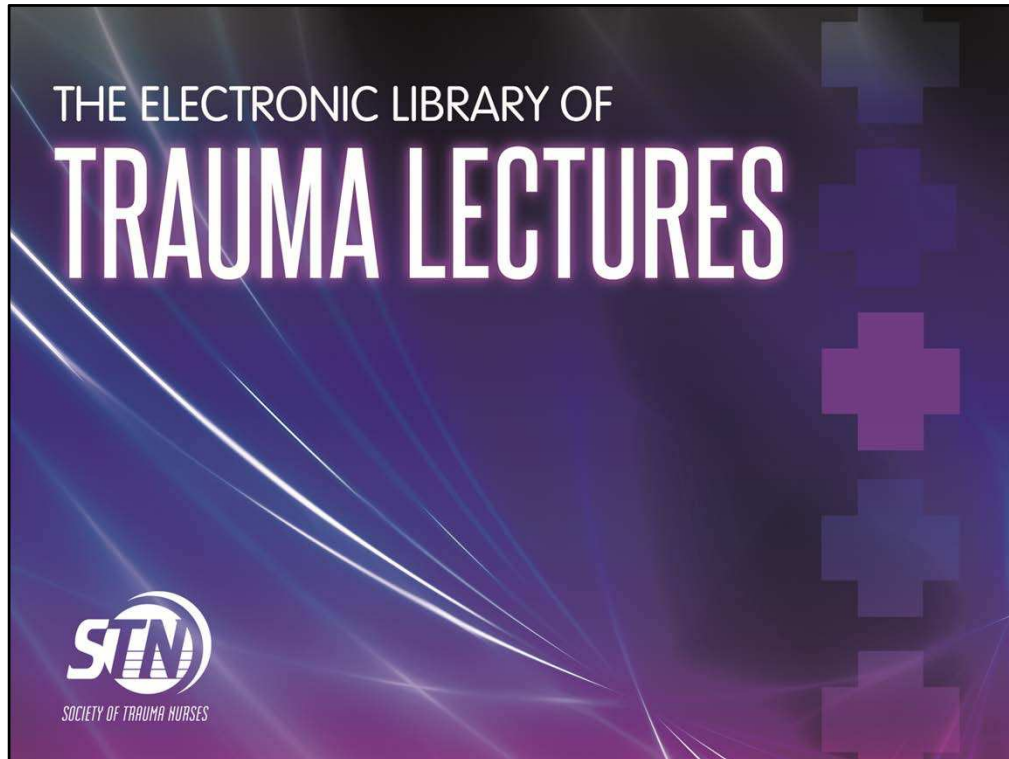
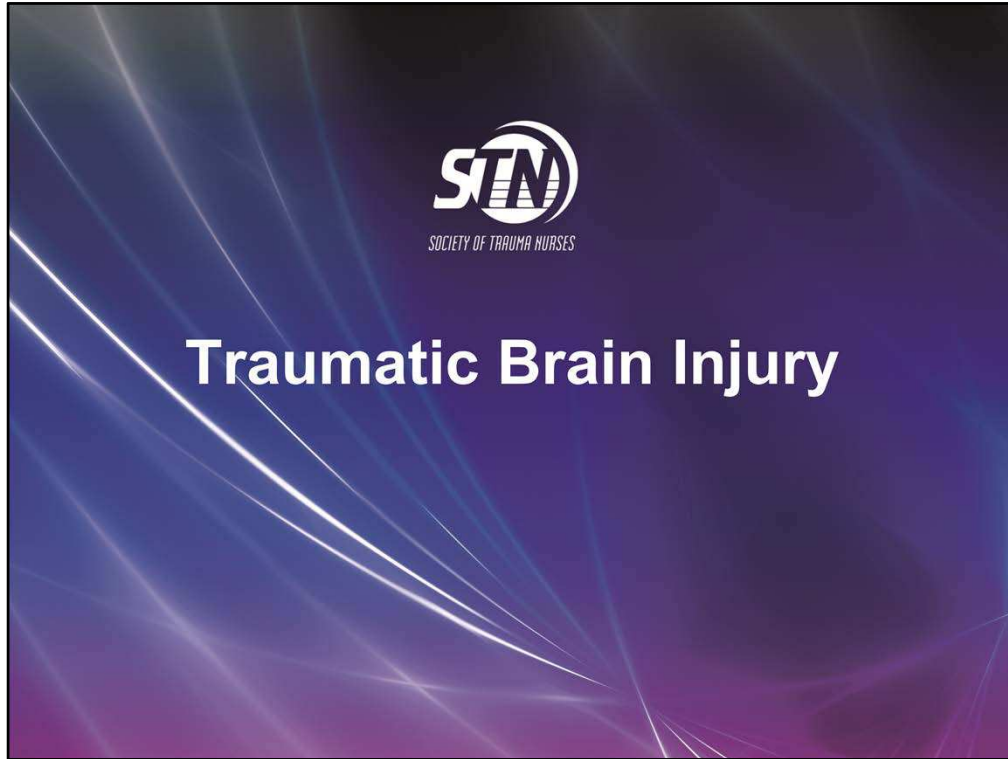






Objectives

At the conclusion of this presentation the participant will be able to:

- Identify the functional anatomy of the brain and the effects of traumatic brain injury (TBI)
- Describe the initial management and neurological assessment of the TBI patient
- Explain management strategies to reduce the risk of secondary injury



Traumatic Brain Injury (TBI)

Definition
Impairment in brain function as a result of mechanical force



- Traumatic Brain Injury (TBI) is a brain dysfunction that can be temporary or permanent, and may or may not result in underlying structural changes in the brain

Epidemiology



- U.S. 1.7 million TBI annually 52,000 die
- 275,000 hospitalized
- 1.365 million treated & released from ED
- 30,000 Children disabled annually
- Costs \$48.3 billion annually

Aspen Photo/Shutterstock.com



- According to the Center for Disease Control (CDC) 2012 statistics, each year an estimated 1.7 million people in the United States sustain a Traumatic Brain Injury (TBI).
- TBI is a contributing factor to a third (30.5%) of all injury-related deaths in the United States
- About 75% of TBI's that occur each year are concussions or other forms of mild TBI
- The number of people with TBI who are not seen in an emergency department or who receive no care is unknown
- Approximately 18% of all TBI-related emergency department visits involved children aged 0 to 4 years.
- Approximately 22% of all TBI-related hospitalizations involved adults aged 75 years and older.
- Males are more often diagnosed with a TBI (59%).

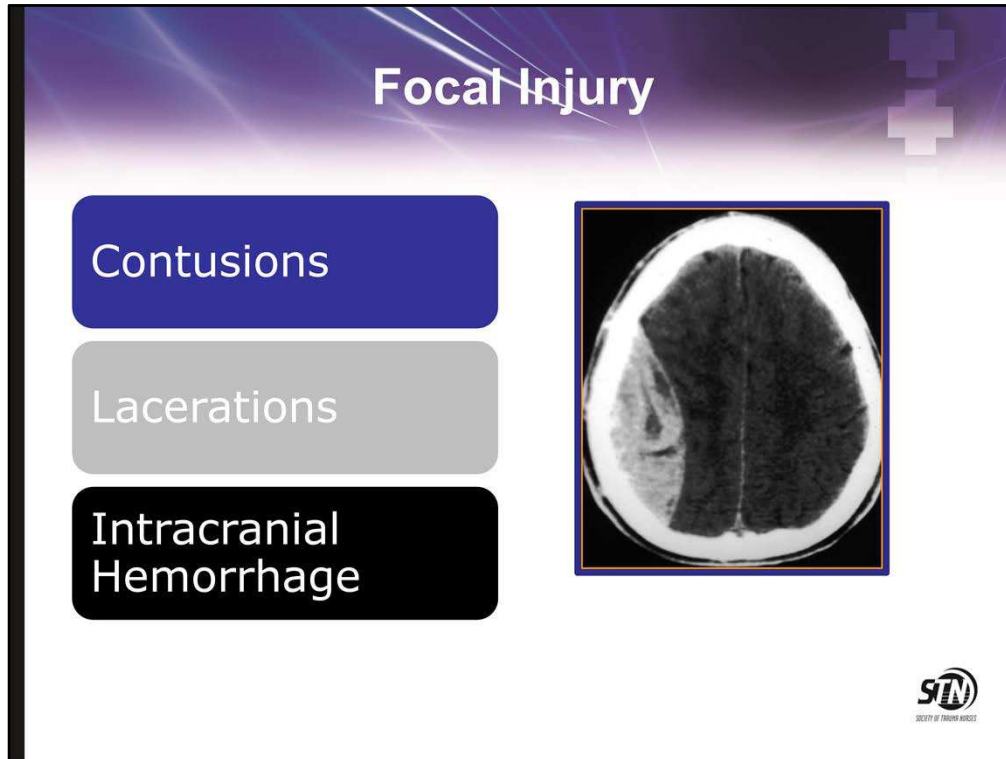
Mechanisms of Injury

- Penetrating
 - Gun Shot Wounds
 - Other penetrating
- Blunt
 - Falls
 - MVC
 - Bicycle Crash
 - Pedestrian
 - Assault



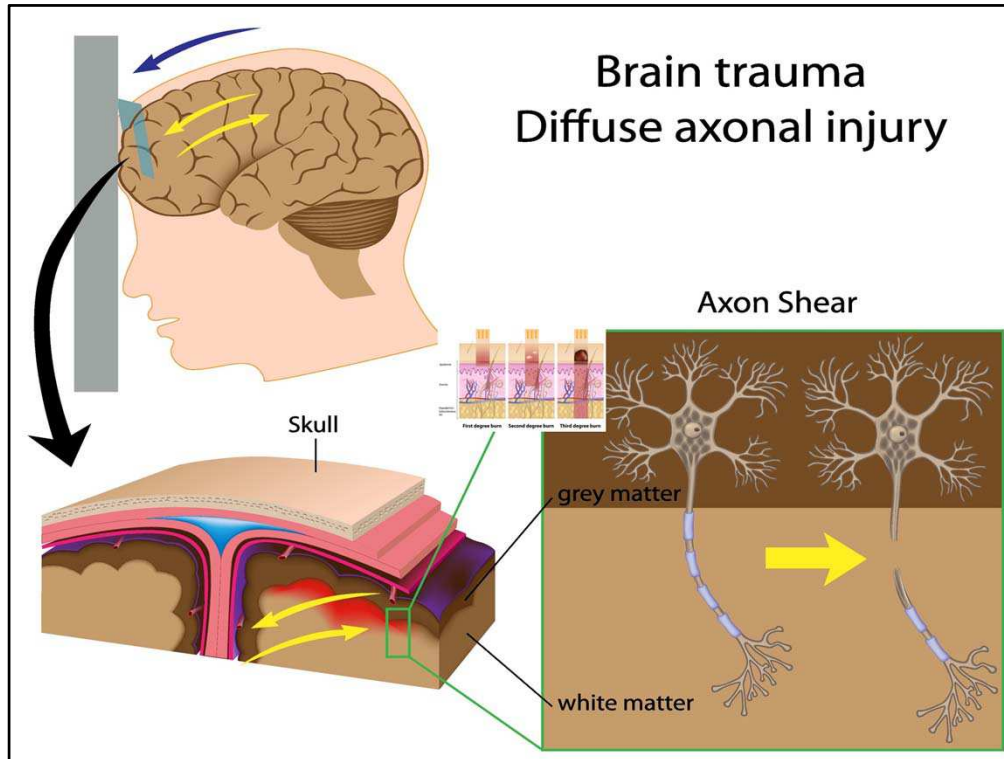
The slide features two images. The top image shows a hand holding a handgun, firing a bullet, with a bright muzzle flash. The bottom image shows a shattered glass surface with a large, circular impact point and significant blood splatters, illustrating a blunt force injury.

- According to CDC 2012 data the leading causes of TBI are:
 - Falls (35.2%);
 - Motor vehicle/traffic (17.3%);
 - Struck by/against events (16.5%); and
 - Assaults (10%).¹
- Falls continue to be the leading cause of TBI in the United States. Falls cause half (50%) of the TBI's among children aged 0 to 14 years and 61% of all TBIs among adults aged 65 years and older.
- Among all age groups, motor vehicle crashes and traffic-related incidents were the second leading cause of TBI (17.3%) and resulted in the largest percentage of TBI-related deaths (31.8%).
- In New York State 58% of all firearm-related deaths are due to a TBI.



- Focal brain injury is confined to one area of the brain.
- In most cases the brain tissue is damaged at the site where the injury occurred.
- Focal injuries can be open or closed depending on the mechanism of injury.
- Contusions are defined as bruising of the brain.
- Bleeding within the skull is called an intracranial hemorrhage.

• **Image is of epidural hematoma**



Diffuse Brain Injury due to acceleration / deceleration

- Diffuse Axonal Injury (DAI)
- Brain Swelling

• Diffuse brain injuries occur throughout the brain over a widespread area. Diffuse Axonal Injury (DAI) falls under the category of a diffuse brain injury and is a result of the brain moving back and forth in the skull as a result of acceleration and deceleration.

• Falls, assaults, automobile accidents, sports related injuries are common causes of DAI.

• Diffuse axonal injury (DAI) is one of the most common and devastating types of TBI. There are extensive lesions in white matter tracts, and is one of the major causes of unconsciousness and persistent vegetative state. It occurs in about half of all cases of severe intracranial head trauma and can also occur in moderate and mild TBI.

The outcome is frequently coma with over 90% of patients with severe DAI never regaining consciousness.. There is often significant impairment in those patients who do wake up.

Traumatic Brain Injury (TBI)

Primary Injury

- Occurs at the moment of impact due to blunt or penetrating mechanism

Prevention

- Includes fall prevention, bicycle helmets, pedestrian safety awareness, gun violence awareness



- Primary Injury occurs as a result of the energy transmitted to the brain tissue at the time of the impact. The only way to prevent primary injury is through injury prevention.
- Following injury prevention guidelines can prevent many disabilities and deaths associated with TBI.
- The Bureau of Injury Prevention maintains several TBI prevention programs including passenger safety, helmet safety and pedestrian safety programs.
- Injury prevention and safety awareness is imperative to prevention of primary injury.
- Despite increasingly effective efforts in prevention TBI remains the leading cause of morbidity and mortality around the world.

