Pittsburgh EMS Pre-Hospital Care Monograph

Closed Head Injuries

This monograph is dedicated to the professional men and women of Pittsburgh EMS.

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Introduction

Head injuries are one of the most common causes of death in trauma related incidents. They are also the leading cause of death for those between the ages of 1 and 44. Severe head injuries are also a major cause of disability. Only half the patients that survive a severe head injury actually return to normal function.

Head injuries may occur in one of two ways:

1. **Closed head injury** - occurs when the moving head is stopped rapidly, as when hitting a windshield, or when it is hit by a blunt object, causing the brain to smash into the hard bony surface inside the skull.

   A closed head injury may also occur without direct external trauma to the head if the brain undergoes a rapid forward or backward movement, such as when a person experiences whiplash, or when babies are shaken.

2. **Penetrating head injury** - occurs when a fast moving object such as a bullet pierces the skull.

This monograph will focus on closed head injuries. Due to the relatively occult nature of some closed head injuries and the high potential for death and disability, it is important for the prehospital care provider to understand their basic pathophysiology and treatment of these injuries. These injuries are often difficult to recognize and therefore, a definitive diagnosis is often only confirmed with a CT scan. Proper identification and treatment in the field may help reduce the mortality and disability from these traumatic brain injuries.

Anatomy and Physiology

In order to recognize the signs and symptoms of a closed head injury, it is first necessary to understand the underlying structures of the head.

Cranium

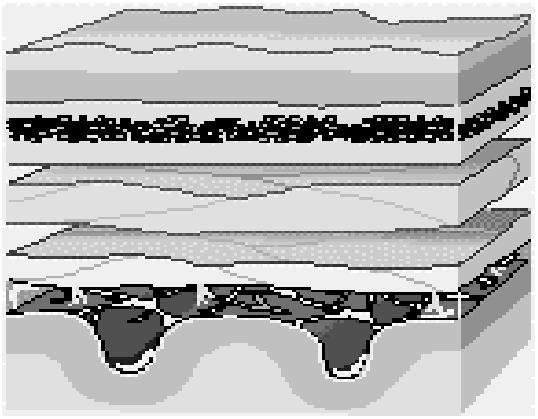
The cranium (skull) is the bony structure that encases the brain. The cranium is rigid and inflexible. It can **not** expand if the brain swells or if a hematoma develops around the brain. Since the skull can not expand, swelling of the brain leads to increased intracranial pressure (ICP) within the skull.

Meninges

The meninges are the three membranes that surround and protect the brain. They consist of the Dura Mater, Arachnoid Membrane, and the Pia Mater.

- *Dura Mater-Latin for "tough mother." The tough outermost layer of the meninges attached to the interior surface of the cranium.*
- *Arachnoid Membrane-The middle layer which is weblike. It suspends the brain within the cranium.*
- *Pia Mater-The thinnest and most delicate layer of the meninges. It directly contacts the brain.*

Layers of the brain starting with the skin



Skin
Skull

Dura

Arachnoid

Pia
Brain

Brain

The brain is essential to human functions such as conscious thought, personality, speech, motor control, and sensory perception. It consists of gray matter and white matter. The brain is supported in place by ligaments called tentorium.

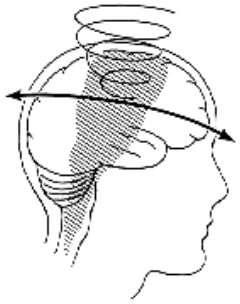
Cerebral Spinal Fluid (CSF)

The CSF surrounds and bathes the brain. The brain "floats" in this fluid. The CSF cushions and buffers the brain against trauma.

