HEAD, NECK CONDITIONS

Henry Katua

BRAIN TUMORS

- Definition.
 - A brain tumor is an abnormal growth of tissue in the brain.
 - The tumor can either originate in the brain itself or come from another part of the body and travel to the brain.

Classification

- Brain tumors may originate from neural elements within the brain, or they may represent spread of distant cancers.
- Primary brain tumors arise from CNS tissue and account for roughly half of all cases of intracranial neoplasms.
- The remainder of brain neoplasms are caused by metastatic lesions.

- In adults, two thirds of primary brain tumors arise from structures above the tentorium (supratentorial).
- In children, two thirds of brain tumors arise from structures below the tentorium (infratentorial).
- <u>- Gliomas</u>, metastases, <u>meningiomas</u>, <u>pituitary adenomas</u>, and <u>acoustic neuromas</u> account for 95% of all brain tumors

World Health Organization classification of tumours of the central nervous system.

- <u>Tumours of neuroepithelial tissue:</u>
 - Astrocytic tumours.
 - Oligodendroglial tumours.
 - Mixed gliomas.
 - Ependymal tumours

- Choroid plexus tumours.

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- Neuronal and mixed neuronal-glial tumours.
- Neuroblastic tumours.
- Pineal parenchymal tumours.
- Embryonal tumours

Tumours of peripheral nerves

- Tumours of peripheral nerves.
- Schwannoma (Neurinoma).
- Neurofibroma .
- Perineurioma.
- Malignant peripheral nerve sheath tumour.

Tumours of the meninges

- Tumours of meningothelial cells.
- Meningioma.
- Atypical meningioma.
- Anaplastic meningioma.
- Mesenchymal, non-meningothelial tumours.
- Primary melanocytic lesions

Lymphomas and haemopoietic neoplasms

- Malignant lymphomas.
- Plasmacytoma.
- Granulocytic sarcoma

Germ cell tumours

- Germinoma.
- Embryonal carcinoma.
- Yolk sac tumour.
- Choriocarcinoma.
- Teratoma.
- Mixed germ cell tumours

Tumours of the sellar region

- Craniopharyngioma.
- Granular cell tumour

Epidemiology

- An increase in the prevalence of HIV infection corresponds to an increase in the occurrence of primary CNS lymphoma.

- Pituitary adenomas are exceptionally common, and they are frequent incidental findings on autopsy.

- Autopsy series of patients with systemic cancer show that intracranial metastases are present in 18-24% of patients.
- Meningiomas and pituitary adenomas are slightly more common in women than in men

- Males are more likely to be diagnosed with brain tumors than females, with a male-to-female ratio of 1.5:1.

- Tumors in the posterior fossa predominate in preadolescent children, with the incidence of supratentorial tumors increasing from adolescence to adulthood.
- Low-grade gliomas, such as <u>astrocytomas</u>, are more common in younger people than in older people.

- High-grade gliomas, such as <u>anaplastic astrocytoma</u> and <u>glioblastoma</u> <u>multiforme</u>, tend to originate in the fourth or fifth decade or beyond.

- In children, brain tumors are the most prevalent solid tumor.

Etiology

- Most CNS neoplasms are thought to arise from individual cell mutations.
- A prior history of irradiation to the head for reasons other than treatment of the present tumor may increase the chance of primary brain tumor.
- A few inherited diseases, such as neurofibromatosis, tuberous sclerosis, multiple endocrine neoplasia (type 1), and retinoblastoma, increase the predilection to develop CNS tumors

- The most common tumors originating from the cerebellopontine angle are acoustic neuroma and meningioma.

