CNS TRAUMA

Head injury

It is injury to the brain and its coverings (meninges, skull bones and scalp). ***Mechanism of injury*** Head injury can be results primarily from two phenomena; contact forces and inertial forces. Contact phenomena appear when the head strikes or is struck by an object, which results in local compressive strains in underlying skull and brain which lead to skull fractures, coup contusion and some of intra or extra axial hematomas. Inertial forces are generated by head motions that occur during the traumatic events which may also accompany contact phenomena. These motions are described by acceleration or deceleration of the head as it is set into motion or is stopped from moving. Concussion, diffuse axonal injury without hematoma, most subdural hematomas and counter coup contusions are produced as a result of acceleration of the head.

Scalp injuries

In cross section, the scalp contains five distinct layers. The first 3 layers (Skin, subCutaneous fascia and galea Apneurosis) adhere firmly to one another and may be considered as single unit. Beneath the galea is a layer of Loose areolar tissue which is easily separated and it is the plane in which scalp flaps are raised and scalp avulsions occur. The final layer is the Pericranium (periosteal layer of the outer table of the skull), which adheres firmly to the skull, particularly at cranial sutures.

Vascular anatomy. Scalp vessels are located in the deep portion of the subcutaneous layer just above the galea.

Nerve supply. Anteriorly the major cutaneous nerves are the supra-orbital and supra-trochlear nerves (branches of ophthalmic division of the 5th. cranial nerve). These accompanied the arteries of the same name and supply the scalp as far posteriorly as a line drawn across the top of the head from one ear to the other. Posterior to this line, the scalp is supplied by greater and lesser occipital nerves (branches of dorsal rami of C2 through C4). ***Lymphatics:*** These parallel the neurovascular supply. The lower portion of the forehead (frontal and temporal areas) drains through the face into submandibular nodes. The upper frontal and parietal areas drain into the superficial parotid groups of nodes. The occipital portion of the scalp drains into the retro-auricular nodes.

Classification of scalp injuries

(1) Scalp wounds Because of the tension in the galea, wounds that include this layer tend to gape considerably. The tendency of the galea to retract offers a form of protection. Since the scalp blood vessels are located in the subcutaneous fascia, they do not contract. If the galea is lacerated, the contraction of this layer will cause retraction of the vessels also. Thus superficial wounds with the galea intact may bleed more profusely than deep wounds with the galea cut. All **examination** of scalp wounds should be performed using sterile technique. Many times an underlying skull fracture can be palpated in the depth of a scalp wound. Most simple wounds can be examined and closed under local anesthesia, but in case of extensive wounds or in very young children, it is necessary to use general anesthesia in the operating room. **Treatment;** because of the rich blood supply of the scalp, extensive debridement is both unnecessary and unwise, but all obviously devitalized tissue should be removed and the wound should be irrigated with normal saline wash. In the absence of an underlying skull fracture, the best technique for stopping bleeding in scalp wound is circumferential pressure of the surrounding scalp against the skull. In very large and gaping wounds, another useful technique is to place clamps (forceps) on the galea and draw it back over the dermis (skin and subcutaneous fascia), there by compressing the scalp vessels. An area of 2-3 cm. around the wound should be shaved. Scalp wounds should be closed in two layers. The first layer approximates the galea and should be closed with absorbable suture (like catgut). The galea is the strongest layer of the scalp and should always be repaired if possible. The second layer is the skin which should be closed with non-absorbable suture (like silk); interrupted or continuous running sutures may be used. If the resulting gap, after removing the devitalized tissue, is large to be closed without tension, undermining of the subgaleal space (manual separation of the galea with the first two layers from the pericranium) is useful in order to a chief wound closure without tension. Following closure, a compression dressing is helpful, particularly if there had been undermining of the subgaleal space. Sutures should be remain in place for 7 days in case of two layer scalp wound closure, but should be extended to 10 days in case of single layer closure (suturing of the skin layer only).

(2) Scalp avulsions This is usually occur in the loose areolar tissue between the galea and pericranium (traumatic separation of the galea with the first two layers from the pericranium) Most scalp avulsions occur when a portion of hair is pulled at a tangential angle. Perpendicular pulling may results in avulsion of hair only.

