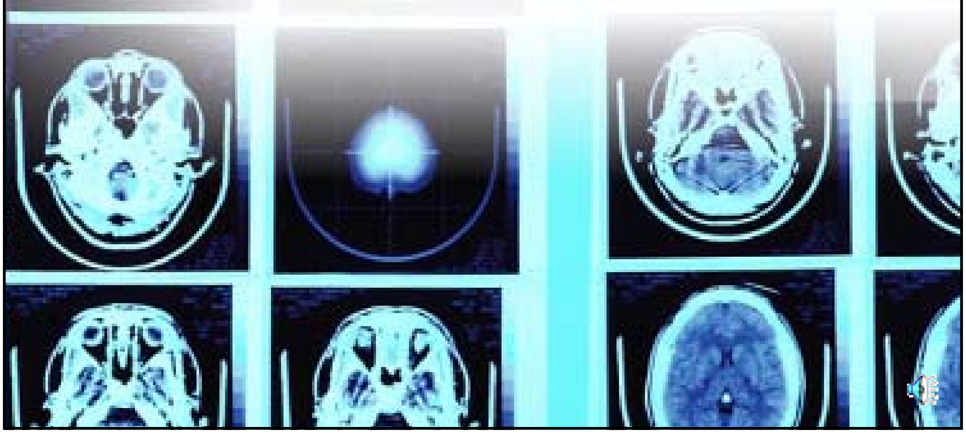
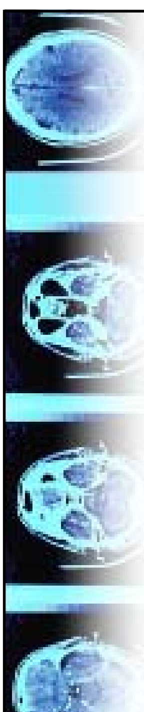
EMC 410
Trauma Management

Chapter 11. Head and Neck Trauma





Unit Objectives

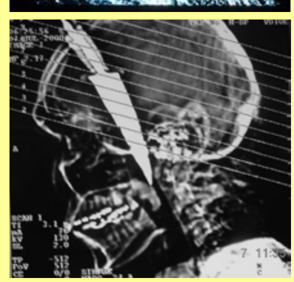
- **Upon completion of this chapter, you should be able to:**
  - Describe the basic epidemiology of head and neck trauma.
  - Describe the concept of primary and secondary brain injury.
  - Discuss the role of initial patient management in reducing secondary brain injury.
  - Discuss the relevant anatomy and pathophysiology of:
    - Increased intracranial pressure (ICP)
    - Loss of consciousness
    - Cushing's response
    - Coupe/contrecoup injuries
  - Describe the anatomical zones of the neck.
  - Describe the major injuries and trauma considerations by zone of the neck.

Unit Objectives continued

- List and describe the pathology and clinical presentations of the major types of intracranial hematomas.
- Contrast concussion and contusion in terms of pathology, clinical presentation, and outcome.
- Discuss the focused assessment of the patient with head and neck injury.
- Describe the Glasgow Coma Scale and its implications in patient care.
- Describe the airway considerations for the initial management of patients with head and neck injury.
- Briefly describe the major research and experimental treatment issues for:
  - Hyperventilation
  - Volume replacement
  - Hypothermia
  - Neuroprotective agents

Epidemiology and Etiology

- **Head**
  - Incidence of 200 head injuries per 100,000 population each year.
  - Half of all head injuries are significant enough to warrant medical care and the majority can be expected to utilize EMS transport.
  - Peak incidence is among males ages 15 to 30.
  - 50% of all traumatic deaths are associated with head injury
  - In MVC deaths, 60%-75% have some form of head injury.
  - Of the 120,000 annual head injuries classified as severe, half will die out-of-hospital with few treatments that could have been rendered to alter outcome.
  - Of these severe head injuries who arrive at the ED alive, most will have or will develop airway compromise and increased ICP.

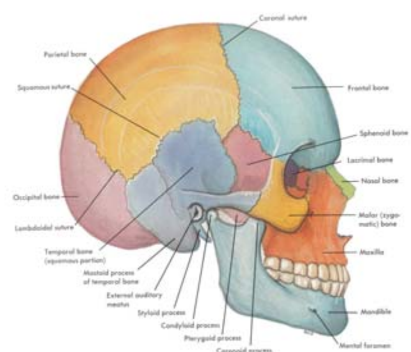


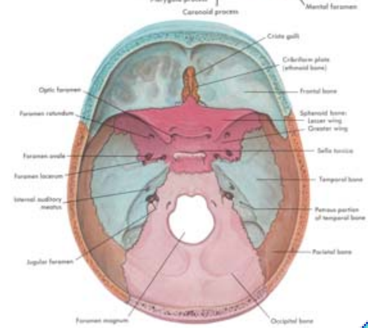
Epidemiology and Etiology continued

- **Neck**
  - Less than 10% of all traumatic injuries involve serious injury to the neck.
  - Although relatively infrequent, neck injuries carry a high risk for morbidity and mortality.
  - Penetrating neck injury tends to be the most common injury, and result mostly from gun or knife wounds.
  - Blunt neck trauma is less common but carries a tremendous risk for airway compromise.
  - Up to one half of neck trauma deaths may be prevented with appropriate early treatment.

