

Attorneys' Textbook of Medicine (Third Edition)
CHAPTER 93 SEQUELAE OF HEAD INJURIES
Excerpt

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P 93.00 NATURE AND SIGNIFICANCE OF HEAD INJURY

The term "head injury" generally refers to traumatic injury to the brain and intimately associated cerebrocranial structures, most prominently the cerebral hemispheres, the brainstem, the intracranial vasculature and the meningeal membranes that envelope the brain. However, other major types of cranial injury can be sustained during acute head trauma, including lacerations of the scalp, skull fractures, cortical contusions and lacerations, and intracranial hemorrhage (Evans and Wilberger, 1999).

Traumatic head injury is a major cause of morbidity and mortality in the United States, especially among the pediatric population and young adults (Evans and Wilberger, 1999; MacKenzie, 2000). Physicians who care for patients suffering an acute head injury must understand the need for timely evaluation and treatment of head injuries and the possible long-term complications of such injuries. The impact of these injuries can be devastating for both the patient and society, since death or permanent disability associated with chronic pain syndromes can result (Evans and Wilberger, 1999). Despite increased understanding of the pathophysiology of brain injury, no new medical treatments have been developed, and supportive care remains the mainstay of therapy. Thus, prevention of acute head injury remains of utmost importance.

P 93.10 ETIOLOGY AND EPIDEMIOLOGY OF HEAD INJURY

Motor vehicle accidents account for the majority of acute head injuries, but recent mandates for seatbelt use and air bags have reduced the incidence of head injuries caused by car accidents. In contrast, gunshot wounds to the head have been increasing, particularly in urban areas, where this is the most common cause of head injury observed in these communities (Evans and Wilberger, 1999). Among children and

adolescents, sports such as diving, gymnastics and football are a major cause of head and neck injuries. Approximately 3 to 25 percent of all traumatic head injuries in this age group are caused by organized sporting activities (Proctor and Cantu, 2000).

For the population as a whole, closed head injury leads to 500,000 hospitalizations per year, resulting in over 175,000 deaths as well as significant disability. Current estimates of the incidence of head injury approach 222 in 100,000 population per year. (Evans and Wilberger, 1999) The peak incidence occurs in men between 15 to 24 years of age and again above 75 years of age.

Head trauma is the leading cause of death and disability in children, accounting for more than 50 percent of deaths in this age group. On average, approximately 100,000 to 200,000 head injuries occur per year among children, with a rate of 193 to 367 per 100,000 (Gedeit, 2001). The incidence peaks early in childhood and again during adolescence, mirroring non-accidental and vehicular causes of trauma, respectively.

[93.11] Mortality

There has been a significant decline in overall mortality associated with head injuries from the mid-30 percent range reported in the 1970s to less than 20 percent in the 1990s. (Evans and Wilberger, 1999) Improved mortality rates have resulted from increased understanding of the pathophysiology of brain injury and the development of aggressive treatment strategies to prevent intracranial pressure elevations and ischemia (lack of blood supply). When secondary insults of low oxygen saturation (hypoxia) and low blood pressure (hypotension) are superimposed on severe head injury, mortality is doubled. Similarly, concomitant elevations in intracranial pressure increases morbidity. Hence, timely treatment to prevent these insults is of paramount importance.

[93.12] Principal Risk Factors for Injury

Principal risk factors for sustaining head trauma include age, gender, race, area of residence, socioeconomic status and occupation. Following injury, the critical determinant of ultimate outcome is the nature of secondary sequelae and the extent to which they develop. Adequate control of these depends on the quality of medical care the patient receives. Males tend to be affected more than females, with a ratio of 2 to 4:1 in adolescents; the highest rate occurring among the lower socioeconomic classes. Advanced age is also an important risk factor for traumatic head injury.

Among the elderly, the increase in frequency of these injuries is related to the increase in frequency of falls (Luukinen, et al., 1999). In rural areas, farm equipment is a major source of injury, while in urban areas, violence, particularly gunshot wounds to the head, is a predominant cause (Evans and Wilberger, 1999). Agriculture, construction, mining, manufacturing and transportation are occupations associated with the highest risk of traumatic injury and death (Evans and Wilberger, 1999).

[93.13] The Cost of Head Injury

Head injuries account for nearly 50 percent of all hospitalizations and are the leading cause of death due to injury as well as disabilities in children and young adults (MacKenzie, 2000). Each year, approximately 80,000 survivors of head injury will incur some disability or require extensive medical treatment for their injury. The direct medical costs for the treatment of traumatic brain injury is estimated to be \$48 billion per year, which includes the costs of hospitalization for acute care and rehabilitation services (ACHPR, 1999). In the pediatric population, the costs of the head-injured patient are even greater. The median costs for a child having a severe head injury is reported to be about \$53,000 dollars, with rehabilitation services accounting for 37 percent of the total costs. The specific costs of head injury vary with the severity of the injury. Early diagnosis and treatment of patients with mild head injury can significantly reduce the costs to patients and their families (Cheung and Kharasch, 1999).

P 93.20 DEFINITIONS

Head trauma is classified as mild, moderate or

severe based on neurologic assessment using the Glasgow Coma Score (which is modified for use in children), with the range between 1 and 15. In general, a Glasgow coma score of at least 13 defines mild head injury, 8 to 12 moderate head injury, and 7 or less severe head injury. (Gedeit, 2001; Evans and Wilberger, 1999) By definition, a concussion is a mild head injury associated with confusion or loss of consciousness (less than one minute). Concussions are not usually associated with structural brain injury or long-term sequelae. The postconcussion syndrome is a distinct entity and will be discussed in detail later on in this chapter. A brain contusion results from direct injury to the brain, either from blunt trauma from external contact forces or from the brain contacting intracranial surfaces resulting from acceleration/deceleration trauma (Gedeit, 2001).

P 93.30 MECHANISMS OF HEAD INJURY

Traumatic brain injury is an insult to the brain by an external force that results in either a transient or permanent impairment of cognitive, behavioral, emotional, or physical function. Traumatic brain injury encompasses a shearing injury, which can result from blunt trauma (such as what may occur in a shaken infant), as well as penetrating injury from a foreign body, such as a bullet (Guthrie et al., 1999).

The sequelae of head injury can be classified three ways:

- (1) with respect to the physical forces that ultimately cause the damage;
- (2) with respect to the types of pathophysiological lesions that result within the cranium, and;
- (3) with respect to the time of injury onset and course.

[93.31] Physical Forces

An understanding of the following principles are necessary in order to understand the physical forces that produce skull and brain injuries. First, a forceful blow to a restful, movable head produces maximum head injury beneath the point of impact (this is known as a "coup" injury). Second, a moving head which collides against a stationary object produces maximum brain injury opposite to the site of cranial impact (also known

as "contrecoup" injury). Such lesions are most common at the tips and the undersurfaces of the frontal and temporal lobes. Third, if a skull fracture is present, the first two principles are not applicable, because the bone itself, whether transiently (e.g., a linear skull fracture) or permanently (e.g., a depressed skull fracture) displaced at the time of impact may directly injure brain tissue (Proctor and Cantu, 2000).

There are also three types of stresses that are generated by an applied force: compressive, tensile (the opposite of compressive, sometimes referred to as negative pressure), and shearing (when the applied force is parallel to a surface). Uniform compressive forces are generally tolerated fairly well by the nervous system, but shearing stresses are very poorly tolerated (Proctor and Cantu, 2000).

The cerebrospinal fluid (CSF) acts in a manner consistent with a shock absorber, protecting the brain by converting focally applied external stresses to a more uniform compressive stress. This occurs because the fluid follows the contours of the sulci of the brain, thus cushioning the brain from damaging shearing forces. Despite the presence of CSF, however, shearing stresses may still be imparted to the brain. If rotational forces are applied to the head, for example, shearing forces may potentially occur at sites where rotational gliding is hindered. These areas are characterized by (1) irregular surface contacts between the brain and skull (which hinder smooth movement); (2) leakage of CSF between the brain and skull, and (3) attachments between the dura mater (tissue that covers the surface of the brain) and the brain itself, which impedes brain motion (Proctor and Cantu, 2000).

The first condition is usually most prominent in the frontal and temporal regions of the brain and is the reason why major brain contusions occur at these sites. The second condition is most commonly associated with the coup and contrecoup injuries. When the head is accelerated before impact (e.g., what occurs in auto racing), the brain lags toward the trailing surface, displacing the CSF, allowing the shearing forces to be maximal at this site. Usually, brain lag thickens the layer of CSF under the point of impact, preventing coup injury in a moving head injury. In contrast, when the head is stationary before the point of impact, brain lag nor a disproportionate distribution of CSF occurs,

which accounts for the absence of contrecoup injury and the presence of coup injury (Proctor and Cantu, 2000).

The scalp also provides energy-absorbing properties. Approximately ten times more force is required to produce a skull fracture in a head with the scalp in place than in one in which the scalp has been removed. In addition, an athlete's head can sustain large forces without brain injury if the neck muscles are tensed at the moment of impact. In the relaxed state, the mass of the head acts as its own weight as it is propelled forward. However, when the neck is rigidly tensed, the mass of the head approximates the mass of the body, enabling the head to sustain the force within minimum impact (Proctor and Cantu, 2000).

In general, the damage done by the various physical forces involved in head trauma is produced through two basic mechanisms: concussive-compressive mechanisms and acceleration-deceleration mechanisms. Most head injuries involve some combination of the two, and their interaction often is complex (Guthrie, et al., 1999).

[1] Concussive-Compressive Injuries-- Concussive-compressive injuries are seen following both blunt and penetrating trauma. Such injuries often result in brain contusions, lacerations, and hemorrhage in the epidural, subarachnoid, subdural, or intracerebral spaces (Amann, 2000).

[1a] Blunt Trauma-- A concussive-compressive type of injury usually results from blunt trauma caused by a blow to the head with a heavy object, such as a baseball bat, hammer or pool cue. As the object impacts the head, the kinetic energy of its motion is transmitted first to the scalp, then to the skull and finally to the brain and surrounding structures. The scalp and skull offer some protection to the brain by dissipating a fraction of the energy. However, a force greater than the elasticity of the skull can accommodate will produce a skull fracture, which can cause direct mechanical tearing of the blood vessels and laceration of the brain, depending on the type and severity of the fracture (Evans and Wilberger, 1999; Amann, 2000).

Whether the skull is fractured or not, some portion of the kinetic energy of the blow is transmitted to the brain and other intracranial contents in the

form of concussive-compressive pressure waves. The force of these waves tends to be concentrated over the area of impact, which usually is relatively small (e.g., the size of a hammer head), but the inertia is propagated to some degree throughout the entire brain. Consequently, the clinical picture is one of local contusion (bruising) and diffuse concussion (jarring shock) (Evans and Wilberger, 1999; Amann, 2000).

Head injury is frequently associated with skull fracture, and the clinical significance of the fracture depends on the type of head injury. With severe head injury, linear fractures are largely insignificant. However, the presence of a skull fracture with mild head injury increases the risk of an intracranial abnormality by as much as four-fold. Basilar skull fractures tend to be complicated by leak of cerebrospinal fluid, infection and cranial nerve palsies. Hence, patients with basilar skull fractures require closer clinical monitoring than those with linear skull fracture. In infants and young children, linear fractures may be complicated by leptomeningeal cysts or "growing" skull fractures. When these lesions occur, a mass may develop in close proximity to the fracture site, as CSF collects from a disruption of the underlying tissue. Appropriate treatment of a cyst requires surgery. For this reason, it is important to follow children for several months for the development of a possible cyst (Evans and Wilberger, 1999).

[1b] Penetrating Trauma-- Although penetrating trauma to the brain is not as common as blunt trauma, the incidence of this type of brain injury has been increasing (Blank-Reid and Reid, 2000; Peek-Asa, et al., 2001) Penetrating injury from a foreign body, such as a bullet or other projectile produces the most destructive concussive-compressive injuries. Damage results from both the transmission of impact forces from the skull to the brain and the passage of the projectile through the brain tissue. Direct structural damage occurs as the projectile crushes the tissue in its path (producing a permanent hole), and to a lesser extent as a result of stretching of the surrounding tissue by waves of kinetic energy. The majority of damage is caused by the crushing force. Mortality rates are higher in patients with penetrating brain injuries compared to those with closed brain injury. In one study, patients with penetrating brain injuries were approximately seven times more likely to die compared to closed brain injured patients (Peek-Asa, et al., 2001).

In the case of penetrating trauma to the head, rapid transport to trauma centers where definitive care can be rendered is essential. Outcome of the injury depends on the nature of the missile tract, the presenting neurologic status, and the extent of tissue destruction. Neurologic deterioration occurs rapidly and outcome results appear to determine the patient's neurologic status at the time of surgery (Blank-Reid and Reid, 2000). Penetrating head injuries are often difficult to manage since the extensive surgery that is required can result in severe morbidity and mortality (Rao, et al., 1998).

[2] Acceleration-Deceleration Forces-- Acceleration-deceleration injury is the type of head injury which is characteristic of falls or motor vehicle accidents (e.g., contrecoup contusions). In both instances, brain tissue is severely injured due to disruption of axonal fibers by shearing forces during acceleration, deceleration and rotation of the head. Diffuse injuries usually result from shearing forces produced by acceleration or deceleration with angular rotation, which commonly occurs when a moving head strikes a stationary object. The resulting injury pattern is known as "diffuse axonal injury" and is often seen in patients with concussion. This is the most common mechanism of injury in young children who experience falls from short distances that impart a linear force to the head (Amann, 2000).

Two case reports in the literature also describe the unusual scenario of patients with cerebral contusions resulting from falling tree limbs hitting the head (e.g., blunt force trauma) (Morrison, et al., 1998) The pathogenesis of these contusions involves a forceful impact resulting from acceleration of the head and brain of a magnitude comparable with that of a motor vehicle accident or fall.

Acceleration or deceleration forces experienced in a motor vehicle accident induce indirect force effect lesions, such as subdural hematomas and subarachnoid hematomas (Richter, et al., 2001) These lesions occur because the collision of a vehicle traveling at high speed with another object brings the vehicle and its occupant to an immediate standstill. This abrupt change in motion results in shear forces that severely damage brain tissue. While the occupant's body and head are brought to a stop by impact with the dashboard (for example), the brain and other intracranial contents

continue to travel at the original high velocity until they are brought to a stop by impact with the inner surface of the skull. In an instant, the brain is subjected to a combination of powerful acceleration-deceleration shearing forces which involve angular acceleration (twisting), differential deceleration of different regions of the brain and rebound forces as the elastic brain tissue snaps back from its traumatic deformation. The tremendous amounts of kinetic energy thus imparted to the brain, its covering membranes (e.g., the meninges) and its vasculature produce widespread and severe damage (Richter, et al., 2001).

Victims of motor vehicle collisions often develop a syndrome consisting of head, neck, and back pain, short-term memory loss, fatigue and a lack of stamina, poor balance, and personality changes. This syndrome is often referred to as "the motor vehicle collision injury syndrome" in the medical literature. Although the pathogenesis of this syndrome is not well understood, it is believed that the collision impact results in an inertial strain injury to the anterior regions of the brain, depressing the functions of the frontotemporal lobes which in turn impairs sensory input to the brain. Early intervention that arrests the metabolic cascade caused by head injury may mitigate the symptoms of this syndrome (Mamelak, 2000).

[3] Combination Injuries and Multiple Trauma-- Every head injury involves both concussive-compressive and acceleration-deceleration mechanisms to varying degrees. In addition to the relatively local concussive-compressive forces that occur from a blow to the head, the force generated by the impact of the object also causes the head to move. When this occurs, the encased brain accelerates as a result of being pushed by the inner surface of the skull. When the head stops moving, the brain decelerates due to the impact with the interior surface of the skull. The brain and associated structures are thus subjected to some inertial acceleration-deceleration shearing forces. For example, when the head of the occupant of a crashing vehicle hits the dashboard or other part of the vehicle, the brain is subjected to local concussive-compressive forces emanating from the site of impact, as well as acceleration-deceleration forces.

Another example of combination head injuries are children who are victims of dog attacks. Children who suffer traumatic head and neck injuries from

a dog attack are subject to both the penetrating component of the bite as well as the blunt nature of the bite (which may represent the most devastating component of the head injury) (Calkins et al., 2001).

In addition to shearing forces, direct mechanical injuries may occur as the brain moves across the bony structures of the interior of the skull and the rigid dura mater (the outermost meningeal membrane enveloping the brain). The brain may be contused (bruised) and brain tissue and/or cerebral blood vessels may be lacerated (cut and mangled). Injuries of this type commonly occur along the sphenoid wing (a wedge-shaped bone) in the base of the skull and along the falx cerebri (the rigid sickle-shaped fold of the dura mater which extends down into the cleft between the right and left cerebral hemispheres). Bleeding from torn vessels can result in a hematoma (a pool of extravasated blood), which can elevate intracranial pressure (Evans and Wilberger, 1999) The resultant compression of the brain tissue leads to ischemic (lack of blood supply) and hypoxic (insufficient oxygen supply) brain damage and, if not relieved, permanent neuropsychological deficits or possibly death.

Intracranial pressure (ICP) also is raised when traumatic force is applied to the head. Any mechanism that creates elevated intracranial pressure can produce a herniation of the brain, which commonly occurs in the foramen magnum (the large opening in the base of the skull through which the spinal cord enters and joins the brainstem). Being the path of least resistance in the otherwise tightly closed cranium, the brainstem will be forced down into that opening as a means of relieving acutely elevated intracranial pressure. Since this region of the brain mediates vital functions such as respiration and blood pressure, herniation damage arising from transmission of shearing forces to the brainstem can produce a variety of systemic insults and possibly death (Evans and Wilberger, 1999).

[93.32] Pathophysiology

The primary pathologic feature of traumatic brain injury is axonal injury. Traumatic forces resulting from blunt or penetrating trauma or accelerating-decelerating mechanisms produce strains and distortions within the brain that disrupt axons (nerve fibers) and small blood vessels. As a

consequence, brain edema (swelling) occurs. Immediate life-threatening complications can result from this axonal disruption, with an alteration or loss of consciousness often being the first manifestation of the disruption. If cerebral impairment is severe, respiratory drive may be affected, and the patient may hypoventilate or become dyspneic (have difficulty breathing) or apneic (stop breathing). If not addressed immediately, cardiovascular function may also be compromised (Gedeit, 2001).

The number of axons injured and the degree of cerebral edema is directly proportional to the severity of the traumatic force. While less severe, axonal injury is usually reversible, whereas more severe injury may be permanent. These forces can also result in gross tearing of the veins, arteries, and dural sinuses, resulting in subdural hematoma, epidural hematoma, and contusion. Lacerations of the vasculature and associated edema can lead to a rapid rise in the intracranial pressure, resulting in compression and herniation of the brain. In turn, compression can squeeze off the cerebral blood vessels, producing ischemic (from lack of blood supply) and hypoxic (from inadequate oxygen supply) brain damage. Intravascular blood clots (thrombi), traumatic vascular spasms and various structural changes in the walls of the blood vessels themselves also can contribute to ischemic and hypoxic injury (Gedeit, 2001).

Primary brain injury from direct trauma is often complicated by secondary neuronal injuries resulting from disturbances in blood flow, capillary permeability, and inflammation. Rarely is structural injury to the brain at the time of impact the sole determinant of outcome. Traumatic brain injury starts a cascade of events leading to impaired metabolism, altered blood flow, and worsening cerebral swelling. Studies have shown that ischemia, abnormalities in glucose metabolism, and abnormal permeability of the blood-brain barrier are factors which increase brain edema (Gedeit, 2001). Inflammation, free radical formation, and influx of calcium can also cause cell damage that worsens edema. These responses initiate a downward spiral beginning with respiratory decompensation, hypotension and cardiovascular collapse that may lead to death. In fact, mortality is doubled when these secondary insults are superimposed on severe head injury (Gedeit, 2001; Evans and Wilberger, 1999).

