University of Basrah
Al-Zahraa Medical College

Ministry of Higher Education
and Scientific Research

PERIOPERATIVE BLOCK

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Approach to head injury

Block staff: (Neurosurgery).
Dr. Wisam Abdullah Jasim
Dr. Ali Mohammed Radhi
Dr. Mulook Gasib
Dr. Zainab Abdul-Muhsin Abood

References:
Essential neurosurgery

Objective:

1. Pathophysiology of head injury and identification of primary and secondary mechanism of head injury.
2. Identification of brain contusions and their types.
3. Intracranial hemorrhage and its types; identification on brain CT scan and management.
4. Concussion and DAI.
5. Skull fractures type and their management.
6. Glasgow coma scale.
7. Management of head trauma and criteria for indication for brain CT scan and admission to hospital.
8. Missile injuries; their categories and mechanisms.
9. Non-accidental head injury.

- **Head injuries** are a major cause of morbidity and mortality in the community. Trauma is the third most common cause of death in the United States, exceeded only by cardio cerebral vascular disease and cancer. **Trauma is the leading cause of death in youth and early middle age** and the death is often associated with major head trauma.
- Head injury contributes significantly to the outcome in over half of trauma-related deaths.

- There is a wide spectrum of head injury from mild concussion to severe brain injury resulting in death. The management of the patient following a head injury requires the identification of the pathological processes that have occurred.

Pathophysiology of head injury

The management of head injury has been based on the concept of **primary** and **secondary** brain injury.

The primary brain injury was defined as the irreversible pathology sustained at the time of the trauma, whereas the **secondary brain** injury has been considered the subsequent or progressive brain damage that occurs due to an evolving pathology following the injury.

It has been the general contention that the primary injury is irreversible, and management should be directed at preventing or treating secondary pathology (such as cerebral swelling, hydrocephalus and intracerebral hematoma).

Most head injuries result from **blunt trauma**, as distinct from a **penetrating** wound of the skull and brain caused by missiles or sharp objects.

The pathological processes involved in a head injury are:

- direct trauma
- cerebral contusion
- intracerebral shearing
- cerebral swelling (oedema)
- intracranial hemorrhage
- hydrocephalus.

Direct trauma. Although penetrating injuries produce most of their damage by direct trauma to the brain this is not the case with blunt injuries, in which the energy from the impact has a more widespread effect.

Cerebral contusion. This may occur locally under the position of the impact and called "coup contusion", although it usually occurs more severely at a distance from the area of impact as a result of a 'contre-coup' injury. As the brain is mobile within the cranial cavity the sudden acceleration/deceleration force will result in the opposite 'poles' of the brain being jammed against the cranial vault. A sudden blow to the back of the head will cause the temporal lobes to slide across the floor of the middle cranial fossa and the frontal lobes across the floor of the anterior cranial fossa, causing contusion on the undersurface of those lobes and to the temporal and frontal poles of the brain as they strike the ridge and frontal bones, respectively. Cerebral contusion consists of lacerated hemorrhagic brain, and a 'burst temporal lobe' may result when the temporal pole has been severely injured.

Shearing forces. Intracerebral shearing forces occur as a result of the differential brain movement following blunt trauma, frequently in conjunction with a contre-coup type of injury. The rotational acceleration following injury will cause shear forces that result in petechial hemorrhages (particularly in the upper brainstem, cerebrum and corpus callosum), and tearing of axons and myelin sheaths. The early pathological changes consist of retraction balls or microglial scars, and if the patient lives for a number of months before death then widespread degeneration of myelin will be apparent at postmortem and this is the mechanism of diffuse axonal injury (DAI).

Cerebral swelling. This occurs following trauma, either in a focal pattern around an intracerebral hematomas or diffusely throughout the cerebrum and/or cerebellum. The nature of the pathological processes are not clearly understood but involve a disturbance of vasomotor tone causing vasodilatation and cerebral oedema. In addition, cerebral contusion and petechial hemorrhages will contribute to the brain swelling.

Intracranial hemorrhage

Intracranial hemorrhage following trauma include:

1. **extradural**
2. **subdural**
3. **intracerebral.**

Extradural haematoma

Extradural hematomas are more likely to occur in the younger age group as the dura is able to strip more readily off the underlying bone.

In patients under 20 years of age, extradural hematomas account for about two-thirds of all traumatic intracranial hematomas, but represent less than 5% of hematomas in patients over the age of 50. Although an extradural hematoma may occur in the presence of a severe head injury and coexist with a severe primary brain injury, the important feature of an extradural hematoma is that it may occur when the injury to the underlying brain is either trivial or negligible.

Distribution of extradural haematomas

The most common sites for extradural hematoma are the **temporal region** followed by the frontal area. Posterior fossa and parasagittal extradural haematomas are relatively uncommon. In most cases the hemorrhage is from a torn middle meningeal artery or its branches but haematomas may also develop from haemorrhage from extradural veins, the superior sagittal sinus, transverse sinus or posterior meningeal artery, the last two being responsible for the posterior fossa extradural haematomas. A fracture overlies the hematoma in nearly all (95%) adults and most (75%) children.