Anatomy

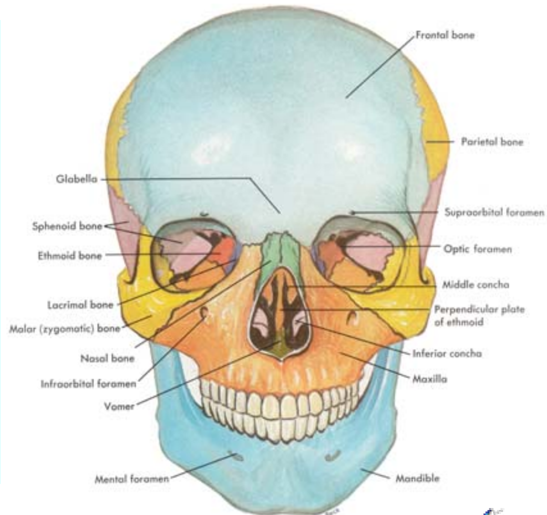
- **Scalp**
  - Composed of 5 highly vascular layers
- **Skull**
  - Cranium
    - 8 bones: frontal, occipital, ethmoid, sphenoid, parietal (2) and temporal (2)
    - All eight bones contribute to form the jagged cranial floor.
    - Forms a closed container of 1900 cc that is completely filled by the brain, spinal fluid, and blood.
    - Foramen magnum forms the large passageway for the spinal cord





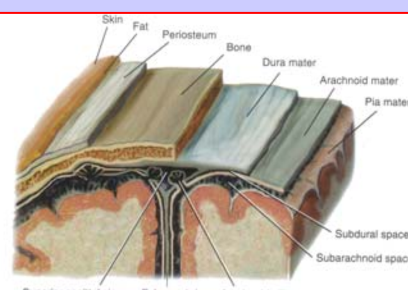
Anatomy continued

- **Face**
  - Maxilla
  - Zygoma
  - Mandible



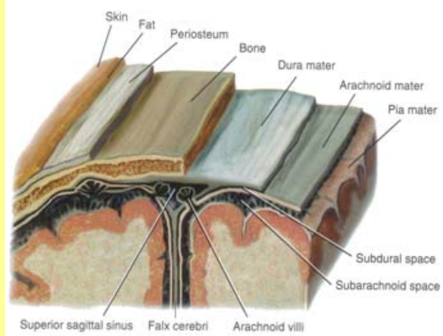
Anatomy continued

- **Meninges**
  - Dura mater
    - Epidural space contains the middle meningeal arteries
    - Dura has 2 projects into the cranial vault:
      - Falx cerebri
      - Tentorium cerebelli
    - Dural sinus are folds of the dura which serve as collecting areas for venous blood
    - Subdural space separates the arachnoid and dura and is crossed by small venous vessels that empty into the dural sinuses



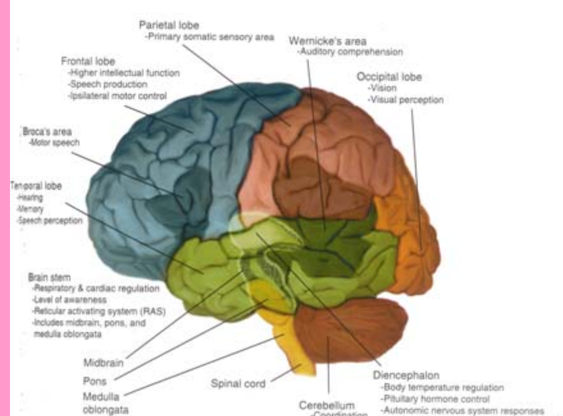
Anatomy continued

- **Meninges continued**
  - Arachnoid
    - Subarachnoid space is filled with CSF
    - CSF is produced by the choroid plexus in the ventricles, circulated through the ventricular system, and reabsorbed by arachnoid villi into the dural sinuses.
  - Pia mater
    - Outermost layer of the brain



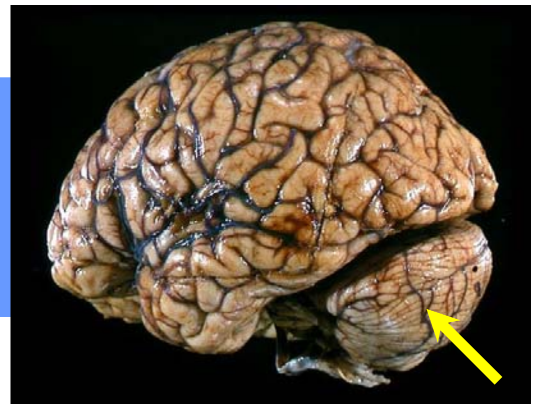
Anatomy continued

- **Brain**
  - Cerebrum
    - Two cerebral hemispheres
      - Frontal
      - Occipital
      - Temporal
      - Parietal
    - Diencephalon - connection and processing point of the two hemispheres
    - Controls higher order activities such as motor control, behavior, speech, and sensation