P 93.40 MAJOR SEQUELAE OF HEAD INJURY

In addition to the aforementioned classifications, head injuries can be classified either as primary or secondary injuries, according to their appearance in the complex posttraumatic chain of events. In general, injuries that result directly from the trauma itself are referred to as primary injuries. The severity and location of the primary brain injury determines the patient's immediate level of consciousness and mental status. Currently, there is no treatment available for primary brain injury. (Savitsky and Votey, 2000; Zink, 2001)

Secondary brain injury occurs subsequent to the trauma causing primary brain injury, and is a result of a large number of interrelated pathophysiologic processes triggered by the primary injury. If left unchecked, these processes may lead to irreversible damage of brain tissue which was partially or totally unaffected by the initial traumatic event. For example, primary brain injury may lead to impaired autoregulation of cerebral blood flow, which in turn contributes to brain swelling. This swelling further exacerbates the injury cascade by causing compression of brain tissue, elevated intracranial pressure, and subsequent brain herniation and death. Because of its devastating consequences, the focus of most current and investigational brain injury therapies is aimed at minimizing secondary brain injury. (Savitsky and Votey, 2000; Zink, 2001)

There is some overlap between these two categories in the actual clinical situation. For example, although some of the vascular lesions (such as hematomas) may be delayed in their appearance or effects, they represent the evolution of a primary injury originating in the traumatic event, not a secondary complication. In addition, a major contribution to the secondary sequelae of head trauma comes from inadequate medical care. Although no specific therapies have proven effective in reversing the devastating consequences of primary brain injury, the prevention of secondary brain injury is possible with emergent resuscitation and acute stabilization of the severely head injured patient (Biros and Heegarrd, 2001).

[93.41] Primary Injuries

Head injury results in different types of primary

injury which occur at the moment of impact including lacerations of the scalp, skull fractures, cortical contusions and lacerations, diffuse axonal injury, and intracranial hemorrhage. Current data indicate that diffuse axonal injury may be the major form of primary brain injury in the posttraumatic persistent vegetative state (Kampfl, et al., 1998).

Types of intracranial hematomas include extradural, subarachnoid, subdural, and intracerebral (Evans and Wilberger, 1999) Other cerebral vascular lesions include cerebral aneurysm and arteriovenous fistula as well as meningeal tears.

[1] Concussion-- A concussion, or mild traumatic brain injury (MTBI) is defined as a clinical syndrome characterized by immediate impairment of neural function such as an alteration in consciousness, disturbance of vision or balance, and other symptoms due to brainstem involvement. More recently, the definition has been expanded to include any alteration in mental status resulting from minor head trauma that may or may not involve a loss of consciousness. A concussion can be accompanied by seizure, vomiting, confusion, headache or lethargy. It is important to understand what constitutes a concussion, as the diagnosis and management are based on this definition. It is also important to realize that if a concussion occurs as a result of traumatic head injury, its symptoms almost always occur immediately and may persist for variable lengths of time (e.g., seconds, minutes, hours, or days) (Amann, 2000; Gedeit, 2001).

Caution should be used when describing a concussion as a "head injury", as this implies injury to the cranial vault. Although an injury to the cranial vault often occurs simultaneously with concussion, it is more appropriate to refer to a concussion as a brain injury, because the neural tissue itself undergoes trauma and physiologic change. In general, patients who experience a concussion are categorized as having a mild impairment (score of 13 to 15) as measured by the Glasgow Coma Scale (GCS). However, the usefulness of this scale is somewhat limited in that many patients with a concussion may have a normal score of 15 and still present with signs and symptoms of mild traumatic brain injury (Evans and Wilberger, 1999; Amann, 2000).

[1a] Concussion Versus Contusion-- The force required to produce only a concussion without contusion and more severe injury cannot be precisely quantified (Amann, 2000). Hence, the popular notion that concussion is a trivial injury from which uneventful recovery always occurs is not valid. Severe concussion can result in prolonged loss of consciousness, headache, amnesia, irritability, depression, hyperacusis (heightened hearing acuity to the point of discomfort), motor weakness, inability to concentrate and personality changes. These symptoms may persist for minutes to months or more. In some cases, concussion can be fatal, such as when the disturbance of brain function results in prolonged apnea (cessation of breathing). Concussion may occur in isolation or in association with contusion, laceration and hemorrhage. In fact, there is a concussive component to all head trauma.

Concussion may result from blunt trauma to the torso or axial skeleton, with the force of the injury indirectly transmitted to the brain. Most concussions, however, occur when the momentum of the head is changed abruptly by either blunt trauma or acceleration-deceleration forces. In adults and adolescents, concussion usually results from motor vehicle accidents, falls, assaults, or sports-related activities. For example, the use of the head for controlling, passing and shooting a soccer ball, otherwise known as "heading" in soccer lingo, and player-to-player contact have reportedly caused concussions in a high percentage of professional soccer players (Barnes, et al., 1998). In children less than two years of age, concussions generally result from motor vehicle accidents and falls which impart a predominantly translational (linear) force to the head (Amann, 2000).

Diagnosis of concussion is clinical and treatment is supportive. Serious changes in cognitive ability following a traumatic event are the most frequent indication of minor brain injury, with impaired directed attention being the most common symptom. Changes in cognition are often subtle and the patient may appear easily distracted, impulsive and irritable. Early recognition of these symptoms leads to early intervention and improved outcomes (Brewer and Therrien, 2000).

[1b] Concussion in Children-- In the United

States, hundreds of thousands of children are treated in emergency departments of hospitals each year for head injury, resulting in 100,000 hospital admissions. Approximately 90 percent of these injuries are due to minor head injury, such as concussion (Savitsky and Votey, 2000). Falls and motor vehicle accidents are the most common causes of mild head injury in children, followed by pedestrian injuries, assaults, and bicycle injuries. There is also a significant gender difference among the pediatric population with regards to head injury, with boys having a higher incidence of traumatic head injury than girls (Savitsky and Votey, 2000).

Since minor closed head injury is one of the most frequent reasons for a physician visit, and consensus is lacking among the medical community as to how children with mild head injury should be treated, the American Academy of Pediatrics (AAP) developed a practice parameter for the management of minor closed head injury in children. According to these guidelines, children with minor closed head injury and no loss of consciousness should undergo a thorough physical and neurologic examination as well as observation for at least 24 hours. The use of cranial computed tomography (CT) scan, skull x-ray (radiograph), or magnetic resonance imaging (MRI) is not recommended for the child with minor closed head injury and no loss of consciousness (American Academy of Pediatrics, 1999).

For children with minor closed head injury and brief loss of consciousness (less than one minute), a thorough physical and neurologic exam should also be performed. Observation in the office, emergency department, in the hospital, or at home may also be used to evaluate children with mild head injury with brief loss of consciousness. In this scenario, cranial CT scan may be warranted, especially if the child exhibits signs and symptoms of brain injury, such as amnesia, lethargy, headache or vomiting at the time of evaluation. Some children with brain injury after mild head trauma do not exhibit any signs or symptoms. Since a delay in diagnosis of intracranial hemorrhage (the major life-threatening complication of mild head injury) increases morbidity and mortality, a CT scan should be obtained in the child with mild head injury with a loss of consciousness, posttraumatic amnesia, or signs and symptoms suggestive of brain injury, such as lethargy, altered mental status, headache,

vomiting or seizure (American Academy of Pediatrics, 1999).

[2] Contusion-- Contusion is produced by forces greater than those that cause concussion. More specifically, a contusion is a bruise on the surface of the brain which occurs when the brain is directly injured (such as in blunt trauma). Areas of focal cortical injury can result from either direct trauma from external contact forces or from contact of the brain with intracranial surfaces with acceleration/deceleration forces (Gedeit, 2001).

The extent of damage from a contusion ranges from mild to severe. Mild contusion may produce localized subpial hemorrhage (bleeding into the space beneath the highly vascularized pia mater, the innermost of the three meningeal membranes). In moderate to severe contusion there is typically more diffuse structural damage, pulping of the brain and frank bleeding and seepage of blood into the cerebrospinal fluid (CSF) space.

Contusions are produced when blood vessels in the parenchyma (underlying brain tissue) are damaged, resulting in areas of petechial hemorrhage and subsequent edema (swelling of the brain due to accumulation of fluid). Contusions develop in the gray matter located on the surface of the brain and taper into the white matter. Blood from the subarachnoid space is often found overlying the involved gyrus (fold on the surface of the brain). Over time the associated hemorrhages and edema of a contusion become widespread and can serve as a nidus (source or focal point) for hemorrhage or swelling, producing a local mass effect. Compression of the underlying tissue can result in areas of ischemia (lack of blood supply to a region) and tissue infarction (obstruction of the blood vessels), if the compression is significant and unrelieved. Eventually, these areas of ischemia become necrotic and cystic cavities form within them (Marx, 2002).

[2a] Site and Mechanism-- The clinical effects and significance of a contusion depend on the region of the brain involved, which in turn depends on the site of trauma and the mechanism involved. By definition, a coup contusion occurs at the site of impact in the absence of a fracture. In contrast, a contrecoup contusion occurs in the brain diametrically opposite the point of impact. Contusions which occur in areas of the cerebral cortex in between, or in the vicinity of the tentorium cerebellum or falx cerebri (portion of the dura

mater in the area of the brainstem and between the two cerebral hemispheres, respectively) are called intermediate coup.

Coup lesions are more common following concussive-compressive injuries (where the head is struck), while contrecoup lesions are more common following acceleration-deceleration injuries (when the head strikes an immovable object).

Temporal lobe contusions often are associated with fractures of the skull and manifest behavioral symptoms that include lethargy, restlessness and/or belligerence. Neurological manifestations include unilateral motor weakness (hemiparesis) and aphasia (disturbance of the power of expression and/or comprehension of speech) when the dominant frontal lobe is involved.

Temporal lobe contusion is not life-threatening unless there is herniation with progressive compression of the brainstem. If neurological deterioration is in progress, it will be most evident on the third day following injury. If the cause of the deterioration is in doubt, a CT scan is indicated to identify the possible formation of a hematoma.

Contusion in the parietotemporal region is associated with visual disturbances, such as homonymous hemianopsia (blindness or defective vision in the right or left halves of both eyes); unilateral motor weakness (hemiparesis) or paralysis (hemiplegia); and disorders of expression or comprehension of language, or both, which may be mistaken for a reduced level of consciousness.

Significant bilateral frontal lobe contusion is associated with reduced consciousness or changes in personality. Contusion of the hypothalamus typically results in coma. Contusion of the motor neurons of the frontal cortex may produce contralateral motor weakness.

Patients with contusion are often delayed in their presentation of signs and symptoms. Although they may have sustained a loss of consciousness for a brief period of time, the duration of symptoms such as confusion and obtundation (altered mental status) is usually prolonged. Most patients with significant contusions have full recoveries, but contusions can also cause significant neurologic impairment, including increased intracranial pressure, seizures, and focal deficits (Marx, 2002).

Surgery for accessible lesions that are greater than 25 milliliters may improve the patient's clinical outcome. Medical measures to control intracranial hypertension (elevated blood pressure within the cranial vault) should also be instituted (Morris and Marchall, 2000).

[3] Laceration-- Laceration is defined as a ragged, tearing wound. It is generally a more distinct lesion than a contusion, and involves direct mechanical damage to the meningeal membranes, the cerebral vasculature and individual neurons. Laceration is associated with less pulping of the brain than contusion, and the pattern of clinically manifest neurological symptoms that arise from it varies with the area of the brain involved.

Laceration can occur in almost any head injury scenario. For example, in severe penetrating head injury, laceration is caused by the projectile and in-driven bone fragments. In blunt traumatic injury of sufficient force to produce a depressed fracture of the skull, laceration results from in-driven bone fragments. In acceleration-deceleration injuries, laceration may be caused by shearing forces and mass movement of the brain over the bony interior of the skull and the rigid falx cerebri of the dura mater.

Scalp lacerations frequently occur after head injury and are a source of significant bleeding because hemostasis is difficult to achieve. Lacerating lesions produce the massive intracranial bleeding that leads to hematoma formation. Stretching and tearing of the axons (the neuronal processes which conduct the nerve impulse) may occur even in the absence of hemorrhage. The resultant disturbance of nerve conduction produces widespread neurological dysfunction, which may be a critical factor in the abrupt rise in intracranial pressure that causes unconsciousness or coma in cases of extensive cerebral contusions.

Methods to control bleeding from a scalp laceration include direct digital compression of the bleeding vessel against the skull, infiltration of the wound edges with a mixture of lidocaine and epinephrine, or ligation of the identified bleeding vessels. If a deep laceration occurs (e.g., the galea is lacerated), surgical techniques are employed to close the wound, after proper debridement and irrigation, and are the most effective methods to stop the bleeding and prevent tissue crush injury should compression be applied for too long a period.

Once hemostasis is achieved, the wound should be irrigated to wash away any debris. Since the peripheral vessels of the scalp drain into the veins of the skull, which in turn drain into the venous sinuses, contaminated scalp wounds have the potential to cause serious intracranial infection. Hence, irrigation of the wound to rinse away debris is extremely important (Marx, 2002).

[4] Vascular Lesions-- The forces that act on the intracranial contents during closed head trauma can produce various types of damage to the intracranial blood vessels. Prominent among these are tearing with resultant hematoma, thrombosis (clotting of a vessel) and infarction; aneurysm; and arteriovenous fistula. Meningeal arteries are injured most commonly in head trauma, but any portion of the cephalic vascular tree may be damaged (Goetz, 1999; Marx, 2002).

[4a] Hematoma-- A hematoma is a blood clot formed by blood extravasated from a disrupted vessel. Hematomas are space-occupying lesions that sometimes grow large enough to displace and locally compress the brain and cause life-threatening ischemic (marked by lack of blood supply) damage (Townsend, 2001).

There are three major kinds of hematoma: intracerebral, subdural and epidural. Intracerebral hematomas occur within the brain, subdural and epidural hematomas occur outside the brain.

An intracerebral hematoma is a clot of extravasated blood which forms deep within the brain tissue. Intracerebral hematomas may form anywhere within the brain tissue, but are found in the frontal and temporal lobes in approximately 85 percent of cases (Marx, 2002). This type of hematoma is caused by shearing or tensile forces that mechanically stretch and tear deep smaller arterioles as the brain is propelled against irregular surfaces in the cranial vault. Small petechial hemorrhages that result from this trauma subsequently coalesce to form intracerebral hematomas. They are frequently associated with extraaxial hematomas, and multiple intracerebral hematomas are often present in many patients. Isolated intracerebral hematomas can be detected in about 12 percent of all patients experiencing severe head trauma.

The clinical effects of intracerebral hematoma depend on the size and location of the hematoma, and whether or not bleeding has been contained. Intracerebral hematomas have been reported with varying degrees of severity of head trauma, with more than 50 percent of patients experiencing loss of consciousness at the time of impact. The level of consciousness of the patient correlates with the severity of the impact and the presence of any coexisting lesions. If a contusion or other concurrent lesion is present, an intracerebral hematoma can produce substantial mass effects and cause a herniation syndrome.

Diagnosis is by CT, since cerebral angiography cannot differentiate intracerebral hematoma from a localized contusion. Many patients with an intracerebral hematoma require emergent medical intervention or surgery to control elevated intracranial pressure. Mortality is low in patients who are conscious before surgery. This is not the case, however, in unconscious patients, in whom mortality approaches 45 percent. Intracerebral hematomas which bleed into the ventricles of the brain or the cerebellum are also associated with a high mortality rate (Marx, 2002).

A subdural hematoma is a clot of extravasated blood which forms between the dura (the meningeal covering of the brain) and the brain. A subdural hematoma results from bleeding from veins after blunt head trauma (acceleration-deceleration injuries), which precipitates movement of the brain within the skull and the shearing off of veins bridging the surface of the brain with the adjacent dural venous sinuses. The blood leaks slowly from the veins, forming a hematoma in the subdural space. A subdural hematoma may be reabsorbed spontaneously or may form an encapsulated hematoma. After approximately two weeks, membranes form around the hematoma and the center of the encapsulated hematoma liquefies due to continuous bleeding from the vascular membrane. Subdural hematomas are more common than epidural hematomas and occur in approximately 30 percent of patients with severe head trauma (Goetz, 1999, Marx, 2002).

Subdural hematomas are classified according to the time signs and symptoms appear. Patients with acute subdural hematomas are symptomatic within 24 hours after the initial traumatic event. They often have a decreased level of consciousness

followed by declining mental status. Patients with a subacute subdural hematoma are symptomatic between 24 hours and two weeks after head injury. These patients usually complain of headache, changes in mental functioning, muscle weakness, or frank paralysis. Chronic subdural hematoma is defined as the appearance of symptoms two or more weeks after trauma. Patients with chronic subdural hematoma may experience a very subtle onset of symptoms or nonspecific symptoms. However, approximately 45 percent of patients will complain of unilateral weakness (weakness on one side of the body) or hemiparesis (paralysis on one side of the body). Approximately 50 percent of patients will also present with an altered level of consciousness, with some patients unable to recall their head injury or describing only a minor injury (Goetz, 1999; Marx, 2002).

The diagnostic method of choice is computed tomography (CT). If a subdural hematoma is not diagnosed early and is left untreated, it may result in severe neurologic impairment or death. Treatment is by surgical evacuation of the hematoma (Goetz, 1999; Townsend, 2001).

Most patients have a good prognosis following surgery. In general, the prognosis is related to the degree of associated brain injury caused by the pressure of the expanding hematoma on underlying tissue. The overall survival rate is 35 percent to 50 percent, with mortality being highest in the elderly, in patients having a measurement of eight or less on the Glasgow Coma Scale, and in patients who present with signs of acute herniation syndrome in the Emergency Department. Overall, the mortality associated with a chronic subdural hematoma approaches 10 percent, with survival decreasing in elderly patients (Marx, 2002).

An epidural hematoma develops when blood collects between the skull and the dura mater (the outermost of the three meningeal membranes that cover the brain) as a result of blunt trauma to the head. Epidural hematomas most commonly occur in the temporal region of the brain, although they may occur in other locations such as the frontal or parietal regions. They usually develop in patients with severe head trauma causing a fracture of the temporal bone and tearing the middle meningeal artery. In 85 percent of cases, epidural hematoma is associated with a skull fracture (Marx, 2002; Goetz, 1999; Evans and Wilberger, 1999).

The classic presentation of epidural hematoma is a decreased level of consciousness followed by a lucid interval (Goetz, 1999; Marx, 2002). There is often a progressive reduction in the level of consciousness as the hematoma enlarges. This lucid interval, however, is rarely seen as most patients are unconscious from the time of injury (Evans and Wilberger, 1999). Only about 30 percent of patients with epidural hematomas follow this classical presentation (Marx, 2002). Most patients with an epidural hematoma complain of a severe headache, increased sleepiness, dizziness, nausea and vomiting. Signs and symptoms develop according to how rapidly the hematoma is expanding. In some instances, a patient with a small epidural hematoma may be asymptomatic, but this is usually rare (Marx, 2002).

Definitive diagnosis is by CT scan. Epidural hematomas require immediate surgical evacuation (Evans and Wilberger, 1999; Goetz, 1999). If the patient's condition appears to be rapidly decompensating, the patient should be taken directly to the operating room for a procedure that is both diagnostic and therapeutic (Goetz, 1999). For a patient who is not in a coma at the time of diagnosis, who receives rapid treatment, the mortality rate approximates zero percent. The mortality rate increases to about 20 percent if the patient is in a coma at the time of diagnosis (Marx, 2002).

The prognosis is excellent when epidural hematomas are rapidly diagnosed and treated surgically. Conversely, severe neurological deficits or even death can result from delayed diagnosis and treatment (Goetz, 1999).

Hematomas may also form in the posterior fossa (the bilateral depression in the posterior floor of the skull) following a wide variety of head injuries. Posterior fossa hematomas are very rare, constituting less than 1 percent of all reported subdural hematomas. Patients usually present with symptoms such as nausea, vomiting, headache, and a decreased level of consciousness. The mass of the lesion can compress the brainstem, resulting in derangement of a number of vital respiratory and cardiovascular functions mediated by that part of the brain. Neurological deterioration of the patient can occur rapidly and without obvious clinical signs. Treatment is by surgical decompression and evacuation of the hematoma. The prognosis is poor, with less than a 5 percent

survival rate (Marx, 2002).

[4b] Thrombosis and Infarction-- In addition to more massive hematomas, smaller blood clots (thrombi) may form within cerebral blood vessels. When these clots occlude the flow of blood to the region of the brain supplied by the affected vessels, they can produce local ischemic and hypoxic damage (infarction) which in turn results in focal neurologic deficits. These lesions may not be apparent for several hours or days, and usually do not produce marked alterations in mental status (Goetz, 1999).

Although carotid dissection (dissection of the main artery supplying blood to the brain) rarely occurs with head injury, it can be the cause of delayed deterioration, particularly when neck injury is involved (Townsend, 2001). In head injured patients, traumatic dissection of the carotid artery is the most frequent cause of infarction, whereas thrombosis of the cerebral artery is extremely rare. An unusual case of posttraumatic thrombosis of the middle cerebral artery has been reported in the literature, in which a teenager suffering blows to the head and face experienced a partial rupture in the arterial wall and subsequent thrombosis and infarction (Bunai, 2001). When either carotid dissection or thrombosis of the middle cerebral artery is suspected, the diagnosis is made by angiography (Townsend, 2001).