(3) Scalp hematomas Caput succedaneum is a localized swelling of the scalp, typically located in the midline posterior parietal area over the part of the cranium that leads the way down the birth canal during delivery. The swelling is intimately within the superficial layers of the scalp and usually consist of tissue fluid (edema) rather than being a true hematoma. These lesions are not associated with skull injury and require no therapy.
13

(a) Cephalhematoma (subperiosteal hematoma); It is the result of bleeding between the periosteum and the skull and is found almost exclusively in the newborn. The presentation is of a localized, initially hard mass. The scalp can be moved over the mass, which itself fixed. Because the blood has collected under the pericranium and the pericranium is attached at the sutures, the mass is limited to one cranial bone and does not cross the suture lines. The initial therapy is to leave the lesion alone. It should not be tapped because of the risk of introducing infection. The hematoma is gradually reabsorbed over several weeks. If the lesion has not been resolved by 6 weeks, plain X-ray will show whether it is calcifying. If significant lump is present and calcifying, it should be removed surgically._____
Caput succedaneum
Subgaleal hemorrhage
Epidnral hemorrhage
Periosteum[1] Skull Dura'
(b) Subgaleal hematoma; It
results from bleeding into the loose connective tissue layer between the galea and pericranium and it can be spread across half the head or even all around the scalp. It is very common after head injury in children. It should not be tapped because of high risk of infection. The treatment is to reassure the parents that the mass will resolved.

Skull fractures

Skull fractures are classified in three ways according to:
(a) Pattern; (1) Linear (2) Diastatic (3) Depressed (4) Comminuted
(b) Location; *(I)* Convexity (2) Base
(c) Skin integrity; (1) opened (compound) (2) Closed
Linear skull fracture: Is a single fracture line in the skull, which passes through the entire thickness of the skull. This requires no stabilization or exploration. Surgery is performed for the associated intracranial lesions (like hematomas).
Diastatic skull fracture: Is a separation of a cranial suture line. The management is the same as for linear skull fracture.
Depressed skull fracture: The fracture is considered depressed if the outer table of one or more of the fracture edges lies below the normal anatomic level of the inner table as determined by the surrounding intact skull. The indications of surgery include;
(1) Cosmetic reasons if the fracture is closed.
(2) Opened fracture which represents a neurosurgical emergency because of the risk of bacterial infection of the cranial cavity. The operation is performed within 24 hours.
(3) There is an underlying surgically indicated intracranial lesion (like hematoma).
Although elevation of bone fragments occasionally improves a focal neurological deficit originating in the cortex directly under depressed bone fracture (presumably by increasing local blood flow), this procedure usually produces no neurological changes, implying that the impact itself produced the cortical damage. The incidence of epilepsy after depressed skull fracture is apparently determined by cortical damage at the time of the impact; since it is not altered by elevation of the fragments. Thus the treatment of depressed skull fracture is based on,
not on initiating neurological recovery or preventing epilepsy; but, rather on correcting cosmetic .deformity as well as preventing infection in open fracture
The process of opening of the cranial cavity by removing parts of the skull bones is known as Craniectomy, while the process of opening of the cranial cavity by creation of reflectable bone flap is known as Craniotomy. The process of closing skull bone defect either by bone or by synthetic material is known as Cranioplasty. Therefore; the surgery for depressed skull fracture as it includes removing or removing and replacement of bone fragments (or synthetic material) is either Craniectomy or .Craniectomy with Cranioplasty
Craniotomy bone flap was created by connecting multiple Burr-holes by using special saw
Comminuted skull fracture: A fracture is called comminuted when more than one linear fracture is present. If the overlying skin is closed; the management is the same as for linear skull fracture, but if it .is opened; the management is the same as for depressed skull fracture
Skull base Fracture: This is usually linear skull fracture. The dura is easily torn which place the subarachnoid space in direct contact with the paranasal sinuses or middle ear structures, providing pathway for infection through CSF leak. Clinical signs of skull base fractures includes; bilateral periorbital ecchymosis (Raccon eyes), anosmia or CSF rhinorrhea for anterior skull base fractures, as

well as hemotympanum (bulging of tympanic membrane due to blood collection), blood in external auditory canal, 7th & 8th cranial nerves palsies, ecchymosis over mastoids (Battle's sign) or CSF otorrhea / otorrhagia (if associated with rupture tympanic membrane) for middle cranial fossa fractures. Management is usually depending on the presence of CSF leak. A patient with basal skull fracture but with no CSF leak is observed for 2-3 days. If he developed CSF leak (or if he had initially CSF leak); then the observation is extend to 7 days. The patient is usually observed by placing a loose-fitting sterile gauze pad over the ear or the nose; the pad is changed every nursing shift and saved as an indicator of the amount of CSF leak. Most traumatic CSF leaks resolved spontaneously within the fist week. If the leak persists beyond 7 days, lumbar taps (punctures) are performed daily for 3 days (removing 30-50 ml. of CSF each time). If lumbar taps fail to stop the leak, then surgery is indicated. The usage of antibiotics is controversy. High does steroid is indicated in presence of facial nerve .paralysis
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Intracranial Lesions