Anatomy continued

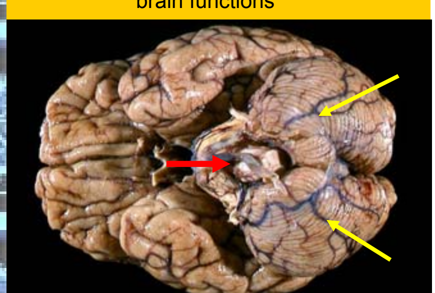
- **Cerebellum**
  - Responsible for equilibrium, locomotion, and coordination

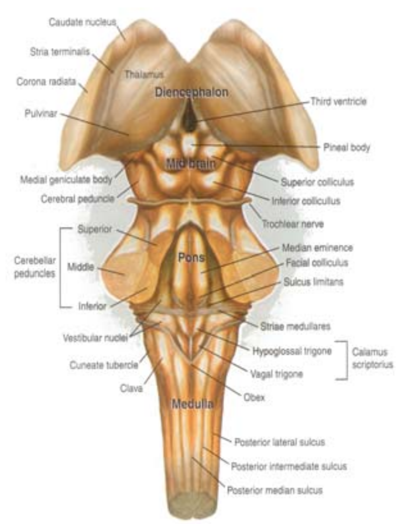


Anatomy continued

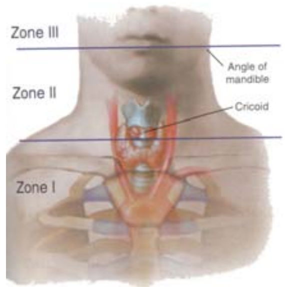
Brain continued

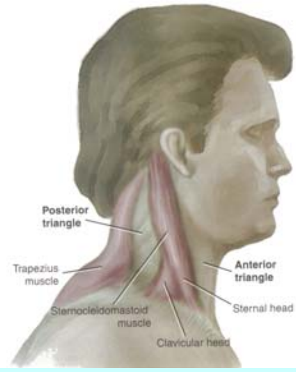
- Brainstem
  - Midbrain
  - Pons
  - Medulla oblongota
  - Unites the spinal cord, cerebellum, and cerebrum, and is responsible for lower brain functions





Anatomy continued

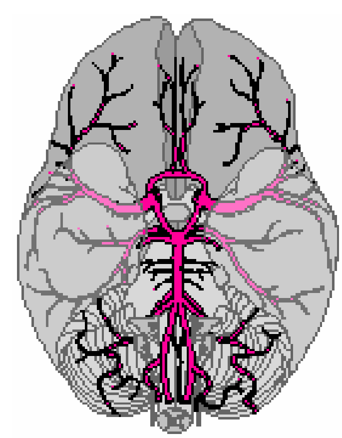




- **Neck**
  - Relatively unprotected, yet contains vital structures such as airway, great vessels, major neurological structures, and the esophagus.
  - Divided anatomically into zones 1-3

Physiology

- **Cerebral Perfusion**
  - Brain consumes 20% of all the oxygen used by the body
  - To meet these demands, the brain has a very high blood flow and oxygen extraction ratio
  - Interruption of blood flow for 5-10 seconds can result in syncope.
  - Carotid and vertebral arteries are the major vessels supplying the brain.
  - Carotid sinuses alter cardiac function to maintain adequate brain perfusion.
  - The brain is also able to maintain cerebral blood flow by altering vessel diameter called autoregulation.
  - Autoregulation works as long as arterial pressure is between 80 - 180 mm Hg

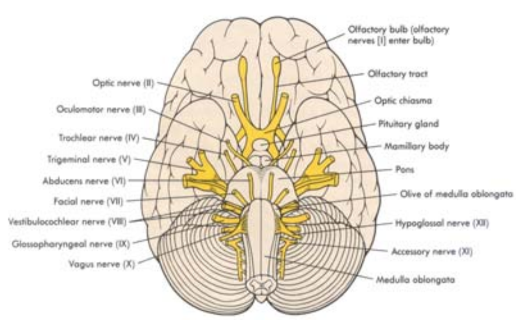


Physiology continued

- **Cerebral Perfusion continued**
  - Carbon dioxide is the trigger to increase cerebral blood flow.
    - Every decrease in $PaCO_2$ of 1 mm Hg, cerebral blood flow decreases by 2 to 4 percent
    - If $PaCO_2$ is decreased to 15 - 20 mm Hg, cerebral blood flow can be diminished to the point of causing cerebral hypoxia.
  - Oxygen levels are a secondary mechanism for regulating cerebral blood flow.
    - High PaO_2 levels cause cerebral vasoconstriction while low levels cause vasodilation.
  - Cerebral Perfusion Pressure (CPP) = MAP - ICP
  - Normal ICP is 2 - 12 mm Hg.
  - CPP must be maintained above 60 mm Hg to prevent cerebral ischemia.