Traumatic Brain Injury (TBI)

Secondary injury caused by events AFTER the primary injury

- inadequate perfusion (hypotension)
- hypoxia

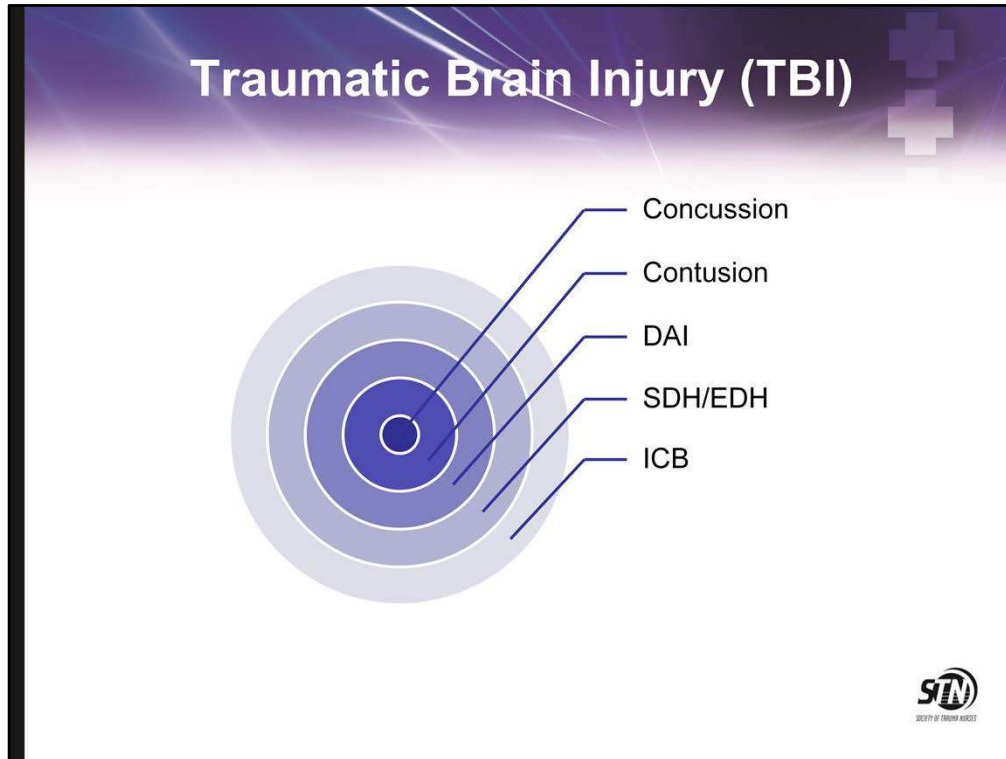


STN
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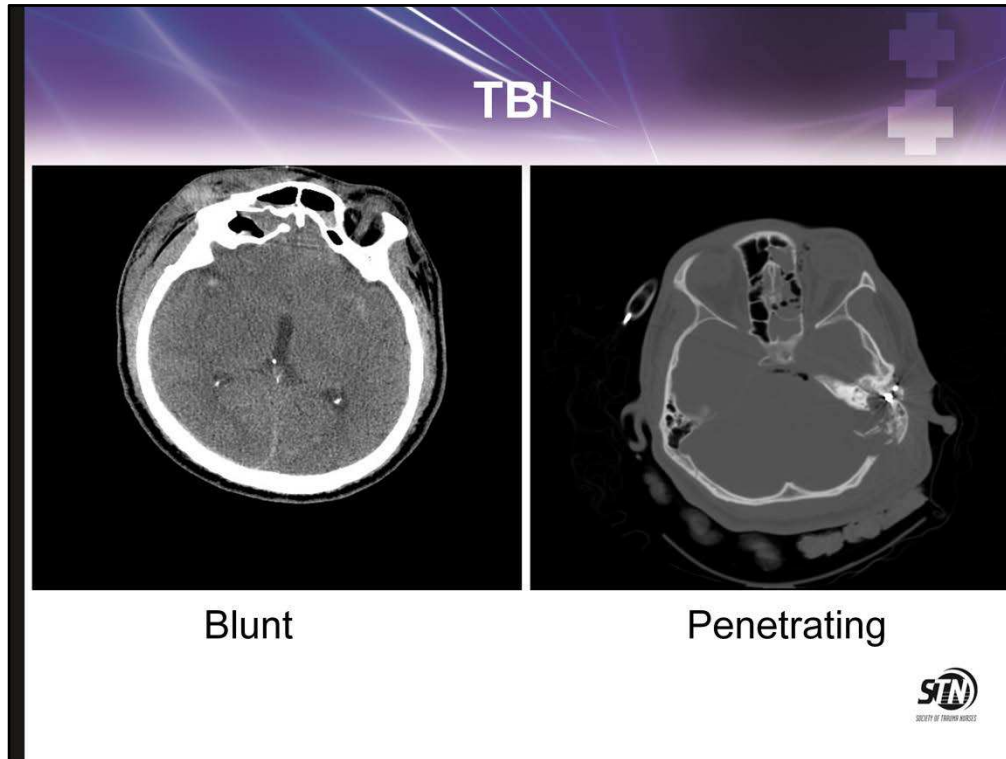
- Secondary Injury results in additional damage to the brain
- Results from:
 - Inadequate perfusion
 - Hypoxia
 - Biochemical changes
 - Other stressors associated with trauma
- Leading to cerebral edema, mass effect, ischemia, cellular death
- Image is cerebral edema. Note lack of sulci/gyri and compressed ventricles.



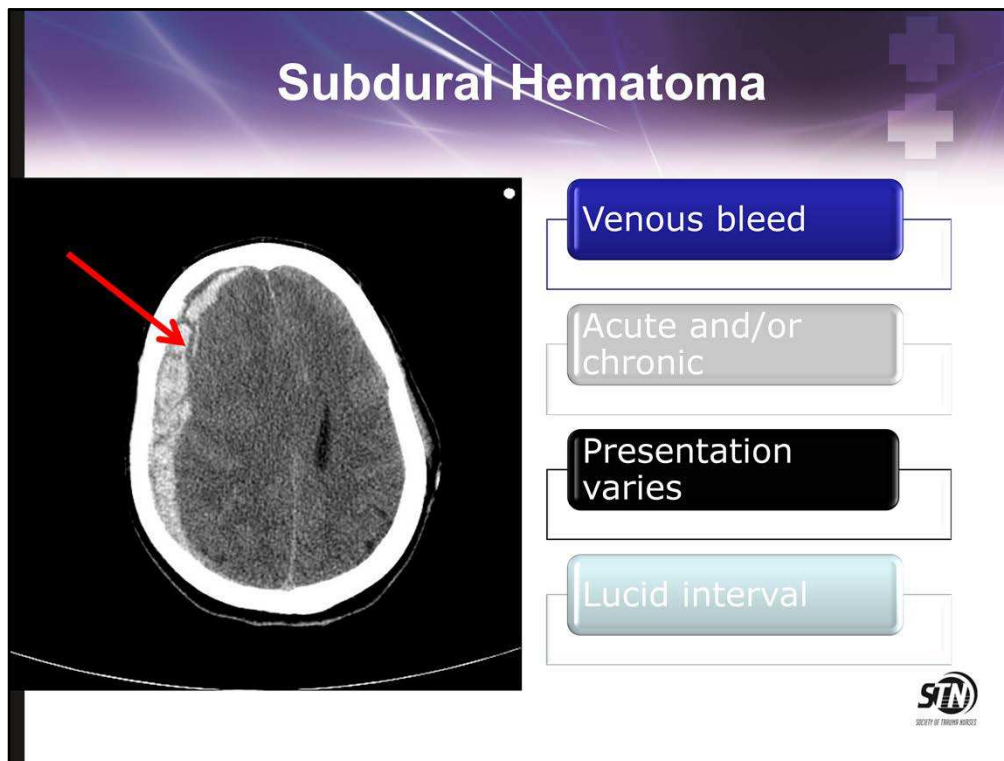
- Key to Improving Survival is PREVENTING Secondary Injury



- The effects of Traumatic Brain Injury can be subtle or catastrophic, and can range from a concussion to brain death.
 - Intracranial lesions are classified as focal and diffuse and often coexist.
 - DAI is a diffuse brain injury that is seen as petechial hemorrhages on CT
 - Focal injuries include epidural hematomas (EDH), subdural hematomas (SDH) and intraparenchymal bleeds (ICB).
 - EDH are rare and occur in 0.5% of patients with brain injuries. The bleed is arterial in origin and appears biconvex in shape on CT scan.
 - SDH are more common and occur in 30% of severe TBI patients. On CT scan a SDH appears to conform to the contours of the brain and can expand to the entire surface of the brain. SDH can be more severe than epidurals due to the underlying brain damage caused.
- (next slides shows images)



- These slides displays head CT scans of 2 patients.
- The image on the left has injuries resulting from blunt impact. There is swelling and a hematoma over the right orbit (appears on image left).
- The image on the right is a patient who sustained a gun shot wound. On this image the skull has been shattered



- **The image displays a right subdural hematoma with a midline shift.**

A subdural hematoma (SDH) is a collection of blood below the inner layer of the dura but external to the brain and arachnoid membrane (see the image above). Subdural hematoma is the most common type of traumatic intracranial mass lesion. These are venous bleeds, often due to a torn blood vessel in the bridging veins (between the brain and the dural sinus)

Generally, acute subdural hematomas are less than 72 hours old.

The subacute phase begins 3-7 days after acute injury.

Chronic subdural hematomas are 21 days (3 wk) or older.

Subdural hematomas may be mixed : acute bleeding into a chronic subdural hematoma.

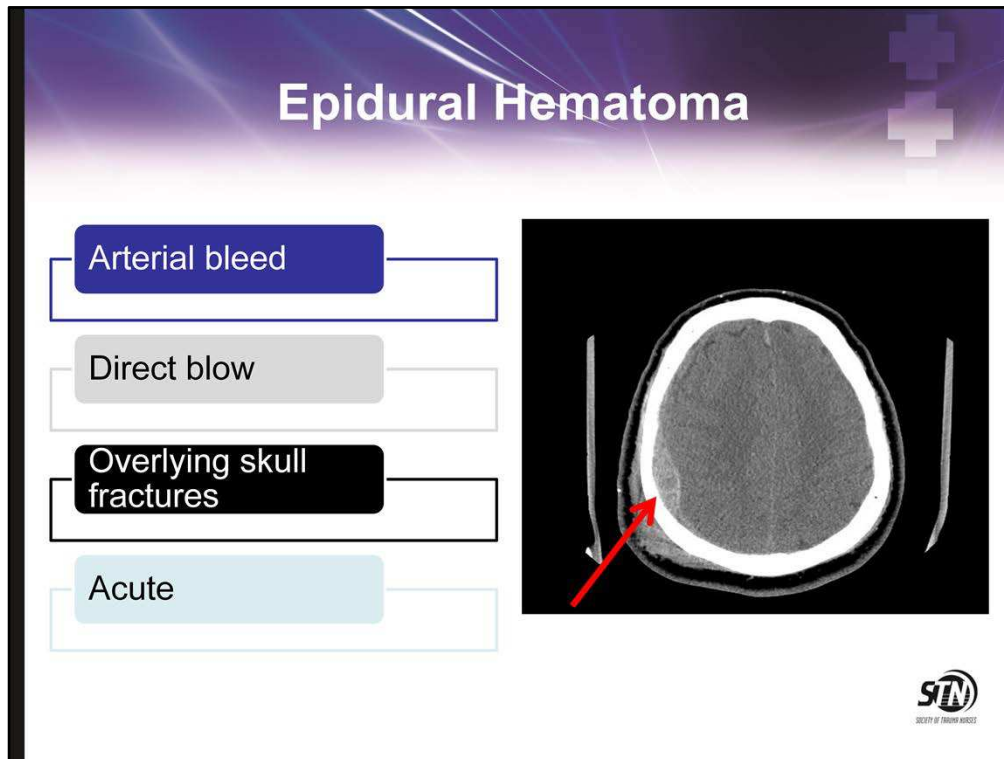
Presentation varies widely in acute subdural hematoma. Many of these patients are comatose on admission. However, approximately 50% of patients with head injuries who require emergency neurosurgery present with head injuries that are classified as moderate or mild (Glasgow Coma Scale scores 9-13 and 14-15, respectively).

Many have lucid intervals and were able to make conversation between the time of their injury and subsequent deterioration.

Significant trauma is not the only cause of subdural hematoma. Chronic subdural hematoma can occur in the elderly or alcoholics after apparently insignificant head trauma. The mechanism of injury may not even be remembered. Chronic subdural hematoma is a common treatable cause of dementia

The head trauma may also cause associated brain hematomas or contusions, subarachnoid hemorrhage, and diffuse axonal injury.

Secondary brain injuries may include edema, infarction, secondary hemorrhage, and brain herniation.



The image is of a right epidural hematoma. Note soft tissue swelling

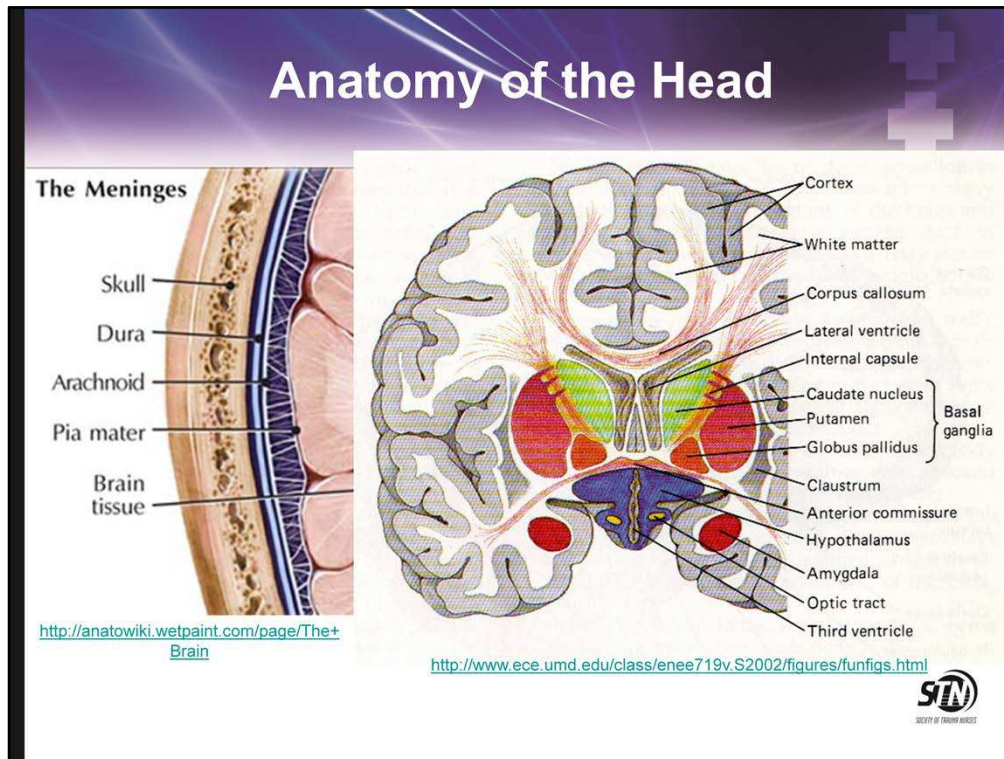
The inciting event often is a focused blow to the head, such as that produced by a hammer or baseball bat. In 85-95% of patients, this type of trauma results in an overlying fracture of the skull. Blood vessels in close proximity to the fracture are the sources of the hemorrhage in the formation of an epidural hematoma.

