).
- (4) Recommended as radiographic imaging technique of choice for children with minor closed head injury (Am Acad Pediatr, 1999).
- (5) Sedation may be necessary to obtain a high quality scan in young, uncooperative stable children (Connors, 1999). Sedation of intoxicated, agitated adults is also frequently necessary (Holger, 1999).

##### b. GLASCOW COMA SCALE = 15:

- (1) Controversial.
  - (a) Many investigators believe that routine CT scanning is not indicated in asymptomatic patients with GCS = 15 and no confounding factors (eg, ETOH, drugs, age <1 year) (Ingebrigtsen, 2000; Miller, 1996; Duus, 1994; Taheri, 1993).
  - (b) Others recommend CT for all patients with a history of loss of consciousness or amnesia (Inamasu, 2000; Nagy, 1999; Jeret, 1993; Stein, 1992).
  - (c) Most agree that CT is indicated in pediatric patients (Kimberly, 1997; Schunk, 1996).
- (2) Intracranial lesions cannot be excluded clinically in BHT patients who have LOC or amnesia, even if GCS score is 15, although <1% of these patients will require neurosurgical intervention (Nagy, 1999; Jeret, 1993; Stein, 1992).
- (3) The American Academy of Neurology recommends CT for patients with grade 2 concussion (ie, transient confusion, no LOC, symptoms >15 min) and worsening symptoms or symptoms for >1 week (Am Acad Neurol, 1997).

(4) In a series of 313 children with GCS = 15, 13 patients had intracranial abnormalities, 4 requiring surgery. Clinical criteria were not accurate for determining which patients had intracranial abnormalities (Schunk, 1996).

(5) In a prospective cohort study of 322 children, 27 intracranial lesions, including one operable lesion, were found in the presence of normal mental status and normal neurologic examination (Kimberly, 1997).

(6) In a prospective study of a consecutive series of 1382 head trauma patients (all ages) with GCS = 15, 0.2% had injuries that required surgery. These patients could be excluded, however, by the absence of headache, nausea, vomiting, and signs of depressed skull fracture (Miller, 1996).

(7) In a retrospective multicenter study, 15% of head injured patients with GCS=15 had an abnormality on CT scan, but only 3% required a craniotomy (Shackford, 1992).

(8) In a prospective multicenter study of 2152 patients with GCS 14 and 15 and LOC or posttraumatic amnesia, the CT scan was normal in 80%, showed intracranial injury in 10%, and was equivocal in 10%; only one patient with a normal CT scan underwent craniotomy (Livingston, 2000).

(9) In a retrospective study of 194 head trauma patients with GCS=15 and presenting 12 h or more after injury, 7 (4%) had significant findings on CT, one of which required neurosurgical intervention (Hemphill, 1999).

(10) In a two-stage prospective study of 1429 head-injured patients with GCS=15 and LOC, CT scans were abnormal in 6.5%; only 6 patients (0.4%) required neurosurgical procedures. All patients with an abnormal CT scan had one of the following 7 clinical findings: age over 60; vomiting; headache; physical evidence of head, face, or neck trauma; drug or alcohol intoxication; short-term memory deficits; seizure (Haydel, 2000).

(11) Despite a GCS = 15, presence of the following may be considered indications for CT scanning (Murray, 1996):

- (a) Penetrating head trauma
- (b) Anticoagulant use
- (c) Bleeding diathesis
- (d) Loss of consciousness >5 minutes
- (e) Depressed skull fracture
- (f) Nausea and vomiting
- (g) Severe headache

c. GLASCOW COMA SCALE  $\leq$  14:

(1) Although radiographic abnormalities are fairly common, the need for neurosurgical intervention is uncommon. Nevertheless, failure to perform CT may result in missing such injuries (Jeret, 1993; Stein, 1992).

(2) Some investigators advocate the use of clinical criteria (ie, cranial soft-tissue injury, focal neurologic deficit, signs of basilar skull fracture, age >60 years) to discriminate those patients who have significant intracranial pathology from those who do not (Borczuk, 1995).

(3) The Miller criteria (ie, severe headache, nausea, vomiting, and signs of depressed skull fracture (Miller, 1996)) are not accurate in predicting presence or absence of intracranial pathology in patients with GCS = 14 (Holmes, 1997).

(4) The American Academy of Neurology recommends CT for patients with grade 2 concussion (ie, transient confusion, no LOC, symptoms >15 minutes) and worsening symptoms or symptoms for >1 week (Am Acad Neurol, 1997).

(5) The Scandinavian Neurotrauma Committee guidelines for the initial management of head trauma recommend routine cranial CT scans for patients with mild BHT (GCS=14 to 15 and history of LOC) and routine CT scans and admission for patients with moderate (GCS= 9 to 13) injuries (Ingebrightsen, 2000).

(6) Despite a GCS >12, the presence of the following may be considered indications for CT scanning (Holmes, 1997; Miller, 1996, Murray, 1996; Borczuk, 1995):

- (a) Penetrating head trauma
- (b) Anticoagulant use
- (c) Bleeding diathesis
- (d) Loss of consciousness >5 minutes
- (e) Depressed skull fracture
- (f) Nausea and vomiting
- (g) Severe headache

## 2. TIMING

- a. Depends on neurologic examination rather than history of impaired consciousness. Serious non-neurologic injuries should be treated prior to obtaining a head CT (Fulton, 1993; Thomason, 1993).
- b. Some authorities recommend obtaining a single midline scan in unstable patients to determine if emergency surgery is required; can be obtained in about 1 min and has >95% negative predictive value (Lewis, 1992).
- c. A series of 212 hypotensive blunt trauma patients suggests that preoperative head CT can be obtained safely in those who respond to initial resuscitation (ie, systolic BP >100 mmHg) (Winchell, 1995).

## 3. FINDINGS

### a. EPIDURAL HEMATOMA:

- (1) Shows a characteristic biconvex mass lesion with obscuring of the ipsilateral ventricle.
- (2) Usual appearance is a hyperdense mass; occasionally a nonhomogenous appearance is noted. May appear to be laminated, with layers of hyperdense and isodense material, or appear as a patchy mixture of isodense and hyperdense material. This appearance has been termed "hyperacute" hemorrhage and suggests active, ongoing bleeding (Greenberg, 1985).
- (3) Air has been noted in association with epidural hematoma; may be due to extension of a fracture into mastoid air cells; presence of air should indicate the possibility of bacterial contamination.
- (4) In one study, hematomas enlarged on serial CT examinations in 2/3 of patients and attained a maximum thickness of 25 mm or greater in 50%, suggesting that epidural hematomas enlarge progressively at rates varying with the condition of the source of hemorrhage (Sakai, 1988).

### b. SUBDURAL HEMATOMA:

- (1) ACUTE: Produces peripheral crescentic zone of increased density that parallels concavity of calvarium (Perini, 1984). It is usually more extensive than epidural hematoma. Bilateral subdural hematomas may be missed because of absence of midline shift.
- (2) SUBACUTE/CHRONIC: Become less dense with age and may require contrast enhancement for adequate visualization. Compression of ventricular system, displacement of midline structures, and effacement of cortical sulci may also be seen because of mass effect of hematoma.

### c. CEREBRAL CONTUSION: Appears as white patches surrounded by lucent areas, giving a "salt and pepper" appearance.

#### (1) HYPERDENSE COLLECTION:

- (a) A concave area between the inner skull and brain is often seen in acute SDH and sometimes in subacute/chronic SDH.
- (b) With the passage of time, an initially hypertense hematoma becomes isodense, then hypodense (D'Costa, 1990).

#### (2) HYPODENSE COLLECTION:

- (a) Usually appears as a hypodense area between the skull and brain with a chronic SDH.
- (b) Rarely, the rebleeding into a hypodense hematoma leads to an isodense appearance due to the fresh blood (D'Costa, 1990).

#### (3) ISODENSE COLLECTION:

- (a) Up to one third are isodense; indirect signs (ventricular mass effect, midline shift, sulcal space effacement, medial membrane enhancement) must be sought.
- (b) An isolated head injury without other evidence of multiple trauma and blood loss will not affect the hemoglobin value. A hemoglobin level in the range of 8 to 10 g will cause the cerebral tissue to be isodense with the blood. When a CT scan is done on patients with this range of anemia, even an acute hematoma may not be visualized (Smith, 1981).

#### (4) MIXED DENSITY: Subdural collections of mixed density, with areas of both high and low attenuation, may be seen in cases of chronic SDH with acute rebleeding. Similar findings have been reported in acute SDH, possible due to ongoing or recurrent hemorrhage (Greenberg, 1985).

#### (5) MASS LESIONS: Indirect signs of mass effect should be sought, including compression or obliteration of one or both ventricles, shift of midline structures, and effacement of sulci.

### d. CEREBRAL HEMORRHAGE: Acute hemorrhage (subarachnoid, cerebral laceration) will appear as area of increased density.

### e. ELEVATED INTRACRANIAL PRESSURE:

- (1) Appearance of cisterns, size of subdural hematoma, ventricular size, status of subarachnoid hemorrhage or cerebral contusion, magnitude of midline shift, and ventricular index contribute to predicting ICP (Mizutani, 1990).
- (2) In patients with severe diffuse head injury, compression of third ventricle and basal cisterns on CT scan closely correlate with ICP >20 mmHg, clinical signs of midbrain dysfunction, and poor prognosis (Teasdale, 1984).

#### 4. ACCURACY

- a. Extremely accurate in diagnosis of traumatic intracranial lesions. Most significant area in which accuracy of CT scan has been questioned is in diagnosis of subacute subdural hematomas and nonhemorrhagic traumatic lesions.
- b. Contrast enhancement or MRI should be considered if these are suspected and their diagnosis will affect patient management (Gentry, 1988; Kelly, 1988).
- c. Delayed intracranial bleeding, typically subdural hematoma, can occur after an initially normal CT scan. Although thought to be rare, delayed intracranial bleeding should be considered in patients who present with persistent symptoms after an initially normal CT (Snoey, 1994; Davis, 1995).
- d. In a retrospective study of 173 children with moderate and severe head trauma, a second CT scan scheduled 24 to 36 h after admission identified findings that resulted in no changes in management in any patient (Tabori, 2000).