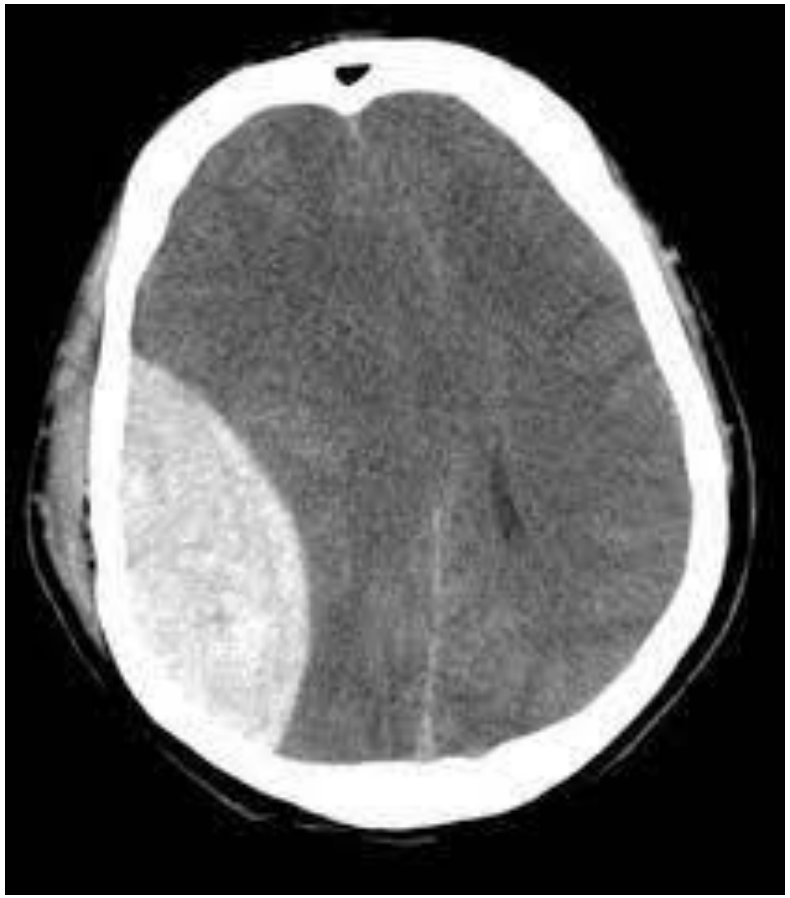
Clinical presentation of EDH

an extradural haematoma may occur as a result of a severe head injury and the haematoma will then become manifest as a further deterioration of the neurological state, particularly with lateralizing features involving a 3rd nerve palsy (dilatation of the pupil) and progressive hemiparesis.

More frequently the extradural hematoma occurs following a head injury that has resulted in only a transient loss of consciousness and in approximately one-quarter of cases there has been no initial loss of consciousness. In these patients the most important symptoms are:

- headache
- deteriorating conscious state
- focal neurological signs (dilating pupil, hemiparesis)
- change in vital signs (hypertension, bradycardia).

The “ lucent interval” is pathognomonic for EDH.



Brain Ct scan show **biconvex** hyperdense lesion consist with acute EDH.

Subdural haematoma

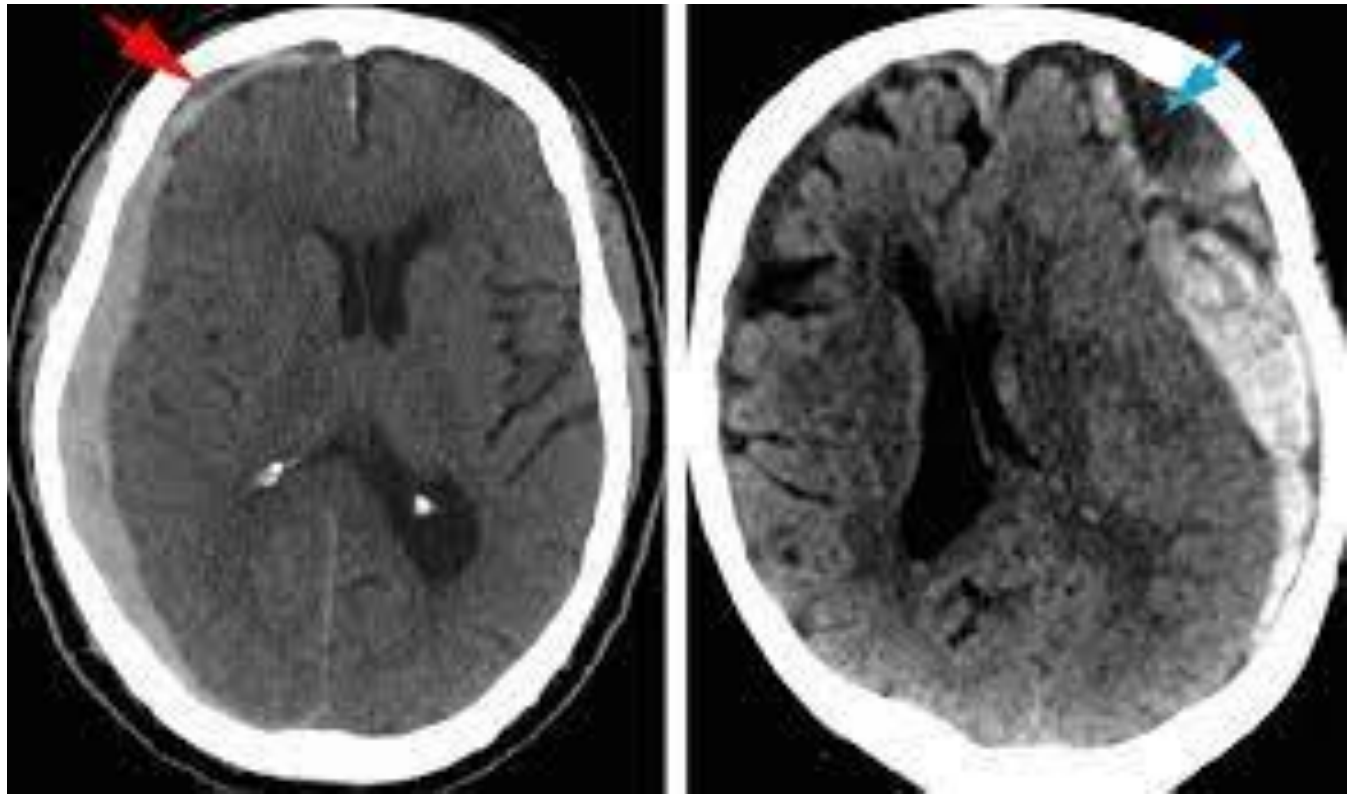
Subdural haematomas have been classified into acute, subacute and chronic, depending on the time they become clinically evident following injury:

- acute subdural haematoma—less than 3 days
- subacute subdural haematoma—4–21 days
- chronic subdural haematoma—more than 21 days.

The CT scan enables a further classification depending on the density of the haematoma relative to the adjacent brain. An acute subdural haematoma is hyperdense (white) and a chronic subdural haematoma is hypodense. Between the end of the 1st week and the 3rd week the subdural haematoma will be isodense with the adjacent brain.

Acute subdural haematoma

The acute subdural haematoma frequently results from severe trauma to the head and commonly from cortical lacerations. However, an acute subdural haematoma can result from a less severe trauma caused by rupture of a bridging vein or focal tear of a cortical artery, especially if the patient has been anticoagulated for other medical reasons (e.g. for atrial fibrillation). Cases of spontaneous acute subdural haematoma have been reported and in these patients it is essential to exclude a ruptured aneurysm or bleeding diathesis as a cause. Acute subdural haematomas are bilateral in approximately one-third of cases, in comparison with less than 3% of extradural haematomas. The features of deteriorating neurological state—decrease in conscious state and/or increase in lateralizing signs— should raise the possibility of a subdural haematoma. The CT scan will show the characteristic hyperdense haematoma, which is **concave(crescent)** towards the brain, Over 80% of patients with acute subdural haematomas have a fracture of either the cranial vault or the base of the skull, which may be evident on the bone 'windows' of the CT scan.A craniotomy is nearly always necessary to evacuate an acute subdural haematoma

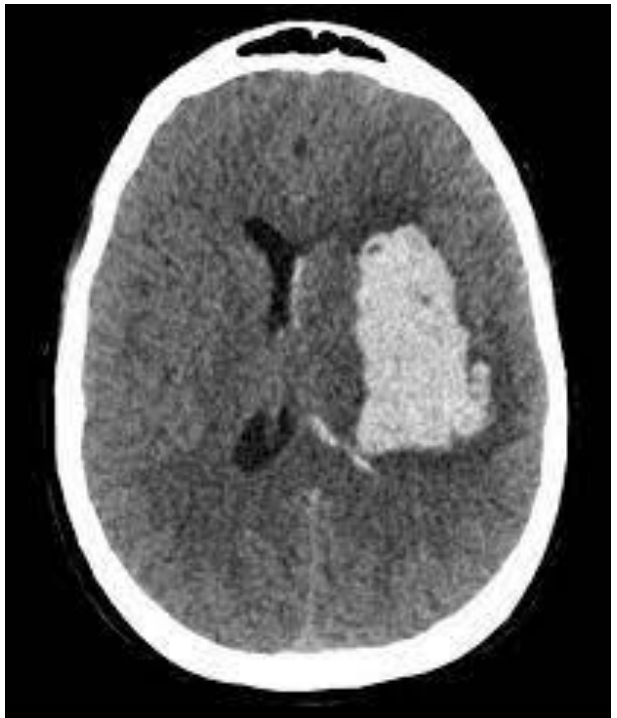


Acute SDH

Intracerebral haematoma

Traumatic intracerebral haematomas occur as a result of a penetrating injury (such as a missile injury) or a depressed skull fracture, or following a severe head injury. Intracerebral haematoma is frequently associated with subdural haematoma.

The size of the haematoma varies considerably and multiple haematomas are frequently seen onthe CT scan following a severe head injury.



ICH + IVH

The intracranial haematoma or cerebral swelling may cause the types of cerebral herniation. The medial surface of the hemisphere may be pushed under the falx (subfalcine), the uncus and hippocampal gyrus of the temporal lobe may herniate through the tentorium causing pressure on the 3rd nerve and midbrain, or there may be a caudal displacement of the brainstem and/or cerebellum, herniating into the foramen magnum.

Concussion

Concussion involves an instantaneous loss of consciousness as a result of trauma.

Associated injuries

Cranial nerves

The cranial nerves may be injured as a result of either direct trauma by the skull fracture, movement of the brain, or cerebral swelling.

- *The olfactory nerves*. These are the **most** commonly affected and this may be as a result of either a fracture through the anterior cranial fossa, directly the tracts, or tearing of the delicate nerve rootlets passing through the cribriform plate caused by the sudden brain movement, particularly from a **blow to the back** of the head.
- *The 8th nerve*. Damage to this nerve is often associated with a fracture of the petrous temporal bone. Deafness may be conductive, due to a hemotympanum, or sensorineural as a result injury to the inner ear or to the nerve itself. Vertigo and nystagmus are due to vestibular nerve or end-organ damage and usually resolve within a few months of the injury.
- *Facial paralysis*. This is usually associated with a fracture through the petrous temporal bone.
- *The 6th cranial nerve*. This has a long course from the brainstem to its entry into Dorello's canal and the nerve is easily damaged by torsion or herniation of the brain.