Because the underlying brain has usually been minimally injured, prognosis is excellent if treated aggressively. Outcome from surgical decompression and repair is related directly to patient's preoperative neurologic condition.

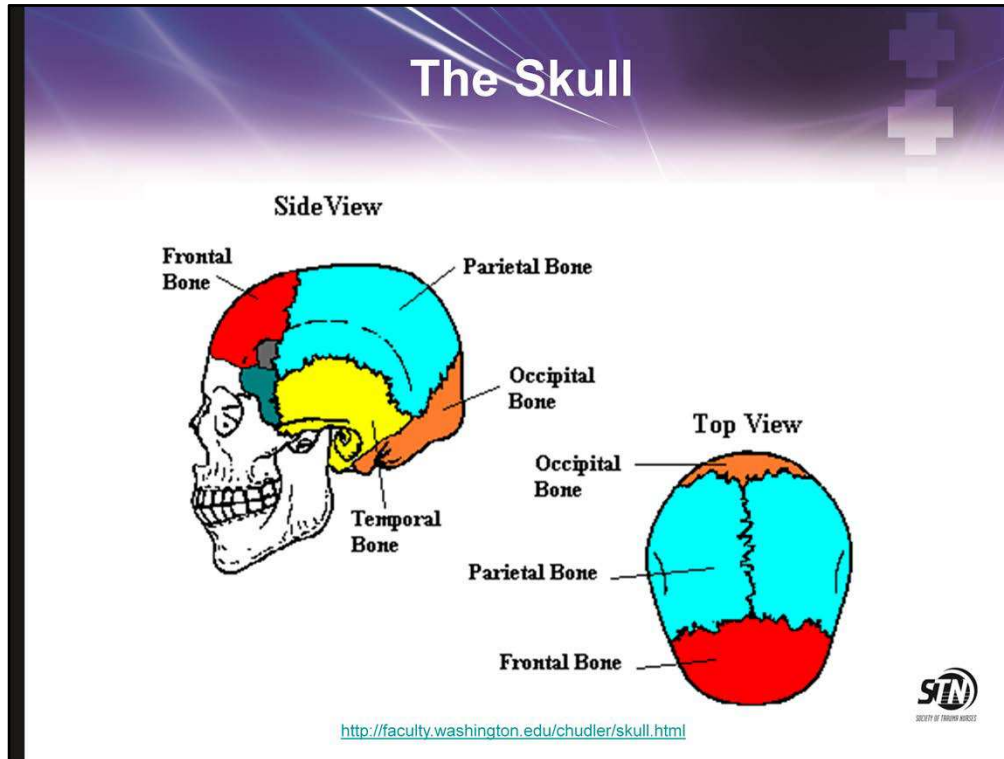
Approximately 70-80% of epidural hematomas (EDHs) are located in the temporoparietal region where skull fractures cross the path of the middle meningeal artery or its dural branches. Frontal and occipital epidural hematomas each constitute about 10%, with the latter occasionally extending above and below the tentorium.

Expanding high-volume epidural hematomas can produce a midline shift and subfalcine herniation of the brain. EDH may also cause uncal (lateral, transtentorial) herniation. Compressed cerebral tissue can impinge on the third cranial nerve, resulting in ipsilateral pupillary dilation and contralateral hemiparesis or extensor motor response.

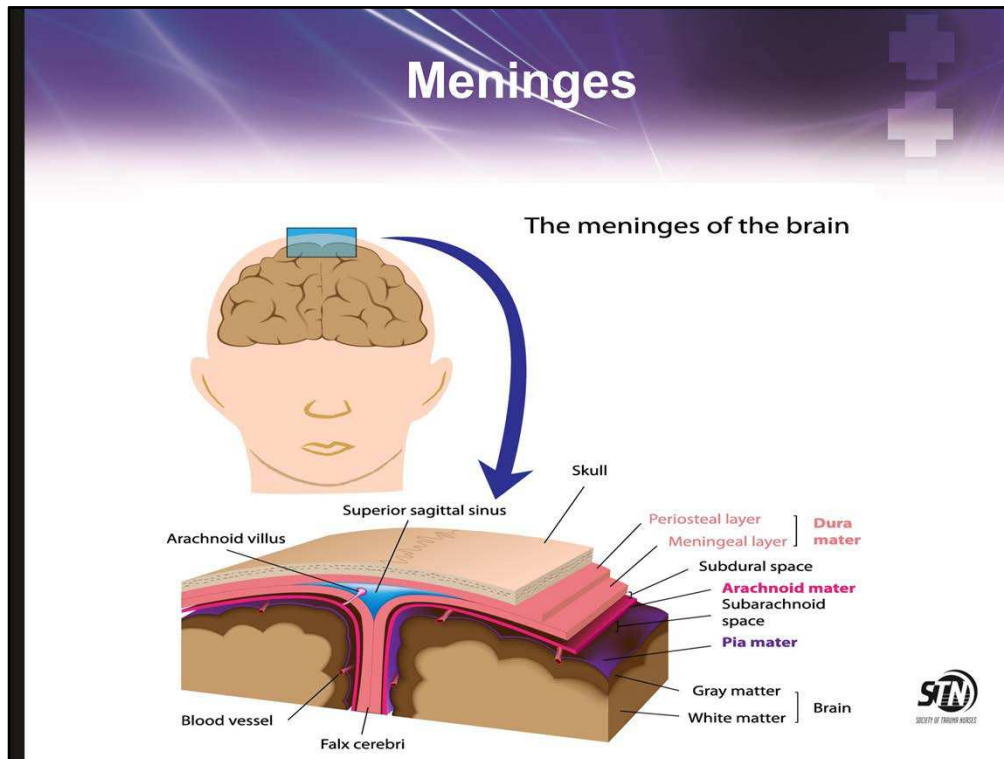
Epidural hematomas are usually stable, attaining maximum size within minutes of injury.



- The major structures of the head consists of the scalp, skull, meninges, brain, ventricular system and tentorium.
- The scalp consists of skin, connective tissue fibers, blood vessels and nerves. The scalp has a rich blood supply therefore lacerations to the scalp can be bleed profusely and lead to severe blood loss in trauma.
- The brain is composed of gray and white matter.
- The gray matter consist of neuron cell bodies and dendrites
- The white mater consists primarily of myelinated axons that aid in the transfer of nerve impulses.
- The brain is divided into three main parts: the cerebrum, brain stem and cerebellum.
- The outer layer of the cerebrum is known as the cerebral cortex. The cerebral cortex is arranged in folds known as gyri and depressions known as fissures or sulci.
- The cerebrum is divided into to hemispheres and connected in the midline by the corpus callosum.



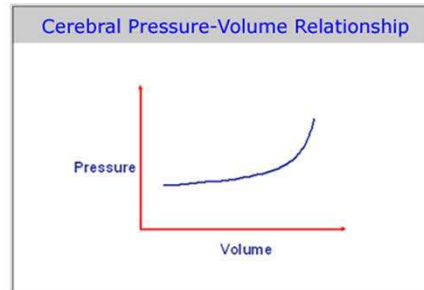
- The skull of the head is known as the cranium.
- There are 8 major bones of the skull
- The bones are frontal (1), ethmoid (1), parietal (2), sphenoid (1) temporal (2) and occipital (1).
- The cranial bones are rigidly jointed together by articulations known as sutures and they form the protective housing for the brain.
- The cranial sutures between bones initially serve as expansion joints but eventually fuse, which halts expandability in adults.



- The meninges are three layers of protective tissue called the dura mater, arachnoid mater and pia mater
- The outermost layer, the dura mater, is firmly adherent to the inner skull and has fixed attachments at the cranial sutures.
- Underneath the dura mater is a thinner connective tissue layer called the *arachnoid mater*.
- The arachnoid mater is loosely adhered to the pia mater, which makes possible the potential subarachnoid space.
- The pia mater is closely associated with the grey matter of the brain and is the innermost layer. Between the arachnoid and the pia is the subarachnoid space, where CSF circulates.
- In the average adult, there is 150 mL of CSF surrounding the brain and spinal cord.
- Approximately 500 mL of CSF is produced in the choroid plexus of the lateral ventricles each day.
- The brain consumes 20% of the body's total oxygen requirement and 15% of total cardiac output.
- The brain is exquisitely sensitive to ischemia and low-oxygen states.

Pressure-Volume Relationships

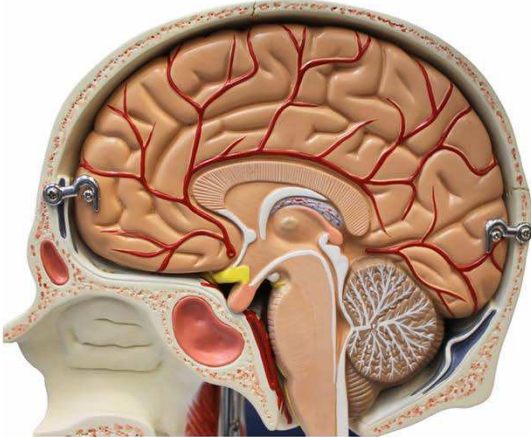
- MONROE- KELLIE DOCTRINE
- The cranium is a **non expansible** vault



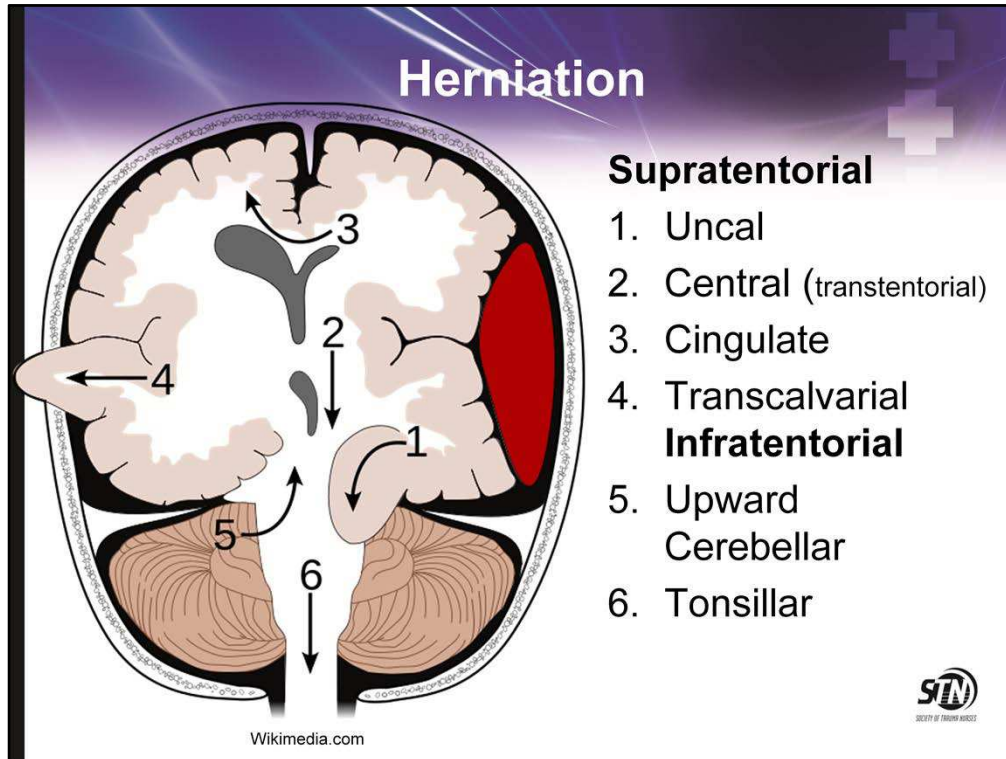
- The Monroe-Kellie doctrine describes pressure-volume relationships within the intracranial cavity
- The cranium as a non expansible vault.
- An increasing intracranial pressure (ICP) warns us that contents under pressure will try to relieve that pressure by finding an exit in the area of least resistance.
- Unlike a volcano the cranium does not erupt, therefore a build up of pressure will result in brain herniation if not treated.

Monro-Kellie Doctrine

- The total volume of intracranial contents must remain constant
 - Brain - 80%
 - Blood - 10%
 - CSF - 10%
- An increase in one causes a decrease in one or both of the remaining two



- Simple concept vital to understanding intracerebral dynamics
- This concept holds that the skull is a rigid compartment that contains 3 components: brain tissue, arterial and venous blood, and cerebrospinal fluid (CSF).
- Under normal circumstances, these components are balanced in a state of dynamic equilibrium.
- If an increase occurs in the relative volume of one component, such as brain tissue, the volume of one or more of the other components must decrease or an elevation in ICP will result
- In a brain with disease or injury, small increases in volume may result in large increases in ICP.

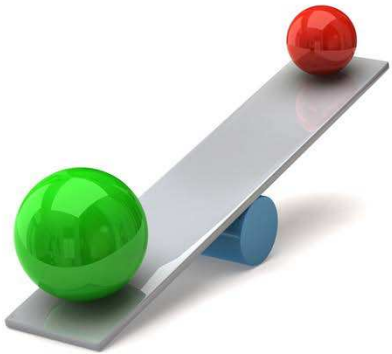


The abnormal protrusion of brain tissue through an opening when there is increased intracranial pressure.

This is a disastrous complication of increased intracranial pressure and is often fatal.

Cerebral Blood Flow (CBF)

- **AUTOREGULATION**
The intrinsic ability of the cerebral blood vessels to dilate or constrict in response to changes in the brain environment
- Enables cerebral blood vessels to maintain cerebral blood flow in presence of wide fluctuation in mean arterial pressure



- Cerebral autoregulation refers to the properties of the brain vascular bed to maintain cerebral perfusion despite changes in blood pressure
- To maintain stable cerebral blood flow (*CBF*), the brain's vasculature must respond to changes in arterial blood pressure (*BP*) or intracranial pressure
- Normally, autoregulation maintains a constant blood flow between Mean Arterial Pressure (MAP) 50 mmHg and 150 mmHg.