#### 5. PROGNOSTIC VALUE

- a. May be of assistance in predicting outcome (Uzzell, 1987; Macpherson, 1990; Costeff, 1990; Eisenberg, 1990).
  - (1) Presence or absence of lesion, as well as its size, are important factors. About 80% of patients who have normal CT scan will have good outcome (Lobato, 1986).
  - (2) The larger the lesion the worse the outcome (Lipper, 1985).
  - (3) In patients with severe BHT, CT findings of midline shift, compression or obliteration of the mesencephalic cisterns, and presence of subarachnoid blood are predictive of abnormal ICP and death; diffuse hemispheric swelling is associated with early episode of either hypotension or hypoxia (Eisenberg, 1990).
  - (4) Patients with a normal CT scan, GCS of 14 or 15, and normal neurologic examination are at minimal risk of deterioration (Stein, 1990; Livingston, 1991, 1991a).

#### 6. CT VS MRI

- a. Superior to MRI for showing hemorrhagic components during the acute phase of trauma and for fractures. Primarily effective in the evaluation of potentially reversible intracranial hematomas (Hesslink, 1988; Doezema, 1991).
- b. Less sensitive than MRI for detecting brain contusions, especially during the subacute and chronic stages of head injury (Hesslink, 1988; Orrison, 1994).
- c. May be insensitive to small lesions resulting from focal cortical contusions and diffuse axonal injury.
- d. CT remains procedure of choice in acute situation principally because of its speed and ease of monitoring (equipment for monitoring may disrupt the MRI magnetic field) and also because MRI is relatively insensitive in detecting acute subarachnoid hemorrhage (Snow, 1986; Gentry, 1988; Hesslink, 1988).

#### B. COMPUTED TOMOGRAPHY, SPINE

1. Patients with severe head trauma (GCS 3 to 6) often have associated cervical spine trauma. In a prospective study of 202 such patients, 5% had fractures of C-1 or C-2 and 4% had occipital condyle fractures that were not visualized by plain radiographs (Link, 1995).
2. If supported by independent studies, routine CT of the cervicocranial junction may be indicated in this patient population.

#### C. COMPUTED TOMOGRAPHY, SINGLE PHOTON EMISSION

1. NOT INDICATED in acute management of blunt head trauma. May reveal abnormalities of cerebral metabolism after head injury, in a manner similar to that of positron emission tomography (PET) scanning. The significance of these findings, however, has yet to be determined (Abdelayem, 1998; Packard, 1994; Jacobs, 1994).

### 3.6 ULTRASOUND

#### A. ULTRASONOGRAPHY, DOPPLER, TRANSCRANIAL

1. NOT INDICATED in acute management of blunt head trauma. May detect evidence of mild head injury, such as changes in cerebral blood flow velocity, that would not otherwise be evident on conventional CT or MRI. The significance of these findings, however, has yet to be determined (Packard, 1994).
2. Has shown some promise as a noninvasive measurement of cerebral perfusion pressure in the ICU setting in severely brain injured patients (Czosnyka, 1998).

#### B. ULTRASONOGRAPHY, ABDOMEN

1. In blunt multiple trauma patients, the decision to proceed to emergent craniotomy versus further evaluation of the abdomen is difficult. ED abdominal ultrasonography, when immediately available, may be helpful in the assessment of blunt abdominal trauma (Huang, 1995).

### 3.7 MAGNETIC RESONANCE IMAGING

#### A. IMAGING, MAGNETIC RESONANCE, HEAD

1. INDICATIONS: If neurological deficits continue or become worse despite a normal CT scan (Benedetti, 1996). Rarely needed in ED management of acute BHT (Yealy, 1991; Murray, 1996).
  - a. May demonstrate areas of brain injury not visualized by CT (Levin, 1985; Hadley, 1988; Gentry, 1988; Kelly, 1988; Mark, 1992; Yokota, 1991), especially in subacute or chronic situation (more than 72 hours after initial injury). Also useful in evaluation of child abuse (Yealy, 1991).
  - b. Ultra-low-field MR imaging can identify anatomic brain lesions in an additional 10% of minor head injury patients who are currently being discharged from the ED (Doezema, 1991). However, this probably would not alter management (Pitts, 1991).
  - c. May be of value in predicting who will have persistent symptoms or cognitive dysfunction (Doezema, 1991; Murray, 1996).
  - d. May suggest the need for neuropsychological testing or information that the injury may interfere with normal functioning, enabling patients to improve their adaptability (Doezema, 1991).
  - e. Small cortical hemorrhages are detected best with MRI techniques such as T2-weighted gradient echo (Yanagawa, 2000).
2. MRI VS CT:
  - a. May be superior to CT scan in detection of subacute injuries, including shearing injuries, hemorrhagic and nonhemorrhagic contusions, and subdural hematomas (Zimmerman, 1986; Snow, 1986; Wilberger, 1987; Gentry, 1988; Benedetti, 1996).
  - b. More sensitive than CT in detecting diffuse axonal injury (shearing injury), subcortical gray matter injury, brainstem injury, (Firsching, 1998) corpus callosum injury, extra axial fluid collection (Doezema, 1991), and brain contusions (especially during subacute and chronic stages of head injury) (Hesselink, 1988).
  - c. CT scans may be insensitive to small lesions resulting from focal cortical contusions and diffuse axonal injury.
  - d. CT remains procedure of choice in acute situation principally because of its speed and ease of monitoring (equipment for monitoring may disrupt the MRI magnetic field) and also because MRI is relatively insensitive in detecting acute subarachnoid hemorrhage and facial, temporal bone, and orbital fractures (Snow, 1986; Gentry, 1988; Hesselink, 1988; Murray, 1996).

### 4.0 DIAGNOSTIC AIDS

#### 4.1 ELECTROCARDIOGRAM

- A. Nonspecific changes have been reported in EKGs of patients sustaining head trauma, including ST segment and T wave abnormalities and prolongation of QT interval (Hersch, 1961).
- B. Transient third-degree AV block has been reported (Wirth, 1988).

#### 4.2 MISCELLANEOUS

##### A. RING SIGN

1. Helps detect CSF leak and diagnose basilar skull fracture. When CSF fluid mixed with blood is placed on filter paper, CSF will separate from blood and produce one or more concentric rings of clearer fluid. Reliable indicator of presence of CSF in blood but is not exclusive for CSF (Dula, 1993).

##### B. ELECTROENCEPHALOGRAPHY

1. NOT INDICATED in acute management of blunt head trauma. Although abnormalities can be detected, their significance remains unknown. Mapping of EEG and evoked potentials in mild head injury is, however, an area of active research (Packard, 1994). However, continuous EEG monitoring may be useful to manage selected severe head trauma patients (Jordan, 1999).

##### C. SPECTROSCOPY, NEAR-INFRARED

1. NOT INDICATED in acute management of blunt head trauma. Has been shown to be useful as clinical monitoring technique for the detection of delayed traumatic intracranial hematomas (Gopinath, 1995).

##### D. TOMOGRAPHY, POSITRON EMISSION

1. NOT INDICATED in acute management of blunt head trauma. PET may reveal abnormalities of cerebral metabolism after head injury. The significance of these findings, however, has yet to be determined (Packard, 1994).

## E. GLASGOW COMA SCALE

### 1. GENERAL:

- a. Permits descriptive and reproducible assessment of patient's state of consciousness; best scale for predicting outcome and is simplest and least time-consuming system. Correlates highly with severity of brain injury (Rocca, 1989; Salluzzo, 1992).
- b. Use of GCS score should replace terms such as lethargy or stupor, which are nonspecific and vary in meaning from one observer to another.
- c. Should be utilized for repeated observation of head-injured patients to help predict prognosis. The GCS score was derived as a prognosticator, not as a treating device. To make an accurate assessment for treatment, other parameters need to be evaluated, eg, vital signs, mechanism of injury, and associated injuries (Kohi, 1984; Winkler, 1984).
- d. CT scan should be obtained in the ED for patients with LOC or amnesia, regardless of GCS score; abnormal in almost 20% of patients with GCS scores greater than 13 (Harad, 1992).

### 2. SCALE:

#### a. BEST MOTOR RESPONSE:

- (1) Obeys = 6
- (2) Localizes pain = 5
- (3) Withdraws = 4
- (4) Flexion to pain = 3
- (5) Extension to pain = 2
- (6) None = 1

#### b. BEST VERBAL RESPONSE:

- (1) Oriented = 5
- (2) Confused conversation = 4
- (3) Inappropriate words = 3
- (4) Incomprehensible = 2
- (5) None = 1

#### c. EYE OPENING:

- (1) Spontaneously = 4
- (2) To speech = 3
- (3) To pain = 2
- (4) None = 1

### 3. INJURY SEVERITY: Rate of change of the GCS may be as important as the absolute value (Schaffer, 1985; Cohadon, 1991):

- a. Mild: GCS between 13 and 15 (High-risk mild head injury = GCS score of 13 or 14, or GCS score of 15 with acute radiographic abnormalities) (Hsiang, 1997).
- b. Moderate: GCS between 9 and 12
- c. Severe: GCS between 3 and 8

### 4. LIMITATIONS:

- a. INTOXICATION: May not be accurate in presence of alcohol or drugs. One study of 257 head-injured patients revealed that alcohol-intoxicated patients had significant depression of their GCS score related to blood alcohol level (Jagger, 1984).
- b. MENTAL STATUS EXAMINATION: GCS score does not provide information regarding mental status.
  - (1) Should be performed on all patients with normal GCS scores and should include determination of orientation to time, person, and place and evaluation of short-term memory (Salluzzo, 1992).
  - (2) Less significant in patients with abnormal GCS scores because they already are in higher risk group for intracranial injury (Salluzzo, 1992).
  - (3) Motor component of GCS score may be equivalent to overall score for mortality prediction and when deciding on triage to trauma center (Ross, 1998).