These may be classified into Focal and Diffuse lesions;
Diffuse lesion: This results from acceleration-deceleration injury to the brain, and it is of two types; **(a) Concussion** It is condition characterized clinically by transient traumatic lose of consciousness for less than 6 hours associated with some degrees of post-traumatic amnesia (amnesia for events related to the injury and afterward). The inertial force causes deeper structures within the brain to deform resulting in wide spread disruption of brain function but most of the strain is insufficient to cause structural damage, **(b) Diffuse axonal injury** It is condition characterized clinically by traumatic lose of consciousness from the time of injury that continues beyond 6 hours. There are microscopic damage scattered throughout the brain including focal axonal changes that lead to focal impairment of axoplasmic transport and disconnection. The brain CT scan is usually negative in diffuse brain lesions that were not associated with focal lesions.
Focal lesion: This include; contusions and intracranial hematomas. Focal lesions that occur underlying the site of impact called coup lesion and those located far from the site of impact called Countercoup lesions.
I - Contusions consist of heterogeneous areas of hemorrhage, brain necrosis and infarction. They appear as salt-and-pepper lesions in brain CT scan. The commonest sites for contusions are frontal and temporal poles. Contusions can over period of hours or days evolve into intra-parenchymal hematomas. The treatment is conservative unless they are complicated by surgically indicated hematoma.
II - Intracranial hematomas are classified according to anatomy of meninges into;
(a) Extradural hematoma: is collection of blood between the inner table of the skull and the dura. It appears as biconvex lesion in brain CT scan. These hematomas results from injury to; (1) meningeal vessels (like middle meningeal artery or vein) (2) Deploic veins (as in Skull fractures) (3) Dural venous sinuses. More than 50% of this hematoma arises from middle meningeal artery injury.
(b) Subdural hematoma. is collection of blood between the dura and arachnoid membrane (subdural space). It appears as crescentic lesion in brain CT scan. This hematoma results from tearing of bridging veins that traverse the space between the cortical surface and venous sinuses. It is also results from injury to the surface of the brain with bleeding from cortical vessels into the subdural space.
(c) Intra-parenchymal hematoma: is collection of blood within the brain parenchyma. It appears as mass lesion in brain CT scan. This results from damage of intra-parenchymal blood vessels or as a complication of contusion.
Intracranial hematomas are considered **acute** if they are present within 3 days of the injury (appear hyperdense lesion in brain CT scan), **subacute** which become manifest 3 days to 3 weeks of the injury (appear isodense lesion in brain CT scan) and **chronic** which do not produce symptoms until 3 weeks from the injury (appear hypodense lesion in brain CT scan).
The indication for surgery in intracranial hematomas is largely depending on the clinical condition of the patient. The surgery either **Craniectomy** (with or without cranioplasty) or **Craniotomy;** and these are followed by evacuation of the hematoma. Chronic hematoma sometimes can be evacuated through single **Burr-hole** (a hole in the cranium of 1 cm. in diameter).

All focal brain lesions could be coup or countercoup except extradural hematoma which is only of coup

Lt. fronto-temporal cerebral contusions	Rt. fronto-temporal acute intra-cerebral (intra-parenchymal) hematoma	Lt. fronto-tempero-occipital acute subdural hematoma	Rt. fronto-parietal acute extradural hematoma

Clinical features of the focal brain lesions:

(A) Conscious level: Patients with these lesions follow one of the following five clinical courses; (1) Conscious throughout (2) Unconscious throughout (3) Initially conscious and subsequently unconscious (4) Initially unconscious and subsequently conscious (5) Initially unconscious followed by lucid interval and then unconscious again. Lucid interval is a period of regaining consciousness after unconscious period and it is proceeds the subsequent lose of consciousness. The initial transient loses of consciousness results from concussion and is followed by a return of consciousness until the growing of intracranial hematoms or development of focal cerebral edema.

(B) Clinical features of rise intracranial pressure

(C) Clinical features of brain herniation (uncal herniation is the commonest type)

Gunshot (missile) wounds of the head

These are classified into:

(1) Tangential Wound: The bullet grazes the skull but does not penetrate it. It may travel in the subgaleal space and exit through or remain in the scalp. In addition to scalp wounds, the bullet can cause varying degrees of damage to the skull (fractures), meningese (hematomas) and underlying cortex (contusions). The effect on the cortex is due to sonic pressure waves of the bullet.

(2) **Penetrating wound:** The skull is penetrated at one side, but the bullet does not have enough energy to penetrate the entire brain or the skull on the opposite side. Sometimes after hitting the inner table of the opposite side of the skull it will ricochet (reflected) with re-entry of the bullet into the brain tissue causing further damage.

(3) **Perforating wound:** The bullet goes through the entire head (entrance and exit). The wound entry is generally smaller than the exit wound.

For penetrating and perforating wounds there are also varying degrees of injuries to all elements of the head. The treatment of gunshot wounds depends on the head elements that were injured (scalp, skull or brain).