Autoregulation - Impaired



- Cerebral Blood Flow varies in changes in Cerebral Perfusion Pressure (CPP) and diameter of cerebral vascular bed
- Autoregulation fails if CPP is < 50 or > 150 mmHg



- The normal cerebral blood flow is 50ml/min/100g.
- There are two essential facts to understand about cerebral blood flow.
- First, in normal circumstances when the flow falls physiological electrical function of the cell begins to fail.
- Second, an increase or decrease in CBF will cause an increase or decrease in cerebral arterial blood volume because of arterial dilatation or constriction.
- Therefore in a brain that is decompensated as a result of major intracranial pathology, increases or decreases in CBF will in turn lead to a significant rise or fall in ICP
- **CPP < 50 mmHg causes hypoperfusion and cerebral ischemia**
- **CPP > 150 mmHg causes hyperperfusion and potentially cerebral edema**



- Secondary injury to the brain is preventable and treatable.
- The goal of care for a TBI patient is to prevent secondary injury specifically hypotension and hypoxia.

TBI Recognition and Management

- Prehospital Care
- ABCDE Management
- Mitigate Secondary Injury



A photograph showing two paramedics in high-visibility blue and yellow uniforms attending to a patient lying on a green stretcher. The patient is secured with yellow straps. One paramedic is kneeling and adjusting the patient's head, which is supported by a red and orange cervical collar. The other paramedic is kneeling and attending to the patient's torso. The scene is outdoors, next to a dark-colored car.



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- Prehospital management of TBI patients can make a difference in patient outcomes.
- Early support of airway, oxygenation and prevention of aspiration can decrease the possibility of secondary injury.

Pre Hospital Care

- Timing and transport
- Oxygenation
- Intubation
- ETCO₂
- Blood Pressure
- Transport decisions



- All regions/states have prehospital guidelines to direct EMS personnel in the care of patients.

Prehospital management of a TBI patient is well defined in Brain Trauma Foundation Guidelines for the pre hospital personnel. Even though there are no Level 1 recommendations in these guidelines the Level III evidence is well researched and supported.

- Field stabilization (less than 10 minutes) and rapid transport to an appropriate facility is critical in a patient with severe TBI, however it is the quality of the resuscitation in the field that impacts the outcome of the patient more than speed.

- Oxygenation and blood pressure management of a TBI patient is crucial in preventing secondary injury.

- Supplemental oxygen should be provided to maintain a SaO₂ greater than 90.

- Pre hospital guidelines recommend intubation if persistent hypoxemia is not corrected by supplemental O₂, inability to maintain an adequate airway and a GCS less than or equal to 8.

- Recent studies have shed a controversial light on in-field intubation and outcomes with some studies showing worse outcomes for those patients intubated in the field. One reason for this may be post intubation hyperventilation.

- The use of capnography is to be used to maintain end tidal CO₂ between 35 and 40 mm Hg.

- Blood pressure greater than 90 systolic should be maintained with isotonic fluids. Hypertonic fluids may be given for patients with GCS equal to or less than 8 if in their local protocols.

- Transport decision made using trauma destination protocols. Current National Guidelines for Field Triage of Injured Patients published in 2011 by the CDC mandate that any patient with a GCS of 13 or less to the highest level trauma center per regional protocol.

Glasgow Coma Scale (GCS)

GCS Score = E + M + V



The photograph shows a person lying on their back on a paved surface next to a dark-colored car. The person is wearing a black jacket and a light blue shirt. There is a visible wound on the person's forehead. The car's door is open, and the person's arm is resting on the door sill. The background is a plain, light-colored wall.

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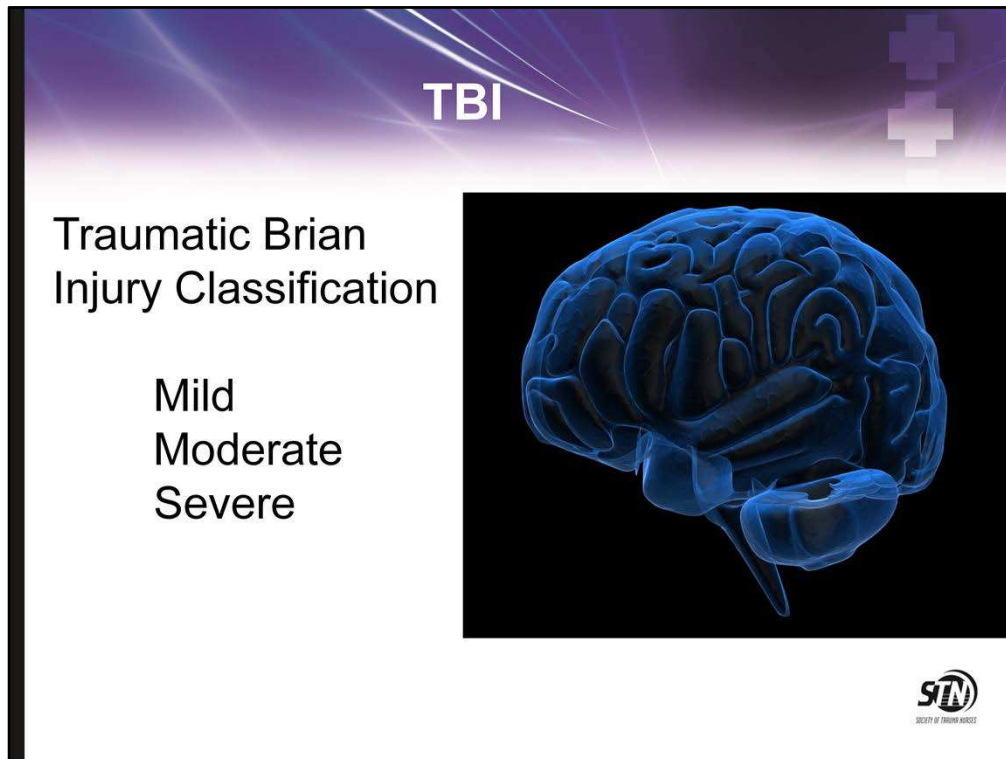
- The GCS is a 15-point scale that is based on a patient's ability to interact with the environment and is divided into three categories: eye opening, verbal function, and motor function.
 - No response scores a 1 in each category (minimum total score of 3).
 - The maximum score is 15
- Should be assessed prior to any medication being given.**

Glasgow Coma Scale (GCS)

Best Verbal Response (5) 1.No verbal response 2.Incomprehensible sounds 3.Inappropriate words 4.Confused 5.Orientated	Best Eye Response (4) 1.No eye opening 2.Eye opening to pain 3.Eye opening to verbal command 4.Eyes open spontaneously
Best Motor Response (6) 1.No motor response 2.Extension to pain 3.Flexion to pain 4.Withdrawal from pain 5.Localizing pain 6.Obeys commands	



- The Glasgow Coma Scale (GCS), a clinical tool designed to assess coma and impaired consciousness, is one of the most commonly used severity scoring systems.
- Persons with GCS scores of 3 to 8 are classified with a severe TBI, those with scores of 9 to 12 are classified with a moderate TBI, and those with scores of 13 to 15 are classified with a mild TBI.



Classification of TBI

- Mild
 - GCS Score 14-15
- Moderate
 - GCS Score 9-13
- Severe
 - GCS Score 3-8

A 3D rendering of a white eggshell that has been cracked open. The crack runs vertically down the center, with the top and bottom halves slightly separated, revealing a hollow interior. The egg is positioned on the right side of the slide, next to the classification list.

- Traumatic Brain Injury is classified as mild, moderate or severe.
- TBI's vary greatly in terms of severity, from mild cases that involve a brief change in mental status, such as confusion, to severe cases that may involve an extended period of unconsciousness.
- The symptoms and management of each classification depend on the extent of damage to the brain.
- Some symptoms can appear immediately after the event and others can appear days or weeks later.
- TBI can result in changes to a person's physical functioning, cognitive or thinking function and behavioral events, and often they are all interrelated.

Mild TBI

- Most Prevalent TBI
- Annual cost of Mild TBI is \$17 billion
- In most cases the patient makes a full recovery
- 15% of patients diagnosed with Mild TBI experience disabling, long-term problems



- Commonly called concussion.
- As many as 75% of all brain-injured people sustain mild TBI
- The cost to the nation each year for Mild TBI is approximately \$17 billion.
- Mild TBI results from direct trauma to the head or from acceleration/deceleration stress to the brain

Signs and Symptoms of Mild TBI



jan kranendonk / Shutterstock.com

- Confusion/ Disorientation
- Loss of consciousness up to 30minutes
- Amnesia near the time of injury
- GCS 14-15



- Mild TBI is also called concussion, minor head injury, minor brain injury, minor head trauma or minor TBI.
- It is one of the most common neurologic disorders.
- Symptoms of Mild TBI include headache, dizziness, attention difficulties, amnesia, orientation problems and sleep disturbances.

Management of Mild TBI



- History
- Exam
- Imaging (CT Scan of Head)
- Admit or Discharge?
- Discharge Education

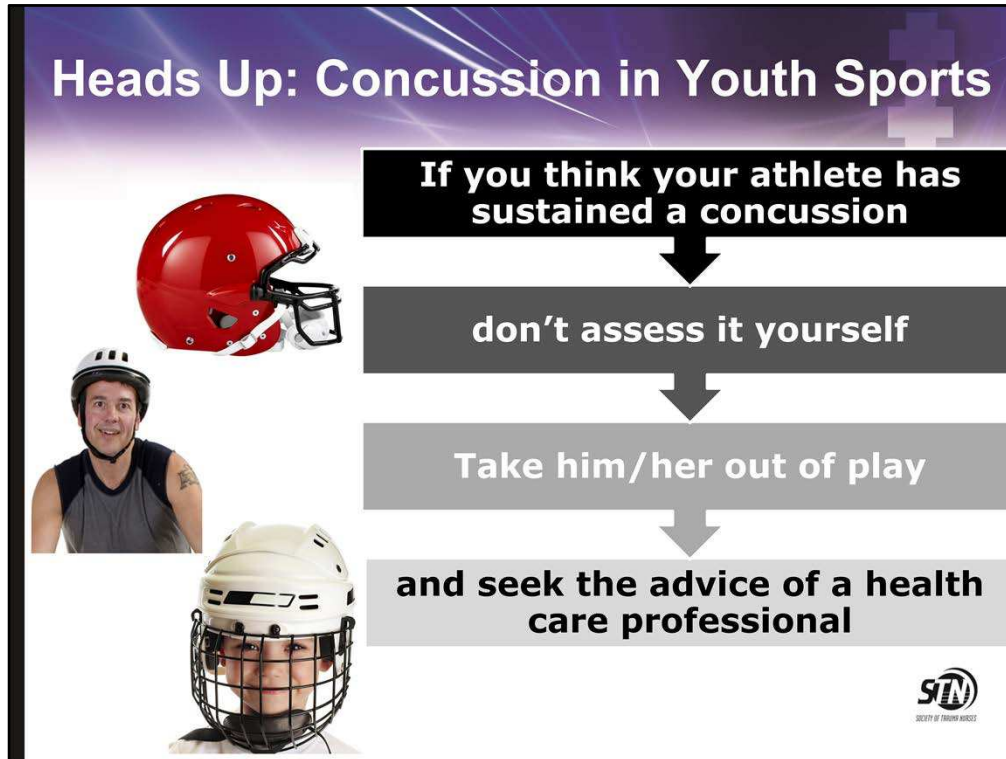


- It is very important to obtain a good history and perform a thorough exam in the patient who has sustained a mild brain injury
- A physical exam , cognitive and behavioral exam should be performed and findings documented.
- A thorough neurological exam is very important including a GCS
- One of the most subtle impairments in mild TBI is cognitive impairments.
- Ranchos Los Amigos or Mini Mental Test can be performed to detect any cognitive impairments
- A head CT scan is indicated for patients with a loss of consciousness or post traumatic amnesia in the presence of symptoms
- A head CT should be considered for patients with no loss of consciousness or post traumatic amnesia in the presence of symptoms
- In the absence of availability to perform a head CT scan patients or if the patient has a positive CT scan then the patient should be admitted for overnight (greater than 12 hours) for observation and hourly neurological assessments.
- All mild TBI patients and their designated caregiver should receive explicit education and discharge instructions regarding post concussive symptoms, follow up and what to expect and when to seek emergency care. Discharge instructions should emphasize rest and sleep

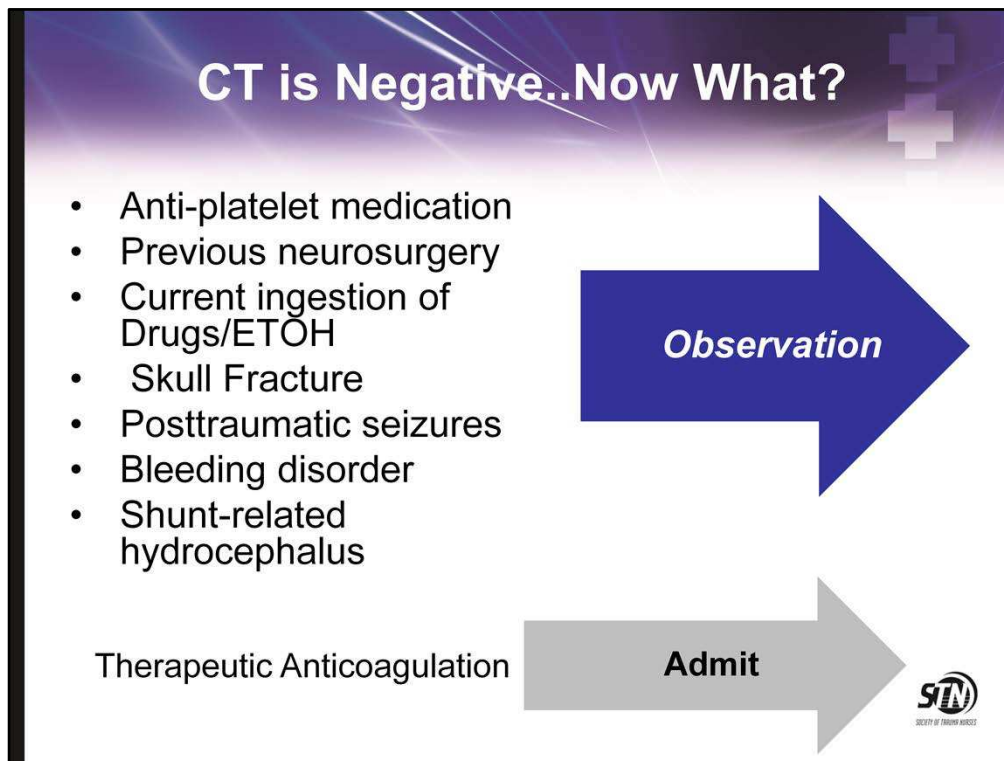
Mini Mental Exam	
The Mini-Mental Status Examination (MMSE)	
	Points
Orientation	
Name: season/date/day/month/year	5 (1 for each name)
Name: hospital/floor/town/state/country	5 (1 for each name)
Registration	
Identify three objects by name and ask patient to repeat	3 (1 for each object)
Attention and calculation	
Serial 7s: subtract from 100 (e.g., 93-86-79-72-65)	5 (1 for each subtraction)
Recall	
Recall the three objects presented earlier	3 (1 for each object)
Language	
Name pencil and watch	2 (1 for each object)
Repeat "No ifs, ands, or buts"	1
Follow a 3-step command (e.g., "Take this paper, fold it in half, and place it on the table")	3 (1 for each command)
Write "close your eyes" and ask patient to obey written command	1
Ask patient to write a sentence	1
Ask patient to copy a design (e.g., intersecting pentagons)	1
TOTAL	30



The **mini-mental state examination (MMSE)** or **Folstein test** is a brief 30-point questionnaire test that is used to screen for cognitive impairment. Any score greater than or equal to 25 points (out of 30) indicates a normal cognition. Below this, scores can indicate severe (≤ 9 points), moderate (10-20 points) or mild (21-24 points) cognitive impairment.