## 5.0 DIFFERENTIAL DIAGNOSIS

### 5.5 METABOLIC

#### A. HYPOGLYCEMIA

1. May present with alteration in mental status. Initial history may be inadequate to distinguish hypoglycemia from blunt head trauma, but there is often history of underlying medical illness.
2. PHYSICAL EXAMINATION:
  - a. Will generally not show signs of significant head trauma, eg, ecchymoses, hematomas, or abrasions.
  - b. Focal neurologic signs may be present in metabolically-induced coma, but pupillary light reflexes are usually preserved, despite length of coma.
  - c. Rostrocaudal deterioration of brain stem function is not present.

### 3. LABORATORY DATA:

- a. May point to metabolic cause. If performed, CT scan will not show significant lesion.
- b. If the diagnosis is questioned, rapid bedside glucose determination should be performed. If unable, 50 grams of dextrose in water should be administered. (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: HYPOGLYCEMIA)

### B. KETOACIDOSIS, DIABETIC

1. May present with alteration in mental status. Initial history may be inadequate to distinguish DKA from blunt head trauma, but there is often history of underlying medical illness.
2. PHYSICAL EXAMINATION:
  - a. Will generally not show signs of significant head trauma, eg, ecchymoses, hematomas, or abrasions.
  - b. Focal neurologic signs may be present in metabolically-induced coma, but pupillary light reflexes are usually preserved, despite length of coma.
  - c. Rostrocaudal deterioration of brain stem function is not present.
3. LABORATORY DATA: May point to a metabolic cause. If performed, CT scan will not show significant lesion. (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: DIABETIC KETOACIDOSIS)

### C. HYPOXIA

1. May present with alteration in mental status. Initial history may be inadequate to distinguish hypoxia from blunt head trauma, but there is often history of underlying medical illness.
2. PHYSICAL EXAMINATION:
  - a. Will generally not show signs of significant head trauma, eg, ecchymoses, hematomas, or abrasions.
  - b. Focal neurologic signs may be present in metabolically-induced coma, but pupillary light reflexes are usually preserved, despite length of coma.
  - c. Rostrocaudal deterioration of brain stem function is not present.
3. LABORATORY DATA: May point to metabolic cause. If performed, CT scan will not show significant lesion.

## 5.6 VASCULAR

### A. STROKE, THROMBOEMBOLIC

1. Vascular disease of cerebral cortex presenting with focal neurologic signs, eg, cerebral embolism, may suggest diagnosis of chronic subdural hematoma.
  - a. History of trauma is usually absent.
  - b. Complaints of headache and fluctuating levels of consciousness generally do not precede development of focal neurologic deficit.
2. PHYSICAL EXAMINATION:
  - a. Will not show signs of significant head trauma, eg, ecchymoses, contusions, or abrasions.
  - b. Evidence of hypertension or cardiovascular disease is often present.
3. Cerebral angiography and/or CT scanning will accurately identify the lesion. (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: THROMBOEMBOLIC STROKE)

## 5.7 NEOPLASTIC

### A. NEOPLASM, CEREBRAL

1. TYPES:
  - a. PRIMARY: Include gliomas (50%), meningiomas, pituitary adenomas, and neurofibromas.
  - b. METASTATIC: Most common source is carcinoma of the lung; other primary sites are breast, kidney, and GI tract.
2. CLINICAL PRESENTATION: Characterized by generalized or focal disturbances of cerebral function, or both, and signs and symptoms of increased ICP.
  - a. GENERALIZED:
    - (1) May include personality changes, intellectual decline, emotional lability, seizures, headache, nausea, malaise, slowly progressive weakness on one side, visual changes, aphasia, vomiting, mental changes. Papilledema occurs in 25% of patients and may not be early sign; vital signs are normal.
    - (2) Increased ICP may cause herniation, most commonly tentorial, characterized by ipsilateral pupillary dilatation, followed by stupor, coma, decerebrate posturing, and respiratory arrest.
  - b. FOCAL: Due to localized destruction or compression of nerve tissue or to altered endocrine function; depend on tumor location.
3. DIAGNOSIS: Neuroradiologic evidence of space-occupying lesion. CT or MRI may detect lesion and also may define its location, shape, and size; extent to which normal anatomy is distorted; and degree of any associated cerebral edema or mass effect.

## 5.8 TOXICOLOGIC

### A. POISONING, DRUG

1. May present with alteration in mental status. Initial history may be inadequate to distinguish drug overdose from blunt head trauma, but there often is history of underlying medical illness.
2. PHYSICAL EXAMINATION:
  - a. Will generally not show signs of significant head trauma, eg, ecchymoses, hematomas, or abrasions.
  - b. Focal neurologic signs may be present in metabolically-induced coma, but pupillary light reflexes are usually preserved, despite length of coma.
  - c. Rostrocaudal deterioration of brain stem function is not present.
3. LABORATORY DATA: May point to metabolic cause. If performed, CT scan will not show significant lesion.

### B. ALCOHOL ABUSE

1. Ethanol intoxication is common among trauma victims and presents a diagnostic problem when associated with altered mental status. Under such circumstances, it is difficult to determine with certainty if altered mentation is due to head trauma, sedative effect of ethanol, or combination of both factors.
2. Focal neurologic signs are uncommon in ethanol intoxication. If present, careful and prolonged observation will demonstrate improvement in neurologic signs.
3. Blood ethanol level is elevated, but this does not rule out significant head injury.
4. CT scan is normal in ethanol-intoxicated patients, but it is impractical to perform this procedure in all intoxicated patients.
5. Intoxicated patients with a strong suspicion for significant head trauma, patients whose mental status is markedly out of proportion to their alcohol level, and patients who fail to improve their mental status within a reasonable period of observation should undergo cranial CT.

## 5.10 MISCELLANEOUS

### A. SEIZURES

1. Patients with epilepsy who seize may sustain head trauma during the episode. Alterations in mental status or focal neurologic signs may be manifestation of postictal state or complication of head trauma.
2. CT scan will be normal or unchanged from baseline.

## 6.0 TREATMENT

### 6.1 TREATMENT SUMMARY

#### A. GENERAL:

1. After ABCs are addressed, management is aimed at controlling and preventing complications of increased intracranial pressure and at rapid neurosurgical decompression of surgically correctable lesions.
2. Protection of cervical spine must be accomplished, as well as priority management of other associated life-threatening injuries.
3. Classic signs and symptoms of head injury can be masked in intoxicated, pediatric, elderly, and multitrauma patients.

B. CERVICAL SPINE IMMOBILIZATION: Any patient with head trauma and altered mental status should be considered to have possible cervical spine injury; immobilize neck until the spine is cleared radiographically.

C. AIRWAY MANAGEMENT: Ensuring a patent airway and initiating effective ventilation is critical.

1. INTUBATION: If airway management is needed, endotracheal intubation is procedure of choice. Rapid-sequence intubation and paralyzing agents are recommended for optimal resuscitation of acutely head-injured patients with suspected increased ICP.
  - a. CERVICAL SPINE CLEARED: Oral intubation may be performed.
  - b. CERVICAL SPINE STATUS UNCERTAIN: Oral intubation in conjunction with in-line stabilization or nasotracheal intubation may be used based on skill and experience of intubationist.

D. IV FLUIDS: In multiply-injured patients, hypotension should be assumed to be secondary to blood loss. Begin NS to maintain BP above 90 mmHg systolic. Avoid routine use of dextrose-containing solutions (potential risk of worsening CNS lactic acidosis and cerebral edema).

1. Begin NS to maintain BP above 95 mmHg systolic.
2. Avoid routine use of dextrose-containing solutions (potential risk of worsening CNS lactic acidosis and cerebral edema).

E. ANTICEREBRAL EDEMA MEASURES: Treatment modalities may include head elevation, osmotic therapy, diuretics, rapid sequence intubation, barbiturates, hyperventilation, mechanical decompression.

F. NASOGASTRIC TUBE: Indicated to prevent gastric dilatation and to decrease incidence of regurgitation.

G. INTRACRANIAL PRESSURE (ICP) MONITORING: Consider in patients with severe head injury (GCS 8 or less).

H. SEIZURE PROPHYLAXIS: Controversial; decreases incidence of seizures in early post-traumatic period but has no long-term benefit.

I. HEMATOMA EVACUATION: Provides definitive therapy for subdural and epidural hematoma; should be performed as soon as possible.

J. OBSERVATION:

1. Patients with normal mental status and neurologic examination may be observed at home with serial neurologic checks by a responsible adult.
2. Patients with altered mental status require hospital admission for close observation and frequent neurologic checks to detect the development of elevated ICP; consider cranial CT scan.

K. TRIAGE IN MULTIPLE TRAUMA: Decision to proceed to emergent craniotomy vs laparotomy is difficult; ED diagnostic peritoneal lavage or abdominal US, if immediately available, may be helpful in the assessment of BAT.

## 6.2 NON-PHARMACOLOGIC TREATMENT

A. AIRWAY MANAGEMENT

1. OVERVIEW

- a. Establishing a patent airway and effective ventilation are critical in the initial management of head injury. Apnea often occurs with concussive head injury; the duration of apnea is directly proportional to the energy delivered to the brain. Prolonged apnea and poor ventilation result in hypoxia and hypercarbia, which can lead to massive cerebral edema (Atkinson, 2000).
- b. In children with severe head injuries, early intubation prior to transport was associated with improved survival (Suominen, 2000).
- c. Prophylactic hyperventilation is no longer recommended in patients with head trauma and should be avoided during the first 24 hours after injury to avoid secondary brain injury due to regional brain ischemia (Marshall, 2000; Sauthoff, 1998).

