Trauma: Head/Brain Injuries
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Purpose The purpose of this course is to explain different types of traumatic head/brain injuries, including primary and secondary injuries, and diagnostic and treatment options.

Goals Upon completion of this course, the healthcare provider should be able to:

- Describe the primary anatomy of the brain.
- Discuss incidence of traumatic head/brain injury.
- Describe injuries related to contact and those related to motion.
- Differentiate between primary and secondary injuries.
- Explain the difference between coup and contrecoup injuries.
- Discuss at least 4 types of skull fractures.
- Identify "raccoon eyes" and Battle's sign.
- Explain the differences among epidural/extradural hematoma, subdural hematoma, subdural hygroma, intracerebral hemorrhage, and subarachnoid hemorrhage.
- Explain the difference between contusion and concussion.
- Discuss diffuse intracranial injuries.
- Describe at least 4 different types of vascular injuries.
- Describe the difference between gunshot wounds and stab wounds.
- Discuss implications of cerebral blood flow and hypotension.
- Discuss cerebral edema and increased intracranial pressure (ICP).
- Describe herniation syndromes.
- Describe 3 assessment tools.
- Describe the ABCDE steps of primary assessment.
- Describe secondary assessment.
- Discuss imaging, cerebral perfusion assessment, and ICP monitoring.

- Discuss ongoing management of mild head injury, moderate head injury, and severe head injury.
- Describe treatment goals for managing traumatic head/brain injuries.
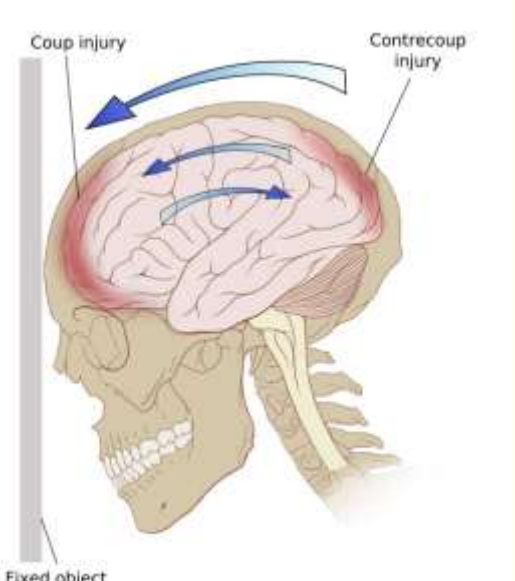
Introduction
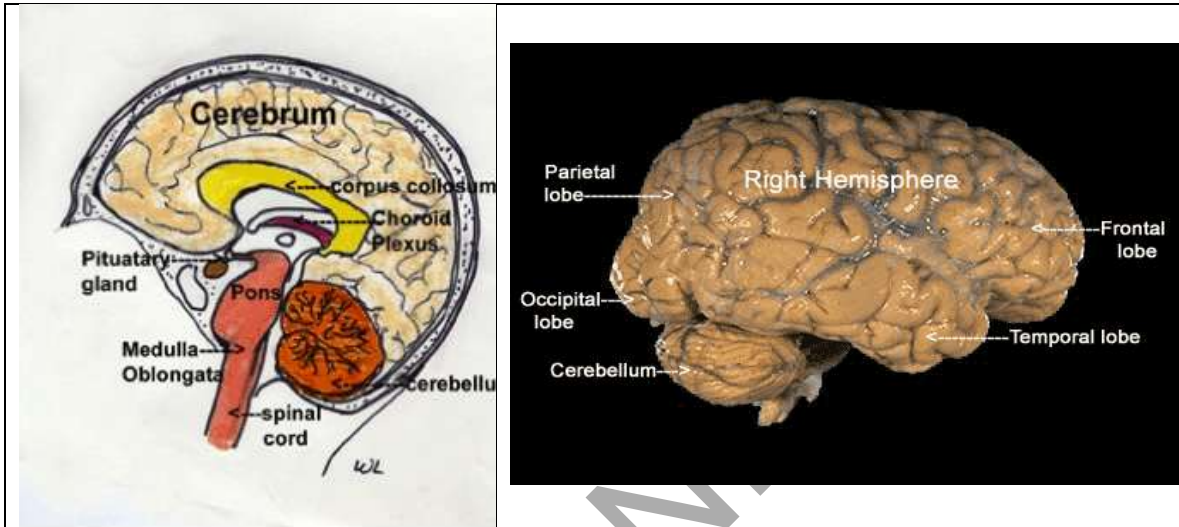
Head injuries are commonly seen in trauma medicine and are the most common cause of trauma-related deaths. About 2 million people suffer traumatic head injuries each year in the United States, resulting in about 50,000 deaths. About a third of head injuries result from motor vehicle accidents, still a high number although the use of seatbelts and airbags has reduced this number considerably. Other common causes include falls, especially in adults >70, and assaults. In general, head injuries are most common between ages 15 and 35 and >70. Head injuries may also occur in children as the result of accidents or abuse.
[See CE course: Abusive Head Trauma in Children.]

In some urban inner cities, up to 40% of head injuries may result from assaults, including gun shot wounds, while in other areas, assault injuries may be relatively rare. Head injuries include a wide range of injury patterns and severity. Head injuries result from blunt or penetrating trauma to the head, usually with a period of altered consciousness following the injury.

Injuries may vary depending on the type of mechanical injury involved:

Mechanical type	Typical injuries
Contact	Skull fractures

|	Epidural hematoma
	Vault, basilar fracture
	Coup contusion
	Contrecoup contusion
	Intracerebral hemorrhage
	Epidural hematoma
Motion Acceleration/deceleration Coup injury — Contrecoup injury — Fixed object	Subdural hematoma Contrecoup contusion Intermediate coup contusion Concussion syndromes Diffuse axonal injury Intracerebral hemorrhage Hemorrhage related to tissue tear

Primary injuries

Head injuries may be classified according to the type of injury as well as the severity.

Scalp/Bone **Scalp injuries** range from minor to large lacerations to extensive degloving injuries. Any type of scalp injury should arouse suspicions of more serious underlying head injury.

Laceration	Degloving
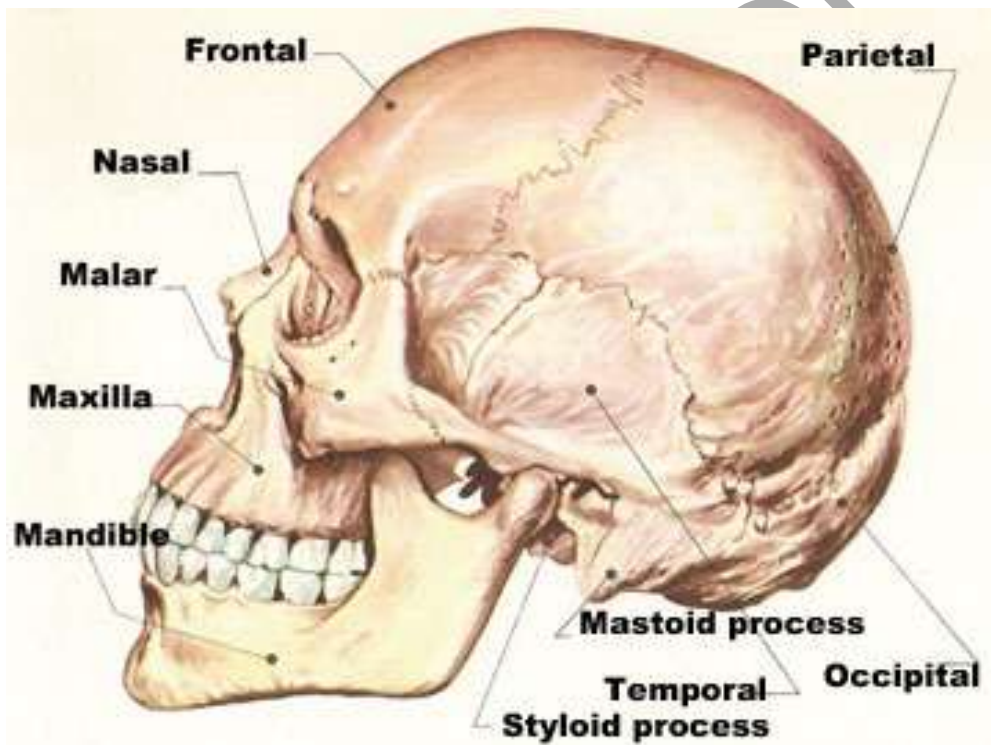
Bone injuries may occur in the cranium or base of the skull, with or without brain injuries. Types of fractures include linear, comminuted, depressed, or misplaced, closed or compound. Diastasis, traumatic opening of sutures, may also occur, but primarily in children. Skull fractures are present in about 80% of fatal head injuries.