- Concussion in sports is widely discussed these days.
- The CDC has developed a major prevention and treatment program that includes education for coaches and parents, athletes.



•If the patient has a negative CT and no other injuries that require admission and has someone who will be with them to monitor behaviors the discharge is appropriate with really good discharge instructions.

•**Are your ED discharge instructions sufficient and easy to understand?**

•If any of the risk factors are present the patient is admitted for observation (q 2 hour neurological checks) and repeat Head CT Scan in 6 Hours. **There is controversy about a repeat CT. The studies show that if there is no change in GCS/behaviors, then there is no need for repeat CT. What are your protocols or practices?**

•

•Close consideration is taken with patients who are taking therapeutic anticoagulation (e.g. warfarin). These patients are at risk of bleeding and should be admitted for neurological observation.

Moderate TBI

- 8-10% of all TBI's
- Confused/somnolent
- Still able to follow commands
- Loss of consciousness lasting a few minutes to a few hours

Management

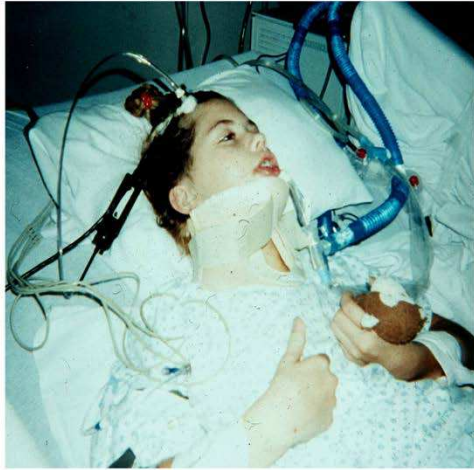
- CT Scan of Head
- Admit for neurologic checks
- Repeat CT scan
- Labs



- Moderate TBI's account for 8-10% of all TBI's.
- Patients may present with a GCS 9-13
- They may still be able to follow commands.
- The goal in managing a moderate TBI is to prevent secondary injury!
- Management of a moderate TBI should follow Airway, Breathing, Circulation, Disability, Exposure/Environment (ABCDE's) approach to the assessment.
- History should include mechanism of injury , medical history, medications and possible use of drugs or alcohol
- Labs should include Basic Metabolic Panel, Complete Blood Count, PT/PTT/INR.
- Management includes
 - Admit for hourly neuro checks
 - Repeat CT scan if exam changes
 - Repeat CT scan 6-12 hours
 - Obtain cognitive evaluation prior to discharge
- Discharge instructions and education are the same as for mild TBI.

Severe TBI

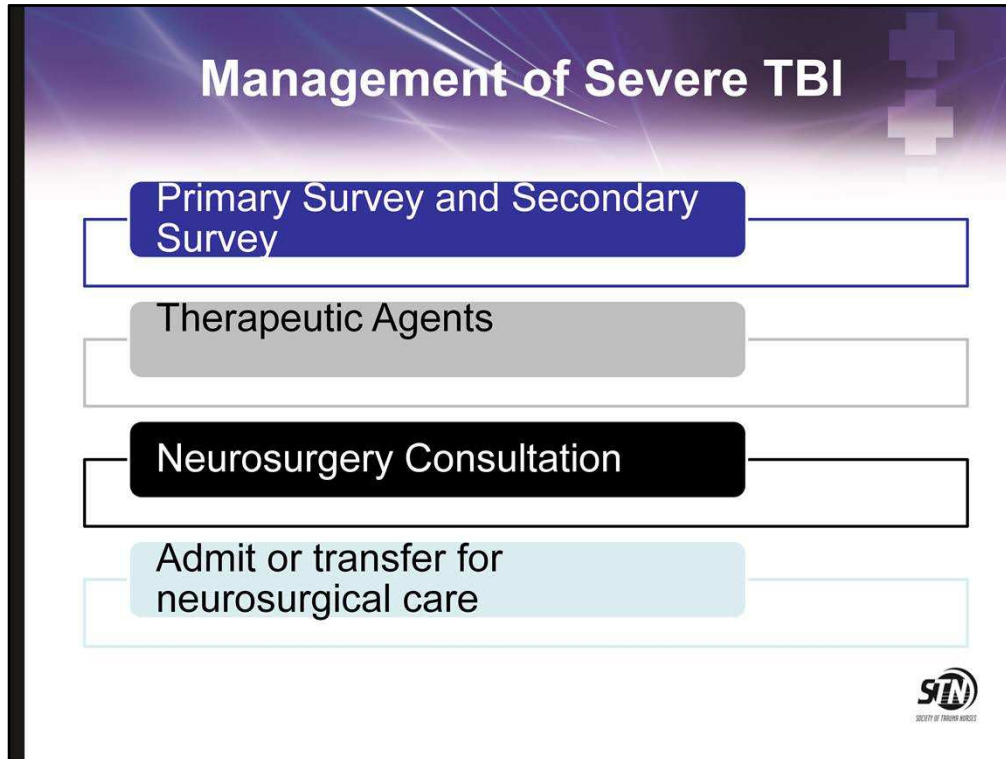
- 10% of all TBI's
- GCS 3-8
- Prolonged loss of consciousness
- Unable to follow simple commands



- Patients with a severe TBI account for 10% of all TBI's
- These patients present with a GCS of 3-8
- The primary goal for severe TBI patients is to prevent secondary injury.
- Other critical goals for the patient include:
 - Identify treatable mass lesions
 - and Identify and treat any life- threatening injuries.



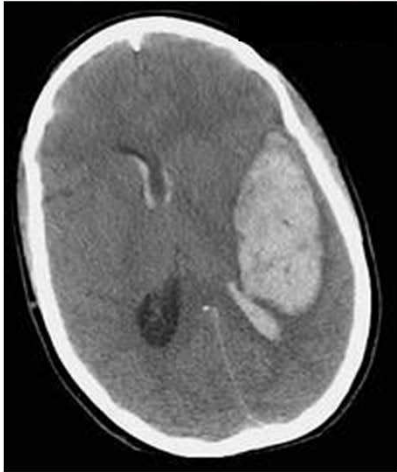
- Prompt diagnosis and treatment of patients with a severe TBI is extremely important
- Initial management can greatly impact the patient's morbidity and mortality.



- Advanced Trauma Life Support guidelines should be followed in the assessment and initial management of the patient.
- Primary and Secondary Surveys should be performed (further explained)
- History should include mechanism of injury, medical history, medications and possible use of drugs or alcohol
- Neurosurgery consultation does not need to be considered for all patients however if definitive neurosurgical care is required and can not be provided at the receiving hospital, patients should be transferred as soon as possible to a center that provides neurosurgical care. .

Initial Management of Severe TBI Patient

- Initial assessment/management
- Primary survey and resuscitation
ABCDE's
- Secondary survey
Head to toe assessment
AMPLE



- Management of a severe TBI should follow Airway, Breathing, Circulation, Disability, Exposure/Environment (ABCDE's) approach to the assessment.
- The goal of the primary survey is to identify any life threatening injuries and managed.
- The secondary survey is more detailed with a head to toe assessment performed with patient history.
- The secondary survey includes reassessment of the patients vital signs and a thorough examination of the patient
- Adjuncts to the secondary survey include X Ray, CT scan, ultrasound and other diagnostic procedures.
- Prehospital providers are critical in providing valuable information when patients are unable to provide a history
- The mnemonic AMPLE can be used to obtain necessary information that helps provide a better understanding of the patients current circumstances.



- The airway is always assessed first for obstruction. Look for foreign objects and listen for air movement / stridor.
- Cervical spine immobilization should always be maintained during assessment and management of the airway

A- AIRWAY

Goals

- Airway protection and C-Spine immobilization.
- Endotracheal Intubate for GCS ≤ 8
 - Rapid Sequence Intubation (RSI)
 - Maintain head and neck in straight alignment



- With all airway assessment the C-Spine must be addressed and C-Spine immobilization and stabilization performed.
- In Patients with severe TBIs indications for intubation include a GCS less than or equal to 8. This low GCS score indicates the patient is unable to protect their own airway and respiratory arrest can occur
- Rapid Sequence Intubation is an important method used for airway management.



RSI includes 5 P's

Prepare equipment/patient (ambu bag, (BVM), suction, pulse oximeter, EKG leads/monitor, endotracheal handle and blade, ET tube, stylet/bougie, syringe and ETCO2 detector)

Preoxygenate – with 100% O₂ until a SaO₂ greater than 90%

Premedicate - Lidocaine

- suggested use in increased ICP patients
- lowers ICP and suppresses the cough reflex

Etomidate:

- causes little CV depression and is cerebroprotective (decreases ICP, maintains MAP and increases cerebral perfusion pressure)

Paralysis – short acting agents are recommended in order to follow a neurological examination

Post Intubation – confirmation of the ET tube placement is obtained by examination and X-ray

Initial Management - ABCDE

B- Breathing

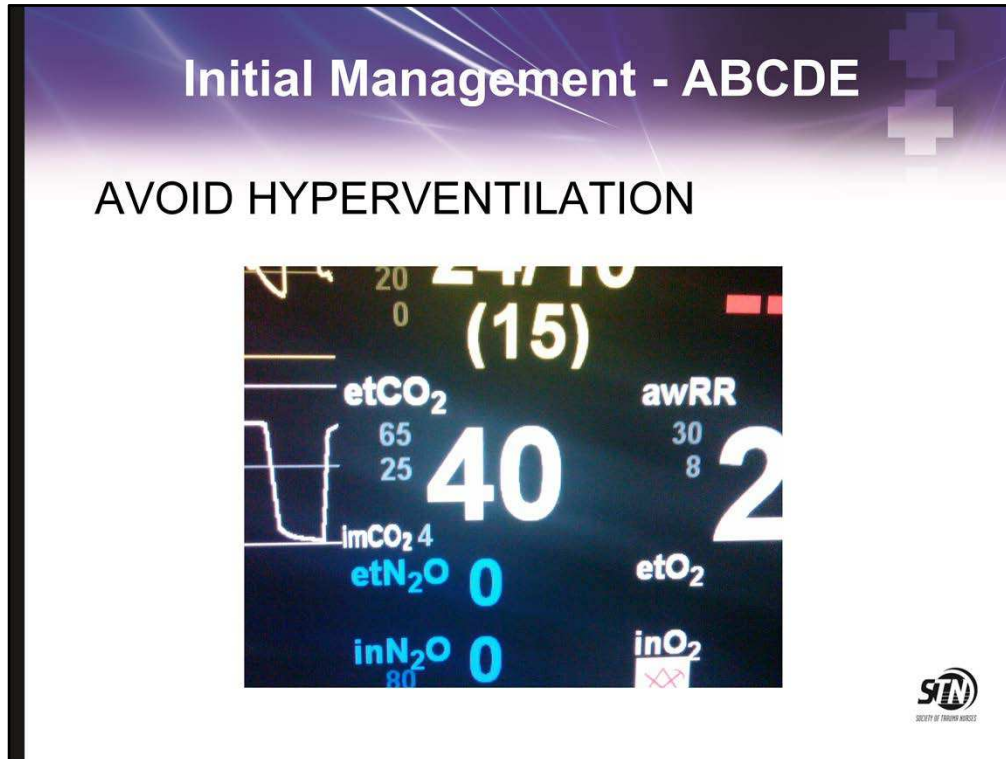
Goals

- Pa O₂ >60mmHg,
- O₂ sat > 98%
- ETCO₂ ~ 35mmHg



STN
SOCIETY OF TRAUMA NURSES

- Breathing assessment includes look, listen and feel approach (inspect, auscultate and palpate)
- Goal for severe TBI patients is to maintain an O₂ saturation greater than 90% and a paO₂ greater than 60mmHg to prevent hypoxia
- The patient should receive 100% O₂ until the Arterial Blood Gas measurements are obtained and oxygen delivery can then be adjusted based on the results.
- Pulse oximetry should be continuously monitored for assessment of patient oxygenation. The pulse oximeter measures oxygen saturation of hemoglobin. The goal is to maintain above 98%
- ETCO₂ gives information regarding adequacy of circulatory flow
- ETCO₂ measures CO₂ at the end of an **exhaled breath**
- ETCO₂ monitoring at all times to detect changes in patient condition and identify need for alterations in ventilatory management.
- Avoid manual ventilation with BVM – if you can. If you must use BVM during transport, continuous monitoring of ETCO₂ is critical.



- Hyperventilation causes vasoconstriction by reducing PaCO₂ therefore causing regional ischemia.
- This compromises cerebral perfusion during a time when cerebral blood flow (CBF) is already reduced.
- Vasoconstriction and impaired cerebral perfusion can occur if PaCO₂ falls below 30mmHg
- It is preferable to keep the PaCO₂ at 35mmHg or above.