2. INTUBATION, RAPID SEQUENCE

- a. DEFINITION: Definitive airway management technique in which a potent sedative or induction agent is administered virtually simultaneously with a paralyzing dose of a neuromuscular blocking agent to facilitate rapid tracheal intubation.
- b. CAUTION: Physicians performing rapid sequence intubation must possess training, knowledge, and experience in the techniques and pharmacologic agents used to perform RSI. (FOR DETAILED REVIEW, SEE CLINICAL REVIEW: RAPID SEQUENCE INTUBATION)

3. INTUBATION, NASOTRACHEAL

- a. INDICATIONS: Indicated early in management of head-injured patient with altered mental status. Guidelines for intubation are any patient with GCS score of 3 to 8.
  - (1) Oral intubation should be done if C-spine is clear of injuries. If intubation is required before C-spine is cleared, oral or nasotracheal intubation may be considered. Strict in-line stabilization of the spine is required for both procedures and the choice of methods should depend on the skill and experience of the intubationist.
  - (2) Nasal intubation may be attempted in patients without midface injuries but is contraindicated if there is question of cribriform plate fracture.
  - (3) Cricothyrotomy may be considered when C-spine has not been cleared or if there are nasal or facial injuries (Fink, 1987).
  - (4) Does not markedly increase complications in patients with skull base fractures when performed in the field by trained and experienced personnel (Rhee, 1993).
- b. ADULT TUBE SIZE: The tube should have high volume/low pressure cuff. Largest tube size possible should always be used, especially in patients with chronic lung disease.
- c. PEDIATRIC TUBE SIZE: Estimate tube size for pediatric patient using following formula: age (in years) divided by 4, plus 4; or, use tube the size of child's small finger. 3-mm tube is smallest size tube allowing tracheal suction.
- d. Cuff should be inflated to pressure <25 mmHg and should allow small air leak during peak inspiration. Cuff should not be deflated periodically as this leaves airway unprotected and permits aspiration.

e. TUBE PLACEMENT:

- (1) After intubation, it is mandatory to check for equal breath sounds over lateral chest, especially on left.
- (2) Tube placement must be confirmed via chest x-ray; tip should be 1 to 2 cm above carina.

4. INTUBATION, OROTRACHEAL

a. INDICATIONS:

- (1) Tracheal intubation is indicated early in management of head-injured patient with altered mental status.
- (2) Oral intubation should be done if C-spine is clear of injuries. Oral or nasotracheal intubation with strict C-spine in-line stabilization may be done when the status of the C-spine is unclear; cricothyrotomy also may be considered. Choice of methods is dependent on the skill and experience of the intubationist.
- (3) In children with severe (Abbreviated Injury Scale score of 4 or greater) head injuries, early intubation prior to transport was associated with improved survival (Suominen, 2000).

5. VENTILATION, MECHANICAL

- a. GENERAL: Used as a temporizing measure to provide ventilatory support. May be administered invasively (eg, endotracheal tube, tracheostomy) or noninvasively (eg, face or nasal mask). Modalities used may include assist/control, intermittent mandatory, synchronized intermittent mandatory, and pressure support ventilation. Adjuncts include continuous positive airway pressure, positive end expiratory pressure, permissive hypercapnia, and tracheal gas insufflation.
- b. CAUTION: Physicians managing mechanically ventilated patients must be experienced and knowledgeable in the monitoring and therapeutic modalities used. (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: MECHANICAL VENTILATION)

6. VENTILATION, PERCUTANEOUS TRANSTRACHEAL

- a. DEFINITION: Temporary airway management technique that is utilized until a more definitive airway can be secured. High-pressure (50 or 60 psi) oxygen is delivered in intermittent jets, usually at 12 to 20 breaths/minute.
- b. GENERAL:
  - (1) Provides immediate oxygenation and ventilation via a large bore (12- to 16-gauge) IV catheter inserted transtracheally through the cricoid membrane. Use in various clinical settings has demonstrated effective pulmonary gas exchange with minimal complications (Depierraz, 1994; Nakatsuka, 1992).
  - (2) In general, this is a temporary procedure; oxygenation can be maintained for approximately 30 minutes; beyond this point, progressive hypercapnia due to inadequate ventilation occurs (Manning, 1995).
- c. INDICATIONS:
  - (1) Alternative airway management when oral or nasal intubation is time-consuming, dangerous, unsuccessful, or contraindicated (Barash, 1992; Nakatsuka, 1992; Benumof, 1994).
  - (2) Various endoscopic procedures involving the glottic or subglottic area (Depierraz, 1994).
- d. CONTRAINDICATIONS: Relative coagulopathy; complete airway obstruction, although this is being debated, especially if large (8.5 Fr) catheter is used (Manning, 1995).
- e. TECHNIQUE (Manning, 1995):
  - (1) Palpate space between inferior surface of the thyroid cartilage and the upper surface of the cricoid; insert tip of 12- to 16-gauge over-the-needle plastic or Teflon IV catheter at a 30- to 45-degree angle caudally through cricoid membrane.
  - (2) Aspirate with syringe during insertion; air return indicates entrance into laryngotracheal lumen.
  - (3) Thread catheter in (to hub of catheter); connect catheter to pressurized (approximately 50 psi) oxygen. If high pressure oxygen not available, may use an oxygen-regulator system at maximum (15 l/min) flow rate (suboptimal but acceptable).
  - (4) Provide 1-second inflations at a rate of approximately 12 per minute.
- f. COMPLICATIONS: Subcutaneous emphysema; hemorrhage, most often involving the thyroid; aspiration; esophageal perforation with gastric dilation; catheter kinking, and laryngeal pneumatocele.

7. CRICOTHYROTOMY

- a. INDICATIONS: Severe maxillofacial injuries that would contraindicate nasotracheal or orotracheal intubation; inability to intubate by these routes.
- b. Oral intubation is preferred once C-spine is cleared (Fink, 1987).

B. IMMOBILIZATION, SPINE, CERVICAL

1. Victim of head trauma must be assumed to have associated cervical spine injury until proven otherwise (Rubsamen, 1974).

2. Unless patient is alert, denies neck pain, and has good range of motion, neck should be immobilized until cervical fracture is ruled out by good quality radiographs showing all seven cervical vertebrae.
3. The neck should be immobilized with sandbags, a cervical collar, or four-poster collar until the cervical spine can be cleared radiographically.
4. Cervical collars that are excessively tight may produce jugular venous obstruction and impede cerebral blood flow (Ferguson, 1993).

#### C. NASOGASTRIC TUBE

1. Indicated to prevent gastric dilatation or regurgitation of gastric contents.
2. Uncommon but potentially serious complication is passage of tube into cranial cavity in patients with maxillofacial fractures. Oral route should be chosen in this setting (Fremstad, 1978).

#### D. CATHETERIZATION, FOLEY

1. Place Foley catheter in patients with depressed level of consciousness to monitor urinary output and to prevent bladder distention.

#### E. ELEVATION, HEAD

1. INDICATIONS: Head, back, and neck should be elevated to 30 degrees providing cervical spine is not injured (Butterworth, 1989; Feldman, 1992).
  - a. This is especially true if PEEP is needed to improve oxygenation.
  - b. Because elevation of head and neck may produce postural hypotension, caution is advised in patients with potential for decreased blood pressure (Rosner, 1986;).
2. EFFICACY (Feldman, 1992):
  - a. NORMAL ICP: Head position does not greatly affect ICP; these patients can be positioned either horizontally or with head elevated to 30 degrees.
  - b. INCREASED ICP: Effective in reducing ICP; significantly reduces ICP in majority of patients without reducing cerebral blood flow or cerebral perfusion pressure.

#### F. MONITORING, INTRACRANIAL PRESSURE

##### 1. OVERVIEW

- a. INDICATIONS: Essential to accurately measure effects of therapeutic interventions aimed at controlling intracranial pressure (Bullock, 1996).
  - (1) Posttraumatic coma, GCS score of 3 to 8, or abnormality on CT scan (Marshall, 2000; Benardete, 1998; Salluzzo, 1992; Clark, 1989).
  - (2) Development of secondary deterioration in brain function (if CT scan rules out mass lesion);
  - (3) Use of agents that suppress CNS activity and prevent adequate monitoring by other means.
  - (4) Severe head injury combined with multiple associated injuries and hemodynamic instability requiring immediate operation (Lillehei, 1990).
  - (5) Nonoperative management of patients with intracranial hematomas to detect early development of mass effect (Patel, 2000).
- b. MODALITIES:
  - (1) Ventriculostomy is traditional modality for monitoring ICP (Pieper, 1996); preferred in head trauma and other neurosurgical conditions. Other techniques are felt to be adequate for other "medical" conditions.
  - (2) Several technical advances (eg, Camino fiberoptic catheter, transcranial ultrasound Doppler) also have been developed (Hall, 1997; Saunders, 1988).
- c. GOAL: Maintain intracranial pressure as close as possible to normal value of 15 mmHg. Values above 25 mmHg are associated with >70% mortality (Miller, 1977; Saul, 1982). Treatments to lower ICP should be initiated when ICP is 20 mmHg or greater (Marshall, 2000).
- d. EFFICACY: ICP monitoring leads to specific interventions in the majority of patients with serious head injuries (GCS 8 or less).
- e. COMPLICATIONS: Major complication is infection, incidence of which is related to duration of monitoring, requirement for serial monitors, and concurrent infections at other sites (Clark, 1989).
- f. CT SCAN: Gives reasonably accurate ICP for initial stage of head injury. Appearance of cisterns, size of subdural hematoma, ventricular size, status of subarachnoid hemorrhage or cerebral contusion, magnitude of midline shift, and ventricular index contribute to predicting ICP (Mizutani, 1990).
- g. PASG: Intermittent pneumatic leg compression results in no significant changes in ICP or CPP in stable patients with BHT who have ICP controlled medically (Davidson, 1993).
- h. CAUTION: Physicians managing cerebral edema must be experienced and knowledgeable in the monitoring techniques and therapeutic modalities used to treat cerebral edema. (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: CEREBRAL EDEMA)

## 2. CATHETER, VENTRICULAR

- a. Preferred for monitoring ICP in head trauma and other neurosurgical conditions.
- b. Catheter is introduced into ventricle through small hole drilled into skull at coronal suture in line with pupil.
- c. ADVANTAGES:
  - (1) Most accurate method of measuring ICP.
  - (2) Major advantage is ability to decrease elevated ICP through therapeutic CSF drainage (Fortune, 1995).
  - (3) Allows measurement of volume-pressure response (VPR) of brain and prediction of impending intracranial hypertension.
  - (4) Facilitates control of cerebral perfusion pressure (CPP), which may be superior to ICP as a monitoring parameter (Rosner, 1995).
- d. DISADVANTAGES:
  - (1) Insertion is technically difficult, especially if patient has small ventricles.
  - (2) Blockage of catheter can occur with collapse of ventricle.
  - (3) 3% to 5% infection rate.