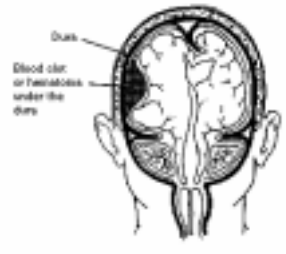
Injuries

Concussion

A concussion is a transient trauma induced alteration of mental status that may or may not involve loss of consciousness. With a concussion, the patient relatively quickly returns to a totally normal mental function. A concussion occurs immediately after a non penetrating event and involves shaking of the brain. Concussions cause a temporary disruption of brain function.



Concussions are the most minor type of head injury. After a concussion, a patient can have retrograde or antegrade amnesia (amnesia of events leading up to or just after the incident) or memory deficits. This amnesia is usually short lived, lasting approximately five minutes to one hour. This is followed by a complete return of motor functions with no lasting damage. CT scans of the head are normal in these patients.

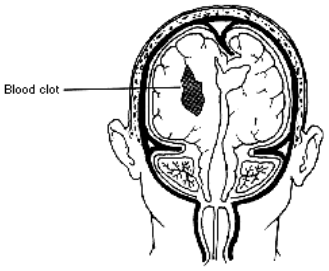


Contusion

A contusion involves bleeding and bruising of the brain. It causes a more significant cellular damage to the brain. The injured tissues swell, increasing intracranial pressure. The patient may have neurological symptoms such as persistent vomiting, headache, blurred vision, extremity weakness, etc. Contusions can be diagnosed by CT scan.

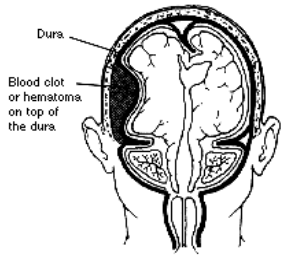
Intracranial Hemorrhage

Intracranial hemorrhage is most often associated with a penetrating injury to the head but can occur secondarily to blunt forces or shaking. Intracranial hemorrhage is characterized by bleeding directly into the brain tissue. Cerebral edema occurs because blood irritates the nervous tissue. The clinical presentation is equivalent to a stroke and occurs quickly. The signs and symptoms progressively worsen over time.



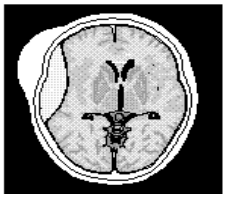
Epidural Hematoma

An epidural hematoma is an accumulation of blood above dura mater. (epi = above, i.e., epidermis) The source of bleeding is usually arterial (85%) in etiology. The bleeding may progress rapidly and lead to an altered mental status and eventually unconsciousness. However patients may experience a "**Lucid Interval**."



After an initial period of unconsciousness the patient may regain consciousness. (**lucid interval**). The patient may become conscious and coherent. However, as the hematoma enlarges the patient's level of consciousness declines. This is characterized by the so-called talk-and-die patient. The lucid interval may be longer if the source of the hematoma is venous in nature (under low pressure). Generally the lucid interval doesn't last for more than 1-2 hours.

Epidural hematomas account for 1-3% of all major head bleeds. Approximately 80% of the time an epidural hematoma is associated with a skull fracture. Motor vehicle accidents are the most common cause. Epidural hematomas are more common in the second and third decades of life. The male/female ratio can be as high as 4:1. In the hospital, epidural hematoma are identified by CT scan.



Epidural Hematoma

The **lucid interval** has implications for prehospital providers. The patient may be alert and oriented, look fine but then decline. The patient may have very minor symptoms initially. In the field it is difficult to identify the patients that may develop complications. Have a very low threshold for transporting patients that have suffered a closed head injury.

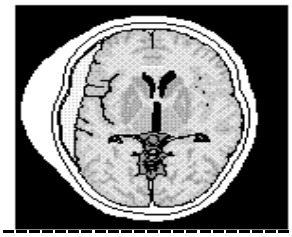
Subdural Hematoma

A subdural hematoma is a collection of blood between the brain and the dura mater. Subdural hematomas are usually the result of venous bleeding. Motor vehicle accidents are the most common cause.