Depressed	Linear

Compound/depressed	Penetrating fractures/injury
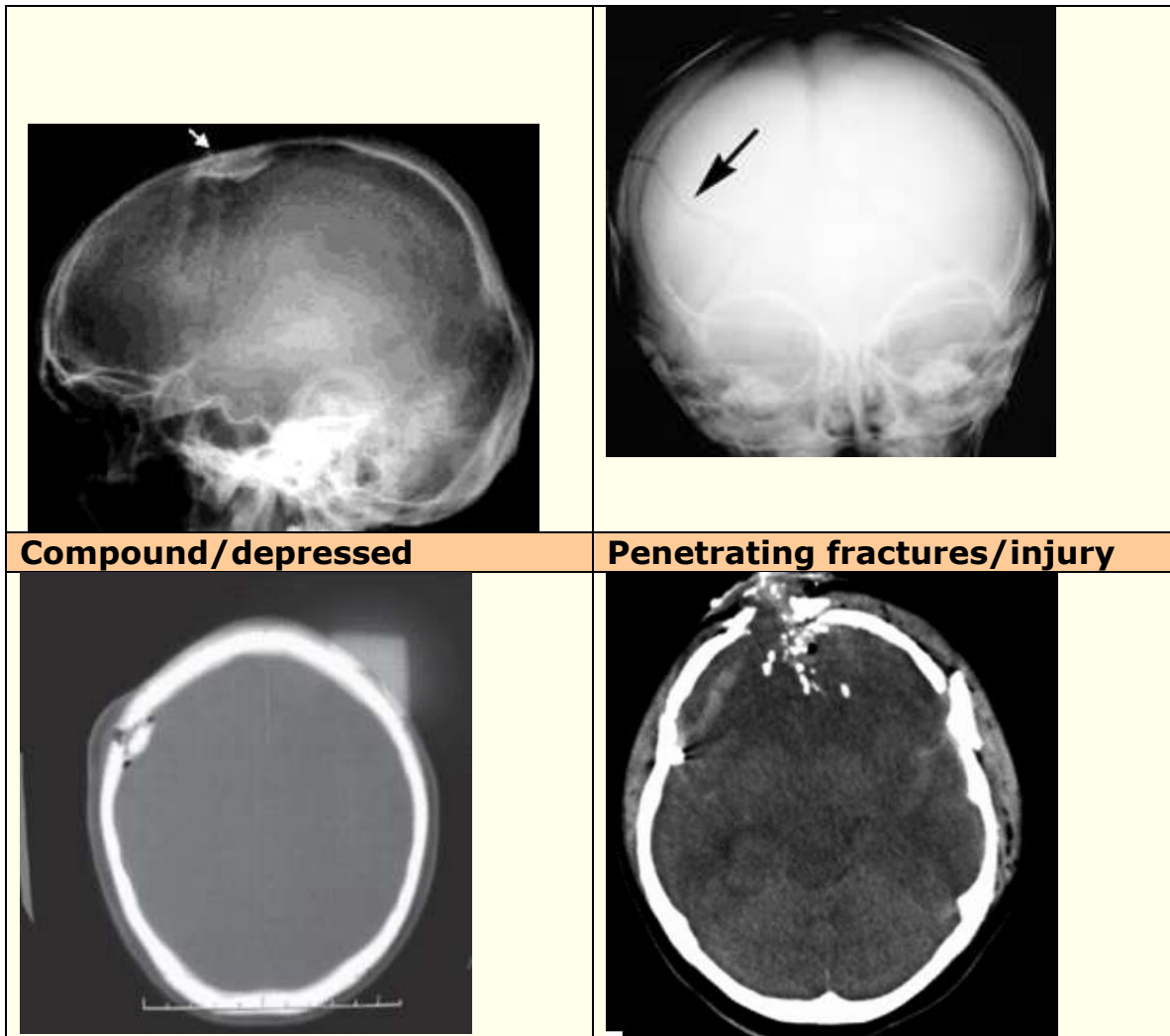
Undisplaced fractures with the scalp intact (closed) usually require no operative management but the patient should be evaluated for underlying injuries. Linear skull fractures associated with scalp lacerations should be cleansed and closed. Open comminuted fractures may require surgical debridement and dural repair. Depressed fractures are significant if displaced to a depth more than the thickness of the skull.

Thin plated bone, such as found in the parts of the temporal region, is more susceptible to fracture than thicker bone. Open depressed fractures should always be explored and debrided because of the risk of infection. Compound fractures that result in dural tear markedly increase the danger of infection.

Basilar skull fractures (BSF) may extend from cranial vault fractures or from impacts to the occipital or mandibular areas. BSFs may result

in damage to the cranial nerves. Fractures of the cribriform plate and anterior cranial fossa (which holds the projecting frontal lobes) may cause damage to the olfactory and optic nerves. With this type of injury, insertion of an NG tube may cause intracranial displacement with severe morbidity.

Additionally, a BSF through the anterior cranial fossa may result in bilateral "raccoon eyes." Other indications of a basal skull fracture include rhinorrhea/otorrhea and positive Battle's sign, which is bruising over the area of the mastoid process.

| Raccoon eyes	Battle's sign

Damage to the petrous (dense portion) temporal bone may result in facial or vestibulocochlear nerve injury. If the dural membrane is torn, then otorrhea or rhinorrhea of cerebrospinal fluid (CSF) may occur.

Focal intracranial These injuries result in visible (macroscopic) parenchymal damage in a focal area, which may be extra-axial (such as subdural/epidural hematoma) or intra-axial (such as intracerebral hemorrhage). Coup, contrecoup, and intermediate contusions may be evident.
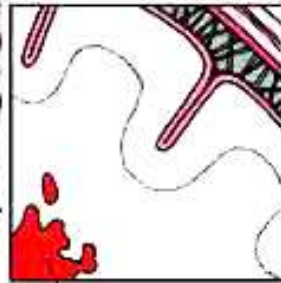
Cross Section of the Brain

- Scalp
- Skull
- Dura mater
- Arachnoid mater
- Subarachnoid space
- Pia mater
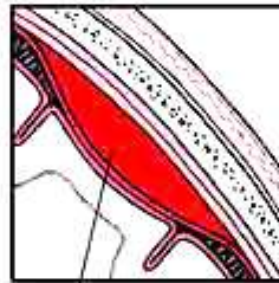- Brain
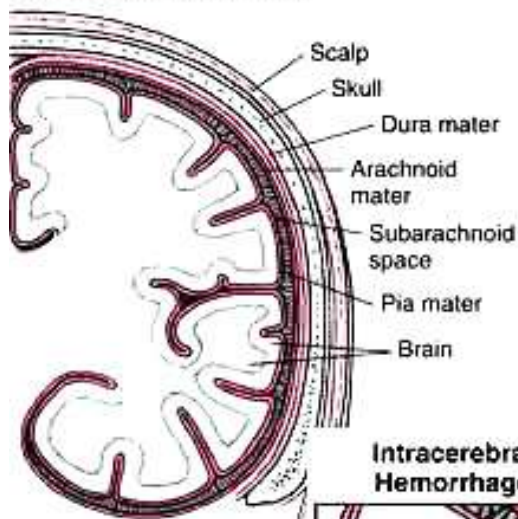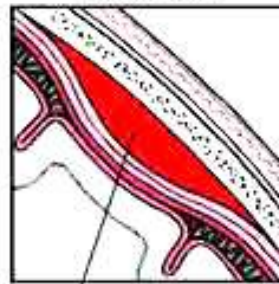
Epidural Hematoma

