TRAUMATIC BRAIN INJURY IN ADULTS

Definitions:

Traumatic brain injury (TBI) can be defined as an insult to the brain caused by blunt (non-penetrating) or penetrating forces. TBI may result in:

1. Concussion: transient brain injury that may or may not permanently affect brain function (Figure 1).
2. Contusion: brain tissue bruising that is akin to bruising of the skin when it is injured (Figure 1, 3).
3. Laceration: brain injury from something entering the skull and tearing brain tissue (Figure 2).
4. Anoxic injury: brain damage from a lack of oxygen (Figure 5)

Most brain injuries (aside from purely anoxic events) are mechanistically caused by the delivery of kinetic energy to the skull's contents. This delivery of energy can come in several forms:

1. Indirect damage to the brain can occur by acceleration/deceleration of the brain tissue. In this situation the head moves rapidly in one or multiple directions. These movements cause the brain tissue itself to move in multiple direction in the skull with injuries being caused by tearing (shearing) and bruising (contusions) of the brain tissue as it strikes the inner surface of the skull. This mechanism of injury can yield concussion and contusion. One can best picture this injury if they were to imagine gelatin containing fruit salad inside a box. If the box is vigorously shaken and then opened one would see the gelatin torn into pieces and the embedded fruit salad shifted from its original position (Figures 1, 3).
2. Direct damage to the brain can occur when a foreign body penetrates the skull and enters the brain. Such injuries are often caused by bullets and knives. Bullets damage the brain by both mechanically tearing brain tissue and by imparting a tissue disrupting energetic shock wave to the brain. The faster and larger the bullet, the more energetic the wave and the more damage that is created (Figure 2).
3. Direct damage to the brain can be caused by the skull itself were it to be fractured. In such situations, bone fragments can travel into the brain much like a foreign body and cause tissue contusion and laceration (Figure 2)

In addition to the mechanism of injury, brain injuries are also categorized as **"open"** or **"closed"**. "Open" injuries indicate that the scalp has been cut, the skull has been fractured and the covering of the brain or the brain itself is exposed to the air (Figure 2). "Closed" injuries refer to brain injuries that occur despite the skull remaining intact (non-fractured). In these cases, the brain is damaged but is not exposed to the outside environment (air) (Figure 1, 3).

Causes of Injury:

Common causes for traumatic brain injury include:

1. Falls (cause 50% of TBI in US; 1.3 million brain injuries/year)
2. Vehicular accidents
3. Weapons (bats, fists, feet, clubs, etc)
4. Penetrating from weapons (knives, bullets, other piercing weapons)
5. Workplace injury (falling materials, tools)
6. Blast injuries (explosions)
7. Domestic violence ("shaken baby syndrome")
8. Anoxic injuries from strangulation, crush injuries of the chest, traumatic cardiac arrest and drowning.

Societal Effects:

1. There are up to 2.87 million Emergency Department visits per year in the US due to TBI of which over 800,000 involve children.
2. Approximately 285,000 US hospital admissions/year are TBI related.
3. Approximately 56,000 deaths/year are TBI related.
4. TBI survivors often harbor permanent physical and/or cognitive disabilities that reduce an individual's quality of life.
5. TBI costs the US economy over $77 billion dollars/year.

TBI Subcategories:

Concussion

1. Defined by the American Association of Neurological Surgeons as a mechanism of injury resulting in immediate yet transient alteration in brain function.
    a. > 3 million concussions/year in the US.
    b. Approximately 300,000 US concussions/year are sports related.
    c. 19% risk of concussion/year from contact sports.
2. Signs and Symptoms include:
    a. Confusion
    b. Headache
    c. Dizziness/imbalance
    d. Nausea/vomiting
    e. Amnesia
    f. Tinnitus (ringing in the ears)
    g. Difficulty concentrating
    h. Light sensitivity (photophobia)
    i. Loss of smell (anosmia)or taste (ageusia)

j. Insomnia
k. Depression
l. Irritability

Contusion

1. Defined as focal tissue death and/or bleeding within the brain that results from external forces which cause the brain to strike internal skull surfaces with subsequent tissue bruising and brain swelling. Contusion can also include tearing of the brain tissue and its communication pathways. This tearing is termed Shear Injury or Diffuse Axonal Injury (DAI) (Figure 4)
2. Signs and Symptoms include:
    a. All Signs and Symptoms seen with Concussion
    b. Focal neurologic deficits:
        i. Paralysis
        ii. Sensory loss
        iii. Loss of significant brain function
        iv. Coma
        v. Death

Penetrating

1. Defined as a brain injury that results from a breach of the scalp, skull, and possibly the brain coverings (dura and pial surfaces) and the brain itself. Such injury is usually caused by the head striking a surface with subsequent displaced skull fracture or by a projectile or other material entering the skull and brain (Figure 2).
2. Signs and symptoms include those seen with Concussion and Contusion.

