Chapter 6: Head Trauma

Objectives:

Upon completion of this topic, the physician will be able to demonstrate the techniques of assessment and explain the emergency management of head trauma.

Specifically, the physician will be able to:

A. Review specific principles of anatomy and physiology related to head injuries.

B. Identify and discuss the principles of general management of the unconscious patient and delayed complications.

C. Outline the method of evaluating head injuries using a minineurologic examination.

D. Identify and discuss management techniques for specific types of head injuries.

E. Demonstrate the ability to assess various types of head, maxillofacial, and neck injuries, using a head-trauma manikin.

F. Discuss clinical signs and outline priorities for the initial management of injuries identified during patient assessment.
I. Introduction

Because a head injury occurs every 15 seconds, and a patient dies of a head injury every 12 minutes, a physician dealing with trauma is confronted with a head-injured patient almost every day. Approximately 50% of all trauma deaths are associated with head injury, and more than 60% of vehicular trauma deaths are due to head injury. Because of the immense importance of head injury, the physician who sees patients soon after injury, but is not expert in the comprehensive management of head injuries, must develop a practical knowledge of the initial care of these patients. A neurosurgeon may not be available immediately when a patient with a central nervous system injury is first seen. Therefore, one of the most important responsibilities of physicians initially evaluating such a patient is in the management of ventilation and hypovolemia, thereby obviating potential secondary brain damage. Therefore, a system that incorporates consultation with a neurosurgeon, whose opinion may modify the management of such a patient, is essential. Early transfer of appropriate patients substantially reduces morbidity and mortality.

When conferring with a neurosurgical consultant about a patient with a head injury, the physician should relay the following patient information:

1. Age of patient and mechanism of injury.
2. Respiratory and cardiovascular status.
3. Results of the neurologic examination, especially the level of consciousness, pupillary reactions, and presence of lateralized extremity weakness.
4. Presence and type of noncerebral injuries.
5. The results of diagnostic studies, if obtained.

Neurosurgical consultation or patient transfer should not be delayed to obtain diagnostic studies, eg, computed tomographic (CT) scan or skull roentgenograms.

II. Anatomy and Physiology

As with all other aspects of medicine, the basis for a rational decision is the integration of the knowledge of anatomy and physiology with the history and physical examination. A short review of these subjects follows.

A. Scalp

The scalp is made up five layers of tissue covering the bone of the top of the skull (calvarium): (1) skin, (2) subcutaneous tissue, (3) galea aponeurotica, (4) a layer of loose areolar tissue, and (5) periosteum, the "pericranium". The loose areolar tissue, separating the galea from the pericranium, is subject to the commonly encountered subgaleal hematomas, large flaps caused by injury and "scalping" injuries. Because of the scalp's generous blood supply, bleeding from a scalp laceration can result in major blood loss, especially in children.
B. Skull

The skull is composed of the cranial vault (calvarium) and the base. The calvarium is especially thin in the temporal regions. The base of the skull is irregular and rough, allowing injury to occur as the brain moves within the skull during acceleration and deceleration.

C. Meninges

The dura is tough, fibrous membrane that adheres firmly to the internal surface of the skull. Because it is not attached to the underlying arachnoid, a potential space (the subdural space) exists into which hemorrhage can occur - usually from veins that traverse the space (bridging veins). In certain places the dura splits into two surfaces forming venous sinuses that provide the major venous drainage from the brain. The midline (superior) sagittal sinus is especially vulnerable to injury.

Meningeal arteries lie between the dura and the internal surface of the skull (epidural). The courses of these arteries may be visible on plain skull roentgenograms, if the vessels groove the inner surface of the skull. Laceration of these arteries may result in an epidural hematoma.

Beneath the dura is a second meningeal layer, the thin transparent arachnoid. The third layer, the pia, is firmly attached to the brain cortex. Between the arachnoid and the pia (subarachnoid space), the cerebrospinal fluid (CSF) circulates. Hemorrhage into this space is, by definition, subarachnoid hemorrhage.

D. Brain

The brain consists of the cerebrum, the cerebellum, and the brain stem. The cerebrum is composed of right and left hemispheres that are separated by the falx, a dural reflection. The left hemisphere, which usually contains the language centers, is often referred to as the dominant hemisphere. The frontal lobe is concerned with emotions and motor function, the occipital lobe with vision, and the parietal lobe with sensory function. The temporal lobe regulates certain memory functions, but can be a relatively silent region on the right side.

The brain can be compared with a funnel. The two cerebral hemispheres comprise the funnel portion. The brain stem, the major neural pathways to and from the hemispheres, comprises the neck of the funnel. The brain stem is composed of the midbrain, the pons, and the medulla. The midbrain and upper pons contain the reticular activating system, which is responsible for an "awake" state. Vital cardiorespiratory centers reside in the lower brain stem, the medulla, which then continues to form the spinal cord. The cerebellum, which controls movement, coordination, and balance, surrounds the pons and medulla in the posterior fossa.

E. Cerebrospinal Fluid

Cerebrospinal fluid is produced by the choroid plexus and is discharged into the ventricles of the brain. The fluid leaves the ventricular cavities to circulate through the
subarachnoid space.

F. Tentorium

The tentorium divides the head into the supratentorial compartment (comprising the anterior and middle fossae of the skull) and the infratentorial compartment (containing the posterior fossa). The midbrain courses from the cerebrum through the large aperture (incisura) in the tentorium. The third cranial nerve also passes along this opening. Any pathologic process that rapidly increases supratentorial pressure, most commonly hemorrhage or edema, can force the medial aspect of the temporal lobe (the uncus) through this opening. The result (uncal or tentorial herniation) causes compression of the third nerve, producing an ipsilateral and dilated, fixed pupil. Contralateral spastic weakness of the arm and leg, caused by compression of the corticospinal (pyramidal) tract within the cerebral peduncle on the side of herniation, is also an integral component of uncal herniation.

G. Consciousness

Alteration of consciousness is the hallmark of brain injury.

Two types of brain injury - bilateral injury to the cerebral cortices and injury to the reticular activating system of the brain stem - can produce unconsciousness. Increased intracranial pressure and decreased cerebral blood flow, regardless of the cause, can also depress the level of consciousness.

H. Intracranial Pressure

Intracranial pressure can be described as a relationship among volumes of CSF, blood, and brain, and expressed by the equation of the modified Monroe-Kellie hypothesis:

$$K_{ICP} = V_{CSF} + V_B + V_Br$$

Constancy ($K$) of the intracranial pressure ($ICP$) relates to the volume ($V$) of the cerebrospinal fluid (CSF), plus the volume ($V$) of blood ($B$), plus the volume ($V$) of the brain ($Br$).

The volume of CSF is altered by varying rates of absorption depending on the intracranial pressure, since CSF production is constant. Intracranial venous blood volume depends on rates of passive drainage influenced by intracranial pressure and intrathoracic pressure. The arterial blood volume is affected by autoregulation, arterial $PCO_2$, and may be decreased by autoregulation with hyperventilation. Brain volume is relatively static, except when cerebral edema occurs.