- *The 3rd nerve*. This may also be damaged by direct trauma or by brain herniation, the herniated uncus of the temporal lobe either impinging on the midbrain or directly stretching the nerve.
- *The optic nerve*. This is infrequently injured by direct trauma.

Skull fractures

Trauma may result in skull fractures which are **classified** as:

- **simple**—a linear fracture of the vault.
- **depressed**—when the bone fragments are depressed beneath the vault.
- **compound**—when there is a direct communication with the external environment. This may result from either a laceration over the fracture or a fracture of the base of the skull which will be compound because there will be a direct connection outside the vault, usually via the air sinuses.

Scalp lacerations

The extent of the scalp laceration does **not** necessarily indicate the degree of trauma to the underlying brain.

Other injuries

The most common associated injuries are to the chest, skeletal and cardiovascular systems.

Initial management of head injury

Immediate treatment involves a rapid restoration and maintenance of an adequate airway, ventilation, essential circulatory resuscitation **(AcBCDE).**

The key aspects in the management of patients following head injury involve:

- accurate clinical assessment of the neurological and other injuries
- determination of the pathological process involved.
- **the concept that a change in the neurological signs indicates a progression or change in the pathological processes.**

❖ Hypotension or hypoxia may aggravate the brain injury, and, if severe, will themselves cause brain damage.

Clinical assessment

It is fundamental to the management of the patient to know of **changes in the neurological condition as soon as possible**. It is essential to ascertain the **type** of accident that caused the head injury and the **time** injury occurred.

Neurological examination

An accurate neurological examination will help to determine the type and position of the pathological process and provide a baseline for comparison with subsequent examinations.

Although a full neurological examination should be undertaken, special emphasis should be given to the:

1. • conscious state with Glasgow coma scale (GCS).
2. • pupillary size and reaction
3. • focal neurological signs in the limbs.

the level of consciousness given as a numerical score—the sum of the three parameters of the Glasgow coma scale. **A score of 8 or less indicates a severe injury**.

Glasgow Coma Scale

Δ Eye opening

Spontaneous	– 4
To speech	– 3
To pain	– 2
None	– 1

Δ Verbal response

Oriented	– 5
Confused	– 4
Inappropriate words	– 3
Incomprehensible words	– 2
None	– 1

Δ Motor response

Obeys commands	– 6
Localises pain	– 5
Flexion to pain	– 4
Abnormal flexion	– 3
Extension to pain	– 2
None	– 1

- Total score : 15
  - Mild head injury : 13–15
  - Moderate head injury : 9–12
  - Severe head injury : < 8 (3–8)

med_life_easy

Pupillary size and reaction

Careful evaluation of the pupil size and response to light is essential at the initial clinical assessment and during further observation. Raised intracranial pressure causing temporal lobe herniation will cause compression of the 3rd nerve, resulting in pupillary dilatation, which nearly always occurs initially on the side of the raised pressure. The pupil will at first remain reactive to light but subsequently will become sluggish and then fail to respond to light at all. As the intracranial pressure increases this same process commences on the contralateral side. A traumatic mydriasis will also result from direct trauma to the eye, and the dilated pupil should not be confused with that due to a 3rd cranial nerve palsy.

- The oculocephalic response should only be performed after a cervical spine fracture has been excluded.

Focal neurological signs in the limbs

Neurological examination of the limbs will assess the tone, power and sensation.

- A hemiparesis will result from an injury of the corticospinal tract at any point from the motor cortex to the spinal cord.
- Following a severe brain injury the limbs may adopt an abnormal 'posturing' attitude:

1. The **decerebrate posture** consists of the upper limbs adducted and internally rotated against the trunk, extended at the elbow and flexed at the wrist and fingers, with the lower limbs adducted, extended at the hip and knee with the feet plantar flexed. There is a continuum of severity of brain injury with the decerebrate posturing response being partial and unilateral and occurring only as a result of a painful stimulus to severe continuing bilateral decerebrate rigidity. The posture probably results from an *upper brainstem injury*.

2. Less frequently, the upper limbs may be flexed, probably due to the injury predominantly involving the cerebral white matter and basal ganglia—corresponding to a **posture of decortication**.

Particular attention must be given to the **patient's ventilation, blood pressure and pulse**. At all times it is essential that care is taken to ensure the patient's ventilation is adequate. Respiratory problems may result either as a direct manifestation of the severity of the head injury or due to an associated chest injury. **Cheyne–Stokes breathing** is due to either intrinsic brainstem damage or raised intracranial pressure causing pressure and distortion of the brainstem. **Bradycardia and hypertension**, the **Cushing response**, are also both indicative of brainstem compression due to raised intracranial pressure.

Pyrexia frequently occurs following a head injury. A temperature lasting for more than 2 days is usually due to traumatic subarachnoid haemorrhage or may occur in patients with a severe brainstem injury or hypothalamic injury.

Radiological assessment

Following the clinical evaluation radiological assessment will be essential unless the injury has been minor. The **CT scan** should be performed where:

1. there is loss of consciousness (post-traumatic amnesia) of greater than 10 minutes
2. the patient is persistently drowsy or has a more seriously depressed conscious state
3. there is persisting nausea or vomiting
4. there are lateralizing neurological signs
5. there is neurological or focal deterioration
6. there is skull fracture
7. there is CSF rhinorrhea
8. there are associated injuries which will entail prolonged ventilation so that ongoing neurological assessment is difficult.
9. Patient on antiplatelet or anticoagulant.
10. History of seizure or epilepsy.
11. Extreme of age, infant or older than 65y.