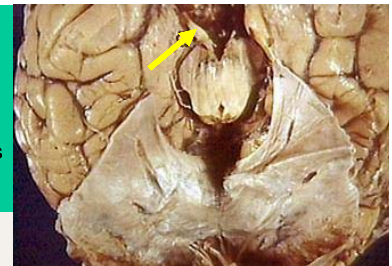
Physiology continued

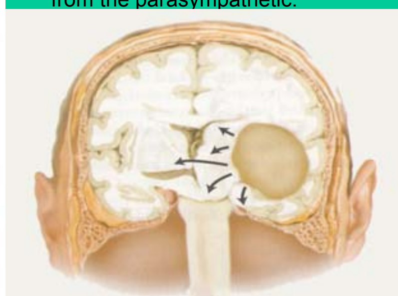
- **Pupil Response**
  - Sympathetic system
    - Dilates the pupil
    - Impulse originates in the hypothalamus and travels down into the spinal cord, exiting at T-1, and return to the cranium by following the internal carotid artery.
  - Parasympathetic system
    - Constricts the pupil
    - Impulse originates in the brainstem and travels within the oculomotor nerve (CN III) to the iris



Physiology continued

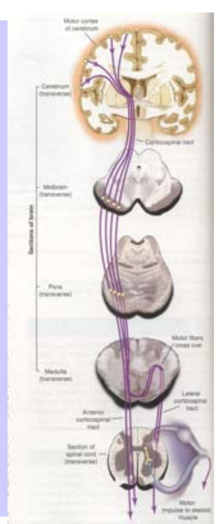
- Herniation of brain tissue in a mass effect causes pressure on the oculomotor nerve. This results in loss of parasympathetic tone resulting in a dilated pupil as the sympathetic impulse reaches the iris by following a pathway separate from the parasympathetic.





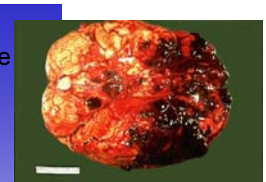
Physiology continued

- **Motor and Sensory Pathways**
  - Each cerebral hemisphere provides voluntary motor control for the opposite side of the body.
  - Motor impulses travel via the corticospinal fibers and cross over in the medulla.
  - Sensory fibers for pain, temperature and touch enter the spinal cord and cross over to the opposite side to form the spinothalmic tract.
  - This crossover of sensory and motor impulses provides the ipsilateral pupil dilatation and contralateral paralysis pattern of head injury.



Pathophysiology

- **Mechanisms of Injury**
  - Acceleration/deceleration injuries produce the coup/contrecoup pattern
  - Penetration injuries may also damage brain tissue via cavitation
  - Primary brain injury
    - Results from the transfer of kinetic energy at the moment of impact
    - Typically requires surgical treatment
  - Secondary brain injury
    - Results from pathological processes that impair cerebral perfusion, oxygenation, and nutrition, and may occur minutes, hours, or even days later.
    - Common mechanisms that lead to additional tissue infarction are hypoxia, systemic hypotension, intracranial hypertension, infection, and seizures.
    - Unlike primary injuries, initial care can significantly alter outcome.



Pathophysiology continued

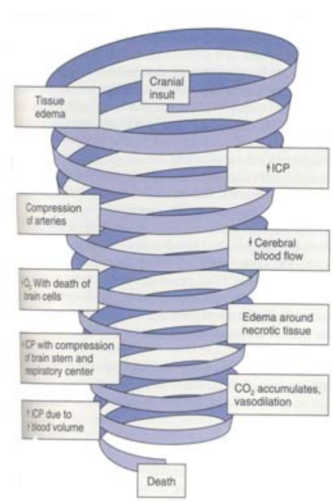
- **Loss of Consciousness**
  - Hallmark sign of significant brain injury.
  - The RAS is located in the brainstem and has widespread neuronal projections into the cerebral hemispheres, which regulates consciousness.
  - The initial loss of consciousness in head trauma is usually due to disruption of the RAS.
  - Typically patients regain consciousness if there is no anatomical damage. Persistent unconsciousness indicates dysfunction of both cerebral hemispheres or the brainstem.
  - In less severe injuries, a wide range of altered levels of consciousness are possible.
  - Patients with significant neurological dysfunction and unconsciousness may demonstrate decerebrate posturing (midbrain) or decorticate posturing (hemispheric dysfunction).
  - Patients with posturing have a 60% to 70% mortality.

Pathophysiology continued

- **Increased Intracranial Pressure**
  - Monro-Kellie principle
    - Limited volume completely filled with brain, blood, and CSF
    - Increases in one component must be compensated by a decrease in another component or the pressure will rise.
  - Initial compensation for volume increase is movement of CSF and venous blood out of the vault.
  - This mechanism can compensate for volume changes of 30 to 50 ml, after which, small increases in volume result in much larger changes in ICP.
  - Normal ICP is 2 to 12 mm Hg and sustained pressures greater than 20 to 30 mm Hg are associated with increased risk of secondary brain injury and poor outcome.

Pathophysiology continued

- **Increased Intracranial Pressure continued**
  - CPP = MAP - ICP
    - As ICP rises, CPP decreases unless the body compensates
    - Cushing's triad (increased blood pressure, decreased pulse, and irregular respirations are the signs of compensatory mechanisms.
  - Patients who are hypotensive are very sensitive to even small increases in ICP
  - Injured brain tissue also loses the ability to autoregulate



Pathophysiology continued

- **Brain Herniation**
  - Shape of the cranium and dural projections form a funnel-like pathway toward the brainstem and foramen magnum.
  - Uncal Herniation
    - Lesion displaces one of the cerebral hemispheres.
    - Displacement forces a portion of the temporal lobe (uncus) to herniate over the edge of the tentorium cerebelli.
    - Herniation compresses the motor tracts resulting in contralateral paralysis or weakness.
    - Compression of the oculomotor nerve results in ipsilateral pupil dilation.
    - Unchecked, ICP will eventually result in pressure on the brain stem and Cushing's triad.