TBI Scoring:

Scoring Systems:

Common scoring systems for documenting TBI severity include (Figure 8):

Glasgow Coma Score (GCS)
FOUR Score (Full Outline of Unresponsiveness Score)
IMPACT Score

GCS is the most widely utilized scale as it assesses Eye Opening, Motor Function, and Verbal Responsiveness with scores ranging from 3 (comatose) to 15 (normal). Scores $\leq$ 8 indicate severe brain injury, 9-12 indicates moderate brain injury and 13-14 indicates mild head injury. A score of 15 denotes a normal level of alertness. A modification of the GCS score is the GCS-Pupil Score which adds a fourth category that takes into consideration pupillary light response.

FOUR Score uses Eye opening, Motor Function, Brainstem Reflexes and Respiratory pattern to provide a score range of 0 – 14 with lower scores indicating worse condition. Some investigators favor the FOUR Score over the GCS because of evidence that shows that by eliminating verbal responsiveness as a measured parameter, the FOUR Score more accurately describes condition and predicts outcome especially in patients with more severe TBI who are intubated.

IMPACT Score is specifically meant to predict outcomes in patient populations with TBI who have a GCS $\leq$ 12 (moderate and severe TBI). The extended version uses data from the first 24 hours after TBI including age, motor exam, pupillary exam, Marshall CT classification (presence of DAI, mass lesion), presence or absence of traumatic SAH, epidural hematoma, hypoxia, hypotension along with glucose and hemoglobin levels. A simpler IMPACT Score uses only motor response, eye opening response and verbal response with a score of 3-8 indicating severe TBI.

Why are standardized scoring systems important?:

Standardized, simple to perform scoring systems serve many important purposes including:

1. Scores provide an objective measure of a patient's condition.
2. Scores provide inter-user reliability when multiple providers serially examine a patient over time.
3. Scores can be easily communicated between providers.
4. Scores are independent of language barriers.
5. Scores can guide care by forecasting outcomes.
6. Scoring can provide reliable serial data for treatment evaluation.
7. Scoring can provide reliable patient comparisons from different care sites over disparate time periods.

GCS and Glasgow Outcome Score (GOS):

The GOS is a 1-5 scale measure of a patient's clinical outcome 6 months following TBI with a higher number denoting a poorer outcome (Figure 8). The attached Table shows the individual scores and their clinical meaning. As discussed above, scoring systems like GCS can be used to prognosticate outcomes. Because such data is based on population statistics, it does not necessarily predict a single individual's clinical course. Despite these limitations, GCS can be used to advise families and guide care.

When populations are studied for relationship between GCS and GOS it becomes clear that the worse the patient's initial neurologic condition, the lower their likelihood of a favorable 6-month clinical outcome. A patient population with a severe TBI (GCS $\leq$ 8) has a 40% mortality, 16% chance if severe disability, 19% chance of moderate disability and 21% chance of a good recovery. In comparison, mild brain injury (GCS $\geq$ 14-15) portends a 9% mortality, near 0%

chance of vegetative outcome, 14% chance of severe disability, 24% chance of moderate disability and > 50% chance of a good recovery.

FOUR Score is also a useful predictor of neurologic outcomes in TBI populations. For every 1-point increase in score the odds of in hospital mortality are reduced by 15% and the odds of poor neurologic outcome are reduced by 18%.

MANAGEMENT OF TBI

Neurosurgical Philosophy for Managing TBI:

1. Little at this time can be done to mitigate damage caused by the initial injury.
2. Secondary injuries/insults (damage that occurs to the brain after the initial injury) must be prevented or at least minimized to improve outcomes. An example of this would be a brain injury with significant blood loss from a scalp laceration. Blood loss induced hypotension and subsequent reduced brain perfusion and stroke would represent a secondary insult.
3. The recovery environment must be idealized.