Perforating wound	Penetrating wound	Tangential wound

Evaluation of patient with head injury

Head injury can be classified according to disturbed conscious level into;

(1) Mild head injury (GCS= 14 or 15)

(2) Moderate head injury (GCS= 9-13)

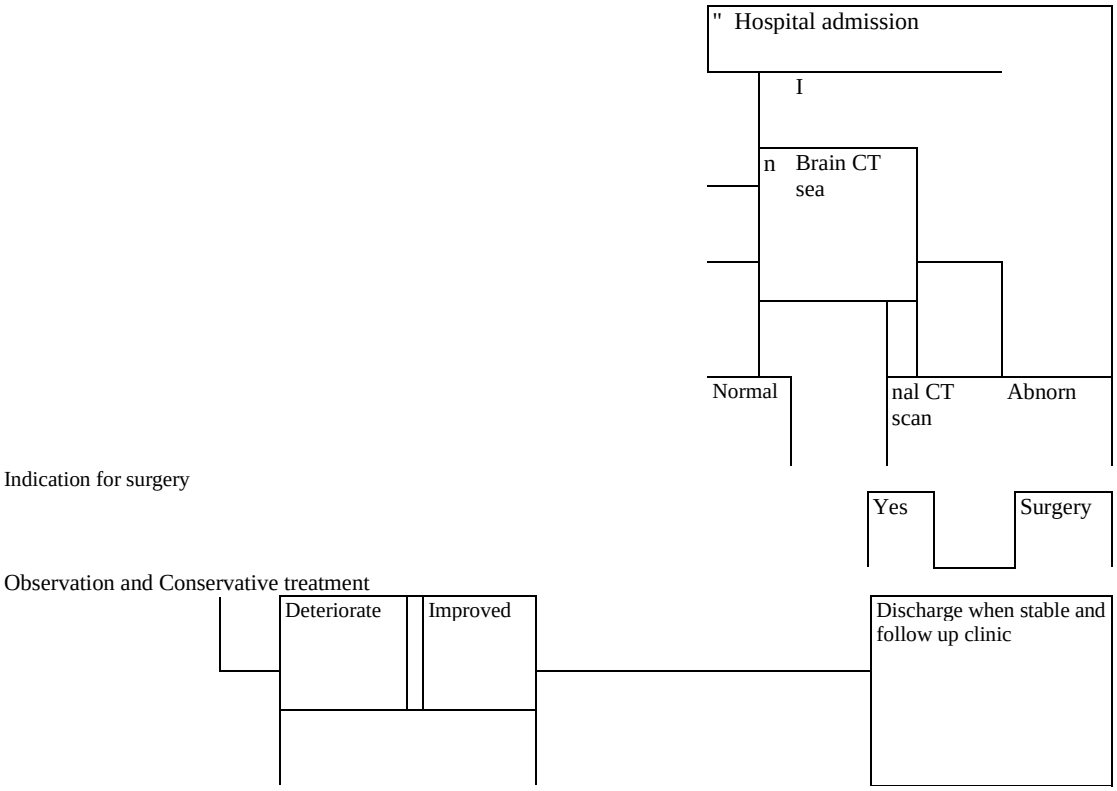
(3) Severe head injury (GCS= 3-8)

Brain CT scan is **indicated** if GCS less than 15 or presence of one or more of the following risk factors: (1) History of lose of consciousness (2) Post-traumatic amnesia (3) Clinical signs of skull fractures (scalp swelling and/or bruising at sit of trauma) (4) Headache (5) Vomiting (6) Focal neurological deficit (7) Epilepsy (8) Coagulation disorder (9) Age < 2 years or > 60 years (10) RTA (Road Traffic Accidence)

Managements of patient with head injury

For **mild and moderate** head injury, the management as following;

GCS=9-14 With or without risk factors



Warning instructions includes; that patient should be brought back again to hospital if he developed deterioration in his conscious level, epilepsy, repeated vomiting or neurological deficit. These instructions should be given to patient's relative. Patient's relative education level and patient's home location relative to the nearest neurosurgical hospital should be considered also.

For severe head injury (GCS= 8 and less) the management as described for comatose patient (page 9).

Neurosurgical ward Patient should be sent for complete blood analysis including cross-matching (if surgery indicated), coagulation studies, electrolytes and blood biochemistry (Blood sugar, blood urea). Chest X-ray is needed also. The following chart should be done for every patient with head injury that was admitted to neurosurgical ward for observation;

Determinant		Time
GCS		
Vital signs	Pulse per minute	
	Blood pressure	
	Breathing pattern	
Pupils (sizes and reaction to light)		
Motor system examination (for weakness)		
Others		

A new brain CT scan is needed if there is deterioration in the above parameters, and possible causes of deterioration are; (1) Intracranial hematomas (2) Brain edema (3) Epilepsy (4) peumocephalus (presence of air in the intracranial cavity)