Pathophysiology continued

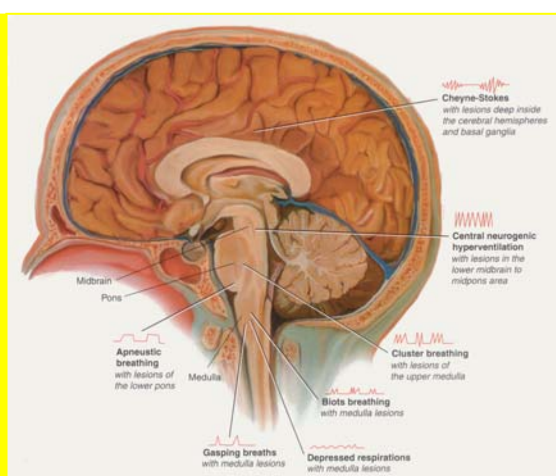
- **Brain Herniation continued**
  - **Central Herniation Syndrome**
    - **Central lesion displaces both hemispheres downward toward the tentorium.**
    - **Results in bilateral muscle weakness or paralysis.**
    - **Associated with progressive loss of function, late bilateral pupil dilation, Cheyne-Stokes respirations, and decorticate posturing.**

Pathophysiology continued

- **Brain herniation continued**
  - Posterior Fossa Herniation
    - Cerebellum can be forced to herniate out of its compartment
    - Because of the early compression on the brainstem, loss of consciousness, respiratory dysfunction, and cardiac dysfunction occur early.

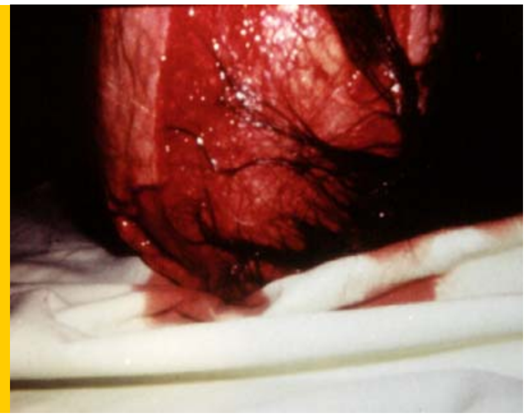
Pathophysiology continued

- **Brainstem Compression**
  - Progression can be traced by breathing patterns



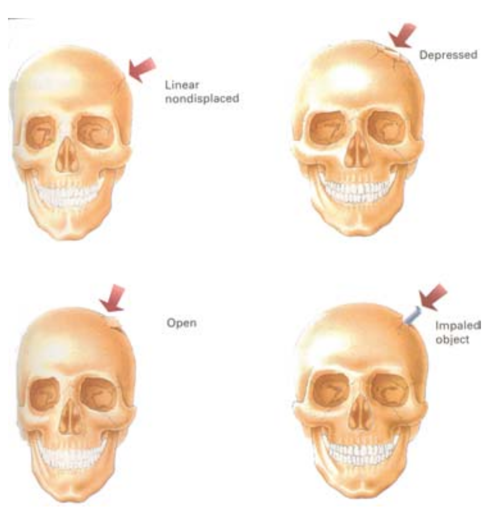
Pathophysiology continued

- **Scalp Injuries**
  - Typically non-lethal, but can bleed profusely
  - Large hematomas can form between galea and periosteum



Pathophysiology continued

- **Skull Fractures**
  - Linear
    - Typically benign unless there is underlying brain injury
  - Depressed
    - More likely to have brain injury and long-term deficits
  - Open
    - Places patient at risk for infection

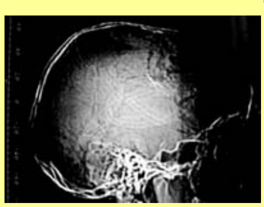


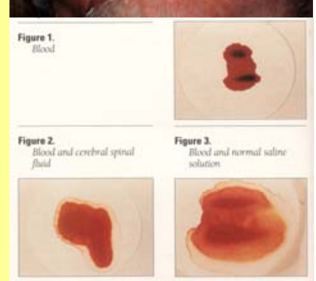
Pathophysiology continued

- **Skull Fractures continued**
  - Basilar
    - 24% incidence among severe head injuries
    - Fracture of the temporal bone can present with blood or CSF in the auditory canal, hemotympanum, and facial (CN VII) or vestibular nerve (CN VIII) dysfunction.
    - Late sign of temporal bone fracture is Battle's sign

Pathophysiology continued

- **Skull Fractures continued**
  - Basilar Fracture continued
    - Fractures of the frontal, ethmoid, or sphenoid bones can present with CSF from the nose or olfactory nerve dysfunction, and later, raccoon's eyes
    - Testing methods for CSF





Pathophysiology continued

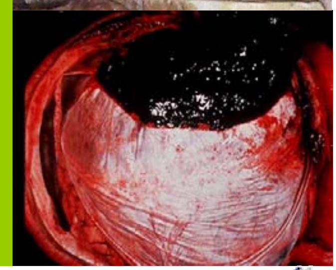
- **Diffuse Brain Injuries**
  - Concussion
    - Disruption of RAS without structural damage
    - Brief period of unconsciousness with rapid recovery and post-concussion mental clouding that clears within minutes to hours
  - Diffuse axonal injury
    - Traditional theory holds that tensile strain pulls white and gray matter at differing rates because of their differing densities, resulting in separation
    - Newer theory suggests that neurons dismantle their cytoskeletal structure, resulting in axonal separation, as a consequence of trauma, hypoxia, and other preventable metabolic derangements.
    - It is believed that barbiturate coma prevents DIA by preventing the secondary axonal destruction.