Bleeding between the dura mater and the skull

Intracerebral Hemorrhage

Bleeding inside the brain

Subdural Hematoma

Bleeding between the arachnoid mater and the dura mater

Epidural/Extradural hematomas result from bleeding between the skull and the dura mater and can cause compression of underlying brain tissue. Epidural/extradural hematomas occur in about 1 to 2% of head trauma patients and 10% of those presenting in a coma. About 90% of adult epidural/extradural hematomas are associated with an overlying skull fracture. About 66% relate to arterial bleeding and 33% to venous.
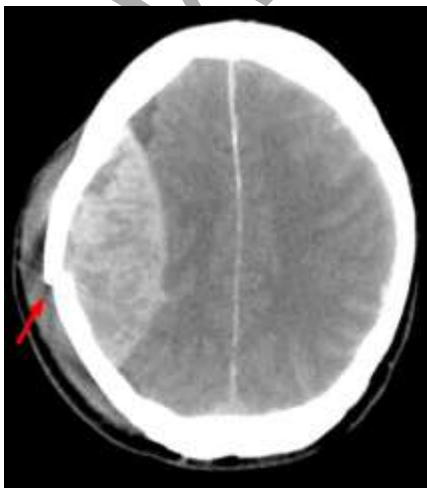
The most common location (70 to 80%) is the temporoparietal area, resulting from a tear in the middle meningeal artery, but epidural/extradural hematomas may occur elsewhere as well. If the compressed tissue constricts the third cranial nerve, patients may exhibit ipsilateral dilation of pupils and contralateral hemiparesis or extensor motor response.

If treated promptly, prognosis is good. However, if the lesion is rapidly expanding

and high volume, a midline shift may occur with resultant herniation. About 90% of patients are <50. Those over 50 rarely suffer this type of injury because the skull and the dura mater are more strongly adhered.

Symptoms are often delayed, so patients may not seek medical help with an initial blow to the head or may appear to have no significant injury; however, as bleeding continues, the patient's condition can deteriorate rapidly. Many patients may initially lose consciousness and then have a period of normal consciousness before condition severely worsens, the "talk and die" phenomenon. With epidural hematomas, there is often no underlying brain injury.

Subdural hematomas, the most common intracranial traumatic lesion, results from bleeding between the dura mater and arachnoid mater, so it is outside of the brain in the layer that usually holds a thin layer of fluid. Subdural hematomas occur in about 30% of severe head injuries. They are often associated with underlying brain injuries, such as contusions and intracerebral hemorrhages, so outcome is often worse than with epidural hematomas.
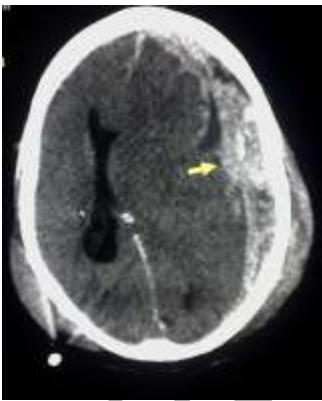
In most cases, the hemorrhage that causes the hematoma is venous. Subdural hematomas may occur in both severe and mild to moderate injuries, especially in the elderly or those on anticoagulation therapy.

Subdural hematoma are characterized according to size, location, and status (acute, subacute, chronic:

- **Acute:** <72 hours after injury. Acute SHs are usually associated with severe head injuries (falls, motor vehicle accidents, assaults), resulting in contusions or lacerations. SH are less common in the young with the average age for acute SH about 41, probably because of brain atrophy resulting in more shear force.

  Most SHs occur ipsilateral to the side where trauma occurred but about 33% are contralateral. Symptoms usually develop over 24 to 48 hours. Symptoms will vary according to the collection of blood. Some patients may exhibit minor or no symptoms for

small hematomas. Symptoms from larger hematomas include changes in levels of consciousness, hemiparesis, pupillary changes, increasing blood pressure with decreasing heart rate and slowing respirations, and coma. Mortality rates are high (up to 90%). Treatment includes immediate craniotomy.

- **Subacute:** 3 to 7 days after injury. Subacute SHs usually result from less severe injuries with clinical manifestations occuring usually between 48 hours and 2 weeks after injury. Symptoms are similar to those of acute SH but develop more slowly. Mortality rates are high without rapid treatment as for acute SH.

- **Chronic:** =/>21 days after injury. Chronic SHs are most common in the elderly and may result from seemingly minor head injuries. The brain atrophy that occurs with aging may result in the brain shifting easily and tearing vessels. Because of the delay between injury and symptoms, patients may have forgotten the original injury, and symptoms may mimic those of a stroke.

After about 2 to 4 days, the collected blood begins to thicken and darken. Eventually the clot calcifies or ossifies. Symptoms may fluctuate and can include severe headache, alternating focal neurological symptoms, personality changes, focal seizures, and cognitive impairment. Treatment includes surgical evacuation of the clot through burr hole suction/drainage or craniotomy.

Subdural hygromas occur in about 6% of head injuries and result from a tear in the arachnoid membrane, allowing CSF to pass into the subdural space.

These usually resolve spontaneously but may be similar in appearance to a chronic subdural hematoma on CT, although less dense.
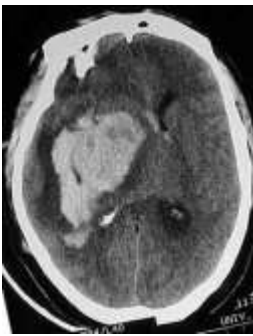

Intracerebral hemorrhages most often occur with trauma when force to the head occurs in a small area, such as can occur with missile injuries, stab wounds,

and gun shot wounds. Two different types of hemorrhage include:

- **Intraparenchymal:** These are focal hematomas >2cm resulting from deep vascular injury, most often in the frontal or temporal areas. This type of injury occurs in up to 3% of severe head injuries. Prognosis is poor because of hypoperfusion to adjacent brain tissue, so prompt surgical evacuation and repair is critical.

- **Intraventricular:** This type of hemorrhage is common with severe head injury, and the prognosis is poor (70%) with persistent vegetive state or severe disability, often related to associated injuries and abnormal presenting GSC score. Intraventricular hemorrhage rarely occurs as a primary injury but is often a secondary injury to acute subdural hematoma or diffuse brain injury. In rare cases, evacuation of a subdural hematoma can result in rupture of subependymal veins as the tamponade effect caused by the subdural hematoma is released. Intraventricular hemorrhage increases risk of posttraumatic hydrocephalus.

Contusions are essentially bruises that occur in the cortical tissue beneath the area of impact, most commonly from a blow to the head. The contusion may occur on the side of impact (coup) or the opposite side (contrecoup). The most common sites for contusion are the frontal and temporal lobes. Contusions result in disruption of cortical tissue and tearing of vessels with edema, which usually causes an increase in intracranial pressure. When extensive contusion occurs with a subdural hematoma it is referred to as a **burst lobe** and has a high mortality rate.
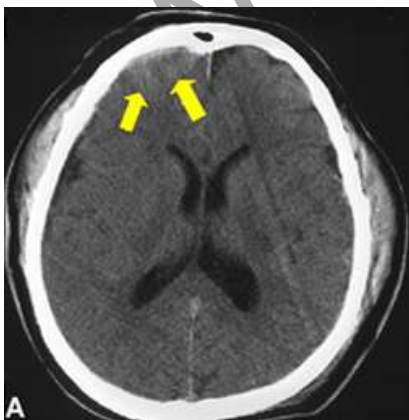
Contusions may be difficult to differentiate from a hemorrhage because both involve bleeding into the cerebral tissue; however, if =/< two-thirds of the involved area is blood, it is considered a contusion and if more than that is blood, it is considered a cerebral hemorrhage. Contusions often have a distinctive wedge shape.