Ischemic thalamic infarctions and strokes have also been reported in children suffering head trauma. The typical clinical presentation of stroke in the pediatric patient (ranging 21 months to 15 years of age in this series) involves a decreased level of consciousness, hemiparesis (paralysis on one side of the body), and aphasia (difficulty in speech). Any child suffering blunt head trauma presenting with these symptoms should be suspected of thrombosis of the cerebral artery and ischemic thalamic infarction, and be evaluated as such (Garg and De Myer, 1995).

Venous sinus thrombosis is rarely associated with head trauma, but can easily be an undetected sequel to head injury. One case report in the literature describes an adult patient with unilateral hearing loss, tinnitus (ringing in the ears) and headache two days after suffering a closed head injury. CT scan revealed a skull fracture and MRI demonstrated sigmoid and transverse sinus thrombosis (Brors et al., 2001). If the diagnosis of

venous sinus thrombosis is suspected, cerebral angiography should be performed promptly and the instillation of urokinase should be considered in any patient with symptomatic occlusion (D'Alise, et al., 1998).

In most cases, infarction is caused by thrombotic occlusion of a vessel supplying blood to the brain, but also may be caused by traumatic vascular compression from a swelling brain. Vessels may be compressed in the cerebral sulci (furrows of the surface convolutions) between edematous gyri (elevated ridges of the surface convolutions), or between the swelling hemispheres of the brain and the skull (Goetz, 1999).

[4c] Aneurysm-- Traumatic intracranial aneurysm (a balloon-like sac in the wall of a blood vessel due to weakening and bulging of the wall) is reportedly a rare event, occurring in less than 1 percent of cases (Tureyen, 2001). This type of aneurysm usually develops after penetrating head trauma or skull fracture, but can occur after blunt trauma as well. When aneurysms rupture, they usually cause devastating subarachnoid or intracerebral hemorrhage. At least three cases of posterior fossa subarachnoid hemorrhage from ruptured cerebellar artery aneurysms have been reported after blunt head trauma (Schuster, et al., 1999).

Although traumatic aneurysms are most common in young adults, the frequency in the pediatric population is relatively high. One case report in the literature describes a posttraumatic aneurysm in a child after falling from a significant height which subsequently spontaneously thrombosed (Loevner, et al., 1998). Another case report describes a traumatic posterior inferior cerebellar artery (PICA) aneurysm not related to skull fracture or penetrating or blunt trauma, but resulting instead from a meningeal tear due to deceleration forces (Sure, et al., 1999).

Approximately 85 percent of ruptures occur within the first three weeks after the trauma (Voelker and Ortiz, 1997). Thus, a high index of suspicion is necessary to promptly diagnose a traumatic aneurysm. When a patient suffers a penetrating head injury or fracture at the base of the skull near major arteries, or exhibits symptoms suggestive of an aneurysm (e.g. a throbbing headache that is persistent) after blunt trauma, an arteriogram is warranted. Treatment is by surgical clipping of the aneurysm (Townsend, 2001). When left untreated,

the mortality rate is between 30 and 70 percent (Tureyen, 2001).

[4d] Arteriovenous Fistula-- An arteriovenous fistula is an abnormal communication between an artery and a vein. The most common site of a fistula following cranial trauma is between the carotid artery and the cavernous sinus (an irregular venous space in the dura mater on either side of the sphenoid bone). Usually, the fistula forms following laceration of the carotid vessel during basilar skull fracture, and occurs in the presence of concomitant damage to the nerves that accompany the carotid artery through this region (namely, the oculomotor, trochlear, abducens, and first division of the trigeminal nerve). Traumatic injury of the subclavian artery can also result in fistula formation between the right subclavian vein and internal jugular vein (Maher et al., 1997). Posttraumatic arteriovenous fistula also may occur in the scalp vasculature.

Traumatic carotid-cavernous fistulas are associated with a large variety of symptoms, including loss of vision, glaucoma, chemosis (excessive swelling of the conjunctival membrane of the eye), exophthalmos (bulging eyeballs), and orbital nerve palsies. In addition, the high pressure in the veins that drain the brain can cause vessels to enlarge and compress adjacent structures, producing neurologic deficits (Wadlington and Terry, 1999). Symptoms usually appear within one month after the traumatic event, but severe head injury may lead to asymptomatic arteriovenous fistula formation after a long period of time. One case report describes a 38-year-old woman who developed an arteriovenous fistula after suffering head injury at the age of three (Fukai, et al., 2001) If a false aneurysm develops as a result of the fistula, this is considered a surgical emergency, requiring early diagnosis and prompt surgical intervention (Maher, et al., 1997).

In diagnosis of the fistula, Doppler ultrasound may be useful because it reveals the vein-arterial blood flow on the fistula side. CT scan and magnetic resonance imaging (MRI) may also suggest the presence of a fistula. The diagnosis is confirmed by arteriography of the involved artery (usually the common carotid artery, although the subclavian or posterior communicating artery may be the primary artery) (Dowzenko et al., 1997). Surgical occlusion of the abnormal communication is the mode of therapy for fistulas (Fukaie et al., 2001; Wadlington

and Terry, 1999).

[5] Meningeal Tears-- The meninges, or three membrane layers that envelop the brain and spinal cord, are the dura mater (outermost), arachnoid (middle) and pia mater (innermost). Cerebrospinal fluid (CSF) circulates within the spaces between these layers. They serve both as a mechanical shock absorber for the brain and a physical barrier against infection.

When the blood vessels which run through the meninges and into the brain are torn, the leakage of blood gives rise to a hematoma. When the meningeal membranes themselves are torn or punctured, leakage of cerebrospinal fluid may form a CSF cyst (fluid-filled sac) in the arachnoid space. Leakage of CSF following head trauma is difficult to diagnose and is of considerable importance as it may cause fistula formation and meningitis. In one study, patients with head injuries and subdural hematoma were found to be at greater risk of developing an unobserved dural tear, with delayed leakage of CSF (Choi and Spann, 1996).

In children, the dura mater may be torn by a linear fracture. The resultant traumatic pulsing of the underlying arachnoid membrane can enlarge the fracture, producing a leptomenigeal cyst (a CSF cyst involving the arachnoid and pia mater). If there is a fracture of the thin bones of the temporal area or of the floor of the skull in the region of the eyes and nasal cavity, CSF may leak from the ears (otorrhea) or nose (rhinorrhea), respectively. Such an injury indicates a communication between the contents of the skull and the outside, which increases the risk of intracranial infection (e.g., meningitis). Any child with serosanguinous (containing CSF fluid) bleeding from the nose or ear should be suspected of having a basilar skull fracture with resultant CSF leakage, and should be followed closely until the dural tear heals or is surgically repaired because of the high risk for developing meningitis (Savitsky and Votey, 2000).

[93.42] Secondary Injuries

Secondary injuries occur subsequent to the primary injury resulting from head trauma and are almost always preventable and treatable. Among the prominent secondary sequelae of head trauma are cerebral edema (brain swelling) and elevated intracranial pressure (ICP), various brain herniation syndromes, cardiorespiratory

complications, spasm of the cerebral vessels, clotting defects (coagulopathies), infection and posttraumatic seizures. Common adverse effects of these secondary complications of cerebral function include ischemia (lack of blood supply) and hypoxia (insufficient oxygen supply).

The presence of hypoxia or shock is associated with a poor outcome, independent of the injury severity. Both occur frequently, not only in the pre-hospital phase but also in patients in intensive care settings. If present, the correction of shock and hypoxia is the first priority of treatment, and any head-injured patient exhibiting signs of poor ventilation should be urgently placed on mechanical ventilation (Townsend, 2001; Zink, 2001).

[1] Elevated Intracranial Pressure-- The skull contains three intracranial components: the brain (80 percent of the volume), blood (10 percent of the volume) and cerebrospinal fluid (10 percent of the volume). Because the skull interior is an effectively closed and rigid cavity, its contents have a specific intracranial pressure (ICP). Normal ICP is 15 mm of mercury (Hg) or less, and is regulated under normal circumstances by compensatory changes in the volume of the three intracranial components.

Following head trauma, the normal balance is disturbed: the limits of the compensatory mechanism are exceeded by the onset of brain swelling or mass lesions such as hematomas caused by intracranial hemorrhaging and pooling of blood. As the traumatic edema or mass lesion increases in size, the brain and its vasculature become increasingly compressed within the unyielding skull, resulting in ischemic (lack of blood supply) and hypoxic (inadequate oxygen supply) brain damage, brain herniation and related insults. Elevated intracranial pressure thus contributes significantly to morbidity and mortality following head trauma.

Evaluation of the severely head injured patient during the acute period following the trauma involves rigorous neurologic assessment as well as frequent monitoring of the patient's intracranial pressure. Clinical signs of rising ICP which may be present in conscious patients include alterations in the level of consciousness, changes in vital signs (blood pressure, pulse and respiration) and papilledema (swelling of the optic disc in the retina of the eye). There are no reliable clinical signs in

comatose patients (Townsend, 2001).

Monitoring of the intracranial pressure is usually initiated after the first CT scan in patients not requiring urgent evacuation of a mass lesion. If the patient is taken to the operating room for urgent surgery, an ICP monitor is usually placed at the end of the operation, since the brain may swell later on as a complication of surgery. In some hospitals, young patients who are comatose, with high Glasgow Coma Scores (e.g., seven or eight) and normal CT scans may not be monitored immediately. In other hospitals, all comatose patients are monitored, regardless of the findings on CT scan. Although the risk of increased intracranial pressure in a comatose patient with a normal CT scan is lower than that of a patient with an abnormal CT scan (having an estimated risk of 10 to 15 percent), it is higher than the risk of inserting an ICP monitor (Townsend, 2001).

Treatment of elevated ICP involves drainage of ventricular fluid by placement of a ventricular catheter or the use of osmotic agents such as mannitol (Townsend, 2001).

[2] Brain Herniation-- Brain herniation syndromes consist of compression and shifting of the brain within the skull secondary to increased intracranial pressure. Increased intracranial pressure may be the result of brain swelling, cerebral edema, expansion of a mass lesion, or any combination of these three factors. During herniation, the brain is forced over the rigid structures of the dura mater (the tough, outermost meningeal membrane) or skull interior. The brain also may be forced down into and impacted in the foramen magnum (the large opening at the base of the skull where the spinal cord enters and fuses with the brain), the only outlet in the otherwise closed skull.

Herniation of the brain is also the most serious complication of lumbar puncture. In a head trauma patient with an intracranial mass lesion, a lumbar puncture can create a pressure gradient between the brain and spinal cord spaces, leading to a downward herniation of the brain through the foramen magnum or the tentorial notch. This results in further neurologic deterioration and death (Naik-Tolani et al., 1999).

Herniation syndromes are divided into four types: uncal, central, transfalcal and tonsillar. Herniation can occur within several minutes or days after

traumatic brain injury. Once the patient exhibits signs of herniation syndrome, mortality approaches 100 percent if temporary emergency measures and neurosurgical treatment are not rapidly implemented (Marx, 2002). Definitive diagnosis of tentorial (pertaining to the dura mater) brain herniations is by cerebral angiography, although special techniques are available to visualize them on CT, MRI and MR angiography. Treatment is by surgical decompression.

[2a] Uncal Herniations-- The uncus (the lower medial portion of the temporal lobe of the cerebrum) rests on a portion of the tentorium cerebelli (the tentlike portion of the dura which covers the cerebellum). The tentorium separates the posterior fossa from the middle cranial fossa. In this region, the brainstem rises through the tentorium by passing through an opening between the tentorium and the sphenoid bone of the skull base (the tentorial incisura). Below this, the brainstem joins with the spinal cord as it passes through the foramen magnum.

Rising intracranial pressure due to an evolving mass lesion can force the uncus over the edge of the tentorial incisura and into the posterior fossa, resulting in uncal herniation. This is the most common herniation syndrome and is a form of transtentorial herniation. Uncal herniation is usually associated with epidural or acute subdural hematomas located in the lateral middle fossa or the temporal lobe of the brain.

Initially, as the uncus herniates, it compresses the third cranial nerve (the oculomotor nerve, which innervates various muscles of the eye). The resulting pressure produces a characteristic constellation of clinical signs, including ptosis (drooping of the eyelid), a sluggish pupillary response to light and impaired extraocular movements. This phase may last from minutes to hours, depending on how rapidly the hematoma is expanding.

As the herniation progresses, contralateral hemiparesis (paralysis on the opposite side of the brain) develops and eventually, bilateral decerebrate posturing occurs.

Direct brainstem compression causes further alterations in the patient's level of consciousness, respiration and cardiovascular function. Mental status changes may be subtle in the initial stages,

and the patient will appear agitated, restless, or confused. Lethargy soon occurs, however, with progression to frank coma. Continued progression of a temporal lobe herniation and compression of the brainstem may lead to rapid changes in the patient's blood pressure and cardiac conduction, and ultimately cardiorespiratory arrest and death (Marx, 2002).

[2b] Central Transtentorial Herniations-- The central transtentorial herniation syndrome is caused by an expanding lesion at the vertex or the frontal or occipital lobes of the brain. This syndrome is less common than uncal herniation. Neurologic and clinical deterioration occurs as central pressure is exerted bilaterally on the brain from above. Initially, the patient may present with a subtle change in mental status or decreased level of consciousness, muscle weakness and pinpoint pupils. As central herniation progresses, both pupils become midposition and lose their responsiveness to light. The patient's respiratory pattern is also affected and he or she may begin to hyperventilate. The patient may begin to yawn or sigh initially, then develop tachypnea (rapid and quick breaths), followed by a pattern of shallow, slow breaths at irregular intervals before collapsing from respiratory arrest (Marx, 2002).

[2c] Transfalcine Herniations-- These lesions are produced by a mass effect on one side of the brain which forces the medial aspect of that cerebral hemisphere beneath the rigid falx cerebri (the ridge of the dura extending down into the cleft between the hemispheres). There is damage to the cingulate gyrus (a key part of the limbic system, which mediates emotions and various regulatory functions), and compression of the cerebral arteries leads to ischemic damage to the involved hemisphere. A vicious cycle of edema ensues, and may combine with the mass effect to produce uncal herniation. Clinically, the patient often presents with an altered level of consciousness and profound weakness of the lower extremities.

[2d] Cerebellotonsillar Herniations-- Herniation of the tonsils (lowest pole) of the cerebellum involves a shift of the brainstem and cerebellum toward the foramen magnum due to an evolving mass effect (e.g., hematoma) in the posterior fossa. In the process, the medulla oblongata (a crucial regulator of respiration and other vital functions) is compressed, and depressed

consciousness and respiratory arrest ensue. Pinpoint pupils and flaccid quadriplegia are characteristic signs and symptoms of this type of herniation. Mortality from cerebellar herniation is high, approaching 70 percent (Marx, 2002).

[3] Cardiorespiratory Complications-- A number of respiratory injuries and complications may be seen following head injury (Marx, 2002). Since the major respiratory centers are in the brainstem, most respiratory problems arise following acceleration-deceleration injuries (in which the brainstem is damaged as a result of mass movement and shearing forces).

[3a] Respiratory Problems-- The types of respiratory sequelae that must be addressed at the scene of the trauma and in the emergency department differ somewhat from complications that present in the intensive care unit (ICU). Common complications during the acute stage of trauma include aspiration pneumonia, apnea (cessation of breathing), and neurogenic (caused by nerve dysfunction) pulmonary edema (fluid in the lungs).

Suppression of the gag and cough reflexes combined with the increased possibility of vomiting following head trauma can result in aspiration of the stomach contents into the lungs and the onset of aspiration pneumonia (infection of the lungs by aspirates). Treatment is by organism-specific antibiotics.

Apnea is potentially the most disastrous sequela and may be the leading cause of death at the scene (Marx, 2002). Although patients have made good recoveries after as long as 8 to 10 minutes of prolonged apnea, the accumulating hypoxemia (deficient oxygen levels in the blood) and hypercarbia (excessive levels of carbon dioxide in the blood) that occur before the respiratory centers resume spontaneous functioning frequently lead to vegetative or even fatal outcomes (Marx, 2002). Treatment is by emergency ventilation.

Apnea usually results from compression of the brainstem or injury after trauma and is one of the most common causes of hypoxia. Hypoxia, defined as an oxygen saturation of less than 60 mm Hg, is a common respiratory complication in head injured patients. In addition to apnea, causes of hypoxia include partial obstruction of the patient's airway caused by blood or debris, injury to the chest wall

and penetrating injury to the lungs. It is difficult to estimate the exact incidence of hypoxia in patients with head trauma, since hypoxia often remains unnoticed in the out-of-hospital setting. When the presence of hypoxia is documented, however, the mortality due to severe head injury doubles (Marx, 2002).

Neurogenic pulmonary edema is defined as pulmonary edema resulting from an insult to the central nervous system that occurs in the absence of cardiac or other disease. This phenomenon has been well documented in the literature for almost a century, and was first described in patients with epilepsy, gunshot wounds to the head, and traumatic intracranial bleeds. More recently, neurogenic pulmonary edema has been reported in children and adults with seizures, closed head injury, intracranial hemorrhage, and penetrating head trauma. At least two case reports describe neurogenic pulmonary edema in young children with inflicted head injuries.

The classic presentation of neurogenic pulmonary edema is one of acute onset, developing instantaneously within minutes to hours after head trauma. Infrequently, a delayed-onset of pulmonary edema can occur hours to days after the precipitating event. Recent research suggests that subclinical pulmonary edema may develop in patients with a normal appearing chest x-ray. In fact, many head-injured patients with an elevated ICP exhibit signs of pulmonary dysfunction long before they develop overt pulmonary edema. Although the exact mechanism of neurogenic pulmonary edema remains unknown, this noncardiac pulmonary edema probably results from changes in hydrostatic forces and capillary permeability directly caused by brain injury. Lowering the ICP appears to reverse the neurogenic cause of the edema (Marx, 2002; Rubin et al., 2001).

Signs and symptoms of neurogenic pulmonary edema include tachypnea (rapid and shallow breathing), dyspnea (difficulty in breathing), chest pain, rales (a wet, bubbly sound characteristic of fluid upon listening to the lungs through a stethoscope), and frothy pink pulmonary secretions. If the head injury is severe, pulmonary edema can be massive, pouring out of the mouth and nose of the patient. Chest x-ray (radiograph) confirms the diagnosis and demonstrates diffuse, butterfly shaped pulmonary infiltrates (Rubin, et al.,

2001).

Common respiratory complications seen in the ICU include hypoxia, atelectasis, bacterial pneumonia, barotrauma, adult respiratory distress syndrome (ARDS) and cardiogenic (caused by cardiac dysfunction) pulmonary edema. Agitation frequently occurs in the head-injured patient in the ICU secondary to hypoxia. The agitated patient will often exhibit continuous movement, characterized by fidgeting or moving from side to side, appear disoriented, and may attempt to remove intravenous catheters. Hypoxemia frequently contributes to agitation and in the ICUs of most hospitals there have been numerous incidences in which hypoxemia has been misdiagnosed as agitation. Hypotension (low blood pressure) has also been linked to agitation and is considered a form of brain injury as a result of hypoperfusion (an inadequate supply of blood and oxygen to the brain) (Critical Care Med, 2002).

Atelectasis (lung collapse) is common following severe head injury and results from a number of disturbances of normal lung function. It can be reversed by various means, including administration of bronchodilator aerosols or bronchoscopic removal of mucous plugs and secretions.

Aspiration can be prevented by proper endotracheal intubation, even if mechanical ventilation is not necessary. There is always some possibility of aspiration, even with the best available devices, so routine nasogastric and oropharyngeal suctioning of collected secretions is a necessary adjunct. However, endotracheal suctioning of head-injured patients can result in significant increases in ICP. Sedation and the use of short suctioning times may aid in preventing this complication (Naik-Tolani et al., 1999).

Of the five types of barotrauma (injury due to positive pressure in the airways as a result of mechanical ventilation), only pneumothorax (air in the chest cavity) normally requires intervention (e.g., a thoracostomy tube).

Cardiogenic pulmonary edema (fluid collection and swelling in the lungs) is seen in patients with preexisting heart disease who have received posttraumatic fluid volume replacement therapy or suffered acute kidney failure. In patients with good kidney function, simple diuretics usually resolve the problem. In more critically ill patients,

intravascular volume reduction may be necessary. In patients with poor heart and kidney function, administration of a diuretic like mannitol dramatically reduces brain swelling, but also dramatically increases intravascular volume.

Adult respiratory distress syndrome (ARDS, one form of which is neurogenic pulmonary edema) is characterized by diffuse bilateral lung infiltrates and hypoxia that responds poorly to supplemental oxygen. It usually is self-limiting but can be fatal. Treatment is nonspecific, and entails supportive care with supplemental oxygenation. Specific treatment is directed at the underlying traumatic cause (e.g., multiple injuries or infection).