Stage 1: Management (Initial Care in the Field)

Emergency Medical Responders (EMS) are generally the first individuals to care for a patient suffering from TBI and their actions are critical to improving outcomes. The goal of EMS care is patient assessment, stabilization, and transfer to hospital for definitive care. Toward this end it is important that responders focus on the following:

1. Maintenance of the patient's airway, breathing and blood circulation (ABCs). This includes oxygen administration, clearing airway obstructions, obtaining intravenous access for administration of fluids and medications, and maximizing cardiac function.
2. In the process of achieving the ABCs, EMS must assume all trauma patients have a spinal column injury and minimize the risk of further spinal cord damage. This can be achieved through rapid immobilization of the neck and back using external fixation devices (collars, boards, straps).
3. Avoid low blood pressure (hypotension) to avoid decreased blood flow to the brain and secondary brain injury. This may require administering intravenous fluids and/or medications to improve cardiac function. This aspect of EMS care is critical since post TBI:
    a. Systolic BP <90 mm Hg is associated with 3x worse outcome
    b. Systolic BP <100 Hg is associated with 2x worse outcome
    c. Systolic BP < 120 HG is associated with 1.5x worse outcome
4. Avoid inadequate blood oxygenation (hypoxia) since post TBI blood oxygen levels (PaO2) lower than 60 mm Hg are associated with worse outcomes. Oxygen saturation levels

should be maintained >90% which often necessitates placement of mechanical airways (intubation) into a patient with altered level of consciousness (LOC) or airway injuries.

5. If possible, EMS should try to avoid over (hyper) or under (hypo) ventilation of intubated patients since ventilation rates affect levels of blood PaCO2 which in turn may decrease blood flow to the brain (hyperventilation and hypocapnia which constricts blood vessels) or increase blood flow to the brain (hypoventilation and hypercapnia which dilates blood vessels). Blood vessel dilation increases the volume of blood in the head which may in turn increase the intracranial pressure (ICP) which may in turn decrease the ability for the heart to deliver blood to the brain (Cerebral Perfusion Pressure; CPP).
6. EMS should make efforts to accurately determine the patient's GCS or FOUR Score as this may influence decisions regarding need for intubation and other ancillary procedures such as ventriculostomy placement for cerebrospinal fluid (CSF) drainage. Establishing a GCS will also allow for accurate documentation of a patient's change in clinical condition over time.
7. EMS should document a pupillary examination
8. EMS should document sensory or motor deficits that could suggest a spinal cord injury

Stage 2: Management in the Emergency Department (ED)

After a TBI patient arrives in the ER, the ABCs discussed above remain of utmost importance with Advance Trauma Life Support algorithms in place. Once the patient is hemodynamically stable, ED personnel should perform the following neurologic examination:

1. Determine GCS or FOUR Score and note changes from the previous EMS documented scores.
2. Intubation should be strongly considered for any patient with a compromised airway or a GCS $\leq$ 8 (severe head injury) as these patients cannot reliably protect their airway from aspiration of saliva or vomitus.
3. Obtain a history that includes age, mechanism of injury, current medications, and co-morbidities.
4. Obtain laboratory tests that document electrolytes (especially sodium), cell counts, coagulation and, in women of childbearing age, pregnancy status. Pertinent abnormalities, especially coagulopathies, should be corrected (if possible)
5. Evaluate the head for:
    a. bleeding scalp wounds which should then be cleaned and sewn or stapled shut to reduce blood loss.
    b. foreign body entry and exit wounds and any signs of leaking brain matter or spinal fluid.
    c. irregularities in the skull contour that might represent underlying depressed skull fractures.
    d. mastoid region and periorbital skin discoloration (Battle Sign and Raccoon Eyes, respectively) and tympanic membranes for retro tympanic blood. These findings indicate the high likelihood of a skull base fracture.

    e. evaluate the eyes to confirm that they can move in all direction and that an extraocular muscle is not entrapped by an orbital fracture
6. Document a pupillary exam for baseline diameter and response to light
7. Document any sensory or motor loss that could suggest a spinal cord injury.
8. Consult Neurosurgery

Stage 3: Radiologic Imaging

Once a patient is pulmonary and hemodynamically stabilized, radiologic imaging of the head and spine can begin. All TBI patients will require imaging of the head and neck. This is generally achieved using CT scanning with or without lateral and AP radiographs of the cervical spine. These studies can determine the extent of the skull and brain injury and determine if there is evidence for cervical spine fracture or vertebral dislocation. Cervical spine abnormalities should be considered unstable until proven otherwise. Patients who are unable to relate whether they have neck pain should remain in a cervical collar and those who report neck pain should be assumed to have spinal column instability until proven otherwise.