Use hyperventilation ***only*** when:
Acute Herniation/Acute Neurologic
Deterioration
Other Therapies Failed

Initial Management -ABCDE

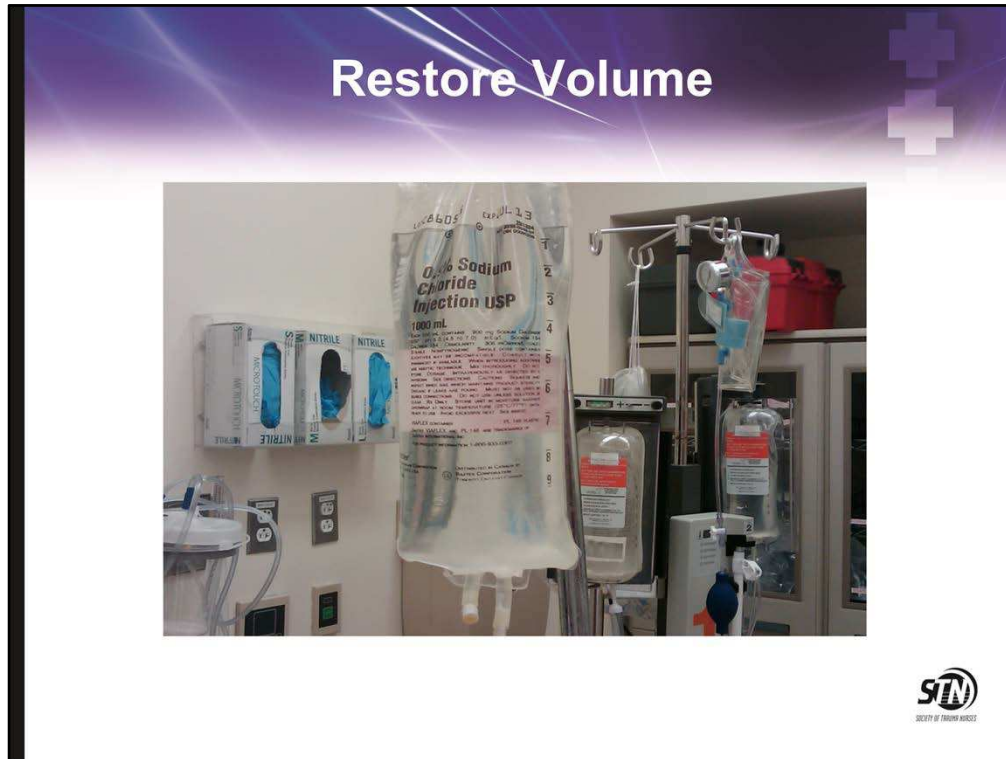
C- Circulation

Goals

- Maintain SPB >90mmHg
- Establish euvoemia
- Stop Hemorrhage
- Restore volume
- Maintain Hct 30%
- CPP > 60mmHg



- Patients who have sustained a TBI often have multiple traumatic injuries
 - Additional injuries can complicate their treatment and prognosis
 - Frequent assessment of vital signs and continuous monitoring (cardiac monitoring , non invasive blood pressure and pulse oximetry) are important to detect changes in a patients condition.
 - In patients with additional injuries, the location of the injuries and hemorrhage should be quickly identified and the hemorrhage stopped.
 - Hypotension is usually associated with severe blood loss and therefore should be treated quickly to maintain a SBP greater than 90mmHg and therefore prevent secondary injury.
 - Goal is to locate the source of hemorrhage and stop the hemorrhage.
 - Hypovolemic shock is not caused by intracranial hemorrhage.
 - Remember – the goal for TBI patients is to prevent Hypoxia and HYPOTENSION!
 - The literature states that one episode of SBP less than 90mmHg can worsen the patients outcome. Hypotension causes secondary brain injury therefore prevention of hypotension is crucial for a TBI patient.
-
- **One single episode of hypotension can double mortality in TBI patients.**
 - **Maintaining a blood pressure affects the patients Mean Arterial Pressure (MAP)**
 - **MAP is a contributor to Cerebral Perfusion Pressure (CPP) later discussed.**
 - **In order to maintain CPP and reduce cerebral ischemia the systolic blood pressure must be maintained above 90mmHg**
 - **In patient who are not bleeding, and are euvoemic , dopamine and norepinephrine can be considered.**



- Fluid resuscitation in a patient who is in hypovolemic shock is crucial in stabilizing and replacing blood loss.
- If the patient is bleeding then blood should be given ASAP.
- Goal is to maintain a hematocrit greater than 30% to adequately perfuse the patient.
- In a patient with an isolated severe TBI injury, the goal is to prevent hypotension
- Hypotonic fluids should not be used as they can cross the blood brain barrier, increase brain water and worsen cerebral edema.
- Normal saline or Lactate Ringers remains to be the first line of fluid choice for resuscitation as per Advanced Trauma Life Support guidelines however current evidence recommends the immediate use of blood products for hemorrhagic shock has shown to improve patient's outcomes.
- Care should be taken not to overload the patient with fluid as this can cause edema of the brain and further worsen the patient's condition

Initial Management - ABCDE

D - Disability

- Neurologic Assessment
- **GLASGOW COMA SCALE**
- Pupils



- Glasgow Coma Scale is a well recognized, internationally used tool for indicating the severity of a head injury
- Useful tool to establish initial head injury severity and evaluate improvement or deterioration over time
- GCS score of 3-5 has at least a 70% positive predictive value for poor outcome
- **A GCS should be performed once the patient's cardiopulmonary status is corrected and PRIOR to sedating and paralyzing the patient . This is important in determining further treatment.**

Initial Management - ABCDE

- Pupil reactivity to light
positive reaction > 1mm constriction
- Pupil asymmetry
significant asymmetry > 1mm difference
- Fixed/Dilated Pupils
pupils that are > 4mm and react < 1mm
- Measurement difference of 1 mm or more is defined as asymmetry



- Pupillary size and reaction to light should be assessed.
- Pupil size and response is an indicator of the severity of brain injury
- Asymmetrical pupil can indicate a mass bleed/lesion and fixed and dilated pupils is a sign of cerebral herniation

ABCDE Considerations for TBI

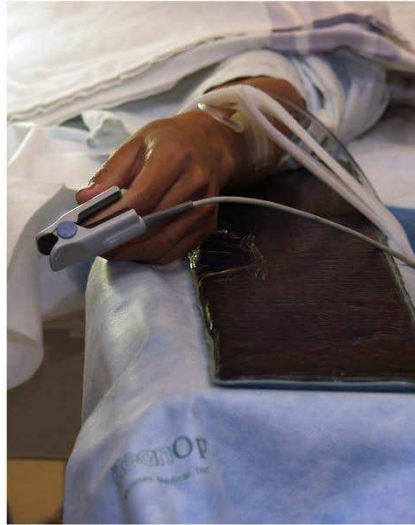
E – Exposure/Environment



- GOAL is to maintain normothermia
- Management of temperature is very important.
- Every degree to temperature elevation increased cerebral metabolic demand by approximately 7%
- Core temperature monitoring can be achieved by either an esophageal sensor or a bladder temperature sensor.

Initial Management – Secondary Survey

- Head to Toe Assessment
- Labs
- Arterial Line
- Pulse Oximetry
- Capnography
- Core temperature



- Labs should include
 - Basic Metabolic Panel – monitoring of sodium is extremely important especially if hypertonic saline is used (discussed in later slide)
 - Complete Blood Count – a HCT above 30% should be maintained to ensure adequate perfusion
 - PT/PTT/INR- prevention of coagulopathy is very important to the patients outcome. Coagulopathy can lead to acidosis and death in a trauma patient. Any coagulopathy would need to be corrected if neurosurgery is indicated
 - ABG (goal is to prevent hypoxia). Maintain a PaO₂ above 60% is crucial to prevent secondary injury.
 - Serum Osmolality (if mannitol is administered then serum osmolality should be less than 300, you do not want to dry out the patient!)
 - Toxicology – should be considered to rule out any other causes for a change in mental status.

Initial Management – Secondary Survey

- Clinical assessment and reassessment
 - Battles Sign
 - Raccoon Eyes



- Initial neurological assessment provides a baseline for further reassessment. In the first few hours of resuscitation frequent reassessment includes q 15 min neurological examination including GCS and pupil exam.
- Skull fractures are classified as linear or depressed fractures. Linear skull fractures are the most common fracture.
- Linear fractures can be vault or skull base fractures
- Basilar skull fractures are linear at the base of the skull
- Clinical signs of basilar skull fractures include Raccoon eyes and Battles sign.
- Raccoon eyes is characterized by bilateral peri-orbital ecchymosis
- Battles sign is characterized by ecchymosis behind the ear
- Vault and depressed fractures can be open or closed.
- Open fractures indicate an open wound and can result in complications such as infection.
- Open and depressed skull fractures require a neurosurgical consult.

Clinical Manifestations of Secondary Injury

- Decreasing consciousness (LOC)
- Alterations in respiratory rate
- Agitation or restlessness
- Headache
- Focal signs (pupillary changes/hemiparesis)
- Nausea/vomiting
- Hyperthermia
- Seizures
- Changes in motor responses or posturing



Every sign and symptom for a TBI patient must be carefully evaluated as this can indicate worsening intracranial pressure.

In a patient who is non intubated, decreasing level of consciousness, agitation, restlessness can indicated signs of secondary injury.

In a patient who is ventilated , monitoring of pupillary changes, seizures and changes to motor response can indict increasing cerebral pressure and worsening secondary injury

Clinical Manifestations of Secondary Injury

- Signs and symptoms of herniation
 - Bradycardia
 - Tachypnea
 - Hypertension
 - Dilated Pupil



An ECG tracing on a blue grid background. The tracing shows a regular rhythm with a slow heart rate, consistent with bradycardia. The QRS complexes are narrow, and the T waves are upright. The STN logo is visible in the bottom right corner of the slide.

- Brain herniation occurs in a TBI patient due mass effect resulting in the brain shifting through or across regions to the other side.
- Symptoms include loss of consciousness, coma, pupillary changes
- Signs include hypertension and bradycardia
- Herniation is a medical emergency and unless treated very quickly by reducing swelling or removing the blood the prognosis is very poor with death likely.

Cushing's Triad

- Increased systolic and decreasing diastolic pressure (widening pulse pressure)
- Bradycardia

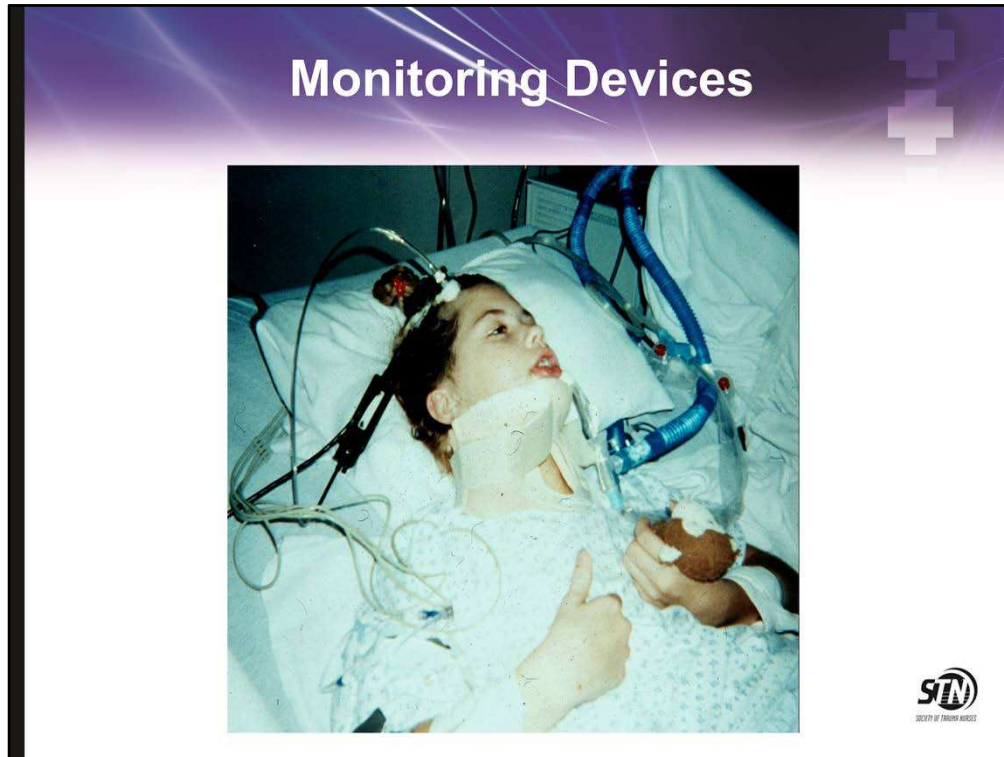
Last attempt of the brain to compensate during the process of herniation



- Cushing's Triad is a late sign of increased ICP and is the last attempt for the brain to compensate during herniation
- Cushing's triad is defined as bradycardia, widening pulse pressure and irregular respirations



- Intracranial pressure (ICP) reflects pressure inside the head
- To measure ICP, ICP monitoring must be established. ICP monitoring requires a probe placed in the brain through a small burr hole which senses pressure inside the skull and brain. The probe is attached to a monitoring device. Some ICP devices can only monitor while others can monitor and drain CSF.
- Insertion of ICP can be done in the Operating Room or at bedside in the ICU under strict aseptic techniques. Insertion of ICP monitoring device can be performed by a neurosurgeon or by a trained Licensed Independent Practitioner (LIP) such as NP or PA.
- Normal ICP is 0-10mmHg
- An increase in ICP represents bleeding, edema or a mass (refer back to the Monroe Kellie Doctrine)
- To decrease ICP you must have to identify, treat and reverse the cause.
- An ICP greater than 20mmHg requires interventions to reduce it below 20 (later described)



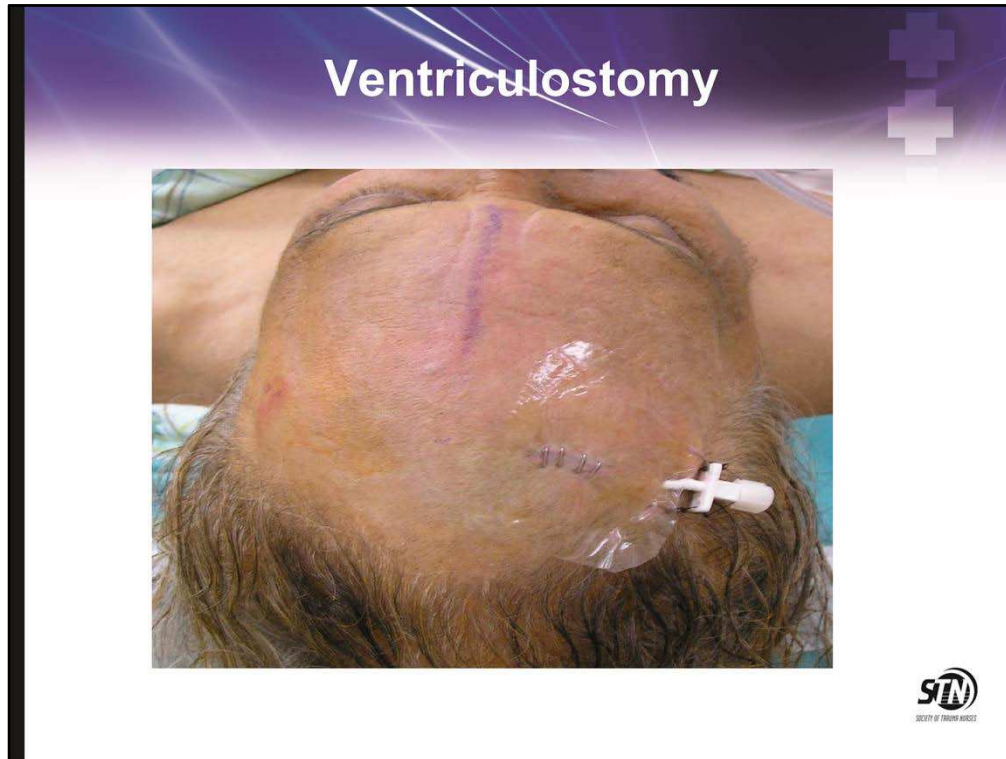
- There are several devices used to measure ICP
- Ventricular catheter is the most accurate monitoring device and is also able to drain cerebral spinal fluid (CSF)
- Subarachnoid screw or bolt and subdural catheter are less accurate
- Subarachnoid bolt is the second choice for monitoring ICP , as they are less invasive however are less accurate than a ventriculostomy.