[3b] Cardiovascular Problems-- Neurogenic hypertension as well as a wide variety of cardiac rhythm abnormalities occur after head injury (Goetz, 1999; Marx, 2002). Cardiac dysfunction can potentially be life-threatening and requires aggressive management. Since the brain is acutely dependent on oxygen, an adequate cardiac output (volume of blood pumped out of the heart upon contraction) is necessary to ensure adequate cerebral perfusion. In head-injured patients, brain injury is a primary cause of cardiac dysfunction. Abnormal cardiac rhythms (dysrhythmias) occur in up to 70 percent of patients with subarachnoid hemorrhage and in more than 50 percent of patients with intracranial hemorrhage.

Although supraventricular tachycardia is the most common cardiac dysrhythmia observed after head injury, many other rhythms have been reported. The electrocardiogram (ECG) reveals large, upright or inverted T waves, prolonged QT intervals, ST segment or depression, and the presence of U waves. The primary goal of emergency treatment of cardiac dysrhythmias after head injury is to maintain adequate tissue perfusion and to avoid hypoxia. Dysrhythmias will often disappear as the ICP is reduced. Standard advanced cardiac life support (ACLS) should be followed after resolution of the dysrhythmia, since cardiac injury may also be present in patients experiencing multiple trauma (Marx, 2002).

[4] Cerebral Vasospasm-- Spasm (involuntary cycles of contraction and relaxation) of the smooth muscles of the walls of the cerebral arteries occur in 30 to 40 percent of patients with posttraumatic bleeding into the subarachnoid space (the space

beneath the innermost of the three meningeal membranes) (Zubkov, 1999). A recent study found that the development of posttraumatic vasospasm was correlated with severe subarachnoid hemorrhage detected on the initial CT scan (Zubkov, 2000). Vasospasm also may be seen in the carotid and vertebral arteries of the neck following head injury.

Neither the etiology nor the pathogenic mechanism of vasospasm are understood as yet. The traumatic release of serotonin (which induces smooth muscle contractions) from certain of the white blood cells which produce and store it is thought to play a role. When spasm chokes off cerebral vessels, cerebral ischemia (lack of blood supply) results. Eventually, this leads to deterioration of the neurons supplied by the vessels in spasm. This in turn leads to delayed neurological deficits, which may progress to coma and paralysis.

Cerebral vasospasm is associated with significant morbidity and mortality. The prognosis after aneurysmal subarachnoid hemorrhage is good if the diagnosis is made in a timely manner and treatment is aggressive (Corsten, et al., 2001). Surgical treatment of the aneurysm involves clipping of the aneurysm or the use of microcoils. Angiography is used to confirm the diagnosis of cerebral vasospasm after surgical treatment for aneurysm. Transcranial Doppler ultrasonography and transcranial color-coded sonography are also useful for accurate monitoring of cerebral vasospasm in the middle cerebral artery (Proust et al., 1999).

[5] Clotting Defects (Coagulopathies)-- Traumatic derangement of the hemostatic mechanism (which maintains the balance between clotting and circulation of the blood) can produce a coagulopathy known as disseminated intravascular coagulation (DIC). This condition involves both excessive clotting and excessive bleeding; it can occur in as many as 90 percent of patients having severe brain injury (Marx, 2002).

As a general rule, the degree of DIC that develops depends on the extent of brain injury and tissue destruction. The injured brain is a source of tissue thromboplastin (a factor involved in the initiation of blood coagulation), which activates the extrinsic clotting system. The central factor in the pathogenic mechanism is thought to be the

traumatic release of thromboplastin (a factor involved in the initiation of blood coagulation), which is present in large quantities in brain tissue.

Disseminated intravascular coagulation (DIC) may develop within hours after brain injury. Early diagnosis is by laboratory measurement of clotting parameters (prothrombin and partial thromboplastin times [PT and PTT, respectively], platelets, plasma fibrinogen levels, and fibrin degradation products). Since awaiting the results of coagulation studies introduces a delay in treatment that allows worsening of the coagulopathy, some clinicians advocate empiric treatment for coagulopathy in patients with severe closed head injuries who present with a Glasgow Coma Scale of six or less (May, et al., 1997).

DIC not only increases morbidity and mortality after severe head trauma, it also increases the risk of delayed intracranial hemorrhage. If a head-injured patient with DIC whose medical condition is stable suddenly begins to deteriorate, a repeat CT scan should be performed to rule out intracerebral hemorrhage (Marx, 2002).

Treatment is medical and directed at the precipitating condition. If treatment is not prompt and aggressive, the probability of serious morbidity or mortality is extremely high.

[6] Syndrome of Inappropriate Secretion of Antidiuretic Hormone (SIADH)-- The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) is a posttraumatic derangement of fluid balance that on rare occasions occurs as a sequela of closed penetrating head trauma (Robertson, 2001). The mechanism involves traumatically increased secretion of antidiuretic hormone (ADH, also called vasopressin) by the posterior pituitary gland. Normal action of ADH takes place in the distal tubules of the kidneys, where it promotes water retention. Following trauma, the normal balance between ADH secretion, water retention and sodium excretion is altered in such a way that sodium is lost from the serum and increased in the urine and water is retained in the cerebral tissues. The net result is an osmotic imbalance between the intravascular serum and the surrounding tissues, which in turn produces an increase in the intracranial pressure due to water retention.

Diagnosis is by laboratory measurement of serum

and urine sodium levels and osmolalities, as well as clinical signs and symptoms. Because sodium is an essential component for proper neuronal function, the hyponatremia (abnormally low serum sodium) presents clinically as neurologic deterioration, which may manifest as seizures. Depending on the magnitude of sodium depletion and rate of development, hyponatremia may also cause mild headache, anorexia, confusion, nausea and vomiting, coma, convulsions and, in the most severe cases, death. In the hyponatremia caused by SIADH, edema and signs of heart failure, cirrhosis, nephrosis, or hypovolemia (low blood volume) are usually absent, unless the patient has one of these disease states as an underlying medical condition (Robertson, 2001).

[7] Brain Abscess-- Although relatively uncommon in current clinical practice, brain abscess remains a serious and life-threatening disease in children and adolescents. Brain abscess results from seeding of microbes into an area of injured brain tissue following the introduction of bacteria into the cranium from a penetrating head injury. Retained bone fragments from a compound skull fracture are associated with an increased risk of abscess formation more frequently than retained metallic fragments or projectiles (such as a bullet). Abscess formation may also complicate anterior cranial fossa or temporal bone fractures, cerebrospinal fluid fistulae, and meningitis.

Patients with brain abscess usually present with systemic toxicity, raised intracranial pressure (ICP) and focal neurologic deficits. The typical clinical presentation is dominated by increased intracranial pressure, with headache occurring early in the course of abscess formation. During the acute phase, drowsiness, confusion and vomiting occur, leading to a slowing in mental acuity and eventually coma. Papilledema occurs rarely and is not a consistent finding on physical examination.

Abscess formation classically evolves in three phases: (1) an initial phase, characterized by temporary malaise (weakness), fever and mild headache; (2) a latent phase, when the patient usually appears well; and (3) a terminal phase, in which the patient presents with worsening headache and vomiting and develops focal neurologic deficits and cerebral herniation. Seizures occur in approximately 25 percent to 50 percent of patients, may be focal or generalized,

and can occur at any time during the course of abscess formation.

Diagnosis of brain abscess is by contrast-enhanced computed tomography (CT). Treatment is surgical and requires the administration of intravenous antibiotics (Cochrane, 1999).

The prognosis of brain abscess is good, with a mortality rate of less than 10 percent, if the abscess is diagnosed early and treated aggressively. Focal residual deficits may remain in some patients and seizures occur in 40 percent to 50 percent of patients following treatment (Cochrane, 1999).

[7a] Bacterial Meningitis-- Bacteria can infect meningeal membranes through gross penetrating wounds or the more subtle openings in the thin nasal or aural bones of the base of the skull following basilar skull fracture. Posttraumatic meningitis is caused by a variety of microorganisms, depending on the portal of entry of the bacteria. Definitive treatment is by the administration of intravenous antibiotics providing adequate coverage against the infecting microorganism (Marx, 2002).

[7b] Osteomyelitis-- Cranial osteomyelitis, or infection of the skull bone, can occur after penetrating injury to the skull and is most often a late sequela of head injury. The patient usually presents with pain, tenderness, swelling and erythema (localized redness and warmth) at the infected site. More than 50 percent of cases can be visualized on plain skull x-ray; technetium bone scans are useful when the skull radiograph is negative. However, false-positive bone scans may occur in patients experiencing previous head trauma or craniotomy. A gallium scan, when added to a skull x-ray or bone scan, will aid in differentiating infection from other causes of a positive technetium scan. Treatment is by surgical drainage and antibiotics (Marx, 2002).

[7c] Subdural Empyema-- The subdural space can be contaminated by a wide variety of microorganisms, usually as a result of facial or sinus fracture, open head injuries, meningitis or neurosurgical procedures. Subdural empyema is often characterized by rapid onset of a severe headache, hemiparesis (paralysis on one side of the body), and cerebral herniation. Seizures occur in approximately 30 percent of cases (Cochrane,

1999). Because of the nonspecific nature of these signs, definitive diagnosis must be made by computed tomography (CT) or magnetic resonance imaging (MRI). Subdural empyema gives rise to brain swelling and rapid rise in intracranial pressure (ICP), in addition to the infective processes, and thus constitutes a life-threatening emergency which must be treated promptly. Treatment is surgical and antibiotics are employed (Cochrane, 1999).

[8] Posttraumatic Epilepsy-- Posttraumatic seizures are among the most serious sequelae of head trauma and are relatively common in the acute or subacute period. The incidence of posttraumatic epilepsy after mild head trauma in the pediatric population is 1 percent to 6 percent (Savitsky and Votey, 2000). In general, the more severe the head injury, the higher the incidence of posttraumatic seizures. Acute posttraumatic seizures usually last for a short period of time and are most likely caused by temporary mechanical and neurochemical changes within the brain. The patient often exhibits no additional seizure activity after the acute seizure. In the subacute period (24 to 48 hours after trauma), seizures are caused by cerebral edema that progressively worsens, small hemorrhages, or penetrating injuries.

Posttraumatic epilepsy is much more likely to occur in patients with severe head injury or injuries resulting in a tear in the dura. High risk injuries include those with intracranial hemorrhage or dural penetration. Other risk factors for posttraumatic epilepsy include a low Glasgow Coma Scale (GCS) score, early seizures (those occurring in the first week after head trauma), and brain CT findings consistent with single temporal or frontal lesions in the acute phase. In addition, an EEG focus one month after head injury was found in one study to be a risk factor four times higher than for patients without this EEG finding (Angeleri, 1999). Depressed skull fractures, penetrating trauma, abscess formation, acute hematomas, prolonged posttraumatic amnesia, prolonged unconsciousness and focal neurologic deficits are also associated with a significantly increased risk of posttraumatic seizures.

Posttraumatic seizures usually are partial (focal) seizures that originate in a site of local brain damage and may either remain confined to the cerebral hemisphere of origin or generalize to both hemispheres. Although potentially disabling and

very difficult to treat, this type of seizure pattern is not in itself life-threatening as a rule. Status epilepticus, on the other hand, represents a life-threatening emergency in adults. When it occurs, it often is seen in the immediate posttraumatic period, and consists of a prolonged, repetitive cycle of seizures without the usual resolution and return to consciousness characteristic of other forms of epilepsy. Its occurrence in adults usually is associated with the worst prognosis in terms of developing chronic late epilepsy. However, status epilepticus has no prognostic significance in children (Marx, 2002).

Patients who make a good recovery from all other injuries may find themselves seriously disabled if chronic posttraumatic seizures develop. The mortality rate is low and was reported to be 14 percent in one study (Barlow, 2000). In addition, neurologic outcome was found to be significantly worse in those with posttraumatic epilepsy than those without (Barlow, 2000).

[93.43] Iatrogenic Injuries Following Head Trauma

The rates of morbidity and mortality for head trauma are strongly influenced by the quality and availability of medical care. Despite advances in diagnosis, medical and surgical therapy and basic scientific understanding of neurophysiological events in head trauma, the proportion of poor or fatal outcomes from head injury remains unacceptably high (Cullen, 2001).

The leading causes of posttraumatic morbidity and death are secondary complications that have their onset and/or evolution while the patient is under medical care (Cullen, 2001). As many as half of the deaths among all patients who sustain more than minor head injury can be prevented; among the most common preventable causes are delay in treating a hematoma, uncontrolled seizures, hypercarbia (presence of high levels of carbon dioxide in circulating blood), coagulopathy, hypotension, meningitis and hypoxia (Zink, 2001).

Iatrogenic causes of secondary brain injury include underresuscitation of shock, overhyperventilation resulting in cerebral ischemia, the use of medications that induce hypotension, and painful procedures or endotracheal suctioning that result in a reflex increase in intracranial pressure (ICP) (Zink, 2001). Other complications of medical

intervention include the administration of too much or too little fluid during fluid resuscitation; a delay in the administration of antibiotics, or inadequate pain control.

Mechanical ventilation of head-injured patients is also associated with risks of perforation of the esophagus and aspiration pneumonia; prolonged endotracheal intubation with stricture and pneumonia, and barotrauma with ventilator-induced lung injury. Minor procedures such as chest tube insertion can be complicated by perforation and hemorrhage. Prolonged resuscitation and invasive monitoring in the ICU can cause hypothermia and coagulopathy and initiate a cascade of events leading to multiorgan failure and Adult Respiratory Distress Syndrome (ARDS) (Cullen, 2001).

Although most patients with head trauma improve and make a good recovery in time, those with moderate to severe injuries are at significant risk of disability and death due to late or missed diagnosis and/or improper treatment. Common errors and omissions include delay in performing diagnostic procedures, inappropriate use or interpretation of skull x-rays, frank misdiagnosis, failure to recognize impending neurological deterioration, inadequate correction of hypotension due to shock and respiratory problems (including simple airway obstruction) and failure to monitor and aggressively treat elevated intracranial pressure, perhaps the leading cause of death in patients with severe head injury (Zink, 2001).

Timely and accurate diagnosis and treatment are crucial for a good outcome. Unfortunately, a major contributor to inadequate care is the simple lack of available trauma care centers outside of major metropolitan areas, with sophisticated diagnostic and monitoring equipment and experienced neurosurgical staffs.

[93.44] Neuropsychological and Neuropsychiatric Sequelae

In addition to neurophysiological sequelae of head injury, numerous neuropsychological and neuropsychiatric sequelae may be seen as well. Although physical disabilities tend to stabilize over time, mood, cognitive and behavioral changes are more central to morbidity, because there may be long-term impairments that adversely affect the patient's quality of life (Rao and Lyketsos, 2002).

Traumatic brain injury is associated with several psychiatric disturbances such as cognitive deficits and mood disorders. Cognitive deficits are very common among head-injured patients, with memory loss being the most common complaint. Almost all moderate to severe head-injured patients suffer from some type of cognitive defect. Disturbances of attention are also common in patients with brain injury, with patients complaining of difficulties with concentration and easy distractibility. In addition, language problems, such as lack of spontaneity of speech and aphasia, are frequent in head-injured patients.

The two most common mood disorders associated with head injury include major depression and bipolar disorder (defined as intermittent periods of major depression and mania). Anxiety is also commonly reported after traumatic brain injury, having an overall prevalence of 29 percent. Another neuropsychiatric disorder frequently experienced by head-injured patients is behavioral dyscontrol disorder (referring to a constellation of cognitive, somatic, and mood symptoms that occur simultaneously) (Rao and Lyketsos, 2002).

The term "postconcussive syndrome" has been used to describe a certain clinical presentation, but is confusing and controversial because it is seen in patients with and without a concussion (Rao and Lyketsos, 2002). The "postconcussive syndrome" refers to a complex of somatic, cognitive, and affective symptoms such as headache, sleep disturbance, memory and attention/concentration problems, irritability, anxiety and depression. Thirty to 80 percent of patients suffering mild head injury report having this complex of symptoms at three months post injury and 15 percent continue to have these symptoms at one year after injury. The percentage of patients reporting symptoms is higher for patients with moderate and severe head injury (Jagoda and Riggio, 2000).

A complete diagnosis should include the type and severity of the brain injury, the associated neuropsychiatric sequelae, other medical illnesses that may be present, and the patient's current functional capacity. Key components in making the diagnosis include obtaining a detailed history from the patient, a review of the patient's old medical records, performing a complete mental status, physical and neurologic examination, as well as a brief test of global cognitive functioning, such as

the MMSE. The MMSE (Mini-Mental Status Examination), however, is not particularly sensitive in determining cognitive loss in mild head injury. The patient's stage of recovery and prognosis for complete recovery should also be noted (Rao and Lyketsos, 2002).

P 93.50 MANAGEMENT OF HEAD INJURY: OVERVIEW

Appropriate management of the head injured patient depends on accurate classification of the head injury. Although a number of classification schemes are used for assessment of neurologic damage following head injury, the most reliable and popular scheme is the Glasgow Coma Scale (GCS). Head injury is classified into three groups based upon the GCS score: (1) mild head injury (score of 13 to 15), (2) moderate head injury (score of 9-12) and (3) severe head injury (3 to 8). However, the management of head injury is complicated by the fact that the GCS score is not always predictive of the presence of an intracranial lesion, such as a subdural hematoma. Indeed, 10 percent to 20 percent of head-injured patients with a GCS score above 12 have positive findings of a mass lesion on CT scan of the head (Jagoda and Riggio, 2000; Cheung and Kharasch, 1999).

From the emergency physicians' point of view, a patient presenting with a GCS score of less than 9 has severe brain injury. Over the past 30 years, mortality from severe head injury has been reduced by half, from about 50 percent to approximately 25 percent due to advances in emergency medical care and diagnostic modalities (Zink, 2001). Increased understanding of secondary brain injury and its impact on neurologic sequelae after brain injury have also lead to improved outcomes. The most critical decision facing emergency physicians today is whether or not to order a CT scan in the patient with mild head trauma (Cheung and Kharasch, 1999).

[93.51] Critical Factors Affecting Outcome

Aggressive early management of head injury improves outcome significantly. The initial evaluation and stabilization of the head trauma patient begins at the scene with aggressive management of the airway, breathing and circulation according to ACLS guidelines. Endotracheal intubation in the field also improves survival in severely head-injured patients by

preventing secondary injury caused by hypoxia and ischemia. If the patient has evidence of increased ICP (such as pupil asymmetry), ICP treatment should be initiated as follows: (1) ensure adequate oxygenation, cerebral venous drainage, and pain relief, sedation or paralysis; (2) hyperventilation (as a temporary measure); or (3) the use of an osmotic agent such as mannitol (Dangor and Lam, 1999).

Three types of injury can be singled out as being both prominent causes of severe disability and death and commonly mismanaged: intracranial hematomas, elevated intracranial pressure (ICP) and cervical spine injury. Acute subdural hematomas occur in about 20 percent of patients with severe head injury, and approximately 50 percent of these patients are unconscious from the time of injury (Evans and Wilberger, 1999) It has been demonstrated that patients with a subdural hematoma who undergo surgery within four hours of their injury have a mortality rate that is three times lower than those who have surgery more than four hours after their injury (Cheung and Kharasch, 1999; Evans and Wilberger, 1999). Hence, a delay in diagnosis and treatment can adversely affect outcome in any head-injured patient with an intracranial lesion requiring surgical intervention.

[93.52] Grading of Head Injuries for Triage and Management

Many physicians consider a systematic protocol for treatment as likely to provide the most effective care. A key element of the overall systematic approach is prioritization of injuries. Those that pose the greatest threat to life and limb must be treated first. Toward this end, triage and initial management are based on assignment of patients to one of four categories according to level of consciousness upon arrival in the emergency department (ED) (Meagher and Narayan, 2000):
Grade I: Mild Head Injury. Patient is alert, oriented and shows no signs of focal neurological deficits; may have headache, nausea, vomiting, and transient loss of consciousness (LOC).

Grade II: Moderate Head Injury. Patient is alert but has focal neurological deficit (e.g. hemiparesis or aphasia), or impaired consciousness (lethargic) but is able to follow simple commands (e.g., hold up two fingers).

Grade III: Severe Head Injury. Patient's consciousness is impaired to the degree that he or she is unable to follow simple commands; may speak, but speech will be inappropriate at best; has variable, often flaccid motor responses.

Grade IV: Brain Dead. No evidence of brain function. (Meagher and Narayan, 2000).