CT scanning of the head will quickly detect the following pertinent findings:

1. Skull fracture location and type (linear; depressed; comminuted; open) (Figure 2)
2. Brain contusions which indicate brain injury and potential for progressive brain swelling and further bleeding (Figure 3).
3. Intracranial air which indicates either an open skull fracture with or without an underlying dural tear or a fracture of the air sinuses with or without an underlying dural tear. Such findings may portend an increased risk of infection or possible spinal fluid leak.
4. Bleeding patterns in the white matter and ascending/descending neuronal tracts that may indicate the presence of shearing forces and diffuse axonal injury (DAI) (Figure 4).
5. Subarachnoid hemorrhage which may indicate minor or major vascular injury.
6. Ischemic brain which may indicate an arterial injury or a period of anoxia (Figure 5).
7. Intraventricular blood which may portend hydrocephalus and increased intracranial pressure (Figure 6).

Once head CT and cervical spine imaging are completed additional radiologic tests may be warranted in an emergent or urgent time frame.

1. The presence of subarachnoid hemorrhage may indicate the need for CT or catheter-based angiography if patterns suggest a traumatic large vessel injury.
2. MRI imaging of the neck may be necessary to visualize the cervical spine ligaments to identify ligamentous injury (elevated T2 signal) and possible spinal column instability. MR imaging can also identify spinal cord injuries, spinal canal hematoma and traumatic disc herniation.

3. If patient have lower extremity weakness or sensory loss, plain X rays, CT, and MRI of the thoracic and lumbosacral spine may be indicated to evaluate for disc herniation, hematoma, spinal column fracture and/or vertebral body dislocation.

It is imperative that any patient with a penetrating injury to the brain from a bone fragment, foreign body, or weapon (knife, bullet) undergo a vascular study to determine the presence or absence of an intracranial vascular injury such as a pseudoaneurysm. If identified, these lesions need to be urgently addressed to reduce the risk of acute intracranial hemorrhage or re-hemorrhage (Figure 7).

After all imaging is performed the physician may be faced with normal studies in a patient with a focal or global neurologic deficit. Practitioners should understand that acutely obtained CT scans of the head may not show radiographic changes such as those from a stroke or DAI. In these situations, the treating physician should entertain the possibility of a vascular injury from traumatic dissection of the carotid or vertebral arteries. Any patient with a neurologic deficit that cannot be explained by the head CT and spine imaging should undergo emergent vascular imaging of the neck and brain. This can be achieved using MRI/MRA, CT/CTA, or catheter angiography. If a symptomatic arterial dissection is identified, it may be addressed with antiplatelet medications and, at times, endovascular repair. DAI diagnosis is not necessary during the acute period. If. CT does not show DAI, MRI imaging of the brain in search of DAI can be performed once the patient is stable. A diagnosis of DAI is useful in terms of guiding care and predicting outcomes, however, it does not generally affect immediate care decisions.

Neurosurgical Decision Making:

Once the EMS/ED evaluation and treatment is completed, the Neurosurgeon must answer several questions.

Question 1: What does the patient's GCS indicate?

Why: If GCS is $\leq$ 8, the patient is considered to have a severe head/brain injury. In addition to being placed on mechanical ventilation these patients are often pharmacologically sedated and paralyzed which makes it difficult to obtain serial neurologic examinations. In lieu of a physical examination, the only way for physicians to detect worsening neurologic condition from dangerous elevations in intracranial pressure (ICP) may be to surgically place an intracranial pressure monitor so that elevated ICP can be identified and treated with medications, spinal fluid drainage, and/or surgery. While it is generally recommended that patients with severe head injury undergo ICP monitor placement, there are instances where CT scans show no clear evidence for increased pressure. In such cases, it may be left up to the neurosurgeon's discretion as to whether an ICP monitor is required. To underline uncertainties in management, numerous studies have shown no difference in outcomes when managing ICP using invasive monitors as compared to management with exam and imaging. While hospital compliance with suggested guidelines for ICP monitor placement vary from 10-65% and for craniotomy between 7-76%, no relationship has clearly been shown between compliance rates and mortality rates.