Subarachnoid hemorrhage, into the subarachnoid space, may result from diffuse brain injury and may be associated with other injuries, such as extensive intraventricular hemorrhage or extensive contusions. Subarachnoid hemorrhage may result in diffuse spread or localized hematoma. Traumatic subarachnoid hemorrhage may cause cerebral vasospasm and hypoxia. Traumatic subarachnoid hemorrhage usually occurs with moderate to severe head injuries and the presentation may vary depending on the other injuries to the brain. Up to 39% of those with severe head injuries show evidence of traumatic subarachnoid hemorrhage.

When patients present with a head injury and subarachnoid hemorrhage, it's important to try to determine if the hemorrhage resulted from the fall or accident or if the accident or fall resulted from rupture of an aneurysm. Vasospasm is more likely to occur with spontaneous subarachnoid hemorrhage rather than traumatic.

Subarachnoid hemorrhage diffuses into the CSF and does not build up into a hematoma that requires evacuation. Therefore, treatment focuses on prevention of vasospasm, which may result in ischemia, and treatment of associated injuries. Vasospasm often occurs around day 2 and peaks at 10 to 14 das after injury. Vasospasm if often treated prophylactically with the calcium channel blocker nimodipine.

Diffuse intracranial Diffuse intracranial injuries cause widespread dysfunction because of damage and disruption to neurons and blood vessels from shearing forces during a traumatic event. Evidence of visible injury on CT may be lacking and symptoms may range from mild concussion to severe traumatic brain injury. Diffuse intracranial injury often results from high-velocity injuries, such as may occur with a motor-vehicle accident, but an also result from falls and blunt assaults.

Diffuse axonal injury (DAI) occurs with widespread damage to axons in the cerebral hemispheres, corpus callosum, and brain stem and global cerebral edema and bleeding. DAI is most common with high-velocity traumatic brain injuries and results in about 33% of deaths

from head injuries and accounts for most severe residual neurological deficits.

Primary brainstem lesions, including contusions, shearing, and tearing, are common with diffuse brain injuries and basilar skull fractures. Severe trauma involving skull or cervical fracture or hyperextension of the head and neck may result in tears in the pontomedullary area of the brain stem. Primary brainstem lesions are lethal.

Concussion A concussion is a type of head injury that involves no apparent structural damage but results in temporary loss of neurologic function, such as a brief period of unconsciousness or confusion following a head injury. Thus, there is no macroscopic evidence on CT scan of injury although neurons may be damaged. Concussions are common sports injuries. With impact to the frontal lobe, patients may exhibit bizarre behavior. With impact to the temporal lobe, patients may experience temporary disorientation or amnesia.

Other typical symptoms of a concussion can include somnolence, severe headache, dizziness, nausea, and vomiting. In some cases, patients may experience difficulty speaking or weakness on one side of the body, but symptoms are usually transient.

Up to 50% of those who suffer mild head injuries may experience post-concussion syndrome for up a period of months. Most recover within a year, but symptoms can include headaches, dizziness, impaired concentration and cognition, dizziness, photophobia, tinnitus, sleep disturbance and sensitivity to noise.

Concussions were once considered mild injuries with no residual effects, but studies in recent years show that permanent residual brain damage can occur, especially with repeated concussions. Residual damage can result in headache, personality changes, cognitive impairment, behavioral changes, and impaired functioning.

According to the American Academy of Neurology, three grades of concussions include:
- Grade 1: Transient confusion without loss of consciousness with symptoms resolving in <15 minutes.
- Grade 2: Transient confusion without loss of consciousness with symptoms resolving in >15 minutes.
- Grade 3: Any loss of consciousness of any duration.

Vascular injuries Vascular injuries to the brain are very common in brain injuries resulting from inertial force, such as high-speed motor-vehicle accidents. Damage may occur in intraparenchymal, intracranial, extraparenchymal, and extracranial vessels and may vary from focal injuries associated with concussions, intracerebral, or subarachnoid hemorrhage to more diffuse injuries, resulting in widespread petechial microhemorrhages.

Patients with vascular injuries often have a delay in onset of neurological dysfunction for days or hours following injury, resulting from ischemia with symptoms related to the site of injury:

- Carotid: hemiparesis, hemisensory loss, and dysphasia, and incomplete Horner's syndrome (meiosis and partial ptosis). Sometimes an audible bruit is evident.

- Vertebral: Vertigo, ataxia, nystagmus, dysarthria, and cranial nerve palsies.

Patients may complain of unilateral pain in the head or neck, without obvious signs of trauma.

Traumatic aneurysms may result from penetrating and non-penetrating head injuries. The aneurysms commonly occur in branches of the external or internal carotid arteries, usually the middle cerebral branches or peripheral branches of the anterior cerebral artery. Traumatica aneurysms are often associated with basal skull fractures.

Traumatic arterial venous fistula may develop from vascular injury. Many patients complain of an audible "whooshing" sound that corresponds with the pulse and is most noticeable when the patient is at rest. An audible bruit may be heard when auscultating the scalp. A carotid-cavernous fistula may result in an orbital bruit, and as the pressure builds pulsatile exophthalmos, ophthalmalgia, chemosis (irritated, fluid filled eye), and vision loss. Carotid-caverous fistulas occur in up to 2% of severe head injuries, often associated with basilar skull fractures, especially those involving the sphenoid bone.

Arterial dissection may also occur in all grades of head injury. The tearing increases the risk for developing thrombosis and distal emboli. Arterial dissection may occur in all areas of the carotid arteries. While some patients may experience headache or neck ache, many patients are asymptomatic.

Penetrating/Perforating injuries Penetrating injuries are those that penetrate the cranium but do not exit. Perforating wounds are those that penetrate and exit, leaving entry and exit wounds. Penetrating wounds may result in:

- Intracranial hemorrhage/hematomas.
- Epidural hematoma.
- Subdural hematoma.
- Contusions.
- Subarachnoid hemorrhage.
- Diffuse axonal injury

Gunshot wounds: The most common type of penetrating/perforating traumatic head injury results from gunshot wounds although knife wounds and other projectiles or severe compound fractures may also cause penetrating injuries. While 50% of all trauma deaths occur secondary to traumatic brain injury, 35% of these are caused by gunshot wounds. In some cities, gunshot wounds to the head are the most common cause of traumatic brain injury.

Gunshot wounds directly injure tissue the missile contacts as well as damaging surround tissue as energy is released. The energy is affected by the mass, shape, and type of bullet as well as the velocity (the most important factor). With low velocity (<300 m/s), the most important factor is the structures the bullet hits, increasing damage if the bullet fragments on impact.

With high velocity (>300 m/s), the energy generated on impact spreads from the point of wounding, resulting in cavitation that can be 10 to 15 times the size of the missile. With perforating injuries, the hole may continue to expand by tearing and stretching tissue and then collapsing inward even after the bullet exits, damaging more structures. With penetrating injuries, the hole on the inside may be much larger than expected according to the entry wound, and dirt, debris, hair, and clothing may have been sucked into the wound. As the bullet traverses the brain and exits, it may push brain matter out through the exit or compress brain tissue.

When assessing a patient with a gunshot wound, the safest assumption is that the bullet was high velocity because in most cases the type of bullet and velocity is not known when a patient arrives at a trauma center.