As the volume of mass from an expanding hematoma or cerebral edema increases, CSF or venous blood (or both) are removed from the intracranial compartment at a greater rate than normal. The result of these adjustments is that the intracranial compartment may accommodate an enlarging mass to a volume of 50 to 100 mL, depending on location and rapidity of enlargement. When all normal compensatory measures are exhausted, further small increases in intracranial mass volume cause very large increases in intracranial pressure,
resulting in diminution of cerebral perfusion pressure. When the mass reaches a critical size, further increases in volume result in a rapid increase in intracranial pressure with brain herniation likely.

Cerebral perfusion pressure is, approximately, the mean systemic arterial pressure minus the intracranial pressure. It is a measure of arterial perfusion of brain tissue, and normally it is greater than 50 mm Hg. Initial elevations of intracranial pressure are often associated with compensatory rises in mean systemic arterial pressure. However, further elevations of intracranial pressure are at the expense of cerebral perfusion pressure (ie, cerebral blood flow), and result in brain ischemia, leading to alterations in the level of consciousness. Brain death occurs when intracranial pressure approaches systemic arterial pressure, thus preventing any blood flow to the brain.

The definitive management of increased ICP is substantially aided by direct measures of arterial, central venous, and intracranial pressures under the direction of a neurosurgeon.

III. Assessment of Head Injuries

A. History

To make the correct management decisions, it is helpful to know what kind of head injury can result from a certain kind of trauma. The cause is usually obvious, but even though a patient may appear to be in good neurologic condition, mechanism-of-injury risk factors should be assessed in deciding how and where to treat the patient (See Chapter 12, Stabilization and Transport.) For example, simply knowing that the patient was injured in a fall instead of vehicular crash quadruples the patient's risk of having an intracranial hematoma.

Prehospital personnel should question observers at the scene about the patient's condition immediately after the incident. Information obtained by ambulance personnel should be documented in writing and provided to the hospital personnel when the patient is delivered to the emergency department.

B. Initial Assessment

The importance of initial assessment is emphasized because it provides the baseline for sequential reassessment, which is the critical basis for many subsequent decisions in patient management.

Because many factors influence the neurologic evaluation, documentation of the cardiorespiratory status must accompany the neurologic examination. Airway, breathing, and circulatory control and management must take priority. Care must be exercised in attributing changes in the patient's mental status to head injury if the systolic blood pressure is less than 60 mm Hg or systemic hypoxemia exists. Similarly, a drug screen for alcohol and other nervous system depressants must be obtained, so that toxic factors that may influence head injury assessment are taken into account.
C. Assessment of Vital Signs

Although brain injury may alter the vital signs, it is very difficult to be sure that changes in the vital signs are due to head injury and not to other factors. Some useful clinical rules include:

1. **Never presume that brain injury is the cause of hypotension.** Although bleeding from scalp lacerations can cause hemorrhagic shock (especially in small children), intracranial bleeding will not produce shock from blood loss alone. Hypotension arising from the brain injury is a terminal event, resulting from failure of medullary centers.

2. The combination of progressive hypertension associated with bradycardia and diminished respiratory rate (Cushing response) is a specific response to an acute and potentially lethal rise in intracranial pressure. In head injury, the course is usually a lesion demanding immediate operative intervention.

3. Hypertension alone or in combination with hyperthermia may reflect central autonomic dysfunction caused by certain types of brain injury.

D. AVPU + Minineurologic Examination

The AVPU mnemonic is supplemented by a minineurologic examination that is directed toward determining the presence and severity of gross neurologic deficits, especially those that may require urgent operation. The AVPU method, completed in the primary survey, describes the patient's level of consciousness. (See Chapter 1, Initial Assessment and Management and Chart 2, Revised Trauma Score in Chapter 12, Stabilization and Transport.)

The minineurologic examination should be conducted repeatedly for any patient with a head injury. The examination assesses: (1) level of consciousness, (2) pupillary function, and (3) lateralized extremity weakness. **Generally, a patient with abnormalities of all three components will have a mass lesion that may require surgery.**

1. **Level of consciousness**

a. **Glasgow Coma Scale**

The Glasgow Coma Scale (GCS) provides a quantitative measure of the patient's level of consciousness. The GCS is the sum of scores for three areas of assessment: (1) eye opening, (2) verbal response, and (3) best motor response. Each is graded separately.

1) **Eye-opening response** (E score)

Scoring of eye opening is not valid if the eyes are swollen shut. This fact must be documented.

a) Spontaneous - already open with blinking (normal): **E = four (4) points**

b) To speech - not necessarily to a request for eye opening: **E = three (3) points**
c) To pain - stimulus should not be applied to the face: \( E = \text{two (2) points} \)

d) None: \( E = \text{one (1) point.} \)

2) Verbal response (V score)

Scoring for verbal response is invalid if speech is impossible, for example, in the presence of endotracheal intubation. This circumstance must be documented.

a) Oriented - knows name, age, etc: \( V = \text{five (5) points} \)

b) Confused conversation = still answers questions: \( V = \text{four (4) points} \)

c) Inappropriate words - speech is either exclamatory or random, but recognizable words are produced: \( V = \text{three (3) points} \)

d) Incomprehensible sounds - grunts and groans are produced, but no actual words are uttered; do not confuse with partial respiratory obstruction: \( V = \text{two (2) points} \)

e) None: \( V = \text{one (1) point.} \)

3) Best motor response (M score)

The best response obtained for any extremity is recorded even though worse responses may be present in other extremities. The worst motor activity also is important but is not used for the GCS; worst response should be noted separately. For patients not following verbal command, a painful stimulus is applied to the fingernail or toenail.

a) Obeys - moves limb to command and pain is not required: \( M = \text{six (6) points} \)

b) Localizes - changing the location of the pain stimulus causes purposeful motion toward the stimulus: \( M = \text{five (5) points} \)

c) Withdraws - pulls away from painful stimulus: \( M = \text{four (4) points} \)

d) Abnormal flexion - decorticate posture: \( M = \text{three (3) points} \)

e) Extensor response - decerebrate posture: \( M = \text{two (2) points} \)

f) No movement: \( M = \text{one (1) point.} \)

b. Patient categorization

The Glasgow Coma Scale itself can be used to categorize patients:

1) Coma

A patient in coma is defined as having no eye opening (\( E = 1 \)), no ability to follow
commands (M = 1 to 5), and no word verbalizations (V = 1 to 2). This means that all patients with a GCS Score of less than eight and most of those with a GCS Score that equals eight are in coma. Patients with a GCS Score more than 8 are not in coma.

2) Head injury severity

On the basis of the GCS, patients are classified as having:

a) Severe head injury if the GCS Score is less than or equal to eight.

b) Moderate head injury if the GCS Score is 9 to 12.

c) Minor head injury if the GCS Score is 13 to 15.

2. Assessment of pupillary function

The pupils are evaluated for their equality and response to bright light. A difference in pupil diameters of more than 1 mm is abnormal. Even though eye injury may be present, intracranial injury must be excluded. Light reactivity must be evaluated for the briskness of response; a more sluggish response may indicate an intracranial injury.