Pathophysiology continued

- **Focal Brain Injuries**
  - Contusion
    - Impossible to distinguish from a concussion in the field.
    - Mildest form of focal injury.
    - Presents with microhemorrhage
  - Cerebral laceration
    - Typically seen on brain surfaces that contact the orbital roof and sphenoid wing
    - 40% mortality rate

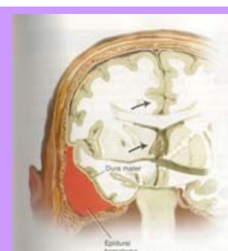
Pathophysiology continued

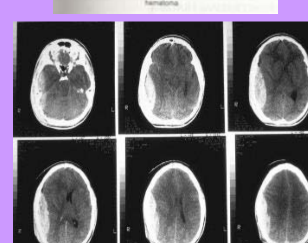
- **Intracranial Hematomas**
  - Epidural
    - Occurs in 1% of all head injuries
    - 90% are the result of fracture of the temporal bone with disruption of the middle meningeal artery with rapid bleeding
    - Requires relatively small force
    - Initially disrupts the RAS, followed by increasing LOC, then decreasing LOC as hematoma expands and ICP rises (1/3 of cases)
    - 1/3 of patients will never lose consciousness and 1/3 will never regain consciousness



Pathophysiology continued

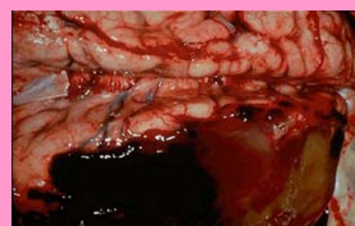
- **Intracranial Hematomas continued**
  - Epidural continued
    - Expanding lesion can result in uncal herniation with ipsilateral pupil dilation and contralateral weakness or paralysis
    - Only 50% will have unequal pupils, and if so, only 80% will be ipsilateral
    - Hemiparesis occurs in 50% - 70% of cases (95% contralateral)
    - Treatment includes moderate hyperventilation, hyperosmotic intravenous fluids, steroids, and burr hole.
    - Prompt treatment has good prognosis; delayed treatment is usually fatal.





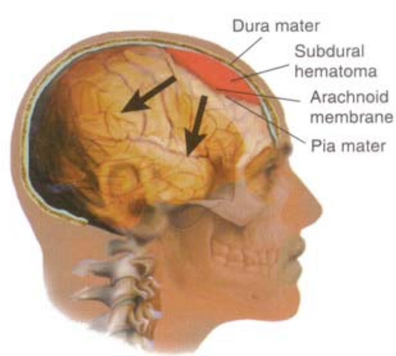
Pathophysiology continued

- **Intracranial Hematomas continued**
  - Subdural Hematoma
    - Caused by shear forces that tear surface blood vessels as the brain moves within the skull
    - Injured vessels are typically bridging veins as they cross the subdural space to empty blood into the dural sinus
    - Elderly and alcoholics are prone to this type of injury



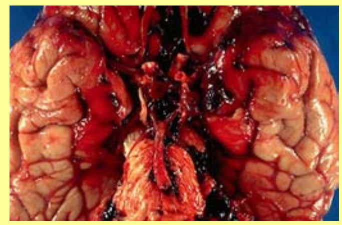
Pathophysiology continued

- **Intracranial Hematomas continued**
  - Subdural hematoma continued
    - Because the injured vessels are venous, hematomas large enough to cause a mass effect may take days to weeks to develop.
    - However, not all subdural hematomas form slowly
    - An enlarged pupil is seen in 50%-80% of cases, and typically on the side of injury.
    - Posturing and hemiparesis on the contralateral side are found in 50% of cases.
    - Mortality rates are high. 12% in patients who regain consciousness, 27% in patients who remain unconscious, and 78% in patients with posturing.



Pathophysiology continued

- **Intracranial Hematomas continued**
  - Subarachnoid Hemorrhage
    - Occur secondary to damage to the small arterial vessels that lie on outermost surface of the brain.
    - Occur as the brain moves across the uneven internal surface of the cranium.
    - Most common type of intracranial hematoma, but also the least significant.
    - Hemorrhage diffuses throughout the CSF causing S&S of meningeal irritation: headache, decreased mentation, nuchal rigidity, confusion, stupor, hemiparesis, and posturing.