The CT scan will clearly show the presence of intracerebral or extracerebral haematoma, as well as cerebral contusion, oedema and infarction.
Small ‘slit’ ventricles and absence of the basal cisterns will indicate generalized brain swelling.
Cerebral angiography is indicated if a caroticocavernous fistula is suspected by the presence of a bruit over the orbit or by pulsating proptosis.
Carotid or vertebral angiography will be necessary if arterial dissection is considered a possibility.
Full radiological assessment of the cervical spine utilizing plain X-ray and CT scan is essential in patients who have sustained a significant head injury, particularly if there are associated facial injuries.

Further management of head injury

Following the clinical and radiological assessment the subsequent management will depend on the intracranial pathology and the extent of any neurological injury.

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Minor head injury

The patient would be assessed as described above. Any patient who has suffered a head injury must be observed for at least 4 hours. The following are the minimal criteria for obligatory CT scan and admission to hospital:

- loss of consciousness (post-traumatic amnesia) of greater than 10 minutes
- persistent drowsiness
- focal neurological deficits
- skull fracture
- persisting nausea or vomiting after 4 hours' observation.
- intracranial pathology noted on a CT scan
- if the patient does not have adequate care at home.

The further management of these patients will be careful observation; the neurological observations should be recorded on a chart displaying the features of the Glasgow coma scale. If there has been a period of significant loss of consciousness, or if the patient is drowsy, then the following measures should be instituted to minimize the development of cerebral swelling:

- elevation of the head of the bed 30-40 degree
- mild fluid restriction to 2–2.5 l/day in an adult.
- Mannitol 20% over 20 min.

Should the patient's neurological state deteriorate an immediate CT scan is essential to re-evaluate the intracranial pathology; further treatment will depend on the outcome.

Severe head injury

The management of a patient following a severe head injury depends on the patient's neurological state and the intracranial pathology resulting from the trauma. In general, the following apply.

1 The patient has a clinical assessment and CT scan as described previously.

2 If the CT scan shows an intracranial haematoma causing shift of the underlying brain structures then this should be evacuated immediately.

3 If there is no surgical lesion, or following the operation, the management consists of(with ICU admission):

(a) Careful observation using a chart with the Glasgow coma scale.

(b) Measures to decrease brain swelling; these include:

(i) careful management of the airway to ensure adequate oxygenation and ventilation. Hypercapnia will cause cerebral vasodilatation and so exacerbate brain swelling.

(ii) elevation of the head of the bed 30-40°

(iii) fluid and electrolyte balance.

Maintenance of isotonic fluid requirements, avoiding dextrose solutions and following resuscitation should be administered until the patient is able to commence nasogastric feeding. Blood loss from other injuries should be replaced with colloid or blood and not with crystalloid solutions. Care should be taken to avoid overhydration, as this will increase cerebral oedema. Avoid hypo or hypernatremia.

c) Temperature control

d) Nutritione

e) Routine care of the unconscious patient including bowel, bladder and pressure care.

f) Antiepileptic medication.

g)More aggressive methods to control intracranial pressure are advisable if:

- the patient's neurological state continues to deteriorate and the CT scan shows evidence of cerebral swelling without an intracranial haematoma.
- there is a posturing (decerebrate) response to stimuli.
- the Glasgow coma score is less than 8.

In these patients an intracranial pressure monitor should be inserted to assess the intracranial pressure as accurately as possible.

The techniques used to control intracranial pressure are as follows:

- Controlled ventilation, maintaining *P*aCO2 at 30–35 mmHg. Reduction of the *P*aCO2 will reduce cerebral vasodilatation and consequently decrease the intracranial pressure.
- If the pressure remains elevated despite hyperventilation CSF can be drained from a ventricular catheter if this has been inserted.
- Diuretic therapy utilizing intermittent administration of mannitol or frusemide can be used if the preceding techniques have failed to control the intracranial pressure. Mannitol is an osmotic diuretic and may also exert its effect by increasing serum osmolality and drawing water out of the brain. The usual dose is 0.5–1.0 g/kg. The serum osmolality should not exceed 320 mosmol/kg.
- Hypertonic saline 3%.
- Barbiturate therapy can be considered if the intracranial pressure is resistant to treatment with the above techniques.
- Decompressive craniotomy.

Management of associated conditions

Scalp injury

Scalp injuries may include:

- abrasion
- confusion
- laceration
- subgaleal haematoma.

A large scalp laceration may result in considerable blood loss. When the patient arrives in the emergency department ‘spurting’ arteries should be controlled with haemostatic clips or suturing prior to the application of a sterile bandage to the head.

The extent of the soft tissue scalp injury may not reflect the severity of the underlying brain injury.

The hair should be shaved widely around the wound, which should be meticulously cleaned and debrided. The closure should be performed in two layers if possible, with careful apposition of the galea prior to closing the skin.