Entry	Exit (different plane)
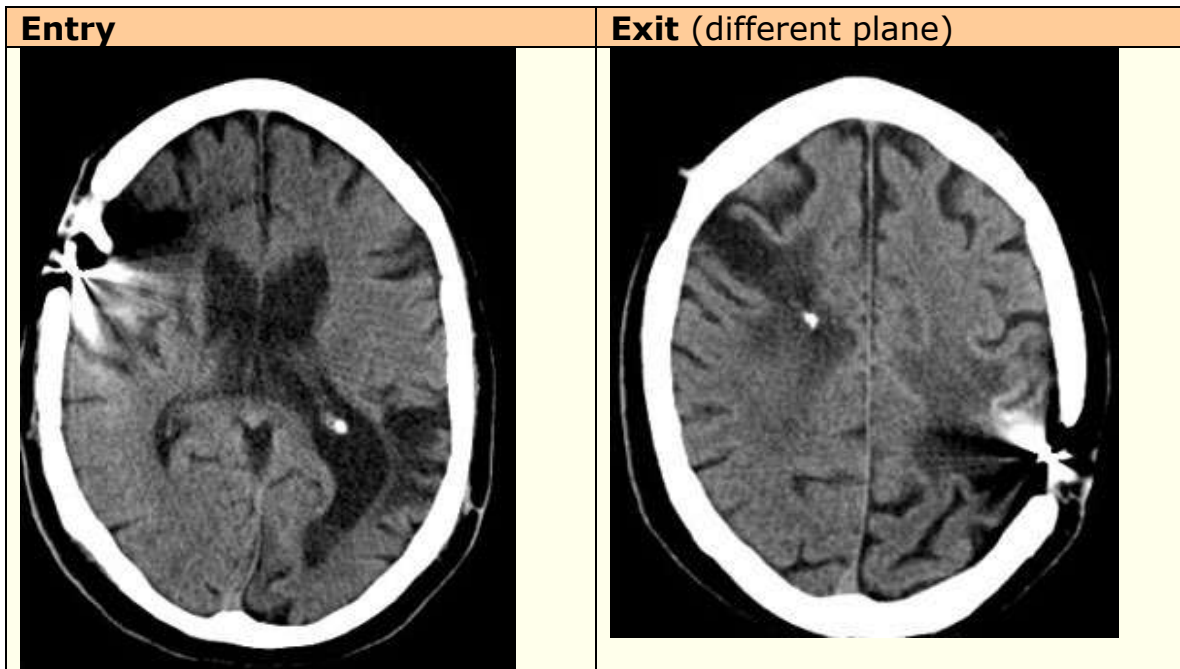
Bullets that are retained in the brain are often left in place because removing them may cause more damage to the brain, but this depends on the location and accessibility.

Stab wounds: Stab wounds typically involve a small impact area and are low velocity wounds, so damage is localized to the area of impact and wound tract. Various instruments can be used for stabbing, including knives, scissors, forks, and spikes. The most common sites for penetration are the orbital areas and squamous temporal areas, where the bones are thinner. However, stab wound to the temporal area are most likely to result in severe neurological impairment.

Mortality rates for stab wounds to the head are about 17% and usually result from damage to the vascular system and massive hemorrhage. Mortality rates are lower (11%) when the stabbing instrument is left in place than when it's removed (26%) prior to emergent care. In some

cases, if the instrument is removed, damage is not evident on scans; however, a slot (narrow, elongated) fracture at the point of entry is diagnostic.

Secondary injuries

Secondary brain injuries result from pathophysiological processes initiated by the primary injury or from extraneous results of trauma, such as hypoxia and systemic hypertension.

Hypoxia
Hypoxia is the most common secondary injury and occurs in 50% of fatal brain injuries. The normal brain responds to hypoxia by vasodilation to increase blood flow and oxygen delivery to the brain; however, with traumatic brain injury, this protective effect is blunted and of shorter duration, resulting in a marked vulnerability to hypoxia. Hypoxia may result from inadequate delivery of oxygen, such as occurs with respiratory dysfunction or impaired ventilation, or increased energy expenditure, such as may occur with seizures.

Hypoxia occurs with an arterial partial pressure of oxygen (PaO2) <60 mm Hg. At this level, the hemoglobin saturation of oxygen begins to drop, reducing arterial oxygen, forcing cells to convert from aerobic to anaerobic metabolism. With any prolongation of hypoxia, tissue damage occurs.

In order to survive, neurons needs a continuous supply of adenosine triphosphate (ATP), and one of the building blocks is oxygen. Without adequate oxygen, ATP must be provided via anaerobic pathways, but this results in acidosis and disturbed cellular function resulting in failures in all areas of cellular homeostasis.

Normal cerebral blood flow (CBF) is about 50 mL/100 g brain/minute, but may vary from 50 to 150 mL/100 g brain/minute. Cerebral ischemia occurs when the CBF falls to 20 mL/100 g brain/minute. Cell death occurs with blood flow of 5 mL/100 g brain/minute.

Cerebral blood flow/ Hypotension
Low CBF may result from increased ICP or systemic hypotension. While CBF is usually autoregulated, the ischemic brain loses the ability to autoregulate blood flow, so this increases the risk of further ischemia and makes the brain very vulnerable to systemic hypotension. Even transient episodes of hypotension can have devastating effects on the brain.

Maintaining cerebral perfusion pressure (CPP), which is the mean arterial pressure (MAP) minus the ICP, is critical for those with closed head injuries. (CPP= MAP – ICP). The goal during active therapy is to maintain the CPP >60 to 70 mm Hg. Rates below 70 mm Hg are associated with higher morbidity and mortality rates.
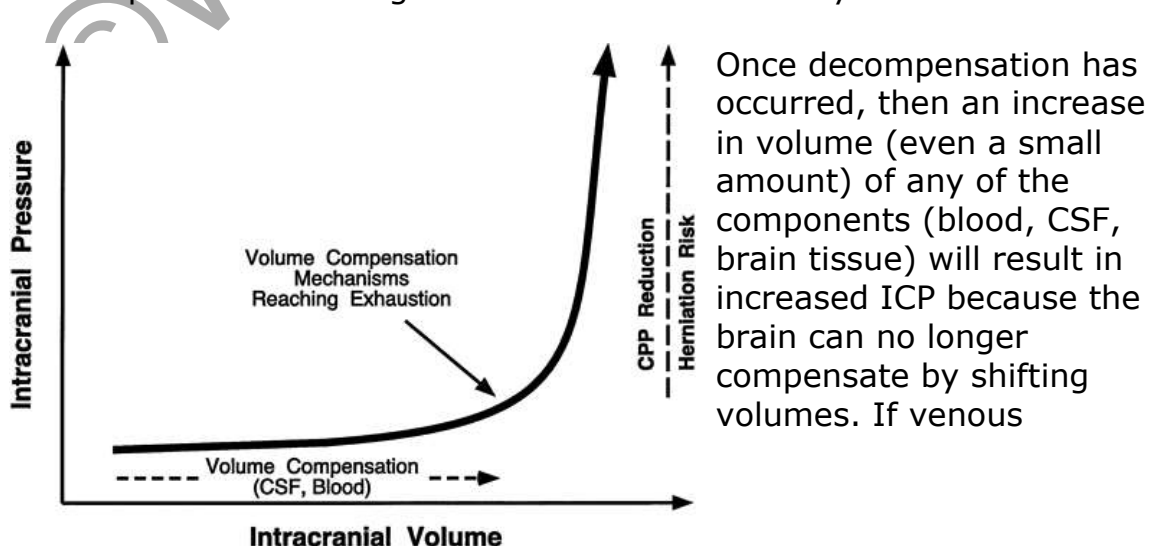
Cerebral blood flow can also be described in relation to CPP and cerebral vascular resistance (CVR): CBF = CPP/CVR. Cerebral blood volume (CBV) is also important because it is one of the three volume components that influence the ICP.

According to the **Monroe-Kellie hypothesis**, in order to maintain ICP within normal range, a change in the volume of one compartment requires compensatory change in the volume of another compartment because the total volume must remain constant (because of the skull). The three components are 1) brain tissue, 2) cerebrospinal fluid (CSF), and 3) blood. Since tissue cannot easily accommodate change, medical interventions must focus on blood flow and drainage of CSF.
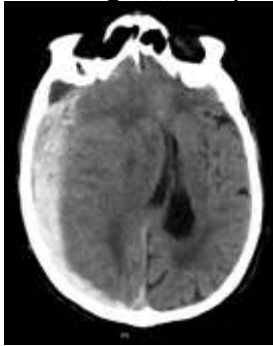
Cerebral edema/ Increased ICP

The normal adult ICP is 10 to 15 mm Hg although higher pressures may be tolerated in a normal brain, but higher pressures are poorly tolerated with severe head injuries. The volume changes possible within the 3 components are limited, so redistribution of CSF is the most common compensatory mechanism. However, with increased pressure, the rate of CSF absorption also increases.

If there is an expanding mass (such as a hematoma) in the brain, the ICP usually stays within normal limits; however, as cerebral edema increases, the CSF and blood volume decrease to compensate until the point of decompensation on the pressure-volume curve is reached, and at that point the ICP begins to increase dramatically.