Pathophysiology continued

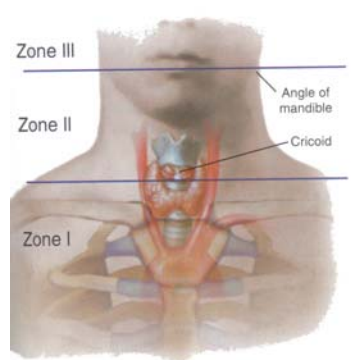
- **Intracranial Hematomas continued**
  - **Intracerebral Hematoma**
    - Surgically correctable cause of secondary brain injury if recognized and treated early.
    - May not present with S&S until days after the initial insult.
    - May produce a mass effect.
    - Mortality rate of 55%

Complications of Head Trauma

- Pituitary injury with diabetes insipidus with massive renal diuresis.
- Decubitus ulcers
- Contractures
- Stress ulcers
- Coagulopathy

Neck Injuries

- **Zone I** - May involve major vascular and intrathoracic structures. Carry the highest mortality rate.
- **Zone II** - Most common and typically the least fatal neck injuries. Carotid artery and airway problems are common.
- **Zone III** - Difficult to assess.



Neck Injuries

- **Vascular Injuries**
  - Immediately life-threatening
  - In addition to hemorrhage risk, injury also decreases blood flow to the brain.
  - Expanding hematomas may compromise the airway.
- **Trachea and Larynx Injuries**
  - Crush injury to the larynx or trachea
  - High risk of complete airway obstruction.
  - S&S

Neck Injuries continued

- **Pharynx and Esophagus Injuries**
  - Typically the result of penetrating trauma.
  - Difficult to detect.
  - Delayed treatment of esophageal injuries have a mortality rate of nearly 100%.
- **Neurological Injuries**
  - Damage to brachial plexus, cervical plexus, phrenic nerve, recurrent laryngeal nerve, and cranial nerves
  - Injury to the recurrent laryngeal nerve may paralyze the vocal cords in a closed position.
  - Phrenic nerve injury may paralyze the diaphragm.

Initial Assessment

- **Airway and cervical spine**
  - Patients with head injuries are 4 times more likely to have spinal injury
  - Neck trauma may produce bleeding directly into the airway
  - Hoarseness, stridor, and voice change are signs of impeding airway obstruction
  - Palpate for subcutaneous emphysema
- **Breathing**
  - Zone I injuries are frequently associated with thoracic injuries that may impair breathing.

Initial Assessment continued

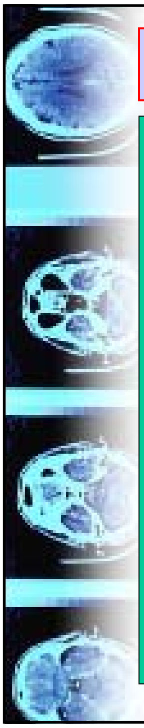
- **Circulation**
  - Do not assume that head injury alone is the cause of shock or hypotension.
  - Autonomic instability is an unlikely source of initial hypotension.
  - Only in infants is intracranial blood loss of sufficient volume to cause hypovolemia.
- **Disability and Exposure**
  - AVPU
  - PMS
  - Pupils
  - Detailed neurological exam is part of the focused assessment.

Focused Assessment

- **History**
  - MOI
  - Loss of consciousness
  - Pre-existing and precipitating medical disorders
  - Intoxication
- **Vital Signs**
- **Neurological Exam**
  - LOC and Mental Status
    - If C&A, increased ICP is unlikely
    - When reporting mental status alterations, simply describe deficits.
  - GCS

Continued Assessment continued

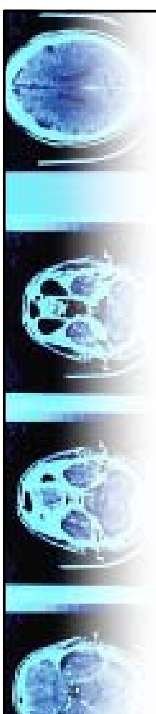
- **Neurological Exam continued**
  - Mental Status Exam
    - Person, place, time, and event
    - Amnesia
    - 3 object recall for testing short-term memory
- **Pupil Function and Cranial Nerves**
  - Pupils
    - Differences in pupil size greater than 1 mm is considered abnormal
    - 5% of normal population have anisocoria
    - 50% of head injured patients may have equal pupils
    - Pupillary response may be altered by drugs, local trauma, and hypotension
    - ICP causes loss of sharp margin to optic disk edge and engorgement of retinal vessels.



Continued Assessment continued

- **Pupil Function and Cranial Nerves continued**

CN I	Olfactory	Ability to smell soap, tobacco, ect.
CN II	Optic	Visual Acuity, Visual Fields, Ophthalmoscopic
CN III	Oculomotor	Pupillary reaction, extraocular movement
CN IV	Trochlear	Extraocular movement
CN V	Trigeminal	Sensory - Corneal reflex, light touch to face.
		Motor - Forced opening of mouth to resistance.
CN VI	Abducens	Extraocular movement
CN VII	Facial	Raise eyebrow, forced eyelid closing, puff out cheeks.
CN VIII	Vestibulo-	Audio acuity
	cochlear	Balance, Romberg
CN IX	Glossopharyngeal	"say AH", elevate palate
CN XI	Spinal Accessory	Raise shoulders against resistance
CN X	Vagus	Swallow, phonation
CN XII	Hypoglossal	Stick out tongue, "say la-la-la"