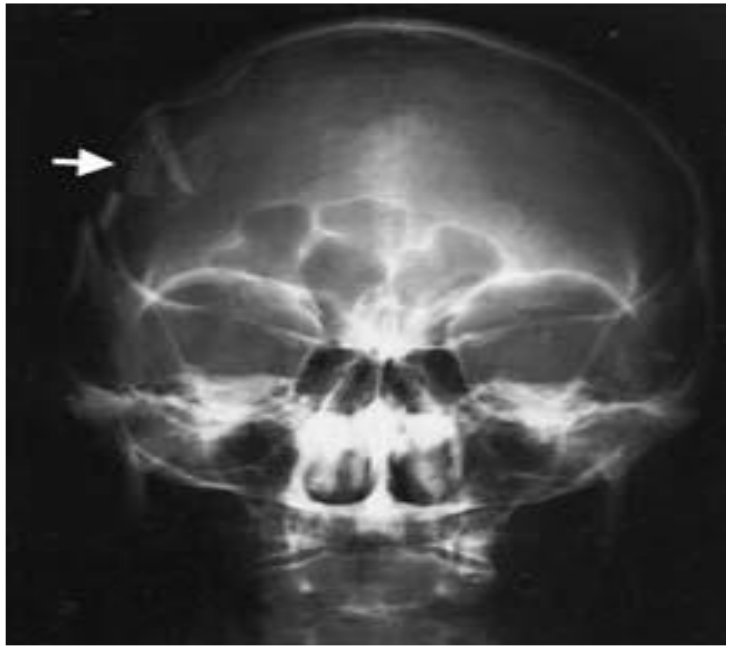
Skull fractures

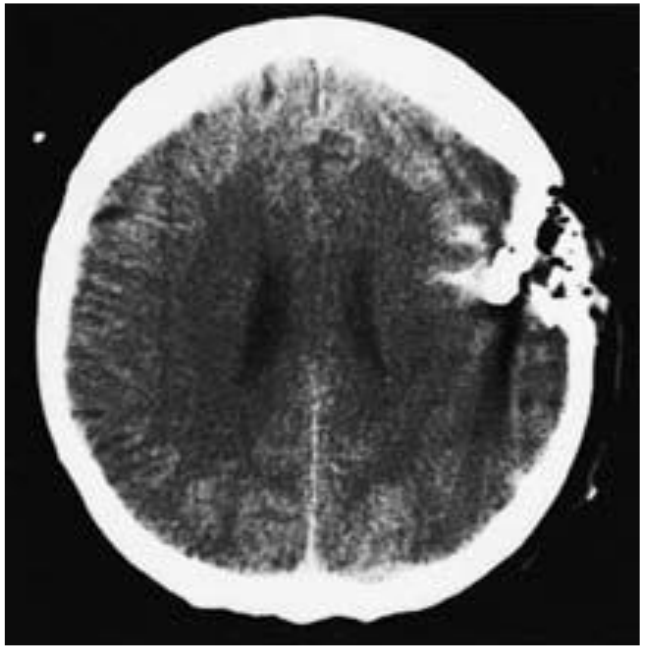
Simple linear fracture. There is no specific management for a simple skull fracture that is undisplaced without an overlying skin injury. However, the presence of a fracture is an indication that the trauma was not trivial and it should provide a warning that a haematoma may develop beneath the fracture. The patient should be admitted for observation and CT scan performed.

Compound fracture. A skull fracture may be compound either because of an overlying scalp laceration or if it involves an air sinus. The scalp wound should be debrided and closed as described above. A short course of prophylactic antibiotics should be administered to reduce the risk of infection.

Depressed skull fracture .(A skull vault fracture is considered to be significantly depressed if the inner table fragments are depressed by at least the thickness of the skull. depressed fracture caused by a non-missile injury usually causes only focal brain damage, so that many patients never lose consciousness. If the underlying injured brain is an eloquent area the patient will exhibit focal neurological signs. Haemorrhage from the bony edges, the dura or underlying brain trauma may resultin an intracranial haematoma

- If the depressed skull fracture is compound, prophylactic antibiotics, antiepileptic and tetanus prophylaxis should be administered and surgery.
- If the depressed fracture is closed there is no urgency in elevating the bone fragments, provided there is no underlying intracranial complication.





In general, the depressed fragments should be elevated if:

1. **there is significant brain compression**
2. **the fracture is compound**
3. **there are cosmetic considerations such as a frontal fracture in a young child.**

Skull base fracture

Anterior fossa fractures may open into the frontal, sphenoid or ethmoid sinuses, often running across the cribriform plate. They present with:

- subconjunctival haemorrhages extending to the posterior limits of the sclera. Periorbital or 'racoon eyes' indicate subgalea haemorrhage and not necessarily a base of skull fracture
- anosmia
- nasal tip paraesthesiae due to anterior ethmoidal nerve injury.
- CSF rhinorrhea.