[1] Management of Grade I Head Injuries-- Minor head injury is defined as brain injury resulting in a GCS score of 13 to 15, with a return to a normal level of consciousness (LOC) within 24 hours (Morris and Marshall, 2000). The majority of patients experiencing minor head injury can be treated on an outpatient basis. If the patient is considered to be neurologically stable after a complete physical and neurologic examination, the patient can be discharged from the ED to home, where he or she is to be observed frequently by a reliable observer. Any change indicating a deterioration in neurologic status, such as an acute change in mental status, loss of consciousness, a worsening headache, or visual disturbances, should prompt immediate evaluation by a physician.

[1a] Use of X-Rays-- Historically, plain radiographs (x-rays) were used to screen for skull fractures and to identify patients at risk for an intracranial injury. The advent of CT, however, has now made the use of x-rays obsolete. Since skull x-rays do not have sufficient sensitivity or specificity to detect skull fractures or intracranial lesions, they are currently not considered clinically useful (Savitsky and Votey, 2000; Amann, 2000).

[1b] Use of CT Scans-- CT scanning is generally reserved for the head-injured patient exhibiting a prolonged loss of consciousness, symptoms that persist over time, or deterioration in neurologic status. In general, CT scanning is the diagnostic method of choice in patients with severe head injuries. Computed tomography (CT) scans are generally not recommended for adult patients with a history of mild head trauma who are considered neurologically stable after diagnostic evaluation (Amann, 2000). Since CT scanning is widely available, it is usually the first diagnostic test performed to exclude intracranial lesions, such as subdural or epidural hematomas.

The use of CT scanning in children is controversial (Savitsky and Votey, 2000). Recent guidelines regarding the use of CT in children have been

published by the American Academy of Pediatrics in conjunction with the American Academy of Family Physicians because of the wide variety of treatment practices regarding this issue. According to these guidelines, a CT scan is not recommended for the initial evaluation and management of children with minor head injury and no LOC. An acute change in neurologic status upon observation, however, such as vomiting, seizure, lethargy or persistent headache in any such child requires a repeat medical evaluation which most likely includes a CT scan (Gedeit, 2001). Children with minor head injury with brief LOC (less than one minute) may undergo a CT scan, if deemed appropriate by their physician (Coombs and Davis, 2000).

[2] Management of Grade II Head Injuries-- Because patients with moderate head injury can deteriorate rapidly to Grade III neurologic status, their management is similar to that of patients with severe head trauma, albeit somewhat less urgent. Similar to the patient with severe head injury, the priority in managing the patient with moderate head injury is to minimize secondary brain injury. To this end, maintenance of adequate oxygenation and hemoperfusion are of critical importance (Gedeit, 2001). Since approximately 10 percent of patients with moderate head injury will either have or develop hematomas, a CT scan is warranted. The presence of a subdural or epidural hematoma requires immediate surgical evacuation to prevent secondary injury. Severe cerebral edema (identified by CT scan) also requires placement of an intracranial pressure monitor. In addition, a patient exhibiting altered mental status may have suffered a spinal cord injury that requires immobilization. Spinal cord immobilization should be continued until a complete diagnostic evaluation is performed (Gedeit, 2001).

[3] Management of Grade III Head Injuries-- Since 1991, there have been significant changes in the management of patients with severe head injury. Current practices parallel the recommendations of evidence-based guidelines. Specific changes include an increase in intracranial pressure monitoring and a decrease in the use of hyperventilation and steroids. Most neurosurgeons agree that patients with severe head injury should also receive treatment at a Level 1 trauma center (Marion and Spiegel, 2000).

In the patient with severe head injury, the ABCs of

emergency medical treatment apply (that is, the establishment of an adequate airway, breathing, and circulation is the first priority). In patients with a GCS of 8 or less, or those exhibiting hypoventilation, apnea, or cardiopulmonary arrest, endotracheal intubation and mechanical ventilation should be performed. It is of utmost importance that hypoxia and hypercarbia (a high carbon dioxide level in the blood) be prevented by using supplemental oxygen and controlled ventilation, since these factors can contribute to secondary brain injury and poor outcomes.

Patients who exhibit signs of circulatory shock (e.g., tachycardia [a rapid heart beat] or hypotension), should receive intravenous isotonic fluid to ensure an adequate blood volume in order to maintain blood flow to the brain. The neurologic status of the patient should also be assessed using the Glasgow Coma Scale to evaluate the patient's response to therapy. The temporary use of hyperventilation or mannitol for patients with impending brain herniation is appropriate until the neurologic evaluation is complete and the patient's condition is stabilized. Seizures are common after severe posttraumatic brain injury and should be treated with benzodiazepines, phenytoin, and phenobarbital as necessary (Gedeit, 2001).

P 93.60 DIAGNOSTIC EVALUATION AND EMERGENCY MANAGEMENT

The diagnosis and treatment of traumatic head injury is a staged, progressive process. At each stage, more diagnostic information is accumulated and further therapy is administered, predicated on the progress of the patient's evolving clinical course.

Damage to the brain that occurs as a result of head trauma is essentially of two types, structural and functional. The location and extent of both types is crucial to emergency management, to monitoring and therapy in the postacute stage of injury and to long-term outcome and rehabilitation (Dangor and Lam, 1999; Cheung and Kharasch, 1999).

Computed tomography (CT) and other radiological diagnostic modalities provide a great deal of information about the structural damage caused by a head injury. It, however, provides virtually no information about the functional damage to the neurons. The fundamental source of information about the functional status of the CNS (central nervous system) neurons is the neurological

examination. However, the ability to define the location and extent of neuronal dysfunction through the neurological exam is limited in patients who are comatose, uncooperative or confused; it is further compromised by the effects of sedative or paralytic drugs administered as part of the patient's treatment.

This situation has led to an interest in using the brain's electrical activity as a more objective source of information regarding the patient's neurological status and any changes that occur in it over time. Two commonly used techniques for noninvasively recording the electrochemical impulses from the brain's nerve cells are electroencephalography (EEG) and sensory evoked potentials (SEPs). Additional factors of enormous significance to the functional state of the neurons are cerebral blood flow (CBF), cerebral metabolism and intracranial pressure (ICP). Various techniques are available for monitoring these functions.

Following severe head trauma, the circulation and metabolic processes of the brain are compromised due to secondary damage such as increased intracranial pressure, arterial hypoxia (insufficient oxygen in the blood), arterial hypotension (due to blood volume loss, for example) and increased blood viscosity. A number of techniques can be used to measure circulatory function (Dangor and Lam, 1999). The ultimate aim of management is to reestablish and maintain the patient's homeostasis in order to provide the best physiological environment possible for the recovering neurons.

Because numerous sequelae of head injury can cause precipitous deterioration and rapid death, triage (prioritization) of the injuries is essential. This in turn requires that all injuries be identified and that the emergency department (ED) physician have a thorough knowledge of the types of injuries that result from trauma and which are most likely to pose a threat to the patient's life. Priorities of care are established by evaluating and managing the patient in the ED according to the following staged approach: history, primary survey, secondary survey, and definitive care (Cheung and Kharasch, 1999; Evans and Wilberger, 1999; Townsend, 2001). Traditionally, the diagnosis of traumatic brain injury is made by history and physical examination (Jagoda and Riggio, 2000).

[93.61] History

The initial evaluation of a patient who has suffered head trauma begins with a thorough history. Since the patient experiencing a concussion is usually unable to give an accurate history, witnesses of the event often provide valuable information. One important aspect of the history is the mechanism of injury; e.g., whether a moving object struck a stationary head, or a moving head struck a stationary object, such as what occurs in a motor vehicle accident. This information often indicates the forces involved in the event (Amann, 2000).

Based on the mechanism of head injury alone, the emergency physician can usually predict the likelihood of significant injury. The highest incidence of intracranial injuries results from unprotected head trauma involving large forces. Pedestrians and bicyclists, for example, have the greatest potential for suffering a severe intracranial injury. Motor vehicle occupants, assault victims, and patients injured from falls have comparable incidences of intracranial injury. Even falls from low heights can result in significant trauma (Cheung and Kharasch, 1999).

Perhaps the most important information to be obtained in the patient suffering blunt head trauma is loss of consciousness. Current emergency medicine practice is to assume that if a patient did not lose consciousness, he or she does not have an intracranial injury. The incidence of intracranial lesions in victims experiencing loss of consciousness ranges from 1.3 to 17.2 percent and rises with increasing time of loss of consciousness (Cheung and Kharasch, 1999). Other important information to be elucidated in the history include the behavior of the patient after the traumatic event, a history of prior concussive episodes, or existing neurologic deficits (Amann, 2000).

[93.62] Primary Survey

The chief objective of the primary survey is to identify those injuries that are immediately life-threatening. The identification and management of these injuries should proceed simultaneously, and the entire primary survey should be completed within 5 to 10 minutes. The stages in the primary survey, outlined below, begin with the "ABCs" (airway, breathing and circulation) and are followed by a brief neurological exam (Townsend, 2001).

[1] Airway, Breathing and Circulation-- Establishing an airway so that oxygen can get to the lungs is of cardinal importance, as the brain is particularly susceptible to permanent damage from oxygen deprivation. At the same time, the possibility of an associated cervical spine fracture must be appreciated, since inappropriate manipulation of the patient during establishment of the airway can cause further injury. Any patient with traumatic brain injury should be assumed to have a cervical spine injury unless proven otherwise, and the patient should be assessed for the presence of other systemic injuries and toxic drug ingestion as well (Dangor and Lam, 1999).

Once an unobstructed airway is established, adequate oxygenation (movement of air into and out of the lungs) must be assured. Inadequate oxygenation rapidly leads to hypoxia, anaerobic metabolism and acidosis and is the most common cause of death in head trauma patients (Dangor and Lam, 1999). Respiratory difficulty is often missed by the physician during the early stage of care when attention tends to be focused most intently on evaluation of the most obvious injuries. Indicators of adequate oxygenation should not be neglected; they include extent of chest expansion, skin color, and respiratory rate. A breathing rate above 30 per minute is suggestive of the patient's need for mechanical assistance in breathing. A respiratory rate above 40 per minute is a definite indication for mechanical ventilation. Minimal chest expansion and cyanosis (blue skin color) are also indications of inadequate oxygenation.

In addition to a patent (open) airway and effective respiration, the patient's cardiac function and circulation must be returned to normal in order to prevent shock and to assure adequate perfusion of the brain and other tissues. Control of bleeding is a major part of this effort (Dangor and Lam, 1999; Townsend, 2001).

[2] Neurologic Exam-- A rapid neurologic assessment includes an evaluation of the patient's level of consciousness (using the Glasgow Coma Scale), pupillary assessment, brain stem reflexes, and focal or lateralizing neurologic signs (Dangor and Lam, 1999; Townsend, 2001). Currently, the Glasgow Coma Scale (GCS) is the most popular system for grading the neurologic status of head trauma patients. This examination assesses the patient's ability to respond to pain, to speak, and to

open the eyes (e.g., motor response, verbal response, and eye opening). When performed in concert with examination of the pupils, the GCS provides an excellent guide to the severity of the injury in the pre-hospital setting. Patients who are unable to follow commands, do not open their eyes in response to noxious stimuli, or are unable to make comprehensible sounds are considered to be in a coma (GCS<<8). All four limbs should be tested for responsiveness, either to verbal command or to pain, so that any focal neurological deficits are not missed (Morris and Marshall, 2000).

Trauma protocols in various hospitals often use an optimal GCS as a measure of reassurance, and this has been deemed as a sufficient reason for discharging a patient to home without further diagnostic evaluation. However, even a perfect GCS of 15 does not exclude the presence of significant intracranial lesions such as subdural or epidural hematomas. In fact, studies show that the incidence of head injuries detected by CT in patients with a GCS of 15 varies between 2.5 percent and 22.5 percent (Cheung and Kharasch, 1999). In addition, up to 6 percent of patients with blunt head trauma and loss of consciousness, a GCS of 15, and normal neurological examinations have been shown to have significant intracranial lesions (Cheung and Kharasch, 1999). Although a perfect GCS score of 15 does not exclude an intracranial lesion that can be detected by CT, a low GCS score does correlate positively with a high incidence of a CT abnormality (Cheung and Kharasch, 1999).

In addition to the GCS, pupillary findings are used to indicate the severity of the head injury. Among the more serious conditions that may be suggested by pupillary findings is herniation of the temporal lobe, which can cause rapid neurologic deterioration and death. When present, the pupils are mildly dilated and respond sluggishly to light. Associated damage to the oculomotor nerve is suggested by pupils that have been dilated from the time of the trauma and weakness of the eye muscles. Pupils that are bilaterally dilated and fixed suggest insufficient cerebral vascular perfusion itself, or secondary to increased intracranial pressure causing diminished blood flow. Bilaterally small pupils (less than 3 mm in diameter) suggest opiate drug overdose.

[93.63] Resuscitation

In a patient with depressed consciousness or coma, resuscitation is accomplished first and foremost by hyperventilation (as a temporary measure) and hyperoxygenation. This can be accomplished either by simple endotracheal intubation or by mask and ventilator if the patient needs assistance in breathing. Since hypoxemia and hypercapnia often occur at the time of initial resuscitation, endotracheal intubation in the field (e.g., outside the hospital, at the scene) has been shown to improve survival in patients with traumatic brain injury by preventing secondary insults resulting from hypoxia and ischemia (Dangor and Lam, 1999; Townsend, 2001).

[93.64] Secondary Survey

Once the quick initial evaluation (primary survey) of the patient's injuries and status has been completed and he or she has been stabilized with respect to cardiopulmonary function, a more complete assessment of the patient's injuries is conducted. The essential components of this secondary survey are: history, physical and neurological examination, laboratory tests, radiological evaluation and any special procedures (Evans and Wilberger, 1999; Townsend, 2001).

[1] History-- During the secondary survey, additional details are added to the basic history of the accident and the patient's injuries acquired during the primary survey and pre-hospital phase. The key points to cover are summarized in the acronym AMPLE: allergies, medications, past medical history, last oral intake, and events surrounding the trauma (Townsend, 2001).

[2] Physical and Expanded Neurologic Exam--

Once the patient's cardiorespiratory function is stabilized, a physical examination should be performed in which the patient is quickly but carefully given a complete body survey. The exam should proceed in an organized fashion and on a regional basis: head, neck, chest, abdomen and extremities. As the examiner proceeds, he or she should coordinate the findings and other aspects of the secondary survey and be constantly integrating all the diagnostic information with management possibilities.

Immediately upon completion of the physical exam, an expanded neurologic exam should begin in patients who are awake and alert following resuscitation. Key components of the exam include

inspection of the cranium for signs of puncture wounds, hemorrhaging behind the tympanic membranes (ear drums), leakage of cerebrospinal fluid (CSF) from the ears or nose and other signs of injury. Any laterality to the neurological findings (such as a dilated pupil or hemiparesis) suggests a focal mass lesion. The cranial nerves mediating vision and ocular motility, hearing, equilibrium, facial sensation and movement and olfaction (sense of smell) should be assessed, since these are often stretched, torn or contused. The higher the GCS score, the more important it is to obtain a detailed mental status examination. For penetrating head wounds, such as gunshot wounds, there should be detailed documentation of entry and exit wounds, powder burns, and foreign bodies (Evans and Wilberger, 1999; Townsend, 2001).

[3] Lab Tests-- Baseline laboratory tests include a complete blood count (CBC), electrolytes, an arterial blood gas, a coagulation profile, blood type and crossmatch, an electrocardiogram, and urinalysis (Dangor and Lam, 1999). Blood glucose levels should be measured as they can indicate or rule out hypoglycemia (low blood sugar) as a cause of depressed consciousness. A toxicology screen including a blood alcohol test should also be ordered as the blood alcohol concentration may affect the GCS score. Studies have shown that a lower GCS can not be attributed to alcohol unless the patient has a minimal blood alcohol concentration of 200 mg/dL (Cheung and Kharasch, 1999).

[93.65] Radiological Investigation

Prior to the introduction of computed tomography (CT) in 1972, radiological investigation of intracranial injuries following head trauma comprised primarily skull x-rays, ventriculography and cerebral angiography. These techniques still have important specific indications, but the radiological diagnosis of intracranial trauma is now dominated by CT.

[1] X-Rays-- In a severely injured patient, x-rays of the cervical spine should be taken first. Since movement of an injured neck can damage the spinal cord and result in permanent paralysis, the patient's head and neck must remain immobilized in a hard (Philadelphia) collar until the radiologist or neurosurgeon has read the cervical films. The one exception to this rule is the patient suffering a

gunshot wound to the head. Indirect spinal cord injury does not occur in a patient having a gunshot wound to the head and cervical spine immobilization may compromise airway management in such a patient (Kaups and Davis, 1998). Skull x-rays have been largely supplanted by computed tomography (CT) scans in the diagnostic evaluation of head trauma.

[1a] Cervical Spine X-Rays-- Cervical spine injury should be suspected in a head trauma patient who has suffered a motor vehicle accident (especially a rear-end collision) or a fall. The cervical spine is most commonly affected because of its mobility. This is followed by the thoracolumbar spine, which forms the junction between the rigid thoracic spine and the flexible lumbar spine. Injury to the cervical spine is caused by flexion, flexion-rotation, vertical compression extension and lateral flexion forces. Approximately one-third of patients with moderate or severe head injury have cervical spine injury (Lida et al., 1999). In addition, patients with upper cervical spine injury are at greater risk of sustaining skull fractures and intracranial hematomas than those with injury to the lower cervical spine. Due to the prevalence of this injury, all patients with traumatic brain injury should be suspected of having a cervical spine injury until proven otherwise (Dangor and Lam, 1999).

Cervical spine injuries are one of the most common missed diagnoses having serious implications for both the patient and physician. The diagnosis of subluxations or spinal cord injuries in the absence of vertebral fractures, especially in the comatose patient, poses a major challenge for the physician (Demetriades et al., 2000). In a conscious patient, pain or tenderness in the neck may alert the physician to the presence of cervical spine injury. Diagnostic hints in an unconscious patient include areflexia (absence of reflexes) and flaccid anal sphincter, diaphragmatic breathing, forearms that can be flexed but not extended, facial grimaces in response to pain above the clavicle (collar bone) only and hypotension in the absence of another clear etiology. Far more objective and reliable evidence, however, must be obtained by x-ray visualization of all seven cervical vertebrae. Lateral, anteroposterior (AP) and open-mouth views are usually adequate, although a "swimmer's view" (with the patient's arm extended above the head) may sometimes be necessary to visualize the 6th and 7th vertebrae. Although lateral cervical spine radiographs show the cervical

spine below the level of C7, they do not necessarily rule out spinal cord injury (Dangor and Lam, 1999). If the diagnosis remains in doubt following the cervical films, a definitive diagnosis can be made by CT, which is superior to plain film x-rays for visualizing spinal injuries in general (Evans and Wilberger, 1999).

[1b] Plain Skull X-Rays-- The use of skull films has dramatically decreased since the advent of the CT scanner. Studies show that skull films are neither sensitive or specific for detecting intracranial injuries. CT is a much better diagnostic modality for the detection of skull fracture, having a sensitivity of 94 percent. Although the presence of a skull fracture increases the likelihood of having an intracranial lesion, approximately 50 percent of all intracranial abnormalities resulting from head trauma are not associated with skull fracture. For this reason, skull films have become superfluous in the management of blunt head trauma. Thus, any head-injured patient with a suspected skull fracture should undergo a CT scan (Cheung and Kharasch, 1999).

[2] Computed Tomography (CT)-- Computed tomography (CT) is a noninvasive radiological technique which allows computer-assisted x-ray visualization of cross-sections of internal soft tissue structures based on subtle differences in their relative densities. A difference in density of as little as 0.5 percent can be revealed on CT (Cheung and Kharasch, 1999).

In essence, computed tomography is performed by placing the patient in the CT scanner and making a series of 10 to 15 sequential "cuts" or "slices" of 1 cm each along the vertical axis of the head. Each slice takes about two seconds to complete. The resulting CT image, reconstructed by computer from the series of contiguous 1 cm cuts, should adequately cover the entire brain as well as the posterior fossa. Different angles of the x-ray beam and patient positions are used as appropriate, providing cuts along different axes. When planning the study, any associated injuries, especially those of the cervical spine, must be taken into account as they will restrict the allowable maneuvers of the patient.

Computed tomography is the initial imaging modality of choice for traumatic brain and spinal injuries (Gean, et al., 1995; Bagley, 1999). Major traumatic lesions identifiable with CT scanning

include fractures and fragments, CSF leakage, edema, contusion, epidural hematoma, subdural hematoma, intracerebral hematoma, subarachnoid hemorrhage, intraventricular hemorrhage, hydrocephalus, posttraumatic infarction and brain abscess.