Subdural hematomas are caused by the movement of the brain within the skull during a traumatic event. This movement is enhanced in patients with decreased cortical mass, i.e., shrunken brains. Brains tend to shrink with age. Alcohol tends to enhance the loss of brain cells. As a result, the elderly and chronic alcoholics are more prone to subdural hematomas due to increased cerebral atrophy. Patients on blood thinners, primarily coumadin, but also aspirin, are at an increased risk of all types of intracranial bleeding. Be extra suspicious with patients on blood thinners or with bleeding disorders, hemophilia, or cancer patients on chemotherapy.

Symptoms of a subdural hematoma can include: persistent headaches, vision changes, nausea, vomiting, abnormal behavior, weakness, decreasing mentation, and unresponsiveness. These signs and symptoms may be acute or they may not be apparent for up to two weeks post incident. The hematoma may develop slowly with few symptoms. This may pose a problem for prehospital providers as the patient may look fine immediately after the event. A patient evaluated in the emergency department may be released from the hospital prior to the onset of symptoms.

As many as 30% or more of subdural hematomas are symptomatic within 24 hours of the incident. This means there are a significant number of patients that are **asymptomatic** early on! The mortality rate is high (60-90%) in comatose victims with subdurals.


Subdural Hematoma

A subdural hematoma may be difficult to detect in the field since the blood is above the pia mater and doesn't cause cerebral irritation initially. In hospital, an initial CT scan may be negative due to the slow build up of blood. This is one of the reasons why patients are discharged from the hospital with specific instructions to return if they develop symptoms.

The alcoholic patient is at increased risk of subdural hematomas for a variety of reasons including their increased incidence of falls and "shunken brains" due to cerebral atrophy. Clinically, a sudural hematoma may be difficult to detect in a patient who is "obviously drunk."

Pathophysiology

Most of the intracranial injuries share a common pathophysiology. ICP builds rapidly because of a hematoma, bleeding or edema. The rising ICP causes a compression of the cerebrum. This compression reduces perfusion to nerve cells.

Common symptoms of an elevated ICP include headache, personality changes, and very late, differing pupillary diameters. In later stages the increasing pressure will cause herniation of brain tissue across the tentorium. This can produce **Cushing's Triad**, respiratory depression, increased blood pressure, and decreased pulse rate.

As the brain swells even more, cerebral perfusion is further decreased and carbon dioxide builds up in the tissues. Hypercarbia dilates cerebral blood vessels causing an increase in intracranial pressure. The patient may also exhibit posturing. Decorticate posturing is an abnormal flexion response. Decerebrate posturing is an abnormal extension response.

Assessment and Treatment

Initial evaluation of the head injury patient should be based on the ABCs. Any problems with ABCs require immediate intervention and transport prior to completing the assessment.

Airway

Establishing a patent airway is a priority. Blood from facial injuries and aspiration of gastric contents could threaten the airway. If the patient is comatose, endotracheal intubation is the preferred method of airway control. Be sure to maintain cervical spine integrity during any attempts at intubation. If possible, administer **Lidocaine** 1mg/kg prior to the intubation attempt. This may reduce the rise of ICP that is seen with manipulation of the airway.

Breathing

Evaluation of respiratory status may also show signs of a possible head injury. Abnormal respiratory patterns are common in patients with severe head injuries. Insure adequate respirations but **do not** excessively hyperventilate the patient. This is a change in our thinking with respect to treating head injuries based on the latest research studies. While hyperventilation may decrease ICP, it also may decrease blood flow to the brain causing ischemia. Adequately cerebral perfusion is the ultimate goal.

Circulation

Assess circulation and blood pressure. Decreased pulse rates and increased blood pressure are also signs of a head injury. Establish IV access. Patient transport should not be delayed to perform interventions that are not immediately life saving.