## 3. SUBARACHNOID SCREW

- a. ADVANTAGES:
  - (1) Easier to insert and has lower infection rate than ventricular catheter; it also does not require insertion into brain. It is inserted at coronal suture in line with pupil.
  - (2) Accuracy is good.
- b. DISADVANTAGES:
  - (1) No possibility for CSF drainage or volume-pressure response measurements.
  - (2) ICP elevations >40 torr may obstruct catheter/transducer and cause highly inaccurate measurement (Winn, 1977).

## 4. EPIDURAL TRANSDUCER

- a. Probably associated with lowest infection rate, but its accuracy is variable.
- b. It is placed via burr hole under bone.
- c. CSF drainage is not available with this method.

## G. OBSERVATION

### 1. LOW-RISK INJURIES:

- a. In patients with normal mental status, close observation with frequent neurologic checks should be performed to detect the development of increased ICP from an intracranial event.
- b. Observation usually is done on an inpatient basis over a period of 24 to 48 hours. Observation in ED for 6 to 8 hours may be utilized for patients who have minor symptoms. In one study of head-injured children, all patients with intracranial hemorrhage had symptoms or signs within 6 hours (Sainsbury, 1984).
- c. Children with minimal symptoms after head trauma and a normal CT, or with an isolated skull fracture, have an extremely low risk of neurologic complications and do not benefit from inpatient observation for neurologic deterioration (Roddy, 1998; Greenes, 1998).

### 2. EPIDURAL HEMATOMA:

- a. Some studies have suggested that certain patients with very small hematomas, who are conscious at the time of presentation and have minimal midline shift, may be managed nonoperatively with serial CT scans and careful neurologic observation (Bullock, 1985; Pozzati, 1986; Knuckey, 1989).
- b. Considerations include the size of estimate of the hematoma, the location of the hemorrhage, and the amount of "acceptable" midline shift seen on CT scan (Bullock, 1985; Pozzati, 1986; Knuckey, 1989).
- c. One study of patients with initially asymptomatic small hematomas found that patients with a fracture overlaying a major vessel or major sinus and/or who are diagnosed <6 hours after injury are at risk of subsequent deterioration and may require surgical evacuation. Conversely, patients without these risk factors have a low risk of delayed deterioration (Knuckey, 1989).
- d. The decision regarding operative vs conservative treatment should be made by the consulting neurosurgeon.

### 3. INTRACEREBRAL HEMATOMA:

- a. Small intracerebral hematomas without evidence of mass effect may be managed nonoperatively with serial CT scans and careful neurologic examinations.
- b. Intracranial pressure monitoring is advocated by many authors to detect early signs of mass effect (Patel, 2000).
- c. Frontal intracerebral hematomas are more prone to early failure of nonoperative management (Patel, 2000).

## H. SURGERY

### 1. BURR HOLES

- a. INDICATIONS: Use of emergency exploratory burr holes is controversial (Wilberger, 1990a; Evans, 1990).
  - (1) Not superior to CT in terms of more rapid diagnosis and treatment or improved outcome. If CT not immediately available, used only if definitive surgical treatment can be undertaken simultaneously if exploration is abnormal, or unless CT is done immediately if exploration is normal (Wilberger, 1990a).
  - (2) May be necessary to partially evacuate hematoma prior to definitive therapy or patient transfer in patients with severe head injuries who continue to deteriorate neurologically (Rinker, 1998).
  - (3) Should NOT be used in patients who have shown evidence of brainstem dysfunction from moment of injury (GCS score of 3) (Evans, 1990; Johnson, 1992).
- b. INTRAOPERATIVE ULTRASONOGRAPHY: Ultrasound imaging of brain during exploratory burr-hole placement prior to CT scanning in patients with postraumatic transtentorial herniation accurately identifies intracerebral hemorrhages and contusions (Andrews, 1989, 1990).

### 2. HEMATOMA EVACUATION, SUBDURAL

- a. INDICATIONS: Definitive therapeutic procedure for treatment of subdural hematoma. Patients with findings suggestive or diagnostic of SDH should have neurosurgical consultations as soon as possible.
- b. EFFICACY:
  - (1) ACUTE SDH: Mortality may be significantly decreased when surgical evacuation of the hematoma is accomplished within 4 hours of injury (Seelig, 1981; Yue, 1982).
  - (2) CHRONIC SDH: Rarely resolves spontaneously (Markwalder, 1981). Burr hole evacuation is usually associated with a low mortality and good recovery (Robinson, 1984; Moussa, 1982). Craniotomy and craniectomy are generally reserved for cases in which the collection reaccumulates, the brain fails to reexpand, or if there is a solid hematoma (Markwalder, 1981, 1985).

### 3. HEMATOMA EVACUATION, EPIDURAL

- a. INDICATIONS: Definitive therapeutic procedure. Neurosurgical consultation should be obtained as early as possible.
- b. EFFICACY: Mortality is significantly decreased when surgical evacuation of hematoma is accomplished within 4 hours of injury (30% vs 90%) (Seelig, 1981).

## I. MONITORING, PULMONARY ARTERY PRESSURE

1. INDICATIONS IN TRAUMA PATIENTS: May alter diagnosis/improve functional outcomes and alter mortality in selected traumatically injured patients when therapeutic objectives are to (Pulmonary Artery Consensus Conference, 1997):
  - a. Determine status of underlying cardiovascular performance.
  - b. Direct therapy when noninvasive monitoring is inadequate or misleading.
  - c. Assess response to resuscitation.
  - d. Potentially decrease secondary injury when severe closed-head or acute spinal cord injuries are present.
  - e. Augment clinical decision-making when major trauma is complicated by ARDS, progressive oliguria/anuria, myocardial injury, CHF, or major thermal injury.
  - f. Establish futility of care.

## J. HYPOTHERMIA, INDUCED

1. Over the past decade, evidence has accumulated that treatment with moderate hypothermia may hasten the recovery of patients who have sustained severe traumatic brain injuries. Moderate hypothermia is thought to limit secondary brain injury by reducing cerebral edema and ischemia, reducing cerebral metabolism, reducing the extracellular accumulation of excitatory neurotransmitters or mitigating the post-injury inflammatory response (Metz, 1996; Marion, 1997).
2. In a randomized, controlled clinical trial, 155 patients with severe closed head injuries (GCS =3-7) were randomized to moderate hypothermia treatment or normothermia (Marion, 1997).
  - a. Hypothermic patients were cooled to 32-33 degrees C, beginning an average of 10 h after injury, and were kept cool for 24 h before rewarming. Mortality and functional outcomes were assessed by a blinded observer at 3, 6, and 12 months.
  - b. Hypothermia did not lead to improved outcome among patients with the most severe brain injuries (GCS =3-4). However, among patients with GCS of 5-7, those treated with hypothermia had markedly improved outcomes at 3 and at 6 months. The differences at 12 months were not statistically significant but still favored treatment with hypothermia.
  - c. No complications were observed.

## K. MANAGEMENT, SPORTS-RELATED CONCUSSION

1. Concussions are graded in three categories. Definitions and treatment recommendations for each category are presented below (Am Acad Neurol, 1997):

### a. GRADE 1 CONCUSSION:

(1) DEFINITION: Transient confusion, no loss of consciousness, and a duration of mental status abnormalities of <15 minutes.

### (2) MANAGEMENT:

(a) The athlete should be removed from sports activity, examined immediately and at 5-minute intervals, and allowed to return that day to the sports activity only if postconcussive symptoms resolve within 15 minutes.

(b) Any athlete who incurs a second Grade 1 concussion on the same day should be removed from sports activity until asymptomatic for 1 week.

### b. GRADE 2 CONCUSSION:

(1) DEFINITION: Transient confusion, no loss of consciousness, and a duration of mental status abnormalities of  $\geq$ 15 minutes.

### (2) MANAGEMENT:

(a) The athlete should be removed from sports activity and examined frequently to assess the evolution of symptoms, with more extensive diagnostic evaluation if the symptoms worsen or persist for >1 week. The athlete should return to sports activity only after asymptomatic for 1 full week.

(b) Any athlete who incurs a Grade 2 concussion subsequent to a Grade 1 concussion on the same day should be removed from sports activity until asymptomatic for 2 weeks.

### c. GRADE 3 CONCUSSION:

(1) Definition: Loss of consciousness, either brief (seconds) or prolonged (minutes or longer).

### (2) Management:

(a) The athlete should be removed from sports activity for 1 full week without symptoms if the loss of consciousness is brief or 2 full weeks without symptoms if the loss of consciousness is prolonged. If still unconscious or if abnormal neurologic signs are present at the time of initial evaluation, the athlete should be transported by ambulance to the nearest hospital emergency department.

(b) An athlete who suffers a second Grade 3 concussion should be removed from sports activity until asymptomatic for 1 month.

(c) Any athlete with an abnormality on CT or MRI brain scan consistent with brain swelling, contusion, or other intracranial pathology should be removed from sports activities for the season and discouraged from future return to participation in contact sports.

## L. ORGAN AND TISSUE DONATION

1. If resuscitation attempts are unsuccessful in preventing death or brain death, the patient may be eligible for organ or tissue donation.

2. Healthcare providers are required by law to make families aware of the option of organ and tissue donation.

3. Organ procurement organizations can provide assistance with donor identification and management, organ recovery, and allocation. (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: ORGAN AND TISSUE DONATION)

## 6.3 PHARMACOLOGIC TREATMENT

### A. INTRAVENOUS FLUID

1. In multiply-injured patients with hypotension, decreased blood pressure should be assumed to be secondary to blood loss.

2. Begin NS to maintain BP >100 mmHg, or age-appropriate norm. Avoid routine use of dextrose-containing solutions (potential risk of worsening CNS lactic acidosis and cerebral edema) (Robertson, 1991; Heiselman, 1991).