Question 2: Does the patient require emergent surgery to evacuate a hematoma, remove damaged swollen brain or to remove a portion of the skull (decompressive craniectomy) to create room for brain swelling to reduce intracranial pressure (ICP)?
Why: Increased ICP may reduce brain blood perfusion and oxygenation. Reduction in brain perfusion may in turn damage remaining normal brain tissue thus worsening outcome from this secondary insult. The answer to Question 2 often depends upon such factors as the patient's age, GCS, type, extent and location of the brain injury, and hematoma volume and location.

Question 3: Does the patient require repair of a skull fracture?
Why: Some skull fractures require elevation depending upon their location, degree of depression, relationship to vascular structures, and effect on the underlying brain.

Question 4: Does physical examination suggest a vascular injury such as arterial dissection?
Why: Some patients have a neurologic deficit due to a trauma induced thromboembolic or occlusive stroke and need to be treated with antiplatelet medications and/or endovascular therapy.

Question 5: Does the patient's examination suggest seizure activity?
Why: Some patients have neurologic deficits that mimic brain injury but are due to seizure activity. It is important to determine the presence of seizure induced deficits to avoid unnecessary surgery.

Question 6: What is the patient's prognosis?
Why: Prognosis, based on GCS, FOUR Score, age, and co-morbidities can help direct appropriate current and future therapy and inform families about likely outcomes. With such knowledge, families can make informed decisions regarding future therapeutic interventions.

Question 7: Does the patient require particular medications?
Why: Depending upon the patient's injury, they may benefit from administration of:

1. High dose steroids for spinal cord injuries.
2. Hyperotonic agents such as mannitol, urea, or 3% saline and/or diuretics to control elevated intracranial pressure.
3. Antibiotics to reduce the risk of certain infections especially with penetrating head injuries.
4. Anticonvulsants to reduce the risk of acute seizures.
5. Antiplatelet agents to reduce the risk of clot formation and stroke.
6. Anticoagulants to reduce the risk of clot formation and strokes.
7. Antihypertensives to reduce the risk of brain hemorrhage.
8. Ionotropic agents (drugs that improve cardiac function) to improve cerebral blood flow.
9. Procoagulants (drugs that improve blood clotting such as DDAVP) especially in patients on anticoagulants.

10. Sedatives, barbiturates, and paralytic agents to reduce intracranial pressure, improve ventilation, reduce cerebral metabolic needs and to provide for patient comfort.

Intensive Care and TBI:

ICU care for TBI is complex and under constant modification. Despite the academic complexities, the basic tenets remain consistent:

1. Avoid hypotension to preserve cerebral perfusion
    a. SBP < 90 mm Hg is associated with 3x worse outcomes.
    b. SBP < 100 mm Hg is associated with 2x worse outcomes.
    c. SBP < 120 mm Hg is associated with 1.5x worse outcomes.
2. Avoid hypoxia
    a. Maintain oxygen saturation > 90% and Hct >25-30 mg/dl to optimize cerebral oxygenation.
    b. Avoid hypocapnia unless ICP is critically elevated and in such cases use hyperventilation only for acute reduction of elevated ICP.
    c. Avoid hypercapnia as it causes intracranial vasodilatation, increased cerebral blood volume and resultant increased ICP which reduces cerebral perfusion.
3. Avoid pyrexia and aim for normothermia (using acetaminophen, cooling blankets, bromocriptine, baclofen)
    a. Fever (Temperature >38.5 C) increases neuronal injury and is associated with worse outcomes.
    b. Fever is present in >80% of patients in the first three days following injury.
4. Maintain cerebral perfusion pressure
    a. CPP = (Mean arterial BP) minus (ICP)
    b. Ideal CPP is 50 -70 mm Hg to maintain cerebral blood flow.
5. Avoid intracranial hypertension > 25 mm Hg using:
    a. normalized PaCO2 or mild hypocapnia (35 mm Hg)
    b. CSF drainage
    c. Pharmacologic sedation/paralysis
    d. Osmotic agents
    e. Diuretics
    f. Pharmacologic paralysis
    g. Normothermia
    h. Head elevation with neck neutral to avoid jugular vein compression
    i. Surgical intervention (decompressive craniectomy, hematoma evacuation, lobectomy)
6. Correct coagulopathies
7. Maintain normal electrolyte concentrations (especially Na)
8. Be vigilant for deep venous thrombosis (DVT)
9. Identify infections