3. Lateralized extremity weakness

Spontaneous movements are observed for equality. If spontaneous motion is minimal, the response to a painful stimulus is assessed. A delay in onset of movement, less movement, or need for more stimulus on one side is significant. Often, the motor component of the GCS can be used to score the best and the worst extremity movement. A clearly lateralized weakness suggests an intracranial mass lesion.

4. Purposes of neurologic examination

The purposes of the minineurologic examination are to determine the severity of the brain injury, and detect any neurologic deterioration. The GCS can be used to categorize injuries as mild, moderate, and severe. Irrespective of the GCS, a patient is considered to have a severe head injury if he exhibits any of the following:

a. Unequal pupils

b. Unequal motor examination

c. An open head injury with leaking CSF or exposed brain tissue

d. Neurologic deterioration

e. Depressed skull fracture.

A decrease in the GCS score of two or more points clearly means the patient's condition has deteriorated. A decrease of three or more points is a catastrophic deterioration.
that demands immediate treatment if the cause is remediable. However, these changes are rather gross and are often preceded by more subtle signs of deterioration. Neurologic deterioration of special concern includes:

f. Increase in severity of a headache or an extraordinarily severe headache

g. Increase in the size of one pupil

h. Development of weakness on one side.

It cannot be overemphasized that the initial neurologic examination is only the beginning. The initial findings are only the reference with which to compare results of repeated neurologic examinations to determine whether a patient's condition is deteriorating or improving. All previous efforts will go to waste if signs of neurologic deterioration are not appreciated. The brain cannot withstand unrelieved compression for very long before brain damage is irreversible. The neurologic assessment is designed to detect this secondary brain damage so timely treatment can be initiated following neurosurgical consultation.

E. Special Assessment

1. Skull roentgenograms

Skull roentgenograms are of little value in the early management of patients with obvious head injuries, except in cases of penetrating injuries. The unconscious patient should have skull roentgenograms only if precise care of the cardiorespiratory system and continuing reassessment can be assured. Physical examination is usually more valuable than skull roentgenograms. (See VII. Scalp Wounds, in this chapter.) Clinical signs of basal fractures are more useful than roentgenograms of the skull base in diagnosing a fracture. Increasingly, skull films are not being obtained on patients with minor head injuries because the information obtained is rarely helpful one way or the other. When in doubt about the patient's condition, the physicians should obtain neurosurgical consultation.

2. Computed tomography

The CT scan has revolutionized diagnosis in patients with head injuries and is the diagnostic procedure of choice for patients who have or are suspected of having a serious head injury. Although not perfect, the CT scan is capable of showing the exact location and size of most mass lesions. Specific diagnosis allows more precise planning of definitive care, including operation. The CT scan has supplanted less specific and more invasive tests, such as cerebral angiography.

Except for patients with minor head injuries, all head-injured patients will require CT scanning at some time. The more serious the injury, the earlier and more emergent is the need for the scan. Consequently, injured patients seen first at facilities without CT capability may require transfer to more sophisticated hospitals.

Once initial resuscitation has been undertaken and the need for a CT scan determined, care must be taken to (1) maintain adequate resuscitation during the scan, and (2) assure the
best possible quality of the scan. The patient must be attended constantly in the CT suite to monitor vital signs closely and initiate immediate treatment should the patient's status deteriorate.

Patient movement results in artifacts and a poor-quality scan. This artifact may mask significant intracranial lesions requiring urgent surgical intervention. Movement artifact can be eliminated by sedating restless or uncooperative patients. However, extreme caution must be exercised to avoid sedating patients whose restlessness or lack of cooperation is a clinical manifestation of hypoxia. Often endotracheal intubation with controlled ventilation becomes necessary if the scan is considered mandatory, and the patient can be rendered motionless only with paralyzing drugs. **Ideally, the neurosurgeon should examine the patient before the patient is iatrogenically paralyzed or has a CT scan.** However, efficient and correct management of the patient, in certain situations, may dictate otherwise.

3. Other tests

Lumbar puncture, electroencephalogram, and isotope scanning have no role in the acute management of head trauma.

Certain reflexes, eg, oculocephalic and vestibulo-ocular, can reflect the integrity of a portion of the brain-stem neural pathways. Although these may permit more specific diagnosis in some instances, their elicitation can be hazardous, their interpretation difficult, and they add little to the emergency management of the patient. They are best left to the neurosurgical consultant.

IV. Specific Types of Head Injury

After initial assessment and resuscitation, the emergency management of the patient with head injury is aimed at (1) establishing a specific anatomic diagnosis of the head injury, (2) assuring the metabolic needs of the brain, and (3) preventing secondary brain damage from treatable causes of elevated intracranial pressure. The need to prevent systemic causes of secondary brain injury, notably hypoxia, ischemia, and hyperthermia, is axiomatic.

Head injuries include (1) skull fractures, (2) diffuse brain injuries, and (3) focal injuries. The pathophysiology, severity, need for urgent treatment, and outcome are different in each group. A review of these head injuries aids in making and simplifying the initial diagnosis.

A. Skull Fractures

Skull fractures are common, but many are not associated with severe brain injury. Likewise, many severe brain injuries occur without skull fracture. Although identifying a skull fracture is important, diagnosing a brain injury does not depend on it. Searching for a skull fracture should never delay patient management. Attention should be directed to the brain injury. The significance of a skull fracture is that it identifies the patient with a higher probability of having or developing an intracranial hematoma. For this reason, all patients with skull fractures should be admitted to the hospital for observation. All patients with skull fractures require neurosurgical consultation.
1. Linear, nondepressed fractures

Linear skull fractures are often seen on the roentgenogram as a lucent line. They may also be stellate. Linear skull fractures require no specific treatment, and management is directed toward the underlying brain injury. Fractures across vascular arterial grooves or suture lines should raise suspicion of the possibility of epidural hemorrhage.

2. Depressed skull fractures

Depressed skull fractures may or may not be neurosurgical emergencies. Management is directed toward the underlying brain injury. To reduce the risk of possible sequelae, such as seizure disorder, any fragment depressed more than the thickness of the skull may require operative elevation of the bony fragment.

3. Open skull fractures

By definition, open skull fractures have a direct communication between a scalp laceration and the cerebral substance, because the dura is torn. This condition can be diagnosed if brain is visible or if CSF is leaking from the wound. Open fractures require early operative intervention, with elevation or removal of the fragments and closure of the dura, to reduce the possibility of infection.

4. Basal skull fractures

These fractures are often not apparent on skull roentgenograms. Indirectly, intracranial air or an opaque sphenoid sinus gives clues.

The diagnosis is based on physical findings such as CSF leaking from the ear (otorrhea) or the nose (rhinorrhea). When CSF is mixed with blood, it may be difficult to detect. An aid is the "ring sign," detected by allowing a drop of the fluid to fall onto a piece of filter paper. If CSF is present, blood remains in the center, and one or more concentric rings of clearer fluid develop.

Ecchymosis in the mastoid region (Battle's sign) also indicates a basal skull fracture, as does blood behind the tympanic membrane (hemotympanum). Cribriform plate fractures are often associated with periorbital ecchymosis (raccoon eyes). These signs may take several hours to appear and may be absent immediately after injury. Whenever a frontal basal skull fracture is present, danger exists of inadvertent intracranial intubation with the insertion of a nasogastric tube. This problem may be prevented by the insertion of the gastric tube through a previously, carefully placed soft, trumpet-style nasal airway, or orally.