Although CT remains the mainstay of evaluation of head trauma, it is not infallible. The incidence of abnormal findings on CT following head trauma varies considerably in published reports, from 37 to 73 percent (Zee and Go, 1998). Accurate interpretation of posttraumatic CT images thus requires appreciation of some of the technique's key limitations and artifacts, most of which can be corrected. Principal limitations include motion artifacts (caused by movement of the patient's head during the procedure) and the partial volume effect (inaccurate interpretation of a finding situated only partially within a "slice"). Motion artifacts are particularly severe in the vicinity of bone, which is where extracerebral hematomas are located. However, CT is superior for analyzing vascular lesions, such as aneurysms, arteriovenous fistulas, and intravascular thrombi.

[2a] Fractures and Fragments-- The extent of depression of a fracture fragment is best demonstrated by CT using bone windows. Other foreign objects, such as bullet fragments, as well as their hemorrhagic track through the brain also are readily identified by CT, as is pneumocephalus (intracranial air).

[2b] Cerebrospinal Fluid (CSF) Leakage-- A CSF leak can be identified by CT scan particularly using bone windows with coronal views (Zee and Go, 1998; Townsend, 2001). Common sites for rhinorrhea (CSF leakage from the nose) include the cribriform plate and the anterior cranial fossa. The common site for otorrhea (CSF leakage from the ear) is through the petrous bone (Zee and Go, 1998; Townsend, 2001).

If sufficient information is not obtained by a standard scan, intrathecal (within the CSF) contrast enhanced CT may be used. In this case, contrast material is injected by lumbar puncture and the scan is performed with the patient tilted head downward 10 to 15 degrees. Any fractures will be revealed as contrast material leaks through into the cavity of the nose or ear (Zee and Go, 1998; Townsend, 2001).

For a patient with clinically diagnosed CSF leakage suspected of having a CSF fistula, the combination of magnetic resonance cisternography and plain high-resolution CT is highly accurate in locating the site of the fistula. This combination of imaging modalities represents a noninvasive alternative to CT cisternography in the diagnosis of CSF fistula (Shetty, et al., 1998).

[2c] Edema-- Edema may be focal, multifocal or diffuse. An edematous area appears as a zone of low density on CT scans, with mass effects manifested as compression, displacement or distortion of the cerebral ventricles. Focal and multifocal lesions can be distinguished by comparison with the somewhat denser white matter of the brain since these lesions, when adjacent to traumatic mass lesions, demonstrate decreased density on CT scans compared with normal tissue (Marx, 2002).

Diffuse edema over the whole brain may be difficult to identify owing to the lack of any region of normal brain density for comparison. In such cases, however, there usually is obvious bilateral ventricular compression. When interpreting the CT scan, it must be kept in mind that the presence of other low density components in the traumatized brain, such as lipids, necrosis or cavitation, can be mistaken for edema or obscure edema that is present.

[2d] Contusion-- Non-contrast-enhanced CT is the diagnostic tool predominantly used to detect contusions early in the posttraumatic period. Contusions appear heterogenous and irregular on CT scan because of mixed regions of hemorrhage, necrosis, and infarction. The surrounding edematous tissue also appears hypodense. By days three and four, following the initial traumatic event, the blood located within the contusion begins to degrade, so that magnetic resonance imaging (MRI) becomes a more useful diagnostic test (Marx, 2002).

Contusions are most commonly located in the anterior frontal and temporal lobes. Contusions often produce a mass effect as well, which manifests as displacement and distortion of the ventricles (although this may be minimal). Since cerebral contusions may evolve into delayed post-traumatic intracerebral hematomas, they should be followed carefully by CT scans (Zee and Go, 1998; Baley, 1999).

[2e] Epidural Hematoma-- An acute epidural hematoma (between the inner surface of the skull and the exterior of the dura mater) generally appears as a biconvex (lens-shaped) region of uniformly high density on CT.

Epidural hematomas are most often located over the temporal or temporoparietal region, and form from extravasated blood from anterior or posterior branches of the middle meningeal vessels that are damaged due to a linear skull fracture (Goetz, 1999). They displace the brain from the midline much more than subdural hematomas do. The development of an epidural hematoma also may be delayed, or may be masked by a large subdural hematoma on the opposite side of the brain. Because an initially hidden epidural hematoma may enlarge rapidly following evacuation of the contralateral subdural hematoma, a postoperative CT scan should be obtained. Since approximately 20 percent of patients with an epidural hematoma have blood in both the epidural and subdural spaces at surgery (or autopsy), it is not always possible to discriminate between epidural and subdural hematomas on CT (Goetz, 1999).

Occasionally, an epidural hematoma will remain undetected in the first CT, which is usually performed a few hours after injury, but will appear on subsequent CT scans as the patient's neurologic status rapidly deteriorates and the hematoma evolves. This is known as a "delayed epidural hematoma" and is a potential for a missed diagnosis by emergency physicians. Although the occurrence of delayed hematoma is rare, 8 percent of patients in a large series were found to have delayed epidural hematoma (Inamasu, et al., 2001). In most of the reported cases, the initial CT scan was taken within 24 hours after the injury and reported to be normal, while the delayed evolution of the hematoma occurred over the next 24 to 96 hours. The progression of symptoms in delayed epidural hematoma are also more gradual rather than acute. Hence, even when the initial CT scan is normal, repeated CT scans should be obtained in patients suffering blunt head trauma with worsening symptoms, to rule out the development of delayed epidural hematoma (Inamasu, et al., 2001).

[2f] Subdural Hematoma-- On CT scan, an acute subdural hematoma appears as a hyperdense lesion over the outer surface of the brain between the inner surface of the skull and dura. Diagnosis is more complex with a subacute subdural

hematoma, since the hematoma appears isodense relative to the brain itself. During the acute period, T1-weighted magnetic resonance imaging may be more useful in detecting a lesion in the subdural space. A chronic subdural hematoma appears as a hypodense area on the CT scan (Goetz, 1999; Marx, 2002). CT can detect a subdural hematoma at a depth of 5 mm; shallower hematomas may be missed due to their close proximity to the skull. A relatively large subdural hematoma also may be missed due to partial volume artifacts if it is located high over the brain and beneath the convexity of the skull.

High subdural hematomas may be detected by scanning perpendicular to the suspected location. Subdural hematomas undergo a decrease in density over time, as the high-density blood components in the clot (such as hemoglobin) disintegrate. In addition, there is probably some transdural migration of cerebrospinal fluid into the hematoma, which also decreases the density of the lesion. Eventually, the hematoma may not be visible. In such cases, major mass effects, such as ventricular distortion and brain shift, may be noted. If these are also absent or minimal, other clues to the presence of a hematoma include obliteration of the sulci (furrows) over the convexity of the involved side and distortion of the ventricle on the opposite side. Contrast enhancement may be useful.

Confusion may exist concerning the classification of subdural hematomas according to CT density. For example, new bleeding into a chronic subdural hematoma will have the appearance of an acute lesion on CT. An acute subdural hygroma (a fluid filled sac or cyst) will have the appearance of a chronic subdural hematoma. Since management decisions can be altered dramatically, information obtained from the history becomes more important (Goetz, 1999).

[2g] Intracerebral Hematoma-- An intracerebral hematoma may be detected on the first CT scan immediately following the trauma, but often is not seen for several hours or days. In contrast to a contusion, intracerebral hematomas are usually located deep within the brain tissue and tend to become well demarcated over time (Marx, 2002). On CT scan an intracerebral hematoma appears as a well-defined hyperdense homogenous area of hemorrhage. Intracerebral hematomas may develop in any part of the brain, but are found most commonly in the frontal and anterior temporal

lobes of the cerebrum (Goetz, 1999). They may be singular, but consist of multiple foci more commonly than any other form of posttraumatic hematoma. This may help to distinguish them from hematomas due to other causes, such as hypertension, aneurysms or arteriovenous fistulas.

Computed tomography can detect an intracerebral hematoma as small as 0.5 cm in diameter.¹⁵ By 3 to 4 weeks following the trauma, the density of the lesion may diminish, making it impossible to visualize except by the use of contrast enhancement. Distortion of the ventricles or displacement of the brain from the midline due to the mass effect of the hematoma also should be noted. The majority of intracerebral hematomas develop immediately following trauma, but may appear later within the first week after injury (Goetz, 1999). On occasion, their development may be delayed for up to 3 weeks.

[2h] Subarachnoid Hemorrhage-- Subarachnoid hemorrhage is detected on the initial CT scan in approximately 33 percent of patients with severe head injury and has an incidence of 44 percent in all cases of severe head trauma. It is the most common CT scan abnormality seen after traumatic brain injury (Marx, 2002). Bleeding into the subarachnoid space (between the arachnoid and pia mater meningeal membranes) appears on CT as high-density linear regions in specific areas of the brain. Subarachnoid hemorrhaging typically is present for only a few days following trauma and commonly is associated with a hematoma (either intracerebral or extracerebral). It may lead to hydrocephalus.

[2i] Intraventricular Hemorrhage-- Bleeding into the cerebral ventricles usually is associated with bleeding within the substance of the brain tissue (parenchymal hemorrhage). Since the advent of CT, traumatic intraventricular hemorrhage has been diagnosed more frequently. In the past, it was associated with a poor prognosis, but intraventricular hemorrhage by itself is rare, suggesting that other lesions that occur concomitantly with it may contribute to a poor outcome (Abraszko, et al., 1995). The uniform high density image of the ventricles (due to the blood therein) is characteristic of the acute stage.

[2j] Hydrocephalus-- The incidence of posttraumatic communicating hydrocephalus (an excessive accumulation of fluid in the ventricles) ranges from less than 1 percent to 29 percent in

patients with severe head injury. If CT criteria for ventriculomegaly are used, the incidence reportedly varies from 30 percent to 86 percent (Guyot and Michael, 2000). The CT scan shows ventriculomegaly (enlarged ventricles) within 10 to 14 days, and sometimes low-density edema around the ventricle borders. Ventriculomegaly after head injury is a subject of current debate, as there is presently no definitive manner to distinguish between posttraumatic hydrocephalus from cerebral atrophy in its presence. The diagnosis is established by using a combination of clinical, neuroimaging results, and physiologic data. For example, the presence of late neurological deterioration associated with increased intracranial pressure confirmed by CT scan findings is more useful in the diagnosis of posttraumatic hydrocephalus than CT scan alone (Phuenpathom, et al., 1999). CSF dynamics are also correlated to changes in ventricular mass during the first three months following severe head injury and are useful in the diagnosis of posttraumatic hydrocephalus (Marmarou, et al., 1996).

Although the diagnosis of hydrocephalus is often problematic, it is most easily made when ventricular dilatation is observed on CT (with minimal dilatation of the sulci) and abnormal CSF pressure is present (Townsend, 2001). The return of the ventricles to normal size following compression by edema or hematoma should not be mistaken for hydrocephalus. The ventricular pathways may also be blocked by a posterior fossa hematoma, giving rise to an acute obstructive hydrocephalus, but this is seen less often (Townsend, 2001).

[2k] Posttraumatic Brain Atrophy-- Posttraumatic brain atrophy is a delayed sequela sometimes seen in a few months to a year following trauma. A diagnostic sign on CT is enlargement of the ventricles. Posttraumatic brain atrophy can be distinguished from hydrocephalus by the presence of enlarged cortical sulci (fissures) in the former. Late magnetic resonance imaging (MRI) findings after severe head trauma also reveal lesions associated with cerebellar atrophy as a consequence of severe head trauma (Soto-Ares, et al., 2001).

[2l] Posttraumatic Infarction-- Mechanical shift of the brain and herniation across the falx and/or tentorium is the predominant cause of posttraumatic cerebral infarction in most head-

injured patients (Server, et al., 2001). Acute posttraumatic ischemic infarction appears as a low density area surrounded by a normal brain (Gean, et al., 1995; Tyagi, et al., 1995; Server, et al., 2001). Infarction usually is first detectable at about 24 hours following the trauma. Sixty percent of these lesions can be visualized by 7 days. An additional 15 percent of infarctions may be visualized by contrast enhanced CT over the subsequent 3-week period, although some contrast-enhanced infarctions may be detected within the first 24 hours after injury. Infarctions smaller than 2 cm may not show up on CT (Gean, et al., 1995; Tyagi, et al., 1995; Server, et al., 2001).

[2m] Brain Abscess-- Abscess is an infectious sequela, usually seen following penetrating head trauma, sinus bone fractures or as a complication of surgery. In one study, the primary cause of brain abscess was reported to be neglected (undiagnosed and untreated) compound depressed skull fractures (Stephanov, 1999). Brain abscess appears as an area of low density on the CT image. With contrast enhancement, the low-density area is circumscribed by a bright ring representing edema. If the abscess has developed in the vicinity of the ventricles, ventricular compression and displacement often is visible.

[3] Ventriculography-- Ventriculography involves x-ray of the brain following removal of cerebrospinal fluid from the cerebral ventricles and replacement with air or other contrast medium. Prior to the introduction of CT, ventriculography was used to determine the degree of posttraumatic supratentorial brain shift (the tentorium is the tentlike process of the dura mater that lies between the cerebellum and the occipital lobes of the cerebrum at the back and base of the brain). Contemporary use of ventriculography is unnecessary since the advent of MRI scans.

[4] Cerebral Angiography-- Despite advances in other imaging techniques, angiography remains the radiographic tool of choice for the evaluation of traumatic vascular lesions (Gaskill-Shiple and Tomsick, 1996). The three primary indications for cerebral angiography are: (1) elevated intracranial pressure (ICP) and shifted or slitlike ventricles (due to compression from a mass lesion) on the ventriculogram; (2) progressive neurological deterioration in the absence of an apparent cause; and (3) suspected vascular injuries. Angiography is particularly useful for detecting traumatic

aneurysms, arteriovenous fistulas and venous occlusions caused by cerebrovascular trauma (Gaskill-Shiple and Tomsick, 1996; Quintana, et al., 1996; Waran and Menon, 2000).

Magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) may also aid in selecting those patients with suspected vascular injuries requiring conventional angiography (James, 1997). In children, MR angiography offers a non-invasive alternative to conventional angiography for the diagnosis of vascular lesions and during follow-up evaluation of anticoagulation therapy (James, et al., 1995).

[4a] Aneurysms-- Enhanced CT (using a contrast medium) may suggest the presence of a cerebral aneurysm, but precise visualization and localization is likely to be impossible without the use of angiography, especially if the aneurysm is associated with an extracerebral hematoma (Townsend, 2001). Patients who deteriorate neurologically when no explanation is forthcoming from the CT scan should therefore undergo angiography. In addition, any patient with a penetrating head injury or skull base fracture near the major arteries should undergo an arteriogram (Townsend, 2001). A new form of angiography, called digital subtraction angiography, is especially useful for studying the extracranial arteries, such as those in the neck (Kuker and Thron, 1996; Mignon, et al., 1999).

[4b] Thrombosis and Infarction-- Unlike hematomas, the smaller clots that form within cerebral vessels and cause infarctions of the tissue do not show up reliably on CT. When this is suspected, definitive diagnosis can be made with cerebral angiography. Because of the decreased use of cerebral angiography following the advent of CT, many of these severely disabling lesions probably go undetected (Gean, et al., 1995; Gebara, 1997; Bagley, 1999). For strokes involving the spinal cord (e.g., spinal cord infarcts), magnetic resonance imaging (MRI) is the best method to visualize the spinal cord and can reveal spinal hemorrhages and infarcts (Gean, et al., 1995; Goetz, 1999; Bagley, 1999). A new technique known as diffuse-weighted imaging has been found to be more sensitive than CT in detecting acute ischemia and can be used to visualize major ischemic change more easily than CT (Barber et al., 1999).

[4c] Arteriovenous Fistulas-- Selective cerebral angiography is performed on the vessels in the region of suspected injury in order to define the extent of the lesion. If a carotid-cavernous sinus fistula is suspected, CT or MRI are the initial diagnostic tests of choice. The final and most decisive diagnostic test is cerebral arteriography of both the internal and external carotid arteries (Gaskill-Shiple and Tomsick, 1996; Goetz, 1999).

[5] Magnetic Resonance Imaging (MRI)-- Magnetic resonance imaging (MRI) is performed by exposing the patient to a magnetic field that aligns the protons in the nucleus of the body's hydrogen atoms along the axis of the magnet. When a pulse of radiofrequency (RF) energy is applied, the protons resonate, re-transmitting the absorbed radio waves in the form of an RF "echo." These signals are detected by an external antenna, and a computer then maps their position and intensity, producing an image of the scanned tissue (Bagley, 1999). Although the technique provides very sharp images, it takes a long time (about 45 minutes), and the patient cannot be monitored during the procedure.

MRI is superior to CT scans for certain types of traumatic brain injuries, particularly those involving the white matter of the brain. MRI is especially useful in the diagnosis of subacute and chronic stages of the neurologic sequelae of head injury (Gean et al., 1995; Bagley, 1999). In one study, MRI identified mass lesions in 80 percent of patients, 6 percent of which were not detected by CT (Jagoda and Riggio, 2000). Lesions which can be identified on MRI range from scattered punctate hyperintensities to the more obvious cerebral hemorrhages.

Although MRI is not typically used in the emergency management of severe head injury, it may provide useful information regarding neurologic outcome. For example, patients with subcortical white matter lesions or brainstem lesions visualized on early brain MRI usually have a longer duration of coma. Likewise, the presence of persistent parenchymal lesions correlates with severe cognitive deficits. Brain MRI performed several months after traumatic brain injury has a better prognostic accuracy than the initial MRI scan (Zink, 2001).

[6] Electroencephalographic (EEG) and Evoked Potential (EP) Monitoring-- Brain Stem Auditory Evoked Potentials (BAEPs) abnormalities have

been reported in mild head injury, but findings do not correlate with the severity or prognosis of the injury. Studies of BAEPs and EEG recordings have shown changes in the brain's electrical activity up to six weeks after injury, suggesting that mild head injury can cause both cortical and brainstem injury (Jagoda and Riggio, 2000).

[6a] Electroencephalography (EEG)-- EEG records spontaneous electrical activity of the brain noninvasively through scalp electrodes. The basic premise on which it functions is that traumatic structural and functional alterations of the brain give rise to EEG patterns in the damaged structures which differ from those produced by intact structures (Jordan, 1999; Guerit, 2000). Accurate assessments depend on good technique, an understanding of the situations in which EEG is most useful and the establishment of acute stage baseline recordings for comparing subsequent changes. Continuous EEG monitoring has recently been found to be beneficial for targeting the management of acute severe head trauma patients. In comatose patients, it can provide diagnostic and prognostic information which is otherwise unobtainable (Jordan, 1999; Guerit, 2000).

[6b] Evoked Potentials (EP)-- While the recording of EP is more involved than recording of EEG, the EP allows more precise localization of functional damage (Guerit, 2000). In essence, the EP is the response evoked from selected CNS neurons by an external stimulus applied to them by the technician. Sensory stimuli commonly used include visual (e.g., strobe light flashes), auditory (clicks or tones) and somesthetic (having to do with the sense of having a body, elicited in EP by depolarizing cranial or peripheral nerves) (Guerit, 2000).

Advances in EP technology permit the noninvasive recording of evoked potentials from scalp and neck electrodes. Clinical interpretation of EP recordings is based primarily on three characteristics of the waveforms: (1) the presence or absence of peaks, (2) the amplitude of peaks, and (3) the latency of peaks. Since the interpretation of EP waveforms in head trauma patients is based on comparison with the recordings from a normal population, strict standardization of technique is essential. Moreover, since there is considerable variation between laboratories with respect to equipment and technique, each EP lab must establish its own normal baseline.

The absence of a peak is generally an indication of frank structural and/or functional damage to the pathway being stimulated. Other reasons for an absent peak must be ruled out, however. Both barbiturates and seizure activity, for example, can cause the transient loss of EP peak, which returns when the drug is withdrawn or the seizure ends (Guerit, 2000). The amplitude of EP peaks is the most sensitive indicator of neuropathological changes, but is also the most variable aspect of normal waveforms.

Three-modality evoked potentials (TMEPs) have been used for many years along with the EEG as both a diagnostic and prognostic tool for patients who are comatose. The use of cognitive evoked potentials is more recent and when used in conjunction with continuous EEG monitoring provide information about the function of the cerebral cortex. Thus, cognitive evoked potentials can complement TMEPs as a prognostic tool. Their presence in comatose patients suggests a high probability (greater than 90 percent) of a complete recovery of consciousness (Guerit, 2000).