10. Prevent pressure sores
11. Institute early nutritional support (TBI leads to catabolic/hypermetabolic state)
12. Avoid hypo and hyperglycemia and insulin insufficiency to reduce risk of acidosis, free radical generation, and increased blood brain barrier permeability.
13. Monitor for increased sympathetic activity which can raise ICP and metabolic needs.
14. Consider need for tracheostomy and feeding tube insertion.
15. Consider need for physical therapy along with upper and lower extremity splinting to limit contracture development.
16. Evaluate swallowing to reduce the risks of aspiration
17. Maintain proper bowel and bladder evacuation.
18. Organize discharge planning
19. Organize family support

FINAL REMARKS

To minimize the risks of Secondary Central Nervous System Injuries, proper care of patients with TBI requires a team approach

First Line

- Emergency Medical Field Services
- Nursing services

Second Line

- Trauma services
- Emergency medicine
- Neurosurgery
- Radiology
- Clinical Laboratory
- Nursing services

Third Line

- Internal Medicine
- Neurology
- Infectious Disease
- Hematology
- Physical therapy/Occupational therapy/Rehabilitation Medicine
- Pharmacy
- Nutritional
- Social Services
- Psychology/Psychiatry
- Addiction Services
- Palliative Care
- Nursing services

Fourth Line

- Skilled nursing facilities
- Rehabilitation facilities
- Nursing services

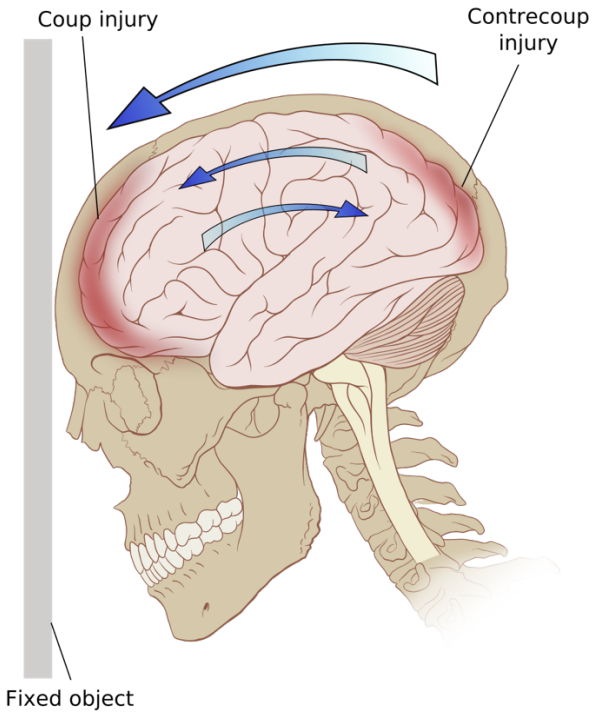

Figure 1: Acceleration/Deceleration forces causing brain to move within the skull and suffer tissue injury.

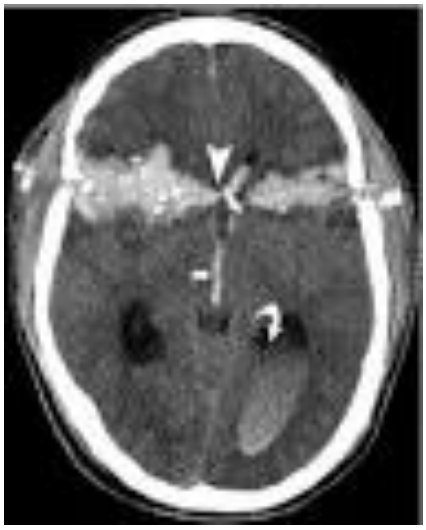

Figure 2: Penetrating TBI secondary to gunshot to head (arrow shows bullet tract)