History

Obtain a history of the events. Identification of the mechanism of injury will also give you an index of suspicion of head injury. Remember to include past history especially, recent trauma, easy bleeding, headaches, weakness, vomiting, altered mental status, etc. Determine the patient's medications and allergies. Is the patient taking blood thinners?

Physical

Establish a baseline level of consciousness as early as possible during patient assessment. This will allow you to identify any deterioration. Remember to complete your head to toe assessment.

When examining the head, check for Battle's sign (mastoid bruising) and Raccoon's Eyes (periorbital ecchymosis). These are late signs of a basilar skull fracture and often take 12 to 24 hours to manifest.

Special attention should be given to the pupils. The pupils are direct windows into the brain and can quickly and accurately tell the condition of the central nervous system. If pupillary reaction is normal (PERRL) presume that the brain is currently well perfused. If pupils are slow to respond this could be the result of drugs, hypoxia or circulatory insufficiency. If pupils are dilated and fixed suspect a severe central nervous system injury or hypoxia.

In a symptomatic patient, with unequal pupils consider increased ICP with brain herniation on the same side as the affected pupil. The pupil dilates due to pressure on the optic nerve. (Note that some people are born with unequal pupils, anisocoria. If a patient is awake and alert AND has unequal pupils, they do have an elevated ICP. They may have anisocoria, a glass eye, a unilateral eye injury or problem, or use eye drops to constrict or dilate their eyes.)

Evaluate the patients level of consciousness. Avoid terms such as stuporous or lethargic. It is best to describe the patient's response to a stimulus in plain English, rather than relying on poorly-defined terms such as stupor, semi-coma, etc. For example, to verbal stimuli patient raises both hands. By documenting the stimulus/response your trip sheet will be understood by any physician performing serial testing.

Begin by trying verbal stimuli: call the patient's name (shout it); say "wake up!" The truly unresponsive patient will make no response. Patients with lesser impairment of consciousness may groan, open their eyes, or even speak. Describe the response.

If there is no response to voice, use painful stimuli (pinch) to the shoulders, arms, and legs. A sternal rub may be used to assess the response to pain. It is inappropriate and unacceptable in the field to use sharp objects (IV catheters) or to apply painful stimuli to the genitalia.

It is essential to observe and accurately describe the motor response to a painful stimulus. The possibilities are:

1. Localizing response: the patient will direct one or more limbs toward the stimulus in an attempt to ward it off.

2. Decorticate posturing: the upper extremities are **flexed at the elbows** and the lower extremities are extended.

3. Decerebrate posturing: the **elbows are extended** and pronated (rotated outward).

4. No response: This may indicate a marked decrease in brain function and/or a spinal cord injury.

Do not forget reversible causes of altered level of consciousness. Check a chemstrip and administer Narcan if appropriate. Apply the Pulse Ox to insure adequate oxygenation and maintain the patient’s blood pressure. Cardiac abnormalities can occur after a head injury. Therefore, all major head injury patients should be placed on the monitor. Remember to immobilize patients with potential spinal injuries.

The most reliable method of evaluating level of consciousness is the Glasgow Comma Scale (GCS). This gives everyone involved with the patient the same scale to evaluate a patient and reduces confusion as to new deficits.

Eyes		**Motor**		**Verbal**	
Open spontaneously	4	Obeys Commands	6	Oriented	5
To Speech	3	Localizes Pain	5	Confused	4
To Pain	2	Withdraws from Pain	4	Inappropriate	3
Unresponsive	1	Abnormal Flexion	3	Garbled	2
		Extensor Response	2	Unresponsive	1
		Unresponsive	1		

GLASGOW COMMA SCALE

Post Injury

Frequently patients with “minor” head injuries are discharged from the hospital to be observed at home by a family member. These patients are given a discharge instruction sheet from the treating facility listing symptoms to be aware of and any follow up instructions. During the research phase of this project we acquired copies of instructions from several hospitals and trauma centers within the City of Pittsburgh. Most of the instructions were fairly standard; telling the patient/family to look for:

1. *Headaches that either worsen or were not improved with pain medication.*
2. *Frequent or repeated vomiting (more than 2-3 times).*
3. *Unequal pupils.*
4. *Confusion.*
5. *Loss of consciousness.*
6. *Slurred Speech.*
7. *Bleeding or drainage from the nose or ears.*
8. *Difficulty seeing.*
9. *Loss of equilibrium.*
10. *Dizziness.*

Most stated that a headache was a normal reaction to a head injury and that there was nothing abnormal unless it worsened or did not subside after 24 hours. All of the instructions stated that the patient should see either his family physician or return to the emergency department should any of the above complications occur.

The instructions failed to state the serious nature of any of the symptoms and did not necessarily stress that the patient should be seen immediately. All of the instructions stated that the patient should only be monitored for the first 24 hours. After reading this monograph you hopefully understand the seriousness of these symptoms and the need to be alert for symptoms beyond the first 24 hours.

Documentation

Consider documenting the following items on the trip sheet on all head injury patients:

- history, mechanism of injury
- use of blood thinners
- any loss of consciousness, seizures

- blood or CSF from ears or nose
- previous head injury
- use of alcohol or drugs
- past history
- symptoms, i.e., headache, nausea, blurred vision, weakness, numbness
- ability or inability to move extremities

Conclusion

Patients seen in the prehospital setting who have recently experienced a traumatic event should be evaluated for the possibility of a closed head injury. Some head injuries may take up to two weeks to manifest. Therefore, a recent traumatic episode should alert you to the possibility of a delayed head injury. Generally the most reliable indicator of an increasing head injury is a drop in GCS. This may be difficult to evaluate in a patient who has been released from the hospital due to inability to establish a base line level of consciousness. Alcohol abuse and old age further increase the difficulty of detection of decreasing GCS. These patients should be transported to the hospital for definitive diagnosis. As a general rule if it was bad enough for them to call 911 then it is bad enough to require transport. When in doubt, consult the physician for guidance.

Even in the Emergency Department, closed head injuries can be difficult to identify. A CT scan is the only definitive way to rule out a closed head injury.

Potential pitfalls in the care of the head injured patient

- Attributing the patient's symptoms (altered LOC, slurred speech, weakness) to alcohol or drugs
- Forgetting about the lucid interval, the so-called talk-and-die patient.
- Failing to insure the ABCs
- Failure to consider spinal cord injury.
- Poor initial assessment
- Failure to obtain an adequate history, including, mechanism of injury, loss of consciousness, seizures, or use of blood thinners.

NAME_____CE#_____ Date_____

Closed Head Injury Quiz

1. The most reliable indicator of a worsening head injury is?
    A. Frequent vomiting
    B. Drainage from ears or nose
    C. Drop in GCS
    D. Slurred speech

2. A 22 year old male is struck in the head by a batted softball. Bystanders state that the patient was unconscious for 8 minutes but now he is awake with a normal neuro exam. He adamantly refuses transport. He drank 3 beers just prior to the accident. Concerns in this patient include:
    A. He may not be competent to refuse treatment and transport
    B. He may be having a lucid interval
    C. Without a CT scan it is difficult to determine the extent of his head injury
    D. All of the above

3. Which of the following closed head injuries may be slow in onset of symptoms taking up to two weeks to manifest?
    A. Subdural Hematoma
    B. Epidural Hematoma
    C. Cerebral Contusion
    D. Intracranial Hemorrhage

4. Which of the following is the most minor of type of head injury with no lasting damage?
    A. Intracranial Hemorrhage
    B. Subdural Hematoma
    C. Cerebral Contusion
    D. Concussion

5. Administration of _____ prior to intubating a patient with a significant head injury may prevent the rise of ICP associated with manipulating the airway.
    A. Cricoid Pressure
    B. Cervical Traction
    C. Lidocaine
    D. Atropine