B. Diffuse Brain Injuries

Diffuse brain injuries are produced when rapid head motions (acceleration or deceleration) cause widespread interruption of brain function in most areas of the brain. Often as with concussion, the disturbance of neural function is temporary. But with more severe injuries (diffuse axonal injury), microscopic structural damage throughout the brain may cause permanent problems. It is important to try to distinguish these injuries from focal injuries,
because diffuse brain injuries do not have mass lesions requiring emergency surgery.

1. Concussion

Concussion is a brain injury accompanied by brief loss of neurologic function. In its milder forms, it may cause only temporary loss of consciousness. The period of unconsciousness is usually short, but more severe forms exist. Many neurologic abnormalities may be described in the first few minutes after concussion, but these disappear quickly, usually before the patient gets to a medical facility. Therefore, any neurologic abnormality observed in the patient should not be attributed to a concussion.

Most patients with a concussion will be awake or awakening when seen in the emergency department, even though some mental confusion may persist. After the confusion clears, the patient may be capable of describing portions of his accident, but may not remember the actual impact. The patient may complain of headache, dizziness, or nausea, but the minineurologic examination will not show localizing signs. These patients should be observed in the hospital and then at home only if their mental state clears completely. Patients sustaining a severe concussion should be admitted for observation, because of the potential for associated serious brain injuries. The decision to admit the patient should be based on the length of unconsciousness and the reliability of the individuals with whom the patient resides. A rule of thumb is that if a patient has been unconscious for five minutes or more, he should be observed in the hospital for 24 hours. The duration of amnesia and/or retrograde amnesia also must be considered. If the patient is under the age of 12 years, it is best to admit for observation. Persistent vomiting and/or convulsions may occur. Any patient whose confusion does not clear completely must be admitted.

2. Diffuse axonal injury

Diffuse axonal injury (DAI) - often called brain-stem injury, closed head injury, or diffuse injury is characterized by prolonged coma, often lasting days to weeks. DAI is a frequent injury, occurring in 44% of coma-producing head injuries.

Overall mortality is 33%. In its most severe form, DAI has a 50% mortality, usually from increased intracranial pressure secondary to cerebral edema. DAI results primarily in microscopic damage that is scattered widely throughout the brain, and therefore does not require surgery. Although the length of coma cannot be determined in the emergency department, recognition of DAI is important because it may mimic other injuries that do require emergency surgery. The diagnosis is justified when an emergency CT scan shows no mass lesion in a patient who remains deeply comatose, often with decerebrate or decorticate posturing. Autonomic dysfunction producing high fever, hypertension, and sweating is common. The patient should be transferred to a facility equipped to care for long-term coma patients.

C. Focal Injuries

Focal injuries are those in which macroscopic damage occurs in a relatively local area. They consist of contusions, hemorrhages, and hematomas. These injuries may require emergency surgery because of their mass effects. Diagnostic efforts during the early
The postinjury period are primarily aimed at diagnosing focal lesions, because they are treatable and frequently require emergency operative intervention.

1. Contusion

Cerebral contusion can be single or multiple, small or large. Therefore, the patient may present in several ways. Most commonly, contusions are associated with serious concussions characterized by longer periods of coma and mental confusion or obtundation. Contusions can occur beneath an area of impact (coup contusions) or in areas remove from impact (contrecoup contusions). The tips of frontal and temporal lobes are especially common sites of contusion. The contusion itself may produce focal neurologic deficit if it occurs near the sensory or motor areas of the brain, but a deficit may not be apparent if the contusion is elsewhere. If the contusion is large or associated with pericontusional edema, the mass effect may cause herniation and brain-stem compression, resulting in secondary or delayed neurologic deterioration. A CT scan of the head determines with certainty the presence, location, and size of contusions.

Patients sustaining cerebral contusion should be admitted to the hospital for observation, because delayed edema or swelling around the contusion may cause neurologic deterioration. Contusions require surgery only if they cause substantial mass effect, but careful observation is necessary to detect patients with delayed bleeding into the contusion. This fact is especially important for patients with measurable blood alcohol levels, because acutely or chronically alcoholic patients have a high incidence of delayed bleeding into their contusions.

2. Intracranial hemorrhages

Intracranial hemorrhages can be classified as meningeal or brain hemorrhages. Due to great variation in location, size, and rapidity of bleeding, there is no typical picture. Computed tomography has made the diagnosis and localization very precise. It must be stressed that epidural hematoma and temporal lobe intracerebral hematoma can mimic each other, because the symptoms and the clinical findings are usually those of tentorial herniation, characteristically seen in epidural hemorrhage.

a. Meningeal hemorrhage

1) Acute epidural hemorrhage

Epidural bleeding almost always occurs from a tear in a dural artery, usually the middle meningeal artery. A small percentage may occur from a tear in the dural sinus. Although epidural hemorrhage is relatively rare (0.5% in unselected head injuries and 0.9% of coma-producing head injuries), it must be considered because it may be rapidly fatal. Such arterial tears are usually associated with linear skull fractures over the parietal or temporal areas that cross the grooves of the middle meningeal artery; however, this injury is not an absolute requirement for the diagnosis.

The signs and symptoms of a typical epidural hemorrhage are (1) loss of consciousness (concussion) followed by an intervening lucid interval (the lucid period may not be a return to full consciousness); (2) a secondary depression of consciousness; and (3)
the development of hemiparesis on the opposite side. A dilated and fixed pupil on the same side as the impact area is a hallmark of this injury. (Less frequently, the dilated, fixed pupil may be on the side opposite the hematoma or the hemiparesis may be on the same side as the hematoma.) Although the patient is lucid, he may not be entirely symptom free, usually complains of a severe, localized headache, and is sleepy.

This injury requires immediate surgical intervention. If treated early, prognosis is usually excellent, because the underlying brain injury is often not serious. Outcome is directly related to the status of the patient before surgery. For patients not in coma, the mortality from epidural hematoma approximates zero. For those in light coma it is 9%, and for patients in deep coma it is 20%. Secondary brain injury occurs rapidly if the hematoma is not evacuated quickly.

2) Acute subdural hematoma

Subdural hematomas also are life threatening and are much more common than epidural hematomas (30% of severe head injuries). They most commonly occur from rupture of bridging veins between the cerebral cortex and dura, but also can be seen with lacerations of the brain or cortical arteries. A skull fracture may or may not be present. In addition to the problems caused by the mass of subdural blood, underlying primary brain injury is often severe. The prognosis is often dismal, and the mortality remains at 60%. However, recent studies demonstrate improved outcome if the hematoma is evacuated very early.