3. Optimal resuscitation of patients with BHT and hypovolemic shock is controversial.

a. Aggressive use of crystalloids has been postulated to worsen brain injury by exacerbating cerebral edema (Olshaker, 1993; Feldman, 1991). The results of one study suggest that maintenance of normovolemia does not predispose BHT patients to uncontrolled increased ICP and that sodium and fluid restriction is not an independent determinant of ICP during initial 72 h postinjury (Schmoker, 1992).

b. A retrospective analysis of 34 patients with severe head injury (GCS 8 or less) and multiple injuries (ISS 16 or greater) demonstrated avoidance of secondary brain injury in 74% of patients and low mortality (6%) when fluid resuscitation was aggressive, based upon maintenance of mean CPP >80 mmHg and systolic BP >90 mmHg (York, 2000).

- c. In a randomized clinical trial of 189 adults with severe head injury comparing two treatment protocols, controlling CPP with use of fluids and pressors caused less secondary ischemic insults than methods used to control ICP alone. However, this did not translate into improvement in neurologic outcome, and the incidence of ARDS was five times higher in the group receiving fluids and pressors (Robertson, 1999).
- d. Early use of hypertonic agents or mannitol may cause hemodynamic deterioration, although some evidence suggests that early use of mannitol may be appropriate despite presence of shock (Feldman, 1991).
- e. A prospective, randomized study of 35 severely head injured children who were resuscitated with hypertonic saline (HTS) or lactated Ringers (LR) demonstrated lower ICP, increased CPP, fewer complications, and fewer ICU days in the HTS patients. Further studies, including assessment of outcome, are needed before this approach can be recommended (Simma, 1998).
- f. A prospective, randomized trial of HTS vs LR in 34 head-injured patients showed more interventions in the HTS group (who had worse injuries at baseline) but improved ICP parameters vs the LR group. No definite conclusions could be made about the utility of this treatment (Shackford, 1998).

## B. OXYGEN

- 1. INDICATIONS: To prevent further cerebral damage due to hypoxemia.
- 2. RECOMMENDATION: 10 liters/minute at a high rate of flow, unless otherwise contraindicated.

## C. ANTICONVULSANTS

### 1. OVERVIEW

- a. Seizures occur commonly after traumatic brain injury, and they may exacerbate cerebral hypoxia, compromise the airway, and complicate management. However, while anticonvulsants may reduce the incidence of early post-traumatic seizures, they do not reduce the incidence of late seizures.
- b. According to the most recent American Trauma Brain Foundation guidelines, prophylactic anticonvulsants are not recommended; anticonvulsants may be used if seizures are observed in the course of acute head trauma (Kirkpatrick, 1997; Am Acad Neurol, 1997; Bullock, 1996).
- c. A randomized, prospective, double-blind, placebo-controlled study of 404 patients with head injuries demonstrated that the incidence of early posttraumatic seizures can be effectively reduced by prophylactic administration of phenytoin; however, mortality was not reduced (Haltiner, 1999). (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: STATUS EPILEPTICUS)

### 2. FOSPHENYTOIN

- a. INDICATIONS: May be given to patients who demonstrate seizure activity following head injury (Lewis, 1993).
- b. RECOMMENDATION (ADULTS): Loading dose: 15 to 20 milligrams of phenytoin equivalents (PE)/kilogram intravenously at a rate not to exceed 150 milligrams PE/minute. Maintenance dose: 4 to 6 milligrams PE/kilogram/day intravenously (at a rate not to exceed 150 milligrams PE/minute) or intramuscularly as a single daily dose.
- c. AVAILABLE FORMS: Cerebyx(R) (injection).
- d. DOSING IN SPECIAL SITUATIONS: Reduce dose in patients with hepatic or renal impairment, the elderly or those with hypoalbuminemia. The safety of fosphenytoin in pediatric patients has not been established.
- e. MAJOR ADVERSE REACTIONS: Hypotension; dizziness; nystagmus; ataxia; drowsiness; vasodilatation; tachycardia; hepatotoxicity; blood dyscrasias; pruritus.
- f. PRECAUTIONS: Contraindicated in patients with hypersensitivity to phenytoin, sinus bradycardia, sinoatrial block, second and third degree heart block, and Adams-Stokes syndrome. Caution in patients with hypotension, severe myocardial insufficiency, liver or renal disease, diabetes mellitus, porphyria, hypothyroidism, leukopenia, thrombocytopenia, anemia, or febrile illness.
- g. MONITORING PARAMETERS: Cardiac, respiratory, and blood pressure monitoring required during IV administration. Serum concentrations (therapeutic levels 10 to 20 mcg/mL, toxic levels > 20 mcg/mL) should not be drawn until 2 hours following IV administration or 4 hours following IM administration.
- h. EFFICACY: Clinical studies evaluating the efficacy of intravenous or intramuscular fosphenytoin for either treatment or prevention of seizures have not been completed. However, fosphenytoin by either route of administration produces reliable and predictable serum levels of phenytoin, which render the agent useful for control of generalized seizures.

### 3. PHENOBARBITAL

- a. INDICATIONS:
  - (1) Usually second-line agent for managing seizures after head injury. Has a slow onset of action, with a peak serum concentration 20 to 60 minutes after administration.
  - (2) Can be used as a first-line medication in children in whom it may become the maintenance drug.

(3) Also used as a first-line medication in patients allergic to phenytoin/fosphenytoin, with cardiac conduction or automaticity problems, or with history of unresponsiveness to phenytoin/fosphenytoin.

b. RECOMMENDATION:

(1) ADULTS: Loading dose: 15 to 20 milligrams/kilogram intravenously at a rate not to exceed 100 milligrams/minute. Maintenance dose: 2 to 3 milligrams/kilogram/day intravenously at a rate not to exceed 100 milligrams/minute as a single daily dose.

(2) CHILDREN: Loading dose: 10 to 20 milligrams/kilogram intravenously at a rate not to exceed 2 milligrams/kilogram/minute. Maintenance dose: 3 to 6 milligrams/kilogram/day intravenously at a rate not to exceed 2 milligrams/kilogram/minute in three divided doses.

c. AVAILABLE FORMS: Luminal(R) (injection); Eskabarb(R) (injection).

d. DOSING IN SPECIAL SITUATIONS: Increase dosing interval in renal failure; dose reductions required in patients with severe liver disease.

e. MAJOR ADVERSE REACTIONS: Sedation; hypotension and respiratory depression with IV use; severe skin rash; hepatotoxicity; paradoxical restlessness and excitement.

f. PRECAUTIONS: Contraindicated in acute intermittent porphyria; caution in pulmonary insufficiency, hepatic disease, and pregnancy; withdrawal seizures may occur following abrupt termination of high doses; may reduce efficacy of quinidine and warfarin.

g. MONITORING PARAMETERS: Cardiac, respiratory, and blood pressure monitoring required during IV administration. Serum concentrations (therapeutic, 10 to 40 mcg/mL; toxic, above 40 mcg/mL).

h. EFFICACY: Phenobarbital has demonstrated effectiveness in the treatment of status epilepticus; however, it is slower acting than diazepam and lorazepam and causes more sedation than phenytoin/fosphenytoin.

4. DIAZEPAM

a. INDICATIONS: Useful for rapid suppression of seizures but has no role in prophylaxis because of short duration of action.

(1) LORAZEPAM VERSUS DIAZEPAM: Both agents are effective in terminating status epilepticus; choice depends on physician preference. However, lorazepam may be preferable because it has a longer duration of action on CNS, resulting in more sustained seizure control and possibly a lower incidence of serious adverse effects.

(2) Administration of a long-acting anticonvulsant should be initiated with diazepam because of the short duration of diazepam's anticonvulsant effect.

b. RECOMMENDATION:

(1) ADULTS: 5 to 10 milligrams intravenously at a rate not to exceed 5 milligrams/minute. Dose may be repeated every 10 to 15 minutes up to a maximum dose of 30 milligrams. Therapy may be repeated in 2 to 4 hours if needed.

(2) CHILDREN: 0.2 to 0.5 milligram/kilogram intravenously at a rate not to exceed 1 milligram/minute (maximum single dose, 5 milligrams in infants, 10 milligrams in children).

Although IV administration is preferred, undiluted diazepam IV solution given per rectum is an alternative when venous access is not possible.

c. AVAILABLE FORMS: Valium(R) (injection).

d. DOSING IN SPECIAL SITUATIONS: Reduce dose in elderly patients and patients with hepatic insufficiency; dose reduction not required in renal failure.

e. MAJOR ADVERSE REACTIONS: Apnea with rapid IV injection (greater than 5 mg/min) especially in patients receiving barbiturates; venous thrombosis and phlebitis at injection site; physical dependence and other adverse reactions with prolonged use.

f. PRECAUTIONS: Contraindicated in acute narrow-angle glaucoma; caution in patients with shock, coma, or alcoholic intoxication with depressed respiratory rate; diazepam potentiates effects of other CNS depressants.

g. MONITORING PARAMETERS: Blood pressure and respirations with IV administration.

h. EFFICACY: Various studies have shown effective termination of seizure activity in 69% to 76% of cases.

5. LORAZEPAM

a. INDICATIONS: Useful for rapid suppression of seizures but has no role in prophylaxis because of short duration of action.

(1) LORAZEPAM VERSUS DIAZEPAM: Both agents are effective in terminating status epilepticus; choice depends on physician preference. However, lorazepam may be preferable because it has a longer duration of action on CNS, resulting in more sustained seizure control and possibly a lower incidence of serious adverse effects.

(2) Administration of a long-acting anticonvulsant should be initiated with lorazepam because of the short duration of lorazepam's anticonvulsant effect.

b. RECOMMENDATION:

(1) ADULTS: 0.05 to 0.15 milligram/kilogram intravenously at a rate not to exceed 2 milligrams/minute (maximum single dose, 8 milligrams).