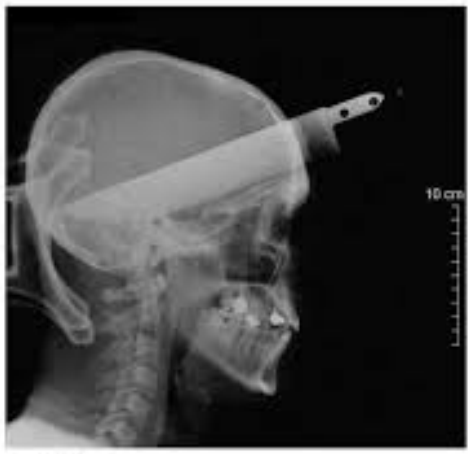

Figure 2: Penetrating head injury due to knife penetration of skull and brain

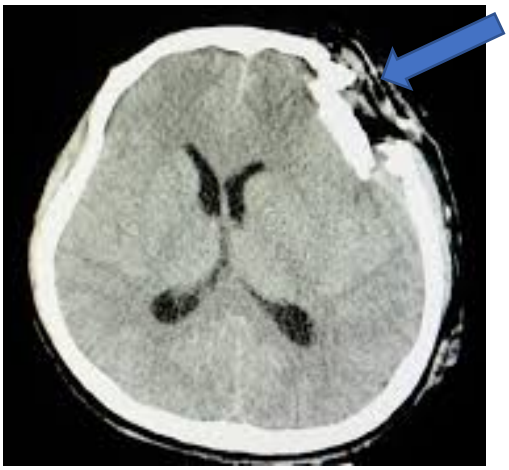

Figure 2: Penetrating injury caused by depressed skull fracture (arrow)

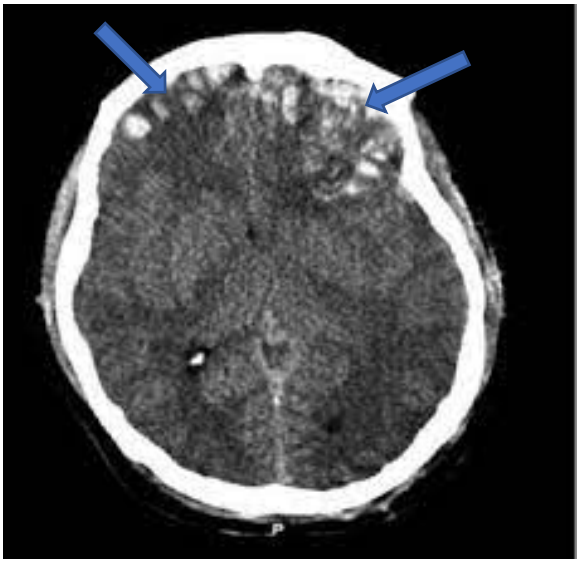

Figure 3: Closed head injury showing contused frontal lobes of the brain (arrows)

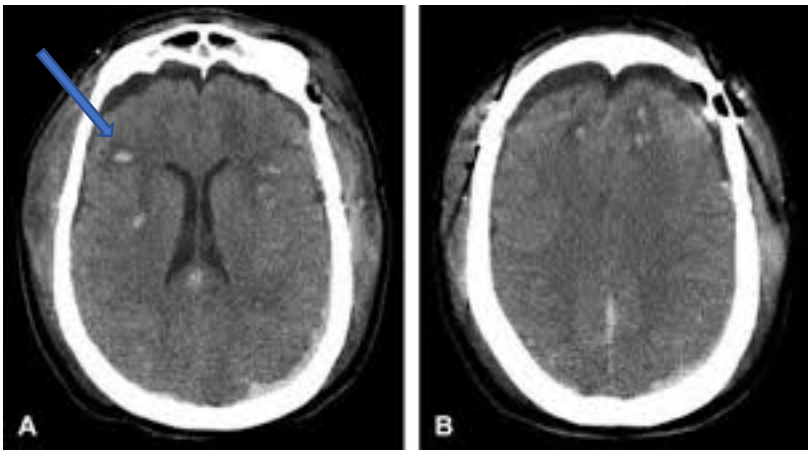

Figure 4: Diffuse axonal injury on head CT. White spots (arrow) in the brain indicate areas where white matter tracts have been torn and bleeding has occurred.