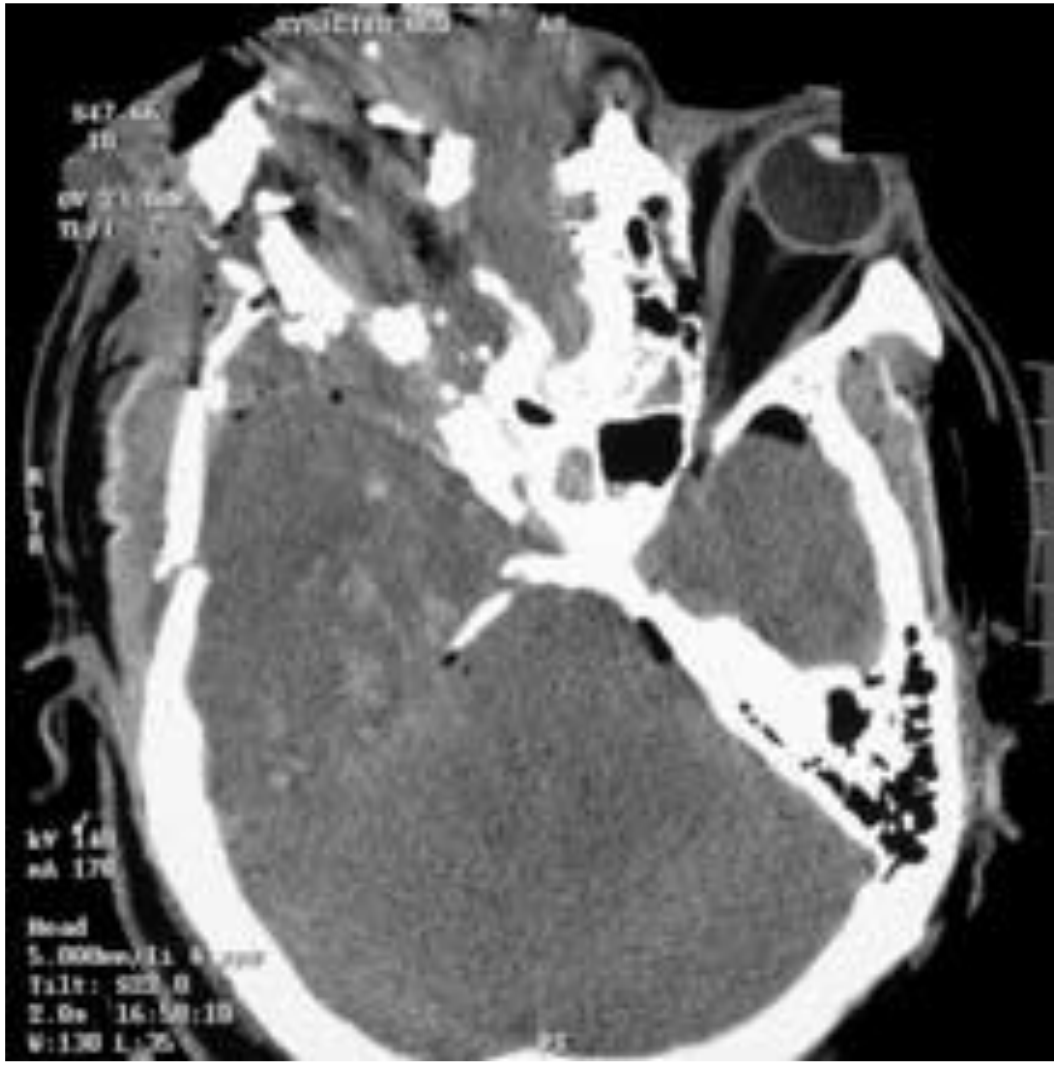
Middle fossa fractures involving the petrous temporal bone present with:

- CSF otorrhea (or rhinorrhea) via the eustachian tube
- deafness due to 8th nerve injury or ossicular disruption
- hemotympanum
- Battle's sign—bruising over mastoid bone
- 7th nerve palsy—often delayed.

❖ The major concern of a dural fistula is the risk of intracranial infection, particularly bacterial meningitis.

Treatment for CSF rhinorrhea or otorrhea is conservative initially with head elevation, bed rest, avoid measures that increase ICP such as coughing or sneezing and the use of antibiotics is controversially. there is general agreement that surgery should be performed if:

- CSF leakage continues for more than 5 days, indicating the fistula is not trivial
- there is another indication for surgery such as hematoma.
- there has been an episode of meningitis.

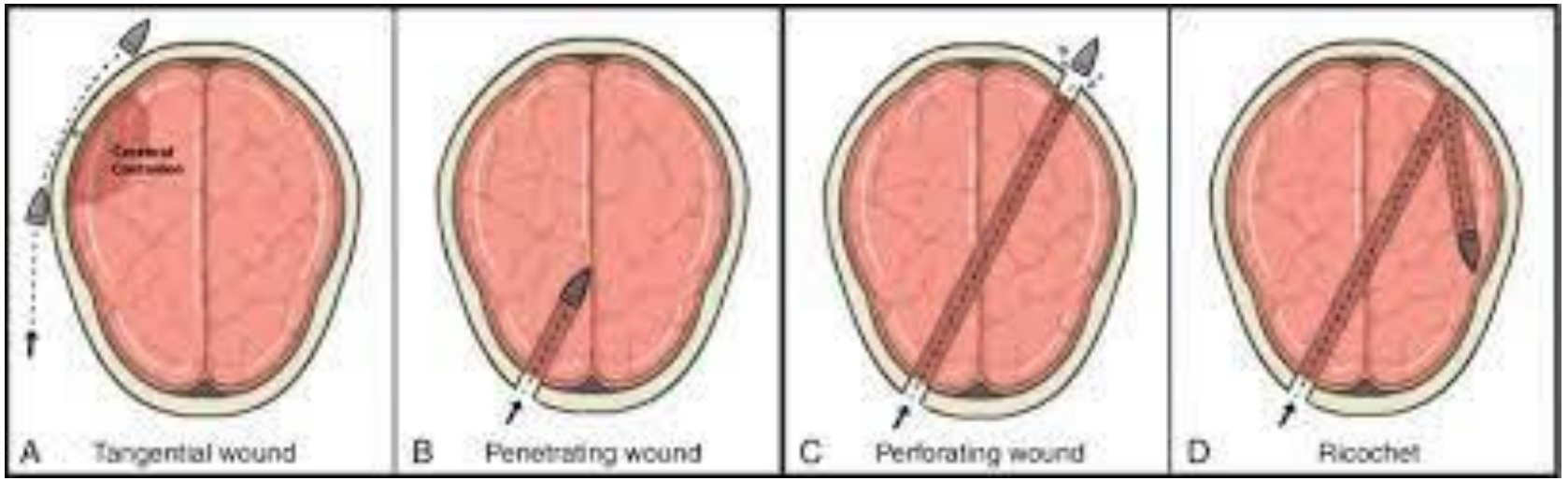


CT showing severe trauma resulting in multiple fractures, disruption of the orbit, intracranial contusions involving the right temporal lobe and intracranial air(ariocele).