[7] Intracranial Pressure (ICP) Monitoring-- ICP monitoring can significantly reduce mortality and morbidity by allowing the administration of appropriate treatment before clinical deterioration of the patient becomes apparent (Morris and Marshall, 2000; Zink, 2001). Although monitoring of ICP requires neurosurgical intervention and the availability of an intensive care unit, it is necessary to properly manage increased ICP in the severely head injured patient with cerebral swelling. Monitoring also allows the clinician to maintain an adequate cerebral perfusion pressure (CPP) by manipulating the ICP and systemic blood pressures (Gedeit, 2001). Invasive monitoring is difficult in patients presenting with a coagulopathy. Three commonly used devices for monitoring ICP include an intraventricular catheter (used in direct ventricular cannulation), a fiberoptic monitor, and the subarachnoid bolt device (Dangor and Lam, 1999; Gedeit, 2001).

[7a] Intraventricular Catheter-- Cannulation of the cerebral ventricles using an intraventricular catheter is the most invasive ICP monitoring procedure. This is the ideal method of measuring ICP because it can be used to measure pressure and drain CSF to reduce pressure (Townsend, 2001). Since this procedure is invasive, it is

associated with a higher risk of infection. Other disadvantages of an intraventricular catheter is that it is difficult to insert in compressed ventricles, and the procedure may have to be discontinued if the ventricle collapses.

[7b] Fiberoptic Monitor-- A fiberoptic catheter can measure ICP epidurally, subdurally, or intraparenchymally and can be inserted at the bedside. It offers the advantage of a lower risk of infection compared to the use of an intraventricular catheter but does not allow drainage of the CSF (Dangor and Lam, 1999; Gedeit, 2001).

[7c] Subarachnoid Bolt-- The subarachnoid bolt can be inserted at the patient's bedside and utilizes the same monitoring sites as ventricular cannulation. Since the device is inserted into the subarachnoid space, the dura mater is penetrated but the cerebral cortex is not. Thus, the rate of infection is low (less than 2 percent versus a high of 20 percent for cannulation) (Dangor and Lam, 1999) and readings are independent of the size of the ventricles. Disadvantages include the lack of access to the CSF and ventricular system, and false readings resulting from occlusion of the lumen caused by the surface of the brain (Dangor and Lam, 1999).

[8] Cerebral Perfusion Pressure-- The maintenance of cerebral perfusion (cerebral blood flow) and lowering of intracranial pressure are the two primary goals of head injury management. By definition, cerebral perfusion pressure (CPP) is calculated as the difference between the mean arterial pressure and intracranial pressure (ICP). For infants and children, a CPP of 50 mm Hg or more is usually adequate; for adolescents and adults, the CPP must be maintained at a level greater than 60 mm Hg (Gedeit, 2001).

Optimization of CPP requires the maintenance of systemic hemodynamics and control of ICP. Since systemic hypotension (defined by a systolic blood pressure of less than 90 mm Hg) is associated with a poor outcome, aggressive fluid management is necessary to maintain the patient's intravascular volume. In addition, the presence of hypotension in the adult head-injured patient should prompt investigation of other sites of blood loss. Hypertension may be a compensatory mechanism to maintain CPP and for this reason, moderate increases in the patient's blood pressure (e.g., up to 160-180 mm Hg systolic) should not be

treated. However, severe hypertension should be treated because it may increase cerebral blood volume and may lead to vasogenic edema and precipitate hemorrhage in injured areas of the brain (Dangor and Lam, 1999).

[9] Lumbar Puncture-- Lumbar puncture is contraindicated in the acute stage of head trauma. If meningitis is a concern due to the presence of cerebrospinal fluid leakage from the nose or ears, or a fever, a CT scan should be taken prior to performing a spinal tap for identification of pathogens, in order to rule out unsuspected early abscess or delayed hemorrhage. Both of these lesions can cause brain herniation and rapid deterioration following lumbar puncture. If CT is not available, empiric administration of antibiotics is preferred to the risk of lumbar puncture (Dangor and Lam, 1999).

P 93.70 DEFINITIVE MANAGEMENT

The management of head trauma begins at the scene of injury (prehospital care by emergency medical technicians) and continues in the emergency department of the hospital. Up to this point, management is essentially acute emergency care, which must be thorough and rapid in order to get the patient into the operating room or the intensive care unit where definitive treatment is provided. In hospital management is a continuation of prehospital (emergency medical services) and emergency department care (Dangor and Lam, 1999).

[93.71] Surgical Management

Surgical treatment of head trauma is typically indicated for evacuation of mass lesions, repair of penetrating wound damage and debridement for control of infection. Specific lesions that necessitate surgery include subdural hematoma, epidural hematoma, large intracerebral hematoma/contusion, posterior fossa hematoma, depressed skull fractures, penetrating wounds and intracranial infection (Dangor and Lam, 1999).

[1] Subdural Hematoma-- Most patients with an acute or subacute subdural hematoma will require surgical evacuation of the lesion. A small subdural hematoma, however, may heal spontaneously without surgical intervention (Goetz, 1999).

The "golden hour" in trauma refers to the concept that the physician has only a short period of time in which to diagnose and treat brain injury before serious harm occurs. In applying this concept to closed head injury, the focus is on early diagnosis of expanding intracranial hematomas, so that surgical evacuation can be performed before secondary brain injury occurs. Studies show that early evacuation of subdural hematoma in adults leads to a decrease in morbidity and mortality associated with head injury (Schutzman and Greenes, 2001). Patients with intracranial hematoma have a better outcome if the hematoma is diagnosed and treated early, before neurologic deterioration begins.

An acute subdural hematoma is surgically removed by means of a large trauma craniotomy flap procedure, which involves excising a large plate of skull bone in the temporal region (side of the head above the ear). Owing to their large size, subdural hematomas cannot be evacuated adequately through burr holes (small holes drilled in the skull to facilitate removal of bone).

In many cases the patient will be undergoing such rapid neurological deterioration that emergency decompression is mandatory. Since burr holes do not provide adequate decompression of a large subdural hematoma, rapid temporary decompression is achieved by opening the incision through the dura as well as the skull along one side (temporal region), rather than taking the time to saw and remove the bone plate before opening the dura. Once the bone plate is removed and the dura fully opened, the clot is gently removed from the surface of the brain by a combination of irrigation, suction, and traction. This should be done with care to avoid causing further damage to the brain. A peripheral clot (on the side of the brain) may be removed using a brain retractor, but no attempt should be made to retrieve small pieces of the hematoma beneath the dura.

A large decompression is advocated for patients with intractable ICP. The decompression should include the dura and expansion of the dura should be accomplished by using a graft. However, the opening should be large enough so that the brain does not herniate through the surgically created hole, extinguishing its blood supply (Townsend, 2001).

If the brain remains swollen after removal of the hematoma, mannitol and increased

hyperventilation should be employed. Contusions on the tips of the frontal and temporal lobes should be resected. Any source of bleeding should be sought. When found, the vessel should be repaired with bipolar coagulation, Surgicel,(R) Gelfoam(R) or Avitene.(R) Control of bleeding should never be attempted by means of injection of saline (physiological sodium chloride solution) into the ventricular/cerebrospinal fluid system to cause brain expansion, as this causes further brain damage.

Once the evacuation and repairs are completed, the dura is closed and tacked tightly to the edges and center of the bone, to minimize the chance of a recurrent hematoma. A second compression of the brain under these circumstances is frequently catastrophic. The bone plate is replaced and secured with wire or sutures, and the scalp incision is closed (Townsend, 2001).

Because bleeding within the subdural space may recur, a surgical drain should be left in place for several days after surgery and the patient monitored carefully for signs of continued bleeding. (Goetz, 1999)

One of the pitfalls of rapid surgical decompression is extrusion of the brain through the craniotomy defect that occurs in response to acute brain swelling. To avoid this complication, a new surgical technique has been developed, in which multiple fenestrations are created in a mesh-like fashion so that clots can be removed through small dural openings. This technique allows for the gradual and gentle release of clots without further disruption of brain tissue (Guilburd and Sviri, 2001).

The treatment of chronic subdural hematomas remains controversial. If the patient with a chronic subdural hematoma becomes symptomatic, most neurosurgeons believe that surgical evacuation of the lesion should be performed (Marx, 2002).

[2] Epidural Hematoma-- In the case of acute epidural hematomas producing neurological deficits, emergency surgical evacuation is the standard treatment. Epidural hematomas are evacuated via the large trauma flap craniotomy used for managing subdural hematomas. Once exposed, the clot is removed and the torn blood vessel repaired. Bipolar coagulation usually suffices. Since additional hematomas not detected on CT often are discovered beneath the dura mater, the dura should be opened when it is made

taut by the bulging brain below. If an epidural hematoma is not found on the side where the craniotomy was made, burr holes should be placed to search for a lesion on the opposite side. When the dura is slack and preoperative CT has not disclosed additional lesions, it is not necessary to open the dura. Once all repairs have been made, the craniotomy is carefully closed (Townsend, 2001).

One recent study examined whether or not patients with epidural hematomas can be safely transported to a neurosurgical unit for decompression or if surgeons must still be prepared to perform emergency craniotomies. The authors of this study found that there is usually enough time to safely transfer patients to a neurosurgical unit, if transport is rapid and anesthesiological services are available during transport. They also concluded that surgeons without training in neurosurgery should not perform emergency craniotomies in local hospitals, but should instead transfer the patient immediately to the nearest department of neurosurgery (Wester, 1999).

[3] Intracerebral Hematoma and Contusion-- Hematomas within the tissue of the cortex are evacuated after craniectomy (excision of a portion of the skull) performed as described for subdural and epidural hematomas. Intracerebral hematomas usually are found within the frontal and temporal lobes. Since the surface of the brain over the intracerebral clot may appear normal, exploration with a blunt needle using ultrasound guidance often is useful. Once located, the clot is evacuated with gentle suction. No bleeding control is usually required. Deep intracerebral clots should not be evacuated unless they are larger than 1 or 2 cm and cause significant mass effects, such as brain shift, pronounced intracranial pressure elevation or neurological deterioration.

Contusions are most often found on the front and lower surface of the frontal and temporal lobes of the cerebral cortex. Those larger than 2 cm in diameter should be resected. The lesion is exposed by craniectomy and suction and retraction is applied to identify surrounding normal brain tissue. The contusion is removed by resecting from the center of the lesion outward, taking care to stay within the area of necrotic (devitalized) tissue. The extent of removal depends on the location of the lesion in the cortex. Extreme care must be taken in removing contusions further back and on

top of the temporal lobe, in the parietal or occipital lobes, or in the area of the central sulcus (the deep fissure separating the frontal and parietal lobes), as motor, language and vision functions are located in these areas. No excess neurological deficit is sustained by removing these lesions, however, as long as resection is strictly confined to the contused tissue (Townsend, 2001).

[4] Posterior Fossa Hematoma-- Hematomas in the posterior cranial fossa (the depression in the back of the base of the skull) are evacuated by a standard suboccipital craniotomy procedure (at the base of the back of the skull). This operation is best performed with the patient prone or in the "reclining park bench" position, as this allows better control of bleeding and prevention of air embolism. In addition, the patient can be maneuvered more readily if a contrecoup lesion, frequently found in association with posterior fossa hematomas, is present in the frontal or temporal lobes (Bozbuga, et al., 1999; Townsend, 2001).

[5] Depressed Skull Fractures-- Surgical treatment of skull fractures is generally reserved for depressed fractures in which the bone fragment is driven into the brain a distance greater than the thickness of the skull itself (about 2 to 6 mm). Depressed fractures are divided into closed or compound (open) types, depending on whether the overlying scalp is intact or has been lacerated. The general objective of surgical repair of depressed fractures is to restore the structural and functional integrity of the skull. Specific aims are prevention of infection, repair of any dural tears and intracranial bleeding and elimination of cosmetic deformities. If laceration of the scalp is extensive in a compound fracture, it may provide an adequate opening to the injury. Otherwise a scalp flap is created. Leveling of the fragment must be avoided, as it is likely to cause further laceration of the meninges and brain. If the bone fragment has not been devitalized, it may be wired in place. Otherwise, acrylic cranioplasty may be required. Dural tears should be repaired and bleeding controlled before closing (Asano et al., 1995; Townsend, 2001).

Traditionally, management of compound depressed skull fractures involved removal of all bone fragments followed by delayed cranioplasty, due to concern over the potential for developing infection. However, one recent study demonstrated that immediate replacement of bone fragments

(within 72 hours of injury) in compound depressed skull fractures is not associated with an increased risk of infection (Wyllen, et al., 1999).

[6] Penetrating Trauma-- In all penetrating projectile wounds of the head there is compound (open) fracture of the skull, dural tearing and laceration of the brain accompanied by bleeding. Consequently, these injuries should be treated as soon as possible to avoid complications from intracranial hematomas and infection.

Following emergency medical care, neurological exam and grading of neurological status as described earlier, all patients except those in Grade IV (brain death) should be prepared for immediate surgical treatment. The emergency diagnostic method of choice is CT. If the path of the projectile passes in the vicinity of a major blood vessel, however, cerebral angiography (x-ray of the blood vessels in the brain) should be performed if the patient's condition allows.

Large intravenous doses of appropriate antibiotics should be given perioperatively, during the operation, and for five days postoperatively to guard against the possibility of infection from the cranial wound and from the projectile.¹⁷ The use of antiepileptics prophylactically in patients with penetrating head injuries for the prevention of posttraumatic epilepsy is controversial. Mannitol should be given to control intracranial pressure from evolving brain edema and/or hematoma formation.

The surgical opening in the skull must be large enough to allow adequate visualization of the dural tear and lacerated brain beneath it. In virtually all civilian injuries, a craniotomy procedure may be used, whereby a plate of bone encompassing the entry site of the projectile is removed and all comminuted (splintered or broken) fragments are removed. This plate may be replaced later in closing. In situations where a significant risk from bone infection will persist, primarily among military casualties of war, a craniectomy (excision of the bone surrounding the entry site) must be used, followed later by cranioplasty (plastic surgical correction of the defect).

Upon opening of the skull, visual inspection will reveal the lacerated dura, pulped brain, clotted blood and bone fragments. After opening and retracting the dura, debris on the surface of the cortex is removed by gentle suction. Next, the

missile tract is debrided. This is the most crucial part of the surgery. If the entire length and bore of the track is debrided of devitalized and necrotic brain tissue (the limits of the debridement being the surrounding healthy white matter), the removal of hematomas and bone fragments as well as pulped brain usually can be accomplished. The projectile at the end of the tract should be removed as well. The number of bone fragments retrieved should be counted and compared with the count taken from the radiological image. If debridement has been thorough, the missile tract will not collapse. If the tract does collapse, either further removal of devitalized tissue is necessary, or a hematoma is present.

If the projectile has passed completely through the head, the exit wound is managed in a similar fashion to that of the entry wound. If the projectile has passed across to the other side of the brain, just short of exiting, the surgeon should not pursue it along the tract. Rather, a separate craniotomy should be performed nearer the site of lodgement. All easily accessible bullets and metallic fragments, especially those which are copper or copper-coated, should be removed at the primary operation. Large intact bullets should be removed as well, as they have a tendency to migrate over time. Small metallic fragments deeply embedded in the brain need not be removed. In all of such cases, a bullet or fragment can serve as a nucleus for the formation of an infectious abscess or hematoma.

Although the prognosis for nonpenetrating head injury has improved, the high mortality rate for patients who are comatose after missile injury appears to be the same. Non-comatose patients suffering penetrating head wounds almost always survive, albeit with neurologic deficits depending on the path of the bullet. In contrast, the mortality rate in the comatose patient approximates 90 percent in most studies. However, despite this high mortality rate, most trauma centers use the same medical management as for patients who are comatose with nonpenetrating injury. For most patients with gunshot wounds to the head, operative therapy of the missile tract is still debated. The current standard is surgical debridement of the first few centimeters of the tract followed by a watertight dural closure. Conflicting reports from the Middle East suggest that simple skin closure of the bullet hole, without any debridement, is not associated with a worse outcome (Townsend, 2001).

[7] Infection-- Surgical management of infection is indicated for osteomyelitis (infection of bone), subdural empyema (accumulation of pus beneath the dura) and brain abscess, all of which are sequelae of penetrating wounds or basilar skull fractures. For osteomyelitis, treatment entails surgical debridement and removal of infected bone and the administration of antibiotics. The choice of antibiotic depends on culture and sensitivity results. The presence of systemic symptoms suggests that the patient has an underlying subdural or epidural empyema. Usually, *Staphylococcus* species, *Streptococcus* species or various anaerobic pathogens are involved (Marx, 2002). Surgical treatment of subdural empyema is by drainage and debridement of the lesion.

Brain abscess is managed medically by intravenous antibiotics or surgically by aspiration and debridement, depending on the size of the abscess. If the brain abscess is relatively small (less than 2 centimeters in diameter), the abscess can be treated successfully with antibiotics without surgical intervention. The initial choice of antibiotic should be directed to the most likely causative microorganism (e.g., *S. aureus*) and be chosen based on culture and sensitivity results. Laboratory diagnosis involves culture of the purulent material obtained from the abscess and antibiotic sensitivity testing. *Staphylococcus aureus* is the most common microorganism causing brain abscess following compound skull fracture and penetrating head injury. Intravenous antibiotics are usually administered for six to eight weeks.

For larger abscesses, surgical aspiration and debridement of the abscess is usually required (Cochrane, 1999). The initial surgical approach to brain abscess is drainage through a burr hole (Townsend, 2001). Excision of the abscess by craniotomy may be necessary in some cases.

Although prolonged, pathogen-specific antibiotic therapy is a crucial component in the management of all these posttraumatic sequelae, antibiotics should not be given prophylactically in the ICU setting in patients with leakage of CSF caused by basilar skull fractures (Marx, 2002).

[8] Aneurysm-- Surgery is the recommended treatment, with surgical clipping of the aneurysm and/or endovascular occlusion being the definitive treatments of choice (Tureyen, 2001, Voekler and

Ortiz, 1997; Schuster et al., 1999).

[9] Arteriovenous Fistula-- Current treatment of choice is endovascular closure of the fistula, especially in patients with rapid neurologic deterioration. Endovascular repair is achieved by percutaneous endovascular balloonization of the fistula ring, although microcoils are also used. For dural arteriovenous fistulas involving the superior sagittal sinus, transarterial intravenous coil embolization is an effective treatment (Fukai, et al., 2001). Surgical closure of the fistula usually results in rapid resolution of symptoms with a good prognosis for complete neurologic recovery (Wadlington and Terry, 1999).

[93.72] Medical Management

The majority of head trauma patients require observation and medical management following emergency department evaluation. Those who are initially treated surgically also require medical management in the intensive care unit (ICU), once neurosurgery has been completed. The two principal causes of death from head injury, in general, are (1) uncontrolled elevated intracranial pressure, and (2) massive systemic insults, such as septicemia (bacterial infection of the bloodstream) and associated septic shock or cardiorespiratory failure (Townsend, 2001).

[1] Elevated Intracranial Pressure (ICP)-- The first line of treatment is drainage of ventricular fluid by placement of a ventricular catheter. If the ICP still remains elevated, hyperosmotic agents such as mannitol or diuretics are used. The role of hyperventilation is controversial, and most clinicians believe this treatment option should not be used as it potentially results in widespread ischemia (Zink, 2001; Townsend, 2001; Naik-Tolani, et al., 1999). Optimum therapy is to maintain the ICP below 20 and the cerebral perfusion pressure (CPP, an index of perfusion, calculated as the difference between the mean arterial pressure and the ICP) above 70 mm Hg. When the ICP remains refractory to treatment, a CT scan should be performed to look for a newly formed or expanding mass lesion. The presence of an epidural or an acute subdural hematoma requires emergency surgical decompression (Townsend, 2001).

[2] Thrombosis and Infarction-- Therapy for these injuries presently is limited. Anticoagulant

therapy is contraindicated in trauma patients and attempts at surgical removal of thrombi (thrombectomy) usually are futile. Treatment is currently directed at control of elevated intracranial pressure, the precipitating factor associated with thrombosis and infarction (Townsend, 2001). The instillation of urokinase should be considered in any patient with venous sinus thrombosis and symptomatic occlusion (D'Alise, et al., 1998).

[3] Clotting Defects-- Therapy for disseminated intravascular coagulation (DIC) is directed at the underlying causes of the excessive bleeding or clotting. Excessive bleeding may be managed by infusion of whole blood, packed red blood cells, crystalloids, fresh frozen plasma or platelet concentrates. Some clinicians have suggested, however, that the introduction of these blood components may further aggravate the condition (Townsend, 2001). Excessive clotting activity has been treated with heparin (an anticoagulant), but this rarely is effective. Moreover, the use of anticoagulant therapy in a trauma patient is a measure many clinicians are uncomfortable with (Townsend, 2001).