(2) CHILDREN: 0.05 to 0.1 milligram/kilogram intravenously at a rate of 1 to 2 milligrams/minute (maximum single dose, 4 milligrams). Rectal administration available when venous access is not possible.

c. AVAILABLE FORMS: Ativan(R) (injection).

d. DOSING IN SPECIAL SITUATIONS: Dose reductions not required in liver or renal disease; reduce dose in elderly patients.

e. MAJOR ADVERSE REACTIONS: Apnea with rapid IV injection (greater than 5 mg/minute), especially in patients receiving barbiturates; venous thrombosis and phlebitis at injection site; physical dependence and other adverse reactions with prolonged use. Because lorazepam has an anti-epileptic duration of action of 4 to 14 hours, late respiratory depression may occur.

f. PRECAUTIONS: Contraindicated in acute narrow-angle glaucoma; caution in patients with shock, coma, or alcoholic intoxication with depressed respiratory rate; lorazepam potentiates effects of other CNS depressants.

g. MONITORING PARAMETERS: Blood pressure and respirations with IV administration.

h. EFFICACY: Various studies have shown effective termination of seizure activity in 75% to 90% of cases.

D. ANTICEREBRAL EDEMA AGENTS

1. MODALITIES: Cerebral edema treatment modalities may include: head elevation, osmotic therapy, diuretics, rapid sequence intubation, barbiturates, hyperventilation, mechanical decompression.

2. CAUTION: Physicians managing cerebral edema must be experienced and knowledgeable in the monitoring techniques and therapeutic modalities used to treat cerebral edema.

3. MANNITOL: Mannitol is effective in reducing ICP but should be reserved for measured intracranial hypertension or clinical herniation. Bolus administration is preferable to continuous infusion (Marshall, 2000). (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: INCREASED INTRACRANIAL PRESSURE)

E. ANALGESICS

1. ACETAMINOPHEN

a. INDICATIONS:

(1) During the first day or so postinjury, most patients with concussion do not complain of much pain from either the head injury or associated injuries.

(2) In general, it is best to treat these patients without the use of analgesics; however, severe pain should be relieved by an analgesic.

(3) Because the administration of any analgesics is documented in the patient's chart, it should not be difficult to interpret the significance of changes in the level of consciousness.

b. RECOMMENDATION:

(1) ADULTS: 650 to 1000 milligrams orally every four hours as needed; maximum 4 grams/day.

(2) CHILDREN: 10 to 15 milligrams/kilogram orally every four to six hours as needed; maximum, 650 milligrams/dose.

c. AVAILABLE FORMS: Tylenol(R) or equivalent analgesic.

d. DOSING IN SPECIAL SITUATIONS: Dose reduction not required in renal failure or geriatric patients.

e. MAJOR ADVERSE REACTIONS: Hepatotoxicity in overdose (adults); thrombocytopenia; hemolytic anemia (rare); adverse reactions during prolonged use.

f. PRECAUTIONS: Use with caution in patients with G-6-PD deficiency.

2. IBUPROFEN

a. INDICATIONS:

(1) During the first day or so postinjury, most patients with concussion do not complain of much pain from either the head injury or associated injuries.

(2) In general, it is best to treat these patients without the use of analgesics; however, severe pain should be relieved by an analgesic.

(3) Because the administration of any analgesics is documented in the patient's chart, it should not be difficult to interpret the significance of changes in the level of consciousness.

b. RECOMMENDATION:

(1) ADULTS: 400 milligrams orally every four to six hours as needed; maximum, 2.4 grams/day.

(2) CHILDREN: 10 milligrams/kilogram orally every six to eight hours as needed; maximum, 40 milligrams/kilogram/day.

- c. AVAILABLE FORMS: Motrin(R) (tablets); Nuprin(R) (tablets, caplet); Advil(R) (tablets); Medipren(R) (tablets), or equivalent NSAID.
- d. DOSING IN SPECIAL SITUATIONS: Increase dosage interval in renal failure.
- e. MAJOR ADVERSE REACTIONS Tinnitus; hearing loss; GI bleeding; cholestatic jaundice; anaphylaxis.
- f. PRECAUTIONS: Contraindicated in patients hypersensitive to aspirin or other NSAID; caution in active peptic ulcer disease, renal insufficiency, hepatic dysfunction, and patients with compromised cardiac function (edema); potentiates effects of warfarin; concomitant antacid administration may reduce absorption.

### 3. ACETAMINOPHEN WITH CODEINE

#### a. INDICATIONS:

- (1) During the first day or so postinjury, most patients with concussion do not complain of much pain from either the head injury or associated injuries.
- (2) In general, it is best to treat these patients without the use of analgesics; however, severe pain should be relieved by an analgesic.
- (3) Because the administration of any analgesics is documented in the patient's chart, it should not be difficult to interpret the significance of changes in the level of consciousness.
- (4) After 24 hours, narcotic analgesics may be given for relief of headache.

#### b. RECOMMENDATION:

- (1) ADULTS: 1 to 2 tablets orally every four hours as needed.
- (2) CHILDREN: 5 milliliters (3 to 6 years) or 10 milliliters (7 to 12 years) elixir orally every six to eight hours as needed.

c. AVAILABLE FORMS: Tylenol #3(R) (tablets); Tylenol with Codeine(R) (tablets and elixir for children) or equivalent analgesic.

d. DOSING IN SPECIAL SITUATIONS: Dose reduction not required in renal insufficiency; reduce dose in hepatic disease.

e. MAJOR ADVERSE REACTIONS: Hepatotoxicity in overdose (adults); thrombocytopenia; respiratory depression; hypotension.

f. PRECAUTIONS: Contraindicated in G-6-PD deficiency, respiratory depression or coma; use with caution in presence of convulsions, shock, asthma or COPD; additive CNS depression with other depressant drugs.

### 4. ACETAMINOPHEN WITH OXYCODONE

#### a. INDICATIONS:

- (1) During the first day or so postinjury, most patients with concussion do not complain of much pain from either the head injury or associated injuries.
- (2) In general, it is best to treat these patients without the use of analgesics; however, severe pain should be relieved by an analgesic.
- (3) Because the administration of any analgesics is documented in the patient's chart, it should not be difficult to interpret the significance of changes in the level of consciousness.
- (4) After 24 hours, narcotic analgesics may be given for relief of headache.

b. RECOMMENDATION (ADULTS): 1 tablet orally every six hours as needed.

c. AVAILABLE FORMS: Tylox(R) (tablets); Percocet(R) (tablets).

d. DOSING IN SPECIAL SITUATIONS: Dose reductions not required in renal insufficiency; dose reductions required in hepatic disease.

e. MAJOR ADVERSE REACTIONS: Respiratory depression; hypotension; addiction with prolonged use.

f. PRECAUTIONS: Contraindicated in patients with respiratory depression or coma; use with caution in the presence of convulsions, shock, asthma or COPD; additive CNS depression with other depressant

## F. ANTIDEPRESSANTS

### 1. INDICATIONS (Horn, 1992):

- a. Have been known to alleviate headaches and, for unknown reasons, appear to help stabilize the physiologic brain dysfunction following brain injury, as in postconcussion syndrome.
- b. May be recommended if there is no relief of postconcussion symptoms from more conventional methods (eg, acetaminophen).

## G. ANTIBIOTICS, PROPHYLACTIC

### 1. OVERVIEW

a. Indicated for open fractures. No clear utility with basilar skull fractures in preventing infectious complications (Villalobos, 1998).

### 2. CEFTRIAXONE

a. INDICATIONS: Open or basilar fractures of the skull; has excellent penetration into CSF fluid (Demetriades, 1992).

- b. RECOMMENDATION: ADULTS: 1 gram (CHILDREN: 50 milligrams/kilogram) intravenously once a day for minimum of three days.
- c. AVAILABLE FORMS: Rocephin Injectable (R) (powder).
- d. SOLUTION PREPARATION (IV):
  - (1) VIAL: For each 250 mg of ceftriaxone powder, add 2.4 mL diluent (sterile water, NS, D5W, D10W); after reconstitution, each 1 mL of solution contains approximately 100 mg of ceftriaxone. Withdraw entire contents and dilute to the desired concentration with the appropriate IV diluent.
  - (2) PIGGYBACK:

Piggyback Bottle Dosage Size	Amount of Diluent to be added
1 g	10 mL
2 g	20 mL

- After reconstitution, further dilute to 50 mL or 100 mL volumes with the appropriate IV diluent.
- e. DOSING IN SPECIAL SITUATIONS: Dose reduction not required in renal failure or hepatic impairment.
  - f. MAJOR ADVERSE REACTIONS: Hematologic abnormalities in up to 14% (leukopenia, thrombocytopenia, eosinophilia; more frequent in children); headache or dizziness; diarrhea or colitis (rare); hepatotoxicity (rare); rash, allergic reaction; renal dysfunction (rare).
  - g. PRECAUTIONS: Contraindicated in cephalosporin hypersensitivity; caution with history of penicillin allergy. Sodium content, 3.6 mEq/g.
  - h. MONITORING PARAMETERS: Therapeutic serum level is 10 mcg/mL or less, which is achieved with 1 to 2g single IV doses over a 24-hour period.

## H. CALCIUM ANTAGONISTS

### 1. NIMODIPINE

- a. A randomized, controlled trial suggests that nimodipine may be beneficial in patients with traumatic subarachnoid hemorrhage (European Study Group on Nimodipine in Severe Head Injury, 1994). Confirmatory studies are needed.
- b. In another trial, 123 patients with traumatic subarachnoid hemorrhage were randomized to receive either nimodipine or placebo. Nimodipine, administered within 12 h of head injury and continued for 3 wk, led to a reduction in death, vegetative survival, and severe disability, all measured at 6 months (Harders, 1996).

## I. ANTIOXIDANTS

- 1. In a cohort of 1120 patients with head injury, the 21-aminosteroid (lazaroid) tirilazad failed to demonstrate an effect on the end points of good neurologic outcome or death. The authors attribute this lack of efficacy on baseline differences between the treatment and placebo group and suggest that further study is required (Marshall, 1998). Tirilazad also failed to demonstrate efficacy in the Third National Acute Spinal Cord Injury Randomized Controlled Trial (Bracken, 1997).
- 2. In a large multicenter trial, 463 patients were randomized to receive either placebo or the free oxygen radical scavenger pegorgotein within 8 h of severe head injury (GCS  $\leq$ 8). At follow-up 3 and 6 months after injury, there were no significant differences in mortality or in neurologic outcome. Patients who received pegorgotein did have a lower incidence of ARDS, although this was not a primary or secondary endpoint of the study design (Young, 1996).