3) Subarachnoid hemorrhage

This type of hemorrhage results in bloody CSF and meningeal irritation. The patient usually complains of a headache and/or photophobia. No immediate treatment is needed, and the hemorrhage alone is not life threatening. A lumbar puncture is not needed, because the diagnosis can be made with a CT scan.

b. Brain hemorrhages and lacerations

1) Intracerebral hematomas

Hemorrhages within the brain substance can occur in any location. Computed tomography provides a more precise diagnosis. Many small, deep intracerebral hemorrhages are associated with other brain injuries (especially DAI). The neurologic deficits depend on these associated injuries and the region(s) involved, the size of the hemorrhage, and whether bleeding continues. Hemiplegia may occur. Occipital hemorrhages can cause visual defects. Intraventricular and intracerebellar hemorrhages are associated with a high mortality rate.

2) Impalement injuries

All foreign bodies protruding from the skull should be left in plate until they can be removed by a neurosurgeon as a formal operation. Roentgenograms of the skull are required to determine the object's angle and depth of penetration.
3) Bullet wounds

The larger the caliber and the higher the velocity of the bullet, the more likely death occurs. Through-and-through and side-to-side injuries, and those lower in the brain, tend to be ominous.

Patients in coma have a very high mortality, especially with an initial GCS Score of less than six. Skull films and a CT scan help plant the surgical approach when operation is deemed necessary. Entrance and exit wounds should be covered with antiseptic-soaked dressings until definitive neurosurgical care is provided. A bullet that does not penetrate the skull may still result in intracranial injury, because of blunt trauma, and such patients require a CT scan.

c. Time factors

The earlier the hematoma is recognized and evaluated, the better the prognosis. Epidural hematomas, being arterial in origin, usually develop more rapidly than subdural hematomas of venous origin. Epidural hematomas may require four to six hours to become clinically apparent, whereas subdural hematomas may take 12 to 24 hours to become evident in the absence of other lesions such as intracerebral hematomas or diffuse cerebral edema. However, both types of hematomas may become evident within as little as one hour after injury, especially in children. Therefore, all patients suspected of harboring a developing intracranial hematoma must be observed closely and transferred to the care of a neurosurgeon as rapidly as possible. **Remember**, the decision to **delay transfer** may be as critical a surgical decision as the decision to operate.

V. Emergency Management of Head Injuries

A. Establishing a Specific diagnosis

The immediate purpose of a specific diagnosis is to determine which patients need an emergency or urgent neurosurgical operation. **This is an issue that demands early participation by the neurosurgical consultant.** Patients requiring emergency surgery are those with massive depressed or open skull fractures and those with large focal mass lesions. The former category is usually obvious, but the latter must be distinguished from diffuse brain injuries where no mass lesion exists.

1. Assessing the need for surgery

Injuries requiring surgery are likely to occur in different circumstances than injuries not requiring surgery. Therefore, a gross estimate of a patient's potential need for surgery can be based on knowledge concerning three factors: (1) whether the patient is comatose; (2) whether the trauma was vehicular or nonvehicular; and (3) whether there is a lateralized motor deficit. (See Table 1, Approximate Percent of Patients Who May Require Surgery.)
Table 1. Approximate Percent of Patients Who May Require Surgery
(Excludes patients with minor injuries who are neurologically normal.)

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<th>Not in Coma</th>
<th>Comatose</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Motor Equal</td>
<td>Unequal</td>
</tr>
<tr>
<td>Vehicular</td>
<td>20%</td>
<td>40%</td>
</tr>
<tr>
<td>Nonvehicular</td>
<td>40%</td>
<td>70%</td>
</tr>
</tbody>
</table>

2. Diagnostic triage system

Based on the minineurologic examination, a practical and systematic approach can be established for diagnosing head-injured patients. It must be understood that this scheme is only a guideline. (See Figure 1, Head Injury Triage Scheme.) Because of the complexity and multiplicity of brain injuries, this system is not and cannot be either foolproof or all-inclusive. However, it offers one useful approach to determining not only the initial diagnosis, but also the severity of injury, so that emergency treatment decisions can be made.

a) The first priority is determining the level of consciousness, based on the GCS. If a patient has a score equal to or less than eight, the physician must immediately determine (1) whether the pupils are unequal, or (2) whether there is a lateralized motor deficit. If either of these exist, a large focal lesion (epidural, subdural, or intracerebral hematoma, or large contusion) must be presumed present. Preparation must be made for emergency operation to evacuate the lesion. Simultaneously, efforts must be taken to decrease the intracranial pressure. Presumptive evidence of a progressive lesion, capable of producing lethal brain herniation (usually an epidural hematoma), is one of the few neurosurgical emergencies in the injured patient that may require operative intervention within minutes.

b) A comatose patient with equal pupils and motor responses may still have a surgical mass lesion but is more likely to have diffuse axonal injury, acute brain swelling, or cerebral ischemia. After immediate measures have been taken to reduce intracranial pressure, an emergency CT is needed to exclude a mass lesion.

c) A patient who is not comatose (ie, GCS > 8) still may harbor an injury requiring operation. If pupillary asymmetry or lateralized weakness is present, the patient must be suspected to have a focal lesion that is not (yet) of sufficient size to compress the brain stem and cause coma. A CT scan is urgently required. If the pupils and movements are equal, the presence or absence of an open head injury must be determined by a search for CSF leaking from the nose, ears, or a scalp wound.

d) If there is no open injury (in the noncomatose patient who has equal pupils and movements), a complete neurologic examination must be performed. If the examination is not normal, a focal lesion must be excluded by CT scan. If the examination is normal, and the patient has been unconscious only briefly and is in the low-risk group for an intracranial lesion (see Table 2, Relative Risk of Intracranial Lesion), discharge from the hospital may be considered after appropriate instructions are given to the patient. If any of these conditions are not met, the patient should be admitted to the hospital for postconcussion observation.
Remember, a normal neurologic examination includes a normal mental status examination. No patient with signs of head injury should be discharged because he is intact except for being intoxicated. Do not presume that altered mental status is due to alcohol. The patient should be observed until his mental status is completely normal, or he should be admitted to the hospital for observation.

Figure 1. Head Injury Triage Scheme

Level of Consciousness GCS ≤ 8:

1. Yes. Pupils Unequal or Lateralized Deficit:
   1.1. Yes.
   **Best Diagnosis:** Large Mass.
   **Neurosurgery:** STAT.
   1.2. No.
   **Best Diagnosis:** Diffuse axonal injury.
   **Action:** Admit. Intubate. Hyperventilate. STAT CT. ICU. Monitor.
   **Neurosurgery:** STAT.
2. No. Pupils Unequal or Lateralized Deficit:
   2.1. Yes.
   **Best Diagnosis:** Possible mass.
   **Action:** Admit. Urgent CT. ICU. Observe.
   **Neurosurgery:** Urgent.
2.2. No.
   2.2.1. Open Injury?
   2.2.2.1. Yes.
   **Best Diagnosis:** Basilar fracture. Penetrating injury.
   **Action:** Admit. Urgent CT. Observe or Operate.
   **Neurosurgery:** Urgent.
2.2.2.2. No.
   2.2.2.2.1. Neurologically Normal?
   2.2.2.2.1.1. Yes.
   **Best Diagnosis:** Concussion, fracture.
   **Action:** Admit. Elective CT. Observe.
   **Neurosurgery:** Selective.
   2.2.2.2.1.2. Yes.
2.2.2.2.1.2.1. No LOC, LOC < 5 Min or Low Risk?
   2.2.2.2.1.2.1.1. No.
   **Best Diagnosis:** Minor injury.
   **Action:** Discharge with instructions.
   **Neurosurgery:** Selective.
**Table 2. Relative Risk of Intracranial Lesion**