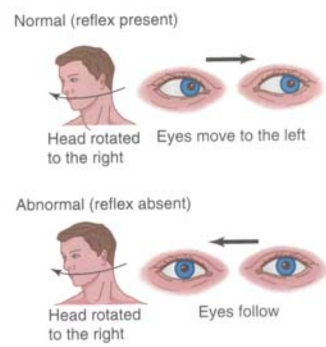


Continued Assessment continued

- **Peripheral Sensory and Motor Function**
  - Sensory and gross motor function of each limb should be assessed for symmetry and strength.
  - Unilateral deficits can by caused by central lesion or peripheral trauma.
    - Upper Motor Neurons
      - Connect motor pathways of cerebral cortex to spinal cord
      - Injury results in spastic paralysis, hyperreflexia and positive Babinski
    - Lower motor neurons
      - Pathway from spinal cord to the muscle
      - Injury results in flaccid paralysis, hyporeflexia, and no Babinski

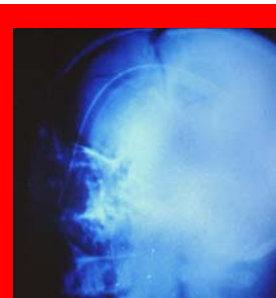
Continued Assessment continued

- **Doll's eyes**
- **Cold calorics**



Treatment

- **Airway**
  - Early intubation and aggressive airway control improve outcome
  - Orotracheal intubation is preferred
  - RSI if indicated
  - Premedicate with 1 mg/kg lidocaine prior to intubation
  - Surgical airways frequently required in neck trauma
  - Laryngeal and tracheal injuries may be worsened by intubation attempts.

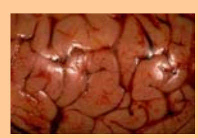


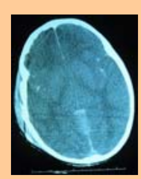
Treatment continued

- **Breathing and Hyperventilation**
  - Maintain minimum PaO_2 of 90 mm Hg
  - Hyperventilation is controversial.
- **Circulation and Fluid Resuscitation**
  - Aggressive fluid resuscitation is indicated in the head-injured patient with hypovolemia and hypotension.
  - Goal is to maintain adequate cerebral blood flow while keeping the patient normovolemic.
  - ICP may result in hypertension. Antihypertensives are not appropriate.
  - Bleeding vessels should not be clamped.
  - IV access for neck injuries should be on the opposite side of the injury, and in zone I injuries, IV access should be both above and below the diaphragm.

Treatment continued

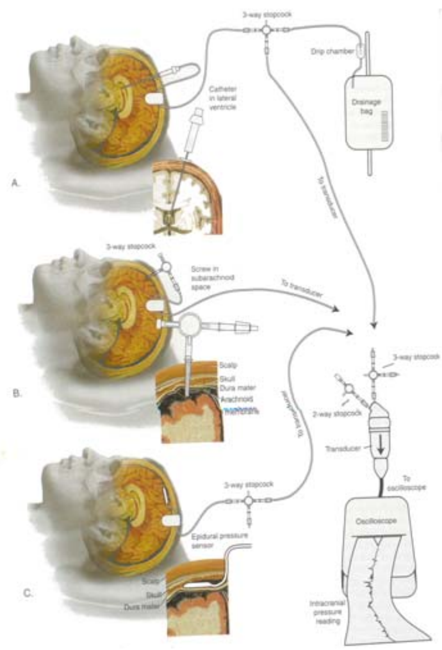
- **Diuretics**
  - Mannitol (1 g/kg over 30 minutes)
  - Lasix (no effect on ICP without mannitol)
- **Patient position**
  - Elevate head 30 degrees if possible
  - May have no effect on CPP
- **Seizures**
  - Early seizures are poor prognostic indicator
  - In paralyzed patients, seizure activity may present as increased pulse, hypertension, and pupillary change.
  - Seizures can result in increased secondary injury from increased ICP and cerebral hypoxia.
  - Seizures treated in the field with valium and phenytoin in the hospital.





Treatment continued

- **Hospital**
  - Radiographic
  - Hemodynamic monitoring
  - ICP monitoring



Current Research

- **Hyperventilation**
  - Autoregulation impaired by injury resulting in variation in cerebral blood flow.
  - Results in increased blood flow into the lesion and ischemic blood flow in previously normal areas, worsening ischemia in normal tissue.
  - Recommendation is that unless attempting to decrease ICP in the face of pending herniation, aggressive hyperventilation should be avoided.
- **Volume Replacement**
  - Some areas have shifted focus from managing ICP to maintaining CPP through volume expansion, vasopressors, and drainage of CSF, and mannitol.
  - Hypotension (< 90 mm Hg) is associated with 150% increase in mortality.

Current Research Continued

- **Hypothermia**
  - Hypothermia decreases cerebral metabolism and improves cerebral ischemia, oxygen consumption, and reduces ICP in head injury.
- **Neuroprotective Agents**
  - Numerous experimental drugs being investigated that are designed to prevent the neurotoxic cascade that follows primary injury.
- **Steroids**
  - Decadron
  - Solu Medrol