Once decompensation has occurred, then an increase in volume (even a small amount) of any of the components (blood, CSF, brain tissue) will result in increased ICP because the brain can no longer compensate by shifting volumes. If venous

drainage is impeded in any way, then the rise in ICP will be very rapid.

Severe cerebral edema or an expanding space occupying lesion may result in displacement of the brain parenchyma (brainshift) from one compartment of the skull to another. This can result in compression of structures, midline shift, and **herniation syndromes** with irreversible brain stem injury.

- Compression of the midbrain: Depending on the area of compression, symptoms can include decreased level of consciousness, ipsilateral pupillary dilatation and loss of reaction to light, contralateral weakness, and homonymous hemianopia.
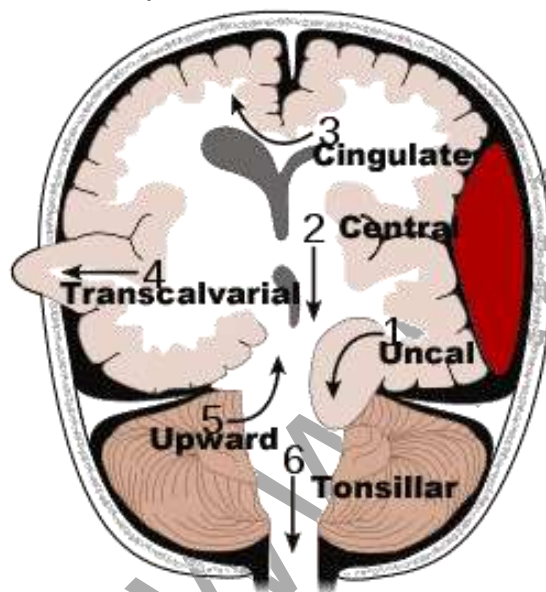
- Supratentorial herniation is above the fold in the dura mater that separates the cerebrum from the cerebellum and includes uncal/transtentorial, central, cingulate/subfalcine, and transcalvarial (through an open skull fracture).
- Infratentorial herniation involves the cerebellum and is upward or tonsillar.

Assessment/Initial management

AVPU   The AVPU assessment is a quick assessment done to determine the patient's level of consciousness. This may be

the first assessment done when initially attending a patient.

AVPU			
A	Alert and awake, aware of person, place, time, and condition.	Yes	No
V	Responds to verbal stimuli.	Yes	No
P	Responds to painful stimuli but not verbal.	Yes	No
U	Unconscious, does to respond to painful or verbal stimuli.	Yes	No

Glasgow coma scale Regardless of the type of injury, head injuries are graded according to the severity of injury. A number of different grading systems are used, but the Glasgow Comas Scale is the most common.

Glasgow coma scale	
Eye opening	4: Spontaneous. 3: To verbal stimuli. 2: To pain [not of face]. 1: No response.
Verbal	5: Oriented. 4: Conversation confused, but can answer questions. 3: Uses inappropriate words. 2: Speech incomprehensible. 1: No response.
Motor	6: Moves on command. 5: Moves purposefully to respond to pain. 4: Withdraws in response to pain. 3: Decorticate posturing [flexion] in response to pain. 2: Decerebrate posturing [extension] in response to pain. 1: No response.

The total possible scores range from 3 to 15, with lower scores indicating increasing morbidity. Injuries and/or conditions are classified according to the total score:
- **Mild** (80%): GCS score 13 to 15 with brief period of loss of consciousness (LOC). Prognosis is good and mortality rates are <1%.

- **Moderate** (10%)**:** GCS score 9 to 12. Patient is usually confused but able to follow simple commands. Patient may have focal neurological deficits. Prognosis is good with mortality rate <5%.

- **Severe** (10%)**:** GCS of =/<8 (coma). Patient is unable to follow commands and requires airway control. ICP is often elevated and the cause of death or disability. Mortality rates are about 33%. Patients who survive usually have significant disabilities.

Note: When documenting the GCS score for patients who are intubated ("tubed), this should be indicated when reporting the score:
- Intubated: 9 [T].
- Intubated and pharmacologically paralyzed 9[TP].

Simplified motor score (SMS) Another simple 3-point scale that is sometimes used, especially by first responders, is the SMS:

SMS	
2 points	Obeys commands
1 point	Localizes pain
0 points	Withdraws to pain or worse.

Primary assessment/ Management

As with most emergency interventions, the first step is to ensure ABCDEs (airway, breathing, circulation, dysfunction/disability, and external exam). The primary survey, a quick review, may be done in as few as 30 seconds to identify the most critical issues, but the secondary and tertiary (following day or days) surveys can be more detailed.

The reality is that with traumatic brain injuries, both primary and secondary assessments may be going on at the same time along with active treatment and laboratory testing, so there is no clear delineation. With a team approach, different members may be assigned different aspects of care, so that one person may insert venous lines while another intubates, and so on.

ABCDEs	
Airway	- Examine the airway for obstructions, such as loose teeth, foreign bodies. Lacerations and bone instability may be obstructive. - Examine the trachea for deviation and observe for signs of circumoral cyanosis (sign of hypoxia).

	Auscultate the airway and listen for turbulence.
Breathing	• Hypoventilation and apnea are common with severe head injuries and require immediate intubation. • Note indications of Cushing's triad (bradycardia, hypertension, abnormal respirations), which indicate herniation with brainstem compression. • Cheyne-Stokes respirations are associated with damage to cerebral hemispheres or diencephalon. • Hyperventilation is associated with damage to rostral brain stem or tegmentum.
Circulation	• Monitor blood pressure, pulse, temperature, color, and indications of cyanosis (circumoral, peripheral), including oxygen saturation continuously. • Use venous access to restore intravascular volume, BP, and perfusion, avoiding hypotonic or glucose containing solutions. • Check arterial blood gases, electrolytes, PTT, hematocrit, and platelet count. • Check serum sodium and osmolality with active therapy. • Evaluate possible causes for hypotension. Hypotension found with bradycardia often indicates spinal cord injury.

Dysfunction/ disability	• Assess responsiveness with alert, verbal, pain, unresponsive (AVPU) system and Glasgow Coma Scale (GCS).
	• Assess pupillary size and responsiveness:
	   o Ipsilateral dilatation with no response to light indicates transtentorial herniation and compression of parasympathetic fibers of cranial nerve III.
	   o Bilateral, dilated pupils unresponsive to light indicate bilateral compression of cranial nerve III or global cerebral anoxia and ischemia.
	   o Nystagmus indicates cerebellar or vestibular injury.
	   o Pinpoint pupils may indicate pontine damage.
	   o Papilledema occurs with increasing ICP.
	• Assess motor ability by observation, pressure to nail bed, or sternal rub:

	- o Decreased spontaneous movement and/or flaccidity may be associated with local injury or spinal cord injury. - o Decerebrate posturing is associated with midbrain damage. - o Decorticate posturing is associated with cerebral cortex, white matter, or basal ganglia damage. • Assess reflexes to determine level of injury and integrity of the spinal cord. • Immobilize patient with rigid backboard and cervical spine collar until spinal cord injury is ruled out. • Provide pharmacologic paralysis and sedation as needed for agitation or combative behavior with short-acting agents: - o Vecuronium bromide, cisatracurium, or succinylcholine. - o Fentanyl or morphine. - o Note: avoid benzodiazepines.
External examination	• Note lacerations, fractures, edema, and bruises.

The primary goals of assessment and treatment are to rapidly identify and evacuate intracranial masses, treat extracranial injuries; prevent secondary injuries, such as hypoxia and hypotension; and enhance cerebral perfusion and oxygenation.

Secondary assessment Secondary assessment includes a comprehensive neurological examination, with the scope of the examination depending on the patient's condition and need for emergent care. The evaluation usually proceeds from head to toe, starting with an examination of the scalp to assess for lacerations and hematomas and careful examination of the face, mouth, nose, eyes, ears, and neck for lacerations, swelling, paresthesia, symmetry, fractures, and drainage.