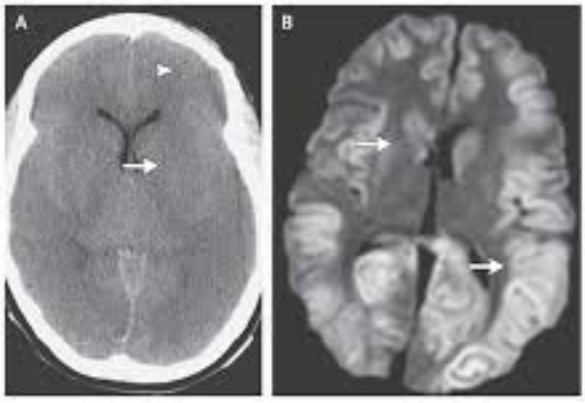

Figure 5: Anoxic brain injury on CT (left) and on MRI (right). Arrows point to brain injured by lack of oxygen (stroke)

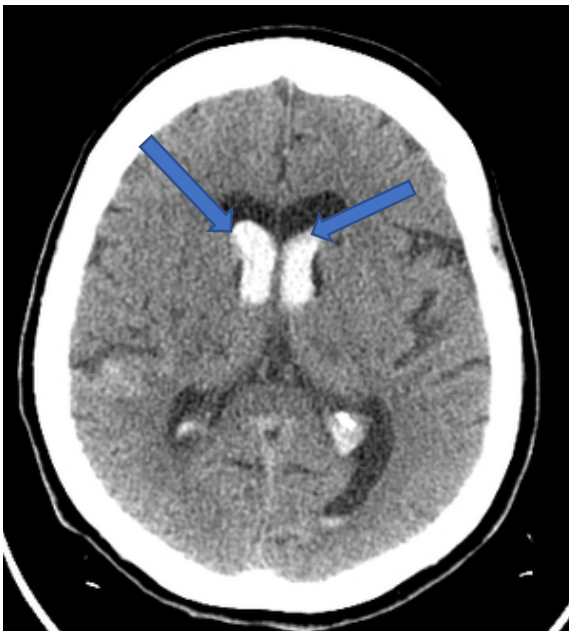

Figure 6: Intraventricular hemorrhage on CT scan (arrows)

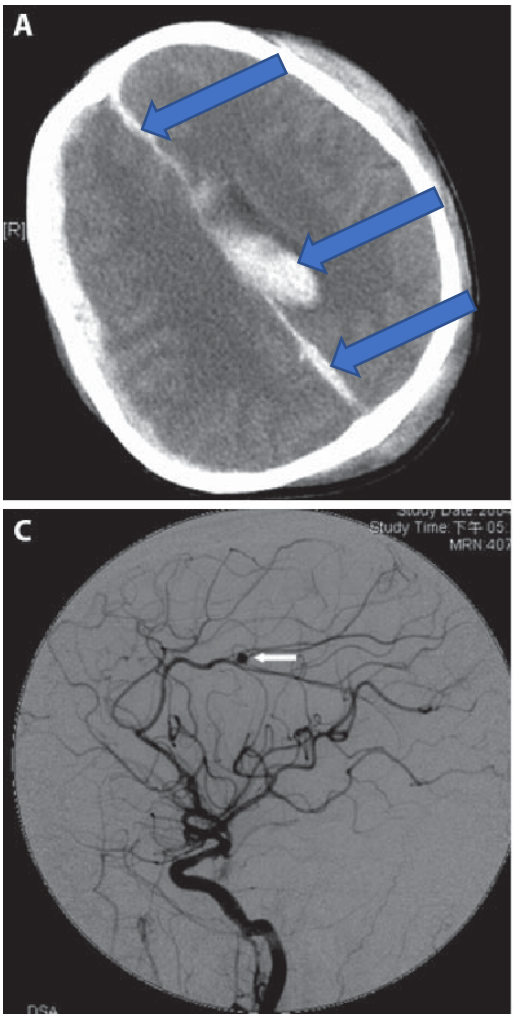

Figure 7: Traumatic aneurysm (white arrow bottom image) with hemorrhage (blue arrow top image)

Figure 8: Glasgow outcome score

Score	Functional status	Description
1	Good recovery	Returned to the original functional level and employment with no deficit
2	Moderate disability	Minor neurological deficit that does not interfere with daily functioning or work
3	Severe disability	Significant neurological deficit that interferes with daily activities or prevents return to employment
4	Persistent vegetative state	Coma or severe deficit rendering the patient totally dependent
5	Death	Self-explanatory

Figure 8: Glasgow coma score and FOUR Score