Image is of young MVC patient with severe TBI and a Camino catheter.

Integra Neuroscience External Drainage Catheters

Types of ICP monitoring devices

Ventrix Ventricular Tunneling Pressure Monitor
Camino Micro Ventricular Bolt Pressure Monitor
Camino Intracranial Pressure Monitor with Licox Bolt
Camino OLM Intracranial Pressure Monitor
Camino Post Craniotomy Subdural Pressure Monitor
Codman Microsensor (subdural, intraparenchymal, and intraventricular)
Gaeltec ICT/B subdural intracranial pressure monitor



- Ventriculostomy is the most frequently used monitoring device
- It allows for measuring of the cranial pressure and drainage of cerebral spinal fluid (CSF). CSF drainage helps to reduce ICP
- Ventriculostomies are at more risk of infection as they are the most invasive.



Indications for Intracranial Pressure Monitoring include

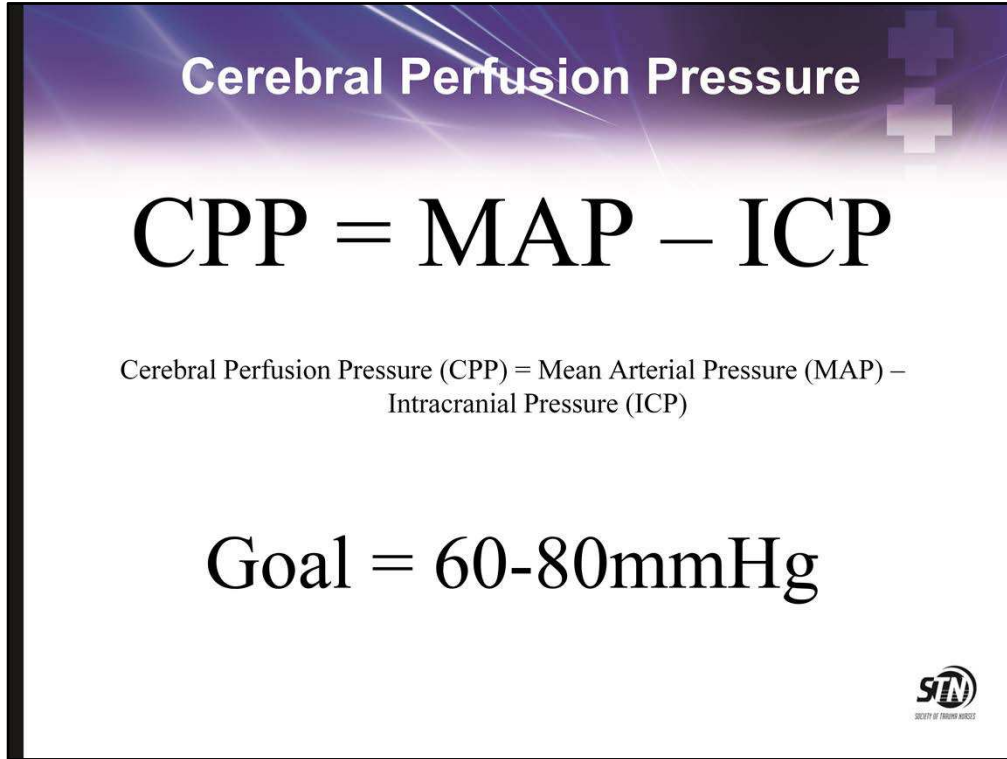
- No eye opening on stimulation (GCS eye =1) + Not following commands (GCS motor less than 6) + Abnormal CT scan **OR**
- Normal CT scan if two or more of the following are noted
- age over 40 yrs
- GCS motor of 1 or 2
- systolic blood pressure < 90 mm Hg at any time.

Abnormal Head CT Findings that do *not* require Neurosurgery consult

Non depressed linear skull fracture
Solitary cerebral contusions < 10mm
Multiple cerebral contusions < 5mm
Subarachnoid blood < 4mm
Isolated pneumocephalus
Subdural hematoma < 4mm

The management of these type of injuries is frequent neurological assessment and reassessment and repeat CT scan

- ICP monitor will be removed when the ICP is below 20mmHg over 1-2 days without CSF drainage



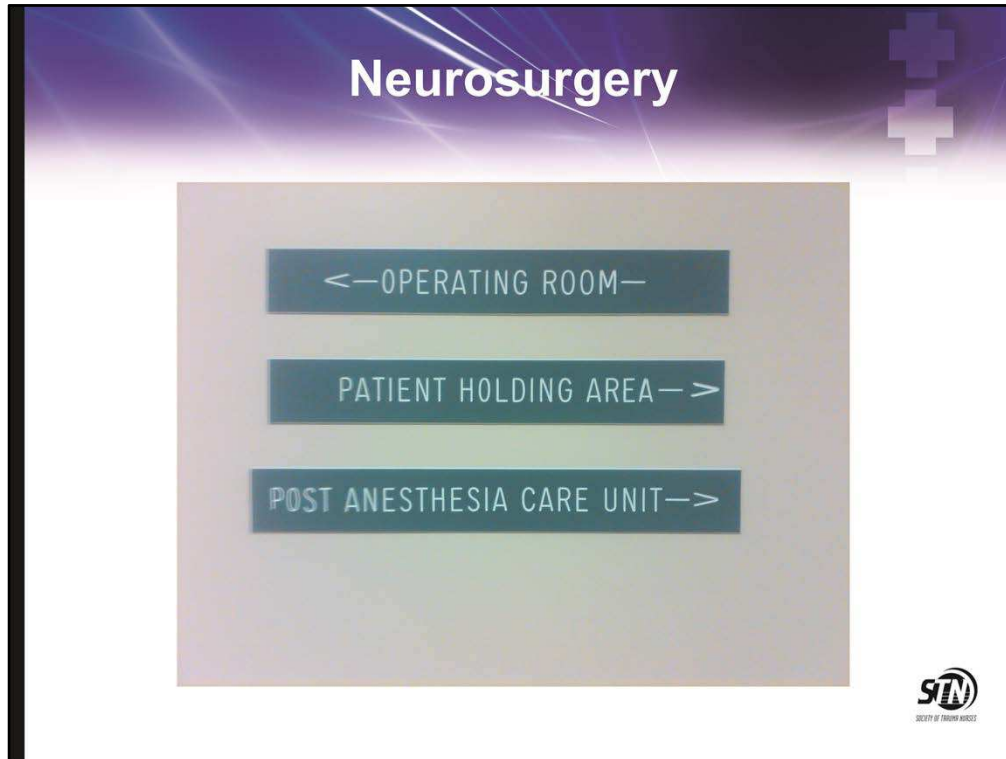
Cerebral Perfusion Pressure

$$CPP = MAP - ICP$$

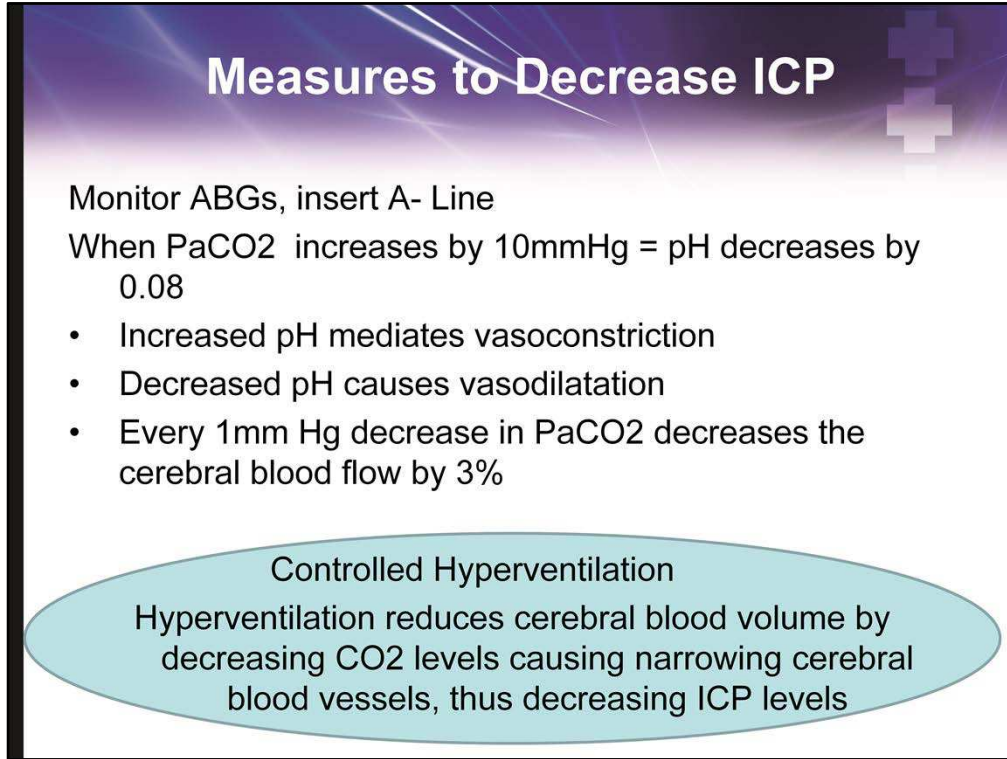
Cerebral Perfusion Pressure (CPP) = Mean Arterial Pressure (MAP) – Intracranial Pressure (ICP)

$$\text{Goal} = 60-80\text{mmHg}$$


- Cerebral Perfusion Pressure (CPP) is defined as MAP minus ICP
- Goal for TBI patients is 60-80mmHg
- CPP represents blood pressure gradient across the brain's vascular bed
- Determines blood flow through the brain



- Neurosurgical involvement should be consulted early for evacuation of intracranial mass lesions, penetrating injuries, and depressed skull fractures
- The crisis within the trauma systems today is the national shortage of neurosurgeons
- The consensus however is that all emergency neurosurgeries should be performed by a neurosurgeon.
- If neurosurgical services are not available the patient must be stabilized and considered for early transfer to a level of care that can provide neurosurgical consultation.



Measures to Decrease ICP

Monitor ABGs, insert A- Line

When PaCO₂ increases by 10mmHg = pH decreases by 0.08

- Increased pH mediates vasoconstriction
- Decreased pH causes vasodilatation
- Every 1mm Hg decrease in PaCO₂ decreases the cerebral blood flow by 3%

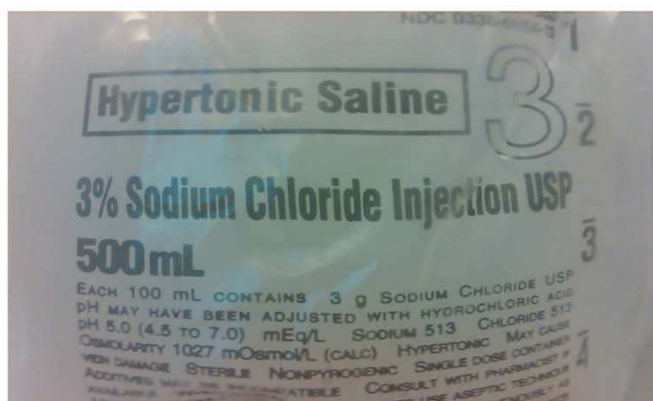
Controlled Hyperventilation

Hyperventilation reduces cerebral blood volume by decreasing CO₂ levels causing narrowing cerebral blood vessels, thus decreasing ICP levels

- For all severe TBI patients the goal is to prevent secondary injury, hypoxia and hypotension
- The insertion of an A- Line is crucial in the management of hypoxia and monitoring of blood pressure
- An A-line allows for frequent ABG draws to monitor PaO₂ and PaCO₂

Hyperosmolar Therapy

- Mannitol
- Hypertonic Saline



- Mannitol is an osmotic diuretic.
 - It can reduce ICP and improve cerebral blood flow
 - It expands plasma volume therefore can reduce blood pressure
 - It is administered by bolus (0.25grams/kg to 1gram/kg) and then continuous infusion
 - The onset is fast (30 mins) and can last up to 6-8 hours
 - It has a diuretic effect therefore monitor blood pressure and intake and output to maintain euvoolemia.
-
- Hypertonic Saline
 - The use of hypertonic saline in TBI has been shown to be beneficial
 - HTS acts by reducing cerebral water content and therefore reducing ICP
 - Can come in different concentrations 3%, 7% 10% and administered as a bolus or continuous infusion
 - Side effects include hyponatremia, therefore monitoring of sodium is important
 - HTS has been associated with inducing pulmonary edema in patients who have underlying cardiac or pulmonary problems.

Measures to Decrease ICP

Seizure Management

- Medications such as Phenytoin and Carbamazepine may be considered for *early* posttraumatic seizure prevention
- Barbiturate therapy may be considered if other treatments have failed



- Posttraumatic epilepsy occurs in 15% of patients with severe TBI
 - One factor associated with posttraumatic epilepsy is seizures occurring in the first week
 - Prophylactic phenytoin or fosphenytoin is recommended to reduce seizures in the first week of injury
 - Therefore antiseizure medication is recommended for patients in the first week.
 - If a patient has prolonged seizures then the antiseizure meds are prolonged
 - The goal is to control seizures quickly as seizures can cause secondary injury.
- Steroids are not recommended in the management of patients with severe TBI

- The evidence has shown that steroid use increases mortality and can increase complications with their use.
- Barbiturates cause hypotension and can increase secondary injury.
- They are not recommended in patients during the acute phase of injury and should only be considered for reducing intracranial pressure when other therapies have failed.