<table>
<thead>
<tr>
<th>Low Risk Group</th>
<th>Moderate Risk</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic</td>
<td>Change of consciousness</td>
<td>Depressed consciousness</td>
</tr>
<tr>
<td>Headache</td>
<td>Progressive headache</td>
<td>Focal signs</td>
</tr>
<tr>
<td>Dizziness</td>
<td>Alcohol and/or other drug</td>
<td>Decreasing</td>
</tr>
<tr>
<td>intoxication</td>
<td>consciousness</td>
<td>Scalp hematoma</td>
</tr>
<tr>
<td>Unreliable history</td>
<td>Penetrating injury</td>
<td></td>
</tr>
<tr>
<td>Scalp laceration</td>
<td>Age &gt; 2 years</td>
<td>Depressed fracture</td>
</tr>
<tr>
<td>Scalp contusion</td>
<td>Seizure</td>
<td></td>
</tr>
<tr>
<td>Scalp abrasion</td>
<td>Vomiting</td>
<td></td>
</tr>
<tr>
<td>Absence of moderate or high-risk criteria</td>
<td>Multiple trauma</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Serious facial injury</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Signs of basilar fracture</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Possible skull penetration</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Possible depressed fracture</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Suspected child abuse</td>
<td></td>
</tr>
</tbody>
</table>

**B. Emergency Management**

Once a working diagnosis is established, treatment must be commenced; urgency of treatment depends on the nature and severity of injury. Emergency treatment of the seriously injured patient is aimed at protecting the brain from further (secondary) insults by (1) maintaining adequate cerebral metabolism, and (2) preventing and treating intracranial hypertension.

1. **Maintenance of cerebral metabolic needs**

Cerebral ischemia or hypoxia result in insufficient substrate delivery to the injured brain. These conditions are present in more than 90% of patients who die of head trauma, are associated with poor outcome, and are the most important preventable complications of head injury.

The principal metabolic requirements of the brain are oxygen and glucose, which are normally used at extremely high rates. The injured brain usually has a lowered cerebral metabolism and therefore requires less oxygen and glucose. However, the damaged brain is more susceptible to the lack of these substrates, and thus temporary severe or prolonged moderate deprivation causes worse damage than in the uninjured brain. Therefore, emergency management of head injury includes maintenance of adequate cerebral metabolic fuels.

The physician must assure delivery of adequate levels of glucose and oxygen to the brain. Delivery of these substrates depends on their arterial concentrations and the blood flow to the brain. Blood glucose concentration is not a common problem in trauma, but if it is, correction with supplemental intravenous glucose is needed. However, recent works suggest that hyperglycemia should be avoided.
Oxygen content depends on arterial hemoglobin and oxygen concentrations. Arterial oxygen concentration can be assessed with blood gases and supplemental oxygen delivered to maintain the arterial PO2 at greater than 80 mm Hg. Blood transfusion may be necessary to provide sufficient hemoglobin to maintain normal oxygen carrying capacity.

Cerebral blood flow is dependent on systemic arterial pressure and arterial PaCO2, especially shortly after head trauma. Normalization of blood pressure and maintenance of PaCO2 at 26 to 28 mm Hg is sufficient to maintain adequate cerebral blood flow under most circumstances. Keep in mind that the PaCO2 cannot be allowed to rise too high as it may aggravate intracranial pressure (see 2.a.).

2. Preventing / treating intracranial hypertension

The purpose of these treatments is to prevent, control, or diminish intracranial hypertension, regardless of its cause. In addition to mass lesions, acute brain swelling (a vascular engorgement phenomena) and brain edema can complicate any primary brain injury by causing or aggravating intracranial hypertension. It is for this reason that, optimally, neurosurgical consultation should be obtained when these measures are instituted.

a. Induced hypocapnia

Arterial carbon dioxide concentration profoundly affects cerebral circulation. When abnormally elevated, cerebrovasodilatation occurs, increasing intracranial blood volume and intracranial pressure. (See II. Anatomy and Physiology, H. Intracranial Pressure in this chapter.) Conversely, reduction of PaCO2 reduces intracranial blood volume and, secondarily, intracranial pressure. Additionally, hyperventilation tends to reduce intracerebral acidosis and increase cerebral metabolism, both of which are beneficial. Therefore, hyperventilation is recommended to reduce arterial PaCO2, maintaining it at 26 to 28 mm Hg. This procedure usually requires endotracheal intubation, controlled ventilation, and intermittent iatrogenic paralysis. Intubation should be performed early for the comatose patient. Care must be taken to assure that injudicious intubation does not cause further intracranial problems due to increased intracranial pressure elevations from coughing or gagging during intubation. (See Chapter 2, Airway and Ventilatory Management.) Continued intubation may require sedation or iatrogenic paralysis. Hypocapnia can reduce cerebral circulation to the point where cerebral ischemia occurs. Blood gases must be monitored closely.

b. Fluid control

Intravenous fluids should be administered judiciously to prevent overhydration, which augments cerebral edema. since the nature and volume of fluids administered will depend on the systemic status of the patient, consultations between the trauma surgeon and the neurosurgeon are necessary. Intravenous fluid used for maintenance must not be hyposmolar, as cerebral edema may likewise follow.

c. Diuretics

Diuretics such as mannitol, which cause diuresis by producing intravascular hyperosmolarity, are widely used for severe brain injury. Mannitol undoubtedly can be very
effective in shrinking brain volume and lowering intracranial pressure. However, other effects may cause major problems. In the emergency department, this agent should be administered only with the consent of a neurosurgeon or to gain time when neurosurgical capabilities will be delayed and the patient's condition is deteriorating. Mannitol is most commonly and effectively used for a suspected surgical mass lesion when time is needed to prepare for operation (ie, hematoma with deteriorating level of consciousness, hemiparesis, and dilating pupil). For the average patient, 1 g/kg, is administered rapidly and intravenously. Care must be taken to prevent subcutaneous infiltration.

The neurosurgeon also may recommend loop diuretics, such as furosemide, (40 to 80 mg IV for adults). These medications act by medically decompressing the brain and may buy up to several hours of reduced intracranial pressure. A urinary catheter is required.

Because of the rapid production of urine, vascular volume may be significantly affected. Therefore, the patient's blood pressure must be closely monitored, especially if the patient has sustained multiple trauma or is a child. Urine volume may be replaced with isotonic fluids in these patients.

d. Steroids

Steroids are not recommended for the treatment of acute head injury.