## J. NMDA RECEPTOR ANTAGONISTS

- 1. Investigational. N-methyl-D-aspartate (NMDA) receptor antagonists appear to have a neuroprotective effect. One such agent, selfotel, is currently in phase III trial (Albanese, 1995).

## K. VASOPRESSORS

- 1. Should be considered to maximize cerebral perfusion pressure (CPP) in patient resuscitated adequately but with suboptimal CPP. Usual choices are norepinephrine or phenylephrine. Only use in conjunction with ICP monitoring and neurosurgical consultation.

## 7.0 DISPOSITION

### 7.1 ADMISSION CRITERIA

- A. MINIMAL HEAD TRAUMA (No LOC or amnesia AND GCS score 15, normal alertness and memory AND no focal neurologic deficit AND no palpable depressed fracture): Admission only if there are other indications (see below) (Ingebrigtsen, 2000; Stein, 1993a).

B. MILD HEAD TRAUMA (Brief (<5 min) LOC OR amnesia for event OR GCS score 13 to 15 OR impaired alertness or memory OR palpable depressed fracture): Obtain urgent CT scan; admission indicated if intracranial lesion on CT scan or other indication for admission; obtain neurosurgical evaluation (Ingebrigtsen, 2000; Stein, 1993a). (NOTE: Not all authors consider brief LOC in absence of other clinical signs and symptoms a definite indication for brain imaging (Miller 1997; Murray 1996; Duus, 1994; Taheri,1996).)

C. MODERATE OR POTENTIALLY SEVERE (Prolonged (<5 min) LOC OR focal neurologic deficit OR post-traumatic seizure OR intracranial lesion on CT scan): Indicated in all cases; obtain urgent CT scan and neurosurgical consultation (Ingebrigtsen, 2000; Stein, 1992a, 1993a).

D. OTHER INDICATIONS (Stein, 1993a):

1. Extracranial injuries requiring admission.
2. Patients <2 years or >65 years with severe headache, nausea, vomiting, etc.
3. No reliable transportation to or reliable observation at home.
4. Therapeutic anticoagulation or medical condition with increased risk (eg, hemophilia).
5. Open, depressed skull fracture. (Patients with basilar skull fracture who have a GCS score of 13 or greater with no intracranial pathology do not require ICU monitoring (Koonsman, 1992).)

## 7.2 HOME CRITERIA

A. All U.S. emergency department patients must be screened, stabilized, and discharged in accordance with the EMTALA (COBRA) law.

B. MINIMAL HEAD TRAUMA (No LOC or amnesia AND GCS score 15, normal alertness and memory AND no focal neurologic deficit AND no palpable depressed fracture): May discharge with head trauma instructions if there are no other indications for admission (see above) (Savitsky, 2000; Ingebrigtsen, 2000; Mitchell, 1994; Stein, 1993a).

1. The AAP/AAFP clinical practice parameter states that children (aged 2 to 20 yr) with minor closed head injury (normal mental status and neurologic examination, GCS=15) who remain neurologically normal and have a normal cranial CT scan can be sent home for monitoring by a reliable observer (Am Acad Pediatr, 1999; Preboth, 1999).
2. The AAP/AAFP clinical practice parameter states that children (aged 2 to 20 yr) with minor closed head injury (GCS=15) and no LOC with a normal neurologic examination can be discharged without radiologic imaging. Those with LOC should be observed for 24 hours by a competent caregiver; cranial CT scan should be considered (Am Acad Pediatr, 1999; Preboth, 1999).

C. MILD HEAD TRAUMA (Brief <5 min) LOC OR amnesia for event OR GCS score 13 to 15 OR impaired alertness or memory OR palpable depressed fracture):

1. Obtain urgent CT scan. May discharge with head trauma instructions if no intracranial lesion on CT scan or other indication for admission (Stein, 1993a).
2. While not current standard of care, a study of pediatric closed head injury patients suggests that home observation may be appropriate for patients with GCS score  $\geq 13$  (Roddy, 1998; Greenes, 1997; Dahl-Grove, 1995).
3. Patients with minimal head injury (GCS 14 or 15) and brief LOC or posttraumatic amnesia who have a cranial CT scan performed on a helical CT scanner that demonstrates no intracranial injury can be safely discharged from the emergency department without a period of observation (Livingston, 2000; Nagy, 1999).
4. Patients with GCS=15 and LOC should undergo cranial CT scan if one or more of the following 7 findings are present: headache, vomiting, age over 60, drug or alcohol intoxication, short-term memory deficits, physical evidence of head, face, or neck trauma, and seizure (Haydel, 2000).

D. SPORTS-RELATED CONCUSSION (Am Acad Neurol, 1997):

1. Concussions are graded in three categories. Definitions and treatment recommendations for each category are presented below. NOTE: These concussion management guidelines remain controversial, with further studies necessary to refine both definitions and recommendations (Collins, 1999); however, careful neurologic evaluation, knowledge of these guidelines, and good clinical judgement should allow sideline physicians to reach rational management decisions (Harmon, 1999).

a. GRADE 1 CONCUSSION:

(1) DEFINITION: Transient confusion, no loss of consciousness, and a duration of mental status abnormalities of <15 minutes.

(2) MANAGEMENT:

(a) The athlete should be removed from sports activity, examined immediately and at 5-minute intervals, and allowed to return that day to the sports activity only if postconcussive symptoms resolve within 15 minutes.

- (b) Any athlete who incurs a second Grade 1 concussion on the same day should be removed from sports activity until asymptomatic for 1 week.
- b. GRADE 2 CONCUSSION:
  - (1) DEFINITION: Transient confusion, no loss of consciousness, and a duration of mental status abnormalities of  $\geq 15$  minutes.
  - (2) MANAGEMENT:
    - (a) The athlete should be removed from sports activity and examined frequently to assess the evolution of symptoms, with more extensive diagnostic evaluation if the symptoms worsen or persist for  $>1$  week. The athlete should return to sports activity only after asymptomatic for 1 full week.
    - (b) Any athlete who incurs a Grade 2 concussion subsequent to a Grade 1 concussion on the same day should be removed from sports activity until asymptomatic for 2 weeks.
- c. GRADE 3 CONCUSSION:
  - (1) DEFINITION: Loss of consciousness, either brief (seconds) or prolonged (minutes or longer).
  - (2) MANAGEMENT:
    - (a) The athlete should be removed from sports activity for 1 full week without symptoms if the loss of consciousness is brief or 2 full weeks without symptoms if the loss of consciousness is prolonged. If still unconscious or if abnormal neurologic signs are present at the time of initial evaluation, the athlete should be transported by ambulance to the nearest hospital emergency department.
    - (b) An athlete who suffers a second Grade 3 concussion should be removed from sports activity until asymptomatic for 1 month.
    - (c) Any athlete with an abnormality on CT or MRI brain scan consistent with brain swelling, contusion, or other intracranial pathology should be removed from sports activities for the season and discouraged from future return to participation in contact sports.

### 7.3 CONSULT CRITERIA

- A. NEUROSURGEON (Gruen, 1998; Bullock, 1990; White, 1992)
  - 1. Obtain prompt neurosurgical consultation on any head-injured patient with a GCS score  $<8$ , persistent or progressive alteration of consciousness, focal neurologic deficit, open or depressed skull fracture, or posttraumatic seizures.
  - 2. Cranial CT findings of mass effect, midline shift, obliteration of basal cisterns, extra or intra-axial hematoma, intracranial air or depressed skull fracture require neurosurgical consultation.
  - 3. Patients admitted with less specific neurologic signs and symptoms, including headache, vomiting, amnesia, brief loss of consciousness, and linear skull fractures, should also be evaluated by neurosurgeon.
- B. ORGAN AND TISSUE DONATION:
  - 1. If resuscitation attempts are unsuccessful in preventing death or brain death, the patient may be eligible for organ or tissue donation.
  - 2. Healthcare providers are required by law to make families aware of the option of organ and tissue donation.
  - 3. Organ procurement organizations can provide assistance with donor identification and management, organ recovery, and allocation. (FOR FURTHER INFORMATION, SEE CLINICAL REVIEW: ORGAN AND TISSUE DONATION)

### 7.4 TRANSFER CRITERIA

- A. All U.S. emergency department patients must be screened, stabilized, and discharged in accordance with the EMTALA (COBRA) law.
- B. INDICATIONS FOR TRANSFER: Patients with serious head injuries (especially those with GCS score  $=3-8$ ) are best managed in centers with appropriate staffing, equipment, and expertise. These patients should be transferred to nearest qualified facility in an expeditious manner.
- C. Formal protocols describing transfer procedures should be arranged between involved facilities whenever practical. Several minimum standards must be applied during transfer process.
  - 1. TRANSFER PROMPTLY: Do not delay for performance of trivial procedures or tests, specifically skull radiography and scalp laceration repair.
  - 2. Initiate life/limb saving procedure prior to transport (Andrews, 1990a): Adequate ventilation and oxygenation, stabilization of cervical spine, and treatment of life-threatening hemorrhage must be attended to before transporting seriously injured patient.
    - a. Hypercarbia, hypoxia, and hypotension are particularly harmful to already injured brain.
    - b. If patient is comatose, tracheal intubation should be performed prior to transport.

3. MAINTAIN LIFE SUPPORT EN ROUTE:
  - a. Hypoxia, aspiration, respiratory insufficiency, and hypotension commonly occur in head-injured patients during interhospital transfer (Gentleman, 1981).
  - b. Transport should be performed only by personnel with expertise in advanced life support and understanding of early management of these patients.
4. Whenever possible, direct communication with a neurosurgeon prior to transfer is useful for patients with severe head injury (GCS 3 to 8).

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