[4] Syndrome of Inappropriate Secretion of Antidiuretic Hormone (SIADH)-- Treatment entails conservative management of water balance. Although SIADH is usually an acute self-limited disorder that resolves within two to three weeks, the condition may be chronic if brain injury is severe. In general, if the patient has minimal symptoms, and the SIADH lasts longer than 24 to 48 hours, restricting the patient's total fluid intake will correct the osmotic imbalance and the SIADH will remit spontaneously. In contrast, if the patient has severe hyponatremia (low serum sodium) that is present for less than 24 to 48 hours, which is accompanied by nausea, vomiting, coma or seizures, the SIADH should be corrected more rapidly by combining fluid restriction with a slow intravenous infusion of hypertonic (3 percent) saline (Robertson, 2001).

Whatever treatment is utilized, the patient's urine output and serum sodium should be closely monitored because the SIADH can suddenly resolve, causing a brisk diuresis that raises the serum sodium more rapidly than what is considered safe. The serum sodium should be raised no faster than 24 mEq/L in 24 hours and to a level no greater than 135 mEq/L. Raising the serum sodium faster is associated with acute

osmotic demyelination, which is characterized by severe neurologic disturbances, including mutism, seizures and movement disorders (Robertson, 2001).

[5] Infection-- Infection is an infrequent complication in head trauma patients as a group. The risk is increased for those with penetrating head wounds and whose injuries, whether penetrating or closed, are severe and require a prolonged stay in the intensive care unit. General hospital-acquired infections are treated by standard antibiotic therapies. The incidence of infections directly related to penetrating craniocerebral wounds has been reduced dramatically by improved diagnostic and surgical procedures and the routine use of antibiotics.

Bacterial meningitis was a significant cause of morbidity and mortality in the past, but has now been practically eliminated by the availability of more effective cephalosporin antibiotics. Patients with a CSF leak that develops from a basilar skull fracture will typically present with early meningitis (occurring within three days of injury) which is usually caused by *Streptococcus pneumoniae*. A third-generation cephalosporin such as ceftriaxone or cefotaxime is the antibiotic of choice, but culture and sensitivities should be obtained due to the increase in resistance of these antibiotics to pneumococci. If sensitivity data are known and the bacteria are highly resistant to this antibiotic regimen, vancomycin should be added.

In contrast, meningitis which develops more than three days after head trauma is usually caused by gram-negative organisms. In this case, a third-generation cephalosporin should be combined with nafcillin or vancomycin to provide adequate coverage against *Staphylococcus aureus*. In children, posttraumatic meningitis is often caused by *Haemophilus influenzae* (Marx, 2002).

[6] Posttraumatic Epilepsy-- In the emergency department, acute posttraumatic seizure prophylaxis is recommended for head-injured patients who will be paralyzed by neuromuscular blocking agents, for purposes of mechanical ventilation, surgery, or transport, because the clinical manifestations of seizures are subdued in these patients. Phenytoin (15 mg/kg) is the anticonvulsant of choice for seizure prophylaxis. Whether or not the head-injured patient is maintained on long-term anticonvulsant therapy during the recovery period depends on the

patient's subsequent clinical course. Not all patients who have suffered posttraumatic seizures in the acute or subacute period require long-term seizure prophylaxis and the role of anticonvulsants for this purpose remains unclear (Marx, 2002).

[93.73] Psychological Testing

The differential diagnosis for psychological sequelae from traumatic head injury must take three possible considerations into account:

- (1) the patient has sustained genuine psychic injury due to organic brain damage;
- (2) the psychic injury existed prior to physical insult and has been aggravated by it; or
- (3) the patient has not sustained psychic injury but feigns it for the purposes of compensation or secondary gain (otherwise known as "malingering").

Every physical injury has emotional consequences, but these must be distinguished from severe, incapacitating and permanent psychic disability. A battery of psychological tests has been devised to aid in the diagnosis of true psychopathology as a consequence of physical insult. By themselves, these tests are not considered diagnostic, but when considered in conjunction with the patient's history, neurological examination, radiological studies and preinjury psychological test results, they can yield significant information useful in the treatment and rehabilitation of the individual with head injury.

In the case of traumatic brain injury, the key issue is the proportionality of injury when performing a neuropsychologic assessment. It is well established that mild and most moderate closed head injuries do not produce alterations in results of tests depending on the patient's overall fund of information such as reading and vocabulary. If a patient that has been involved in a motor vehicle accident claims that he or she has suffered a brain injury, a neuropsychologist can perform tests to determine whether or not changes in mental performance are indeed neurologic. For example, a patient with a Verbal Performance Score which is significantly lower than what was measured before the accident, has most likely not experienced this change due to a mild head injury.

In contrast, if the patient is suffering from short-term memory problems, attentional deficits, difficulties in processing information, and cognitive speed impediments, it is probable that these changes were caused by closed head injury (Boll, 2000).

[1] Objective Personality Tests-- When used in psychological testing, the term "objective" refers to personality tests that employ true-false, yes-no or multiple choice questions that specifically limit the patient's answering options. In this context, objectivity is understood to mean that different persons with similar expertise examining the same patient data would be able to arrive at similar conclusions regarding the examinee's condition. Objectivity does not mean that the results of these tests can be proven to be objectively true. Indeed, a drawback of these tests is that they present patients with answers that are recognizably more socially acceptable, allowing fakers and malingerers to "fake good" or "fake bad."

Personality assessment is not critical because of the widespread availability of validated instruments to assess the role of neurological injury in changes in personality. In fact, one may argue that if a person is neurologically impaired, the results of personality tests may be questionable and may require further assessment using factors for which there are no adequate normative data. Nevertheless, personality assessment can provide valuable information on difficulties in cognitive functioning, which can result in anxiety and depression, and other forms of personality change (Boll, 2000).

[1a] The Minnesota Multiphasic Personality Inventory-- The Minnesota Multiphasic Personality Inventory (MMPI) is the most useful objective personality test. It consists of 13 test scales: three validity scales which are used to identify deliberate faking, and 10 clinical scales each of which assess a particular behavior or personality characteristics (e.g., depression, hysteria, paranoia). These scales are meant to be integrated in such a way that psychopathology indicated by a deviant answer in one scale should be reinforced by similarly deviant answers on other test scales (e.g., elevations of the schizophrenia scale would be supported by the results of the scales used to test depression, paranoia, hypomania and other symptoms).

Of the validity scales, the L scale measures the

patient's willingness to admit to common human frailties such as anger or dislike of others; a high score often points to intent to fake good. The F scale measures answers to outrageous or bland and generally accepted statements; a high score usually indicates intent to fake bad.

The ten clinical scales can be summarized as follows:

Scale 1: assesses a hypochondriacal personality as determined by responses concerning body functions and somatic complaints.

Scale 2: assesses depressed mood through answers to questions regarding vegetative symptoms and feelings.

Scale 3: assesses hysteria as a foundation for somatic complaints.

Scale 4: assesses psychopathic deviance through questions concerned with relationships to authority figures and family, as well as antisocial activity.

Scale 5: assesses whether the patient has traditional culturally defined masculine or feminine interests.

Scale 6: assesses paranoia through questions regarding the individual's perceived treatment at the hands of others.

Scale 7: assesses hypaesthesia, or the obsessive-compulsive syndrome.

Scale 8: assesses schizophrenia through questions regarding how different or isolated the person feels from others.

Scale 9: assesses manic disorder through questions that elicit the persons' level of restlessness, energy and sense of self-importance.

Scale 0: assesses social introversion or shyness.

Scores on all of these scales are synthesized to determine a personality profile of the examinee and a probable diagnosis of psychopathology based on that profile. A high score on any one scale is not considered diagnostic of that particular problem (i.e. a high score on the schizophrenia scale should not be interpreted to mean that the person is schizophrenic).

[1b] Other Objective Personality Tests-- A variety of other personality tests are used either to augment the MMPI or arrive at information other than possible psychopathology. The California Psychological Inventory (CPI) is used to identify personality types, particularly among problem populations such as delinquent students and parolees. The Millon Behavioral Health Inventory employs three scales to measure the coping abilities of persons with organic illness. The Millon Clinical Multiaxial Inventory uses 175 self-descriptive statements to arrive at the same personality profiles as the MMPI.

[2] Projective Personality Tests-- Projective personality tests are open-ended tests that allow a patient to project his or her thoughts onto test material to be interpreted by the examiner. Thus, there is no obvious socially desirable "answer" to these tests. Results of these tests do not meet the requirements of standardization, reliability and validity of clinical diagnostic tests, and interpretation is thus often controversial.

The most widely used projective personality test is the Rorschach Inkblot Test, in which the patient is shown ten cards with inkblot patterns, some in black and white and some in color, and asked to identify what the inkblot looks like. The rationale for the test is based on the assumption that a person organizes various environmental stimuli according to his or her fears, wishes, conflicts, motives and other determinants. Since inkblots are so amorphous in shape, a person's need to organize his or her perceptions is increased to greater than normal levels when asked to interpret this material. After the person describes what the inkblots look like the examiner conducts an inquiry to aid in scoring responses.

Another widely used projective test, the Thematic Apperception Test (TAT) consists of 30 cards depicting ambiguous situations, mostly involving people. The individual is asked to make up a story describing what led up to the scene, what is happening, what the people in the picture are thinking or feeling, and how the situation turns out. The TAT provides information on interpersonal relationships, perception of the environment, personal needs and conflicts.

Projective drawing tests include the Draw-A-Person Test (DAP), the House-Tree-Person Test (HTP) and the Draw-A-Family Test. As their

names imply, these tests ask the examinee to draw a particular type of picture. The results are interpreted to tap into the person's non-verbalized feelings regarding what the scenes depict.

[3] Intelligence Tests-- Intelligence tests measure the capacity of an individual to act purposively and effectively in dealing with his or her environment. The score arrived at reflects the current functioning of the individual as influenced by such factors as hereditary potential, early learning, cultural background, emotional and physical state, motivational level and situational factors operating at the time of testing. Interpretation involves a careful evaluation of scores for the different subsections of the test to assess the degree to which each of these factors influences current functioning. Intelligence quotient, or I.Q., is measured as a percentile rank within a population or on a scale where a mean score of 100 represents normal.

The Wechsler Adult Intelligence Scale - Revised (WAIS-R) is the most widely used test of intelligence among adults. It is comprised of six verbal subtests: information, comprehension, arithmetic, similarities, digit span and vocabulary, and five performance subtests: digit symbol, picture completion, block design, picture arrangement and object assembly. The test generates a verbal I.Q., a performance I.Q. and a full-scale I.Q. A higher verbal than performance I.Q. sometimes indicates organic brain damage. A higher performance than verbal I.Q. may indicate schizophrenia and personality disorders.

Other intelligence tests include the Stanford-Binet test and the Shipley-Hartford test. The Stanford-Binet test measures intelligence levels appropriate for the age of the person. The Shipley-Hartford Test uses 40 vocabulary items and 20 abstract reasoning questions to generate a verbal intelligence score and a conceptual quotient obtained by dividing the abstract reasoning score by the vocabulary score and multiplying by a factor of 100. The Shipley-Hartford test is usually used for detecting organic brain damage or psychopathological emotional disorders.

[4] Neuropsychological Tests-- Neuropsychological tests assess more subtle and important abilities than those measured on most intelligence tests, and thus are more specific for organic brain damage. The WAIS-R is sometimes

considered such a test, since organic brain damage interferes with intellectual function, and thus is one potential indicator of brain damage.

A number of tests exist to measure sensory and perceptual functioning. For example, many complex language tests such as the Boston Diagnostic Aphasia Examination and the Western Aphasia Battery are used to measure difficulties with language functioning. The Halstead Category Test and the Wisconsin Card Sorting Test are two of the most common tests used to assess nonverbal learning, problem solving and mental flexibility. For litigation involving head injury, the Wisconsin Card Sorting Test is useful in the detection of malingering in patients suspected of faking a brain injury, having a reported sensitivity of 82 percent and specificity of 93 percent (Suhr and Boyer, 1999). Similarly, the Word Memory Test, a relatively new computer based test, is useful in measuring biased responding (e.g., malingering) in patients with mild head injuries claiming they have suffered moderate to severe brain injuries (Green, et al., 1999).

In addition to individual tests of cognitive function, a variety of specific battery tests have been developed which provide information across a broad range of known domains of brain behavior relationships. These tests represent the most scientifically validated approach to neuropsychologic evaluation of the head-injured patient (Boll, 2000).

One such battery is the Halstead-Reitan Neuropsychological Battery, which consists of seven subtests of ability to categorize, tactile performance, auditory discrimination and motor speed. Diffuse brain damage is suggested by impairment on three of the seven tests and strongly indicates impairment on four. When localized damage is present a pattern of deficits consistent with clinical examination, history and other test results is usually elicited.

The Luria-Nebraska Psychological Battery is another series of tests composed of 269 items covering various components of neuropsychological investigation: intellectual processes, expressive speech, receptive speech, writing, arithmetic, reading, visual functions, motor functions, tactile function, rhythmic and pitch skills, memory, etc. Scores help determine whether pathology is in the left or right brain hemispheres.

The Bender Gestalt Test is used to determine if organic brain damage is present, as a projective personality test, and as a test for emotional disorder. It consists of nine cards containing simple designs which the subject is asked to reproduce as accurately as possible on a blank sheet of paper. The resulting drawings are then scored in terms of accuracy and general integration. A number of characteristics in the drawings of persons with organic brain damage distinguish their drawings from those of non-brain damaged subjects: perseveration (the subject responds in a way beyond what is required), rotation and reversals (the drawing deviates by more than 45 degrees from the axis of the original or is drawn in reverse) and concretism (the drawn image looks more like an actual object than the more abstract designs on the cards).

Other neuropsychological tests include the Wechsler Memory Scale, in which evidence of a memory quotient lower than the intelligence quotient suggests brain damage, and the Graham-Kendall Memory for Designs, in which a score of 12 mistakes or more in accurately reproducing 15 simple designs from memory is considered a good indication of organic brain damage.

It is important to keep in mind that neuropsychological tests were not designed to predict how a head-injured patient is likely to function in real-world settings, such as vocational settings. Predictions using these tests are much more accurate if the tasks used during testing mimic the patient's everyday personal and vocational demands (Sbordone, 2001).

[93.74] Neuropsychological Management and Rehabilitation

Advances in the medical management of severe head injury have contributed to significant increases in the survival rate, as well as the medical challenges that people face with disabilities. Increased awareness of this problem and its consequences for society has led to extensive growth in the rehabilitation industry. Since clinical rehabilitation services vary widely, many clinicians are interested in the effectiveness of rehabilitation for brain injury on patient outcomes (AJCPR, 1999; Rao and Lyketsos, 2002).

It is generally accepted that supportive individual

and family psychotherapy is important for dealing with the neurobehavioral changes in the head-injured patient. For both patient and family, the patient's cognitive deficits represent one of the greatest challenges to coping and adaptation. The field of cognitive rehabilitation emerged in the early 1980s and has addressed these issues. Since its introduction, this field has shown impressive growth and application. Cognitive rehabilitation involves retraining in various cognitive skills, such as problem-solving, inductive reasoning, abstract thinking, logical thinking, memory, fine perceptual motor skills, practical mathematics, spelling and remedial reading. The ultimate goal is to promote carry-over of these skills to the patient's everyday life.

Since cognitive rehabilitation has grown in popularity, the Agency for Health Care Policy and Research (AHCPR) has examined the effectiveness of cognitive rehabilitation on outcomes for people who sustain a head injury (AHCPR, 1999). According to their report, compensatory cognitive rehabilitation (CCR) reduces the level of anxiety and improves the patient's self-concept and interpersonal relationships. Computer-aided cognitive rehabilitation (CACR) also improves a patient's immediate recall on neuropsychological testing, but the clinical significance of this finding has not yet been validated (AHCPR, 1999).

Other factors that are associated with improved outcomes include early rehabilitation in patients with severe head injury, supported employment in the vocational setting, and appropriate case management. While case management may only have an indirect effect on the patient's functional outcomes such as level of disability, it can directly affect family knowledge of the need for rehabilitation, the level of psychosocial anxiety, and the family's ability to cope with a traumatic brain injury (ACHPR, 1999).

P 93.80 PROGNOSIS

Determining the prognosis following head injury in a given patient is of inestimable value for providing optimal care for the patient and counseling for the family (Zink, 2001). More specifically, prognostic indicators serve three major functions in head trauma:

(1) they allow continuing evaluation of new treatments by comparison with the outcomes

expected from standard therapies;

(2) in the acute (emergency) stage of evaluation and management, they provide triage guidelines which aid physicians in identifying those patients almost certain to die or be vegetative, thus avoiding inappropriate and expensive heroic measures; and in patients for whom intensive life-saving care is warranted, they aid in charting the patient's moment by moment course (deterioration or improvement); and

(3) they allow advance counseling and preparation of family and social service agencies with regard to the kinds of long-term care the patient will need (Zink, 2001).

[93.81] General Considerations and Indicators

Some general indicators of prognosis following head trauma include the following:

Age. The rates for both disability and mortality tend to increase with increasing age.

Length of Coma. In general, there is a direct correlation between the length of coma and the probability of permanent cognitive deficits.

Posttraumatic Amnesia. Posttraumatic amnesia lasting less than 1 week is associated with a good recovery; amnesia that persists beyond the first week is a less favorable sign.

Location and Extent of Brain Lesion. Bilateral lesions in the frontal lobes are predictive of marked personality changes; bilateral lesions in the temporal lobes are predictive of permanent cognitive deficits.

Blood and CSF Findings. Persistently elevated levels of lactic acid in the blood and cerebrospinal fluid is evidence of continued ischemia (lack of blood supply) and hypoxia (insufficient oxygen supply) of the cerebral tissues, conditions likely to result in permanent neurological and cognitive deficits (Zink, 2001).

Responsiveness. Movement or other coherent response to verbal commands is a favorable sign.

Glasgow Coma Scale. Each of the elements of the Glasgow Coma Scale (motor score, eye opening, and verbal score) has a prognostic value in that

lower scores are associated with worse outcomes. Of the GCS components, the motor score component has the most prognostic value in terms of predicting outcome, based on the eventual score achieved with the Glasgow Outcome Scale.

One study examining early predictors of mortality and morbidity after severe closed head injury found that age was the only important predictor of outcome on the Glasgow Outcome Scale, the Glasgow Coma Scale score was the best predictor of neuropsychological function, and pupillary reactivity was the most predictive factor for quality of life. Factors important for predicting mortality (such as intracranial pressure, blood glucose levels, or CT observations) were not correlated with morbidity (Lannoo, 2000).

Serious head injury in children is associated with a poor prognosis. Of the 10 to 15 percent of children hospitalized for a serious head injury, 33 to 50 percent will die. While survivors of severe head injury often have permanent disability, children with mild or moderate head injury are also at risk for long-term cognitive and motor dysfunction (Gedeit, 2001).

To enhance the prognosis in head injury cases, management with ventilation and other supportive measures is indicated for patients in chronic coma. Patients who are conscious but paralyzed require physical and occupational therapy. Patients who are conscious and apparently not significantly impaired in any way need to receive thorough intellectual and psychosocial evaluations prior to release, since many who appear well have in fact sustained subtle alterations of personality and deficits of attention, learning and memory capabilities. Although poor Glasgow Outcome Scale scores and severe cognitive impairments are usually associated with more severe head injuries, moderate head injuries can cause similar problems and the needs of these patients should not be overlooked (Hellowell, 1999).

[93.82] Statistical Prognostic Systems

An objective and highly accurate means of establishing a prognosis for individual patients remains an elusive ideal. Given the complexity of the clinical situation in head trauma, it is unlikely that an infallible system ever will be developed.

Despite the difficulties involved, the application of

various statistical methods to the problem of assessing a patient's probable outcome is seen as a valuable clinical tool by some authors (Zink, 2001). A widely accepted standard system for describing the outcome of head injury is the Glasgow Outcome Scale (GOS) (Zink, 2001). With the GOS, patients are placed in one of 5 categories, ranging from dead to good outcome:

Category	Description
1	Dead
2	Vegetative state
3	Severe disability
4	Moderate disability
5	Good recovery

The GOS score is usually determined at 3, 6, and 12 months after the initial posttraumatic injury (Zink, 2001). A number of statistical methods have been devised for assigning a patient to a given outcome (Zink, 2001). Two of these methods are the linear logistic regression method (LLRM) and the discriminant analysis method (Zink, 2001). Two basic classes of prognostic indicators that may be used in these systems are investigational indicators (e.g., CT scan, evoked potentials, intracranial pressure) and clinical indicators (e.g., age, presence of a mass lesion, pupillary light response).