VI. Other Manifestations of Head Injury

A. Seizures

Seizures, either full-blown convulsions or focal seizures, can occur with any injury. Seizures are common at the time of injury or shortly thereafter, and do not necessarily portend the development of chronic epilepsy. The neurosurgeon may recommend no treatment. Prolonged or repetitive seizures may be associated with intracranial hemorrhage and should be treated aggressively, because they may cause cerebral hypoxia, brain swelling, and increased intracranial pressure. A common treatment regimen consists of 10 mg of diazepam via an intravenous bolus. Respiratory exchange should be closely monitored. If the patient tolerates diazepam and has another convolution, the dose may be repeated once with caution. Phenytoin, 1 g IV at a rate of 50 mg/minute, should be started as soon as possible, while monitoring the patient's blood pressure and electrocardiogram. If the convulsions persist despite diazepam and phenytoin, the patient should be given phenobarbital or an anesthetic. The injured brain tolerates anesthesia well.

B. Restlessness

Restlessness frequently accompanies brain injury and/or cerebral hypoxia. Its development in a previously quiet patient may be the first clinical expression of an expanding intracranial mass. Restlessness can often be controlled by correcting the cause. Primary attention should be directed to the possibility of cerebral hypoxia and its etiology. Pain from injuries, a distended bladder, painful bandages, tight casts, or systemic hypoxia may also induce restlessness. Only when these etiologic factors have been treated or excluded is the use of drugs justified to control the restlessness.
The relief of severe pain is an important part in the management of the trauma patient. Effective analgesia implies the use of intravenous opiates which may adversely affect the patient. They also may cause respiratory depression and mask neurologic signs. Therefore, for these reasons, opiates and other strong analgesics/sedatives should be withheld, if possible, until neurosurgical consultation has taken place. This presents less of a problem for the neurosurgeon if CT scan is readily available. Chlorpromazine, 10 to 25 mg IV, may be helpful for severe agitation. This agent must be administered carefully to avoid hypotension.

C. Hyperthermia

Hyperthermia is disastrous for the patient with an injured brain. Elevations in body temperature increase the rate of brain metabolism and levels of carbon dioxide. The higher the temperature, the greater the risk to the patient. A head-injured patient with hyperthermia should be placed on a hypothermia blanket, and shivering should be controlled with chlorpromazine.

VII. Scalp Wounds

Despite the dramatic appearance of scalp wounds, they are usually tolerated well and cause few complications. Scalp wounds usually heal well if general principles of good wound management are followed. The location and type of injury to the scalp give the physician a better understanding of the force and direction of the energy transmitted to the brain.

A. Blood Loss

Blood loss from scalp wounds can be extensive, especially in children. If the adult patient is in shock, bleeding from the scalp alone is usually not the cause. The general principles of scalp wound care include locating and stopping of the bleeding. In the case of severed large vessels (ie, superficial temporal arteries), the vessels should be clamped with hemostats and ligated. Moderately severe bleeding from a deep scalp laceration can usually be controlled temporarily by applying direct pressure or placing hemostats on the galea and reflecting them backwards. For the unconscious patient, self-retaining retractors can be used.

Most scalp bleeding, with or without direct ligation of bleeding vessels, may be controlled by a snugly applied circumferential head dressing. If necessary, the hair may be clipped to facilitate application.

B. Inspection of the Scalp Wound

One should inspect the wound carefully and search under direct vision for signs of a skull fracture or foreign material. Blind probing with an instrument or the gloved finger should be avoided. Wound inspection also includes looking for CSF leaks, which may indicate not only a skull fracture, but also an arachnoid and dural tear. A neurosurgeon should be consulted before the wound is closed for all cases involving open fractures or depressed fractures of the skull.
C. Repairing the Scalp Wound

The first step in wound care is to irrigate the scalp wound with copious amounts of saline. Debris, including hair, must be removed. **Do not** remove bone fragments, because they may be tamponading intracranial bleeding. The neurosurgeon should remove bone fragments in the operating room. When in doubt, **always consult a neurosurgeon**. If transfer to a definitive-care facility is required, definitive surgical closure of a scalp wound can be delayed. However, temporary repair may be done to prevent further contamination of the wound.

When possible, primary definitive repair should be achieved under sterile technique with meticulous attention to the wound, as **scalp wounds may become infected**. The area immediately around the laceration should be shaved. The galea is closed first, using interrupted sutures. The skin is then sutured and a nonadherent dressing applied. The head wrap, if applied, should be just tight enough to apply mild pressure and prevent subgaleal accumulation of blood.

VIII. Definitive Surgical Management

Within two hours of injury, essential diagnostic studies should be completed. Although meeting this standard is difficult, delay can be extremely costly for the patient. If a neurosurgeon is not available at the facility initially receiving the patient, expeditious transfer of the patient to a hospital with an available neurosurgeon is necessary.

In exceptional circumstances, rapidly expanding intracranial hematomas (usually epidural hematomas) may be imminently life threatening and may not allow time for transfer if neurosurgical care is some distance away. Although this circumstance is rare in urban settings, it may occur occasionally in rural areas. In such circumstances, emergency burr holes can be considered if a surgeon, properly trained in the procedure, is available.

If definitive neurosurgical care is not immediately available, the purpose of an emergency burr hole is to preserve life by evacuating life-threatening intracranial hematomas. The performance of this procedure may be especially important for patients whose neurologic status rapidly deteriorates and for patients who do not respond to nonsurgical measures. However and before proceeding with the procedure, the physician must consider the following problems:

1. Not all severely head-injured patients have hematomas.
2. A burr hole, placed as little as 2 cm away from a hematoma, may not locate it.
3. A burr hole itself may cause brain damage or intracranial hemorrhage.
4. Burr-hole evacuation of a hematoma may not be life saving.
5. A burr hole can consume as much time as getting the patient to a neurosurgeon.

**Performance of emergency burr holes should be done only in exceptional circumstances and only with the advice and consent of a neurosurgeon. The indications**
for a burr hole performed by a nonneurosurgeon are few, and widespread use as a desperation maneuver is not recommended or supported by the Committee on Trauma. The procedure should be employed only if, in the judgment of a neurosurgeon, the burr hole can be performed in a timely manner, and when definitive neurosurgical care cannot be rendered expeditiously. In almost all instances this means that definitive neurosurgical care is many miles or several hours away.

The neurosurgeon usually recommends the burr hole be placed on the side of the largest pupil in comatose patients with decerebrate and decorticate posturing whose posturing does not respond to endotracheal hyperventilation and dehydrating agents (eg, 1 g/kg mannitol). The Committee on Trauma strongly recommends that those who anticipate the need for this procedure receive proper training in the procedure from a neurosurgeon.

IX. Summary

A. Secure and maintain an open airway.
B. Ventilate the patient to maintain oxygenation and to avoid hypercarbia.
C. Treat shock, if present, and look for cause.
D. Except for shock, restrict fluid intake to maintenance levels.
E. Perform a minineurologic examination.
F. Establish an initial working diagnosis.
G. Prevent secondary brain injury.
H. Search for associated injuries.
I. Obtain roentgenograms or CT scan as needed, but only after the patient is stable.
J. Consult a neurosurgeon and consider early transfer.
K. Should the patient's condition worsen, consider other diagnoses and forms of treatment; consult with a neurosurgeon, and consider transfer.
L. Continually reassess the patient (neurologic examination and GCS Score) to identify changes that necessitate neurosurgical intervention.
Skill Station IX: Head and Neck Trauma Assessment and Management

Resources and Equipment

This list is the recommended equipment to conduct this skill session in accordance with the stated objectives for and intent of the procedures outlined. Additional equipment may be used providing it does not detract from the stated objectives and intent of this skill, or from performing the procedures in a safe method as described and recommended by the ACS Committee on Trauma.