Missile injuries

In general the cranial injury is directly proportional to the velocity of the missile. The ‘high-velocity’ injury is defined as resulting from a missile travelling faster than the speed of sound (1050 ft/s), and modern rifle bullets have a muzzle velocity greater than 3000 ft/s. There are **three** categories of missile injury:

1. • **tangential**—the missile does not enter the cranium but causes a depressed skull fracture, lacerating the scalp with an underlying cortical contusion, laceration or hematomas.
2. • **penetrating**—the missile enters the cranium resulting in the deposition of metal, bone fragments and debris within the brain
3. • **through-and-through**—the missile enters and exits the cranium, frequently creating more than one tract due to fragmentation.



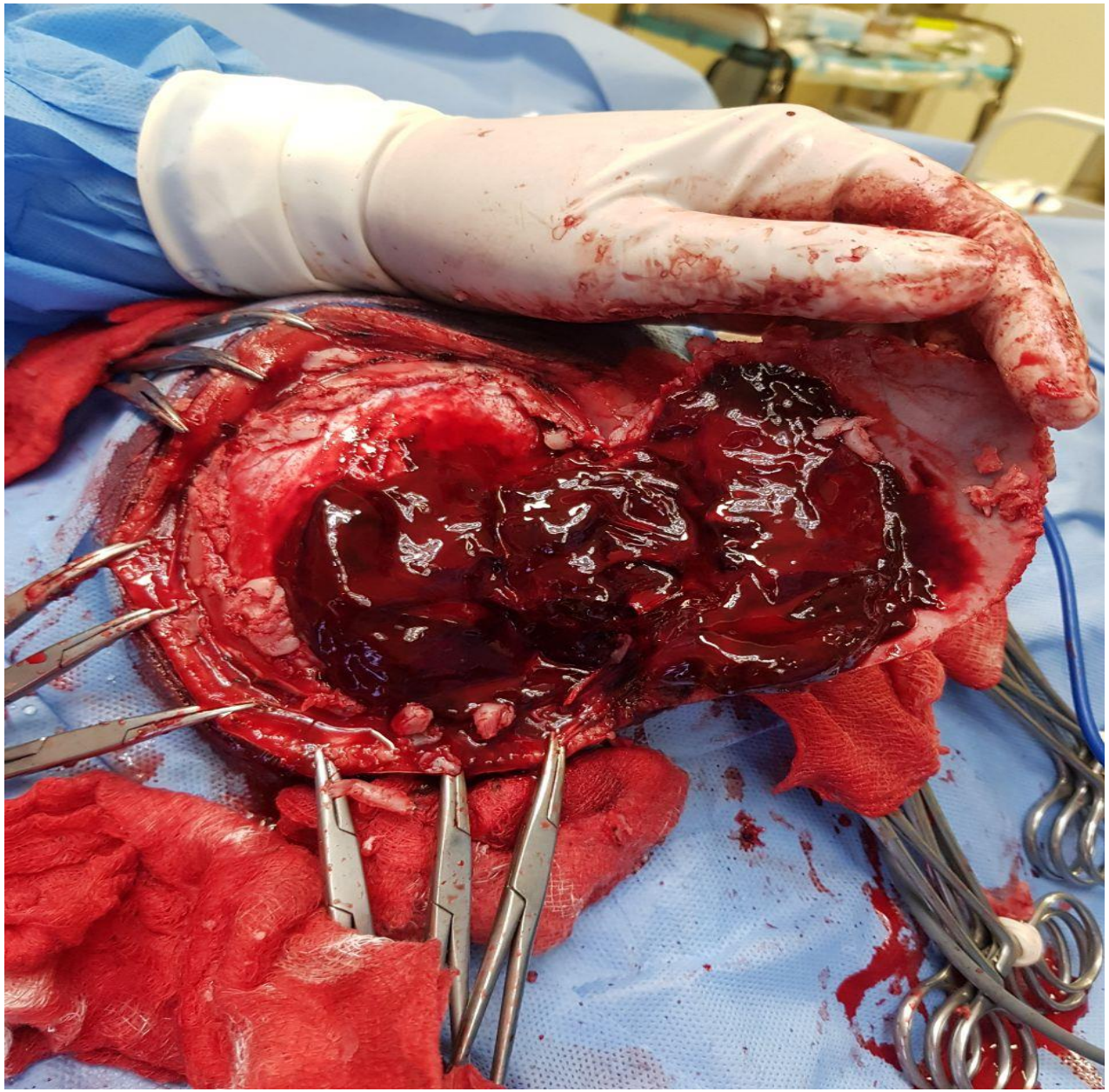
The missile causes cerebral damage via **three** mechanisms:

1. • **mechanical laceration** of brain tissue during transit
2. • the **shock wave** promulgated ahead of the missile
3. • **cavitation** in the wake of the missile.

Non-accidental head injury

The infantile chronic subdural haematoma or effusion is a distinct clinical entity. Birth trauma is a frequent cause but in many cases a past history is inadequate to establish the nature of the injury with certainty. **Chronic subdural haematomas occur in approximately 20% of battered children.**

The violent shaking of the immature brain might be sufficient to rupture bridging veins or cause shearing at the grey/white interface without evidence of external trauma. If an inadequate history is provided in such a setting, it is important to screen for a coagulopathy, **examine the fundi for retinal haemorrhages**, arrange a **skeletal survey** and when appropriate involve a pediatrician and social services. MRI now plays an important role in determining the chronicity of cerebral injuries in infants. Collections of different chronicity, or in unusual locations, should alert the physician to the possibility of non-accidental



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