CSF rhinorrhea indicates an anterior base skull fracture while CSF otorrhea or bloody discharge indicates a temporal base skull fracture. If blood is found in the external auditory canal, a careful examination should be done to determine if blood might have drained into the ear from other injuries, such as facial or scalp lacerations. A sample of discharge should be sent to the laboratory for analysis, but the

target/ring sign may be used at bedside to help distinguish CSF mixed with blood from serous drainage: The blood gathers at the center surrounded by a halo of CSF. CSF has a glucose level that is >30 mg/dL and presence of β-2 transferrin.

The patient should be kept in neutral body alignment if spinal cord injury is a risk and has not been excluded by examination or radiology. The cervical collar will not prevent movement of the spine. At least 3 people should assist in logrolling the patient for examination of the back, maintaining the spine in neutral alignment.

Imaging Standard x-rays serve little purpose in diagnosis and treatment of traumatic head/brain injuries. While they may show fractures, they do not show underlying tissue damage. The rapid non-contrast CT is the imaging method of choice and should be completed as soon as possible after initial stabilization of the patient. However, if the clinical evaluation indicates a need for transfer because the facility does not have the capability for providing necessary care, the transfer should not be delayed for the CT, as time may be critical.

CT scans are often not required with mild injuries. Criteria for CT scanning after mild injury include:
- LOC >5 minutes.
- Post-traumatic amnesia >5 minutes
- Deterioration of GCS scores after initial assessment.
- Worsening or severe headache.
- Persistent nausea and/or vomiting.
- Focal neurological deficits.
- Suspected skull fracture.
- CSF leak (otorrhea, rhinorrhea).
- Post-traumatic seizure.
- Possibility of anesthesia because of multiple injuries.
- Delayed presentation to ED or second presentation.

MRI is rarely indicated in the initial assessment because MRI takes longer and prevents adequate monitoring. Additionally, MRI is not adequate to assess fractures and is less able to detect hemorrhage in the first 3 days than CT. After 3 days, the MRI may be superior to the CT for detecting small extra-axial collections and non-hemorrhagic contusions. Additionally, the MRI is better able to detect lesions in the posterior fossa and better able to differentiate between chronic hematomas and hygromas. MRI and MRA may be more effective in identifying vascular injuries.

Cerebral perfusion assessment

The most commonly used method of screening for vascular lesions of the brain is cerebral angiography, which can detect dissection, traumatic aneurysm, and cerebral vasospasm. MRI and MRA are also increasingly used to detect vascular lesions. Transcranial Doppler ultrasonography (TCD) is a non-invasive method of assessing perfusion and can demonstrate vasospasm, hyperemia, hypoperfusion and impaired autoregulation. Single-photon emission computed tomography (SPECT) scanning is also occasionally utilized.

ICP monitoring

It's important to remember that in a fluid-filled space, the pressure is usually the same wherever it's checked; however, with brain injury, various interfering factors, such as decreased CSF because of brain swelling, may interfere, so that the pressure obtained by the catheter or sensor may reflect only the pressure at that site rather than pressure within the ventricular system (the site of the most accurate CSF pressure).
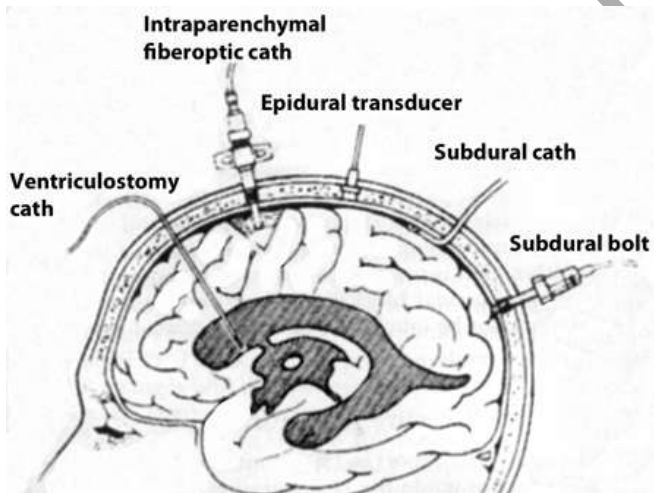
An ICP consistently >20 mm Hg is considered elevated and requires intervention to prevent secondary brain injury. ICP monitoring is not usually done for mild to moderate closed head injuries but may be considered for patients with moderate injuries who must have surgical intervention for other injuries.

Indications for ICP monitoring, along with a brain oxygen monitor, include:
- Severe traumatic brain injury (GCS =/<8) with abnormal CT (hematoma, contusion, edema, compressed basal cisterns).
- Severe traumatic brain injury (GCS =/<8) with normal CT if 2 or more of the following are present:
    - >40 years of age.
    - Posturing (unilateral or bilateral), flexor or extensor.
    - Hypotension with systolic BP <90 mm Hg).

Ongoing Management

Mild head injury (GCS 13-15)
A mild closed head injury requires careful observation and assessment because 10% to 35% will show subsequent abnormalities on CT scan and 2% to 9% will require craniotomy. The usual procedure is to observe the patient for a minimum of 4 hours, keeping the patient NPO with head elevated to 30° in case the condition worsens and the patient requires surgical intervention. If the patient is stable and has minimal, receding symptoms (headache, nausea, vomiting), the patient can be discharged into the care of a reliable person who can monitor the patient.

Patients should be admitted if they have an abnormal CT, deterioration of GCS, multiple traumatic injuries, and or progressive symptoms. Patients should also be admitted if they do not have reliable care on discharge. Patients should be observed closely for at least 24 hours after discharge and should have follow care within 48 hours.

Treatment options	
Analgesia	Acetaminophen. IM or IV opiates (for severe headache or vomiting): Should be avoided if the patient is intoxicated unless there are other painful traumatic injuries (such as fractures).
Antiemetics	May be used to control nausea and vomiting, especially if it is severe.
Tetanus toxoid or immunoglobulin	As indicated if there are open wounds, such as lacerations.

Treatment for **concussions related to sports injuries** includes:
- Grade 1: Observation. Return to sports activities if return to normal within 15 minutes with no residual effects.
- Grade 2: Observation and return to sports activities after asymptomatic for one week.
- Grade 3 (or second grade 2): Observation and return to sports after asymptomatic for two weeks with evaluation by neurosurgeon prior to resuming sports.

Moderate head injury (GCS 9 to 12) Management is similar to that of those with mild head injury except that all patients should have a CT scan and be admitted for observation and treatment. Even patients with higher GCS scores may be classified as moderate or severe with CT abnormalities, such as contusion and subdural or epidural hematoma, so it's important to remember that the GCS score is only a guide.

If the CT scan shows no abnormality, most patients can be observed overnight and should show improvement within 12 hours with increase in GCS score to 14 to 15. An abnormal CT usually indicates a need for surgical intervention or careful observation. The CT should be repeated in 12 to 24 hours or with further neurological deterioration.

Patients who are extremely restless or agitated may need to be intubated and sedated prior to CT scanning. Generally, all patients with moderate head injuries should have repeat CT scans at 48 to 72 hours.

Severe head injury (GCS =/<8 All patients with severe head injury must be intubated and symptoms managed to prevent secondary injury. Patients may be transferred immediately to surgery or admitted into the intensive care unit when stabilized.

Patients require intensive continuous monitoring to prevent cerebral hypoxia and maintain adequate cerebral perfusion. The ICP monitor should be inserted as soon as possible, usually immediately after the CT scans. While various options are available, the external ventricular catheter allows for drainage of CSF to treat cerebral hypertension, and the other monitors do not, so that may be a consideration, especially with hydrocephalus. The fiberoptic parenchymal catheters (bolts) are accurate and allow for direct brain oxygen monitoring and are less likely to result in complications, such as infection, but do not allow for drainage.

Other monitoring should include:
- Arterial blood pressure (arterial catheter preferred over non-invasive monitoring).
- ECG, heart rate, temperature.
- Pulse oximetry.
- CVP or PAC if patient's volume status is a risk.
- Brain tissue O2 (and, if possible, cerebral microdialysis).