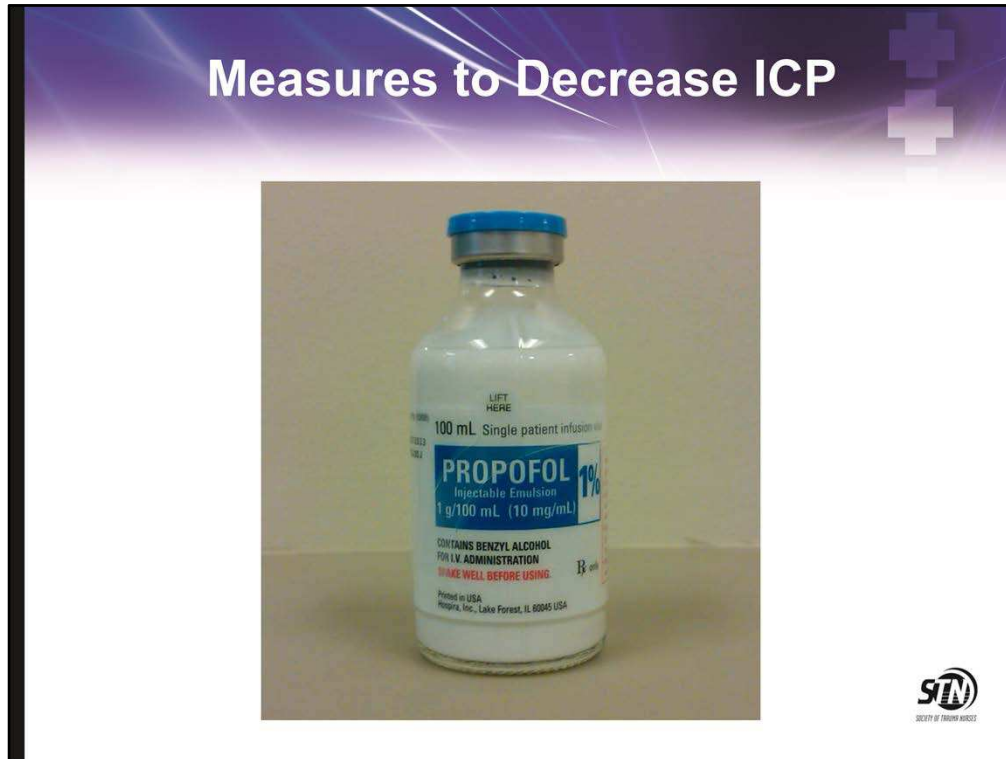
Measures to Decrease ICP



- Sedation and Paralysis
- Always assess for hypoxia/hemodynamic/ ICP changes prior to administration of all agents
- Do not utilize extremity restraints to control patient agitation



- Adequate sedation and pain control are important first steps in managing raised ICP
- Reducing ICP reduces metabolic demand and subsequently cerebral blood flow
- Analgesia should always accompany sedation
- Minimally tolerated doses should be utilized
- A system for monitoring should be utilized (protocols based on use of bedside sedation(Ramsey)/pain scales (Adult Non Verbal))
- At least once a day routine awakening should occur to assess the patient
- Goal is adequate control without obscuring of clinical examination
- The hemodynamic impact must be monitored.



- Propofol is commonly used with TBI pts.
- It is a general anesthetic
- Has no analgesic properties
- Appropriate analgesics and neuromuscular blocking agents must also be administered
- Has amnesic properties
- Benefits: Prolonged cessation of sedation not required to perform neurological assessment
- Side effects of propofol include hypotension, bradycardia and decreased cardiac output.
- TBI patients must be closely monitored for these side effects in order to decrease secondary injury.

Measures to Decrease ICP

Patient Positioning

- Elevate head of bed
- Maintain neutral alignment of head and neck



Elevate the patient's head between 15 -30° to improve cerebral venous outflow - cervical/TL spine should be cleared or stabilized

For every 10° of head elevation, the mean ICP decreases by 1 mm Hg.

Patients with cervical/TL spine injury must be log rolled!

Maintain neutral alignment of head and neck – avoid tight cervical collars and tight taping of ETT as this can cause resistance to cerebral blood flow and venous drainage.

Measures to Decrease ICP

Patient Care Considerations

- Move patient using slow, gentle motions - rapid movement increases ICP
- Turn stopcock off to stop drainage when any nursing activity is performed (turning, suctioning)
- Limit external stimuli



- Nursing interventions must be carefully considered as they can greatly affect potentially increasing ICP
- When moving a patient slow gentle motions should be used as any rapid movement can cause an increase in ICP
- Patient who have an ICP monitor the stopcock should be turned off to drain when nursing activities are being performed.
- Keeping the stopcock open during nursing interventions can result in increase CSF drainage and worsen brain injury (refer to Monroe Kellie Doctrine)
- Maintaining a therapeutic environment for the critically ill TBI patient includes reducing environmental stimuli such as noise, decreasing lighting and regulating visitors

Additional Points



- Early Tube Feeds : Feed within 24 hours to goal within 72 hrs
- VTE Prophylaxis
- Monitor Blood Glucose



- Nutrition is very important in the management of severe TBI patients. The literature has demonstrated that in the first 2 weeks following injury energy expenditure increases.
- Adequate caloric intake has been shown to effect patient outcomes.
- Nutrition replacement should begin no later than 72 hours following the injury and full nutritional replacement within 7 days
- Patients with severe TBI are at risk for developing venous thromboembolic events (VTE).
- VTE prophylaxis includes the use of intermittent pneumatic devices on lower extremities if permitted.
- **Is a vena caval filter indicated?**
- Low molecular weight heparin or low dose unfractionated heparin may be used in conjunction with PCD however there is an increased risk of increasing intracranial hemorrhage.
- Hyperglycemia in conjunction with TBI has been shown to worsen patient outcome
- Hyperglycemia increases metabolic demand and therefore increase risk of secondary injury
- The goal for a TBI patient is to maintain glucose in the therapeutic range (surgical patients 80-110)
- Monitor blood glucose every 2-4 hrs

Other Therapies

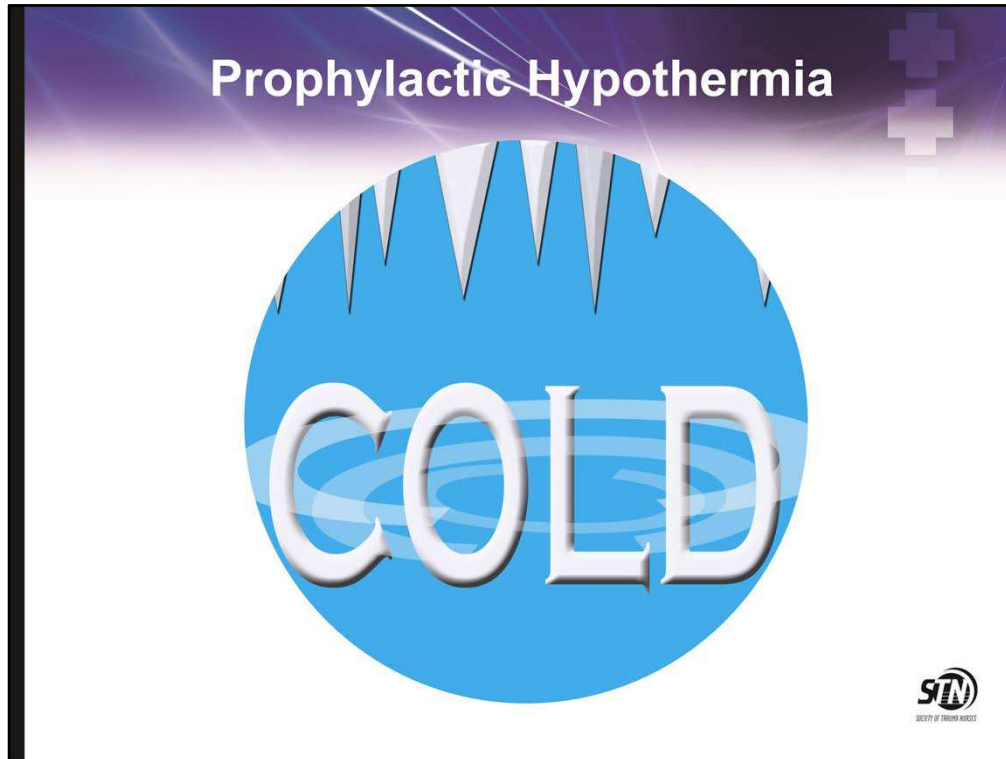
- PbtO₂
- Licox Therapy
- SjvO₂



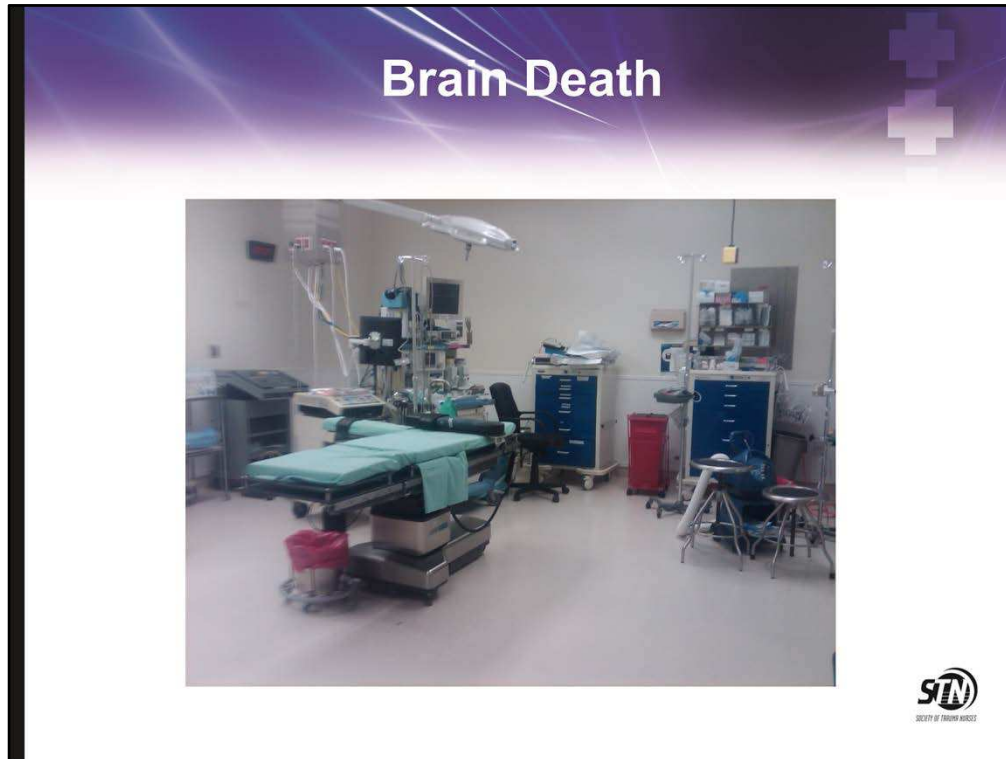
CAMINO® ICP Monitoring Catheter
with Integrated Bolt Fitting
Integralife.com



- Failure to deliver oxygen to an injured brain can be detrimental. Remember the goal is to prevent hypoxia!
- Monitoring the partial pressure of brain tissue oxygen (PbtO₂) can help in avoiding hypoxia and secondary injury
- Brain Tissue Oxygen Monitoring enables the clinician to measure the difference between delivery and consumption of oxygen
- It enables the practitioner to assess levels of oxygenation related to secondary injury and in response to interventions
- Over the last 15- 20 years accurate and reliable methods have been researched
- Licox therapy measure brain tissue oxygenation.
- A catheter/probe is placed in the white matter of the brain and can detect the regional pbtO₂.
- The normal baseline PbtO₂ values range from 25 to 35 mm Hg.
- Mortality rate increases with a PbtO₂ at or below 15 mmHg or with the occurrence of any PbtO₂ values at or below 6 mmHg.
- Recent studies have shown significantly reduced mortality rates in patients who have been treated under a PbtO₂ protocol compared to those with ICP and CPP directed therapy.
- Jugular venous oxygen saturation (SjvO₂) is a measurement of the amount of oxygen in the venous blood taken from a catheter inserted into the jugular venous bulb.
 - It measures the balance between cerebral oxygen delivery and cerebral oxygen consumption
- SjvO₂ is the percentage of oxygen bound to hemoglobin (oxygen saturation). Normal is 55-75%.
- If the SjvO₂ falls below 55-75%, it suggests that the blood flow to the brain is not enough to meet the brain's oxygen requirements, causing the brain to extract a greater percentage of oxygen from the blood.
- If the brain injury becomes so severe that the brain is unable to extract oxygen, the SjvO₂ will rise.



- Prophylactic hypothermia in severe TBI patients has been associated with improved patient neurological outcomes and decrease mortality
- The best available evidence to date supports the use of early prophylactic mild-to-moderate hypothermia in patients with severe TBI
- Hypothermia should be commenced as soon as possible after injury (e.g., in the emergency department after computed tomography) regardless of initial ICP, or before ICP is measured.
- Most studies report using a temperature of 32°-34°C.
- The maximal benefit occurred with a long-term or goal-directed cooling protocol, in which cooling was continued for at least 72 hours and/or until stable normalization of intracranial pressure for at least 24 hours was achieved.
- There is large potential for further research on this therapy in prehospital and emergency department settings.



- The diagnosis of brain death should be considered after all measures have been taken to restore normal physiological parameters.
 - All medications should be reviewed for potential CNS side effects
 - Criteria for brain death include
 - nonreactive pupils
 - GCS = 3
 - absent brainstem reflexes (no gag reflex, Dolls eyes)
 - failed apnea test (no spontaneous ventilations)
- Confirmation of brain death includes
- cerebral angiogram
 - EEG (electroencephalography) showing no activity
- Organ procurement agencies should be notified according to hospital and Procurement Agency. **Review your hospital protocols for Brain Death and Organ Procurement.**

Summary

- TBI can be categorized into mild, moderate and severe according to GCS
- The initial management of the TBI patient are critical to avoiding secondary injury
- There are pharmacologic, monitoring, and nursing care that are instrumental in decreasing intracranial pressure



Useful Websites

Brain Trauma Foundation

<http://www.braintrauma.org/>

National Institute of Neurological Disorders and Stroke

<http://www.ninds.nih.gov/>

Brain Injury Association of America

<http://www.biausa.org/>

Brain Trauma Foundation Learning Portal

<http://www.btflearning.org/go/Home>

American Association of Neuroscience Nurses

<http://www.aann.org/>