1. Mr. HURT - Head trauma manikin.
2. Ophthalmoscope.
3. Otoscope.
4. Table and chairs; or gurney, pillows, and sheet.
5. Semirigid cervical collar.
7. 2 coconuts.

Objectives

1. Performance at this station will allow participants to practice and demonstrate their assessment and diagnostic skills in determining the type and extent of injuries with Mr. HURT (head trauma manikin).

2. Upon completion of this station, the participant will be able to discuss the importance of clinical signs and symptoms found through assessment.

3. Upon completion of this station, the participant will be able to discuss and establish priorities for initial primary management of the patient with head trauma.

4. Participation at this station will allow the student to discuss other diagnostic aids that can be used to determine the area of injury within the brain and the extent of injury.

5. Upon completion of this station, the participant will be able to demonstrate proficiency in the use and application of the cervical traction tongs.
Skill Procedures: Head and Neck Trauma Assessment and Management

Note: Universal precautions are required whenever caring for the trauma patient.

I. Head Trauma Assessment and Management

A. Primary Survey

1. ABCs.

2. Immobilize and stabilize the cervical spine.

3. Perform a brief neurologic examination.
   a. Pupillary response.
   b. AVPU.

B. Secondary Survey and Management

1. Inspect the head.
   a. Lacerations.
   b. Nose and ears for presence of CSF leakage.

2. Palpate the head.
   a. Fractures.
   b. Lacerations for underlying fractures.

3. Inspect all scalp lacerations.
   a. Brain tissue.
   b. Depressed skull fractures.
   c. Debris.
   d. CSF leaks.

4. Perform a minineurologic examination and determine the patient's GCS Score.
   a. Eye-opening response.
   b. Verbal response.
c. Best limb motor response.

5. Palpate the c-spine for fractures and apply a semirigid cervical collar, if needed.

6. Determine the extent of injury.

7. Reassess the patient continuously, observing for signs of deterioration.
   a. Frequency.
   b. Parameters to be assessed.

C. Head and Neck Injuries

1. Open, depressed skull fractures on the right

   This type of injury is indicative of the amount of energy force that has been delivered to the cranium. An open, depressed skull fracture requires early operative intervention, with management directed toward the underlying brain injury.

2. Zygomatic fracture (Le Fort III fracture) on the right

   A Le Fort III fracture is a separation of the mid-third of the face at the zygomaticofrontal, zygomaticotemporal, and nasal sutures, and across the orbital floor. It is a compound fracture with major hemorrhage that jeopardizes the integrity of the airway. Airway control and protection of the airway with concomitant immobilization of the cervical spine is imperative. Inability to intubate the patient may be an indication for surgical cricothyroidotomy, depending on the clinical judgment and skill level of the examiner. Nasotracheal intubation is contraindicated.

3. Maxillary fracture (Le Fort I fracture) on the right

   Traumatic malocclusion is an important sign indicating a major disruption of the facial bones. Associated bleeding also may compromise the airway. Management includes: (1) securing a patent airway, (2) maintaining c-spine immobilization, and (3) hemorrhage control.

4. Mandibular fracture on the left

   This is an important injury to recognize, especially if it occurs in combination with other facial fractures. In the unconscious patient, the tongue falls posteriorly and occludes the airway. Basic airway management procedures, eg, insertion of an oropharyngeal airway, are necessary to prevent airway obstruction.

5. Mandibular/neck laceration on the left

   Penetrating injuries of the neck are located in three anatomic areas: (1) Zone 1 - structures at the thoracic outlet, (2) Zone 2 - between the clavicles and mandible, and (3) Zone 3 - between the angle of the mandible and the base of the skull.
This is an important wound to recognize. In this particular manikin, the platysma has been violated. Active bleeding, external or internal, may be present. Control and protection of the airway is necessary in this unconscious patient.

6. Nasal fracture

Bleeding can be significant from nasal fractures and can place the integrity of the airway in jeopardy, especially in the unconscious patient.

7. Multiple fractures, C-6 vertebra

A step-off deformity, although difficult to palpate, is present. Management includes continued immobilization of the c-spine and obtaining a lateral c-spine roentgenogram.

8. Unequal pupils

A unilateral, fixed and dilated pupil in an unconscious patient is a sign of grave consequence. Appropriate resuscitation and surgical intervention must be instituted rapidly. The inability of the dilated pupil to react to light denotes one lesion. The third cranial nerve on the side of the dilated pupil may be compressed by increased intracranial pressure due to a mass lesion or epidural bleeding. Rapid neurosurgical intervention is necessary to prevent secondary brain injury. Controlled hyperventilation and fluid resuscitation restriction may be required for this patient. The eyes also are exophthalmic.

9. Hemotympanum on the right

This sign denotes a basal skull fracture. Patients with this type of fracture may have significant traumatic CSF leaks. Cribriform plate fractures are often associated with this injury. Insertion of a nasogastric tube and nasotracheal intubation are contraindicated.

II. Application of Cervical Traction Tongs (Optional Skill Procedure)

Note: Differences of opinion exist among neurosurgeons regarding the indications and techniques for the use of cervical traction in the patient with a known cervical spine injury. Therefore, protocols for its use should be established at each local institution in consultation with a neurosurgeon.

This skill procedure is based on the use of Gardner-Wells tongs for the patient with mid-cervical fracture-dislocation, eg, C-4 on C-5.

A. Identify the site for application of the tongs. The tongs are applied above the ears and below the "equator" (temporal ridges).

B. Surgically prepare the scalp with an antiseptic preparation. It is not necessary to shave the scalp. An aerosol spray that coats and penetrates the hair to the skin is recommended. Then rub in the antiseptic.

C. Locally inject an anesthetic and allow time for it to take effect.
D. If using a spray antiseptic, respray the scalp.

E. Place the tong apparatus with the metal instruction plate facing forward.

F. Advance the spring-loaded points until the 1-mm protrusion indicator within its knob shows that the proper squeeze is being exerted. (As the points are advanced, the skin is stretched increasingly snug, effectively sealing the point of entry and preventing bleeding.)

G. When bone is encountered, the stiff spring yields until the outer end of the spring-loaded point barely protrudes beyond the flat surface of the knurled end. (Indicates 30 pounds of "squeeze" being applied.)

H. Tilt the tong back and forth several times to seat the points. Then check the indicator protrusion and retighten it if necessary.

I. Support the tongs with slight, hand-applied traction, parallel to the bed or patient cart, until the assistant attaches the rope and pulley system.

J. Apply five pounds of weight to the rope, and carefully release hold of the tongs.

K. Additional weight can be applied according to protocol or at the direction of the neurosurgeon.

Contraindications

1. Paget's disease of the skull.

2. Neonates or infants.


Complications

1. Localized sepsis at puncture sites.

2. Laceration of a scalp artery.