- Intake and output.
- Arterial blood gases every 4 to 6 hours.
- Electrolytes, glucose, serum osmolality (if receiving mannitol) every 6 hours.
- Hematocrit, PT, PTT, platelets every 12 hours.
- Jugular venous O2 saturation.

Treatment goals	
Mean arterial blood pressure (MAP)	>80 mm Hg.
Oxygen saturation (arterial)	100%
ICP	<20 mm Hg.
CPP	>60 to 70 mm Hg.
PaCO2	33 to 37 mm Hg
Hematocrit	32 +/- 2%
CVP	8 to 14 cm H2O
Jugular venous O2 saturation	>50%
Direct brain O2	>20 mm Hg
Temperature	Within normal range
PT, PTT	Within normal range.
Platelet count	150,000 to 450,000

Note: Routine hyperventilation is no longer recommended as a treatment and should be reserved only with suspected herniation and abrupt neurological deterioration if other treatments have been unsuccessful.

Note: Protocols for treatment may vary somewhat from one institution to another.

Common treatment approaches for specific issues	
Epidural hematoma	- Intubate with rapid sequence induction (RSI), usually with premedication with lidocaine, a sedating agent (such as etomidate), and a neuromuscular blocking agent. - Elevate head to 30° or place in reverse Trendelenburg to increase venous drainage if ICP elevated. - Administer IV crystalloids to prevent hypotension. - Administer 0.25-1 g/kg IV mannitol if MAP >90 mm Hg with signs of elevated ICP. - Administer phenytoin 15 to 18 mg/kg IV to

	reduce risk of early seizures (although does not alter incidence of late-onset or persistent seizure disorder). • Note: Thiopental should be avoided as it may result in hypotension. • Refer to neurosurgeon for evacuation and surgical repair.
Subdural hematoma	• Intubate with RSI with GSC <12 or as needed based on symptoms. • Elevate head to 30°. • Refer to neurosurgeon for decompression and surgical repair if midline shift =/> 5m or hematomas >1 cm thick. • Refer to neurosurgeon for decompression with less shift or thickness if GSC score decreases by =/> 2 points from time of injury, patient's pupils are dilated and fixed, and ICP >20 mm Hg. • Administer mannitol 0.25-1 g/kg/ IV for signs of herniation syndrome. • Place Burr holes if evidence of herniation syndrome and surgical repair is delayed. • Avoid short-acting sedative and paralytics unless needed for ventilation or increased ICP. • Administer phenytoin to prevent seizures, which may increase ischemia. • Provide transfusion of FFP and/or platelets for those on anticoagulants. • Note small and/or asymptomatic acute SDHs may be observed with serial CTs.
Penetrating head wounds	• Type and crossmatch immediately because blood loss may be extensive. • Intubate according to ATLS guidelines. • Avoid NG tubes with anterior skull basal fractures. • Elevate head to 30°. • Administer mannitol 0.25-1 g/kg/IV for increased ICP and signs of herniation syndrome. • Use barbiturate coma if necessary/decompressive craniectomy as needed for uncontrolled increased ICP. • Administer phenytoin (usually 15-18 mg/kg IV bolus and 200 mg IV q 12 hours).

	• Administer broad-spectrum antibiotics for 7 to 14 days. • Administer histamine blocker to prevent stress ulcers. • Refer to neurosurgeon for surgical repair, which usually includes superficial debridement of entrance and exit wounds.
Cerebrospinal fluid leak	• **Note:** May occur after injury or after surgical repair (especially with penetrating injuries). CSF otorrhea is more likely to heal spontaneously than CSF rhinorrhea. • Treat initially with bedrest in position that decreases drainage. • Insert lumbar drain if drainage has not decreased within 24-48 hours. Drain 10 mL per hour for 5-7 days but avoid continuous drainage. (This may increase risk of infection). • Refer to neurosurgeon for surgical repair if drainage persists. • Prophylactic antibiotics should be avoided in those with uncomplicated CSF leak because of the risk of superinfection.
Increased ICP	**Order of first-line interventions:** • Elevate head of bed to 30°. • Administer neuromuscular paralysis (Vecuronium bromide or pancuronium) and narcotic sedation. Short acting benzodiazepines, such as midazolam, and narcotics, such as morphine are commonly utilized. Sedation may result in hypotension. • Provide intermittent ventricular CSF drainage per ventriculostomy (not continuous). • Administer bolus of mannitol 25-50 g/IV every 4 hours if serum osmolality is =< 315 and sodium =/< 150 mEq/L. • Monitor electrolytes throughout treatment. • Administer furosemide 20 to 40 mg IV every 4 hours. • Maintain PaCO2 at 28-30 mm Hg. **Second-line interventions:** • Administer barbiturates (Pentobarbital 400 to 1000 mg IV over a period of 60 minutes followed by 40 to 100 mg per hour with

	continuous EEG monitoring to ensure suppression of cerebral electrical activity. Monitor for hypotension.
	• Utilize hypothermia to core temps of 34 to 36°C.
	• Use intravascular volume control and vasopressors to maintain MAP and ensure that CPP is >70 mm Hg.
	• Utilize hyperventilation for short periods (last resort) to reduce PaCO2 to 25 to 35 mm Hg.
	• Refer to neurosurgeon for decompressive craniectomy.

Conclusion

Traumatic head injuries encompass a wide range of injuries of varying severity but are leading causes of long-term morbidity and a leading cause of death. Head injuries may be classified and graded in a number of different ways.

Key considerations to keep in mind when treating patients with traumatic head injuries include:
- Loss of consciousness is always cause for concern and a primary indication for brain injury.
- The GSC score should be determined as quickly as possible for a baseline.
- Primary goals of therapy should be to prevent hypotension and ischemia and promote cerebral perfusion.
- Mass lesions (such as hematomas) should be promptly diagnosed and referred for surgical evacuation.
- The CT scan should be used as the diagnostic tool of choice for all types of brain injuries.

Because of the high mortality rate (about 36%) with severe head injuries, many patients may be candidates for organ donation after brain death is certified. All healthcare personnel involved in trauma care should be aware of protocol for requesting organ donation and notifying UNOS.

References

- Biros, M.H., & Heegaard, W.G. (2019). Chapter 38: Head injury. In Marx JA, ed. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. Philadelphia, Pa: Mosby Elsevier.

- Dawodu, S.T. (2021, November 10). Traumatic brain injury (TBI): Definition, epidemiology and pathophysiology. *Medscape Reference.* Retrieved from http://emedicine.medscape.com/article/326510-overview
- Flint, L, Meredith, J.W., Schwab, C.W., Trunkey, D.D., Rue, L.W., & Thaeri, P.A. (2018). *Trauma: Contemporary Principles and Therapy.* Philadelphia, PA: Lippincott Williams & Wilkins.
- Kazim, S.F., Shamim, M.S., Tahir, M.Z., Enam, S.A., Waheed, S. (2021). Management of penetrating brain injury. *Journal of Emergencies, Trauma, and Shock.* Retrieved from http://www.onlinejets.org/article.asp?issn=0974-2700;year=2011;volume=4;issue=3;spage=395;epage=402;aulast=Kazim
- McPhee, SM, & Papadakis, MA. (2019). *Current Medical Diagnosis & Treatment.* San Francisco: McGraw Hill Medical.
- Mitchell, EL, & Medson, R. (2005). *Introduction to Emergency Medicine.* Philadelphia: Lippincott Williams & Wilkins.
- Peitzman, A.B., Rhodes, M., Schwab, C.W., Yealy, D.M., & Fabian, T.C. (2008). *The Trauma Manual: Trauma and Acute Care Surgery.* Philadelphia: Lippincott Williams & Wilkins.
- Price, DD. (2020, November 3). Epidural hematoma in emergency medicine. *Medscape Reference*. Retrieved from http://emedicine.medscape.com/article/824029-overview
- Sherry, E, Trieu, L, & Templeton, J., Eds. (2003). *Trauma*. Oxford: Oxford University Press.
- Vinas, F.C. (2021, June 2). Penetrating head trauma. *Medscape Reference.* Retrieved from http://emedicine.medscape.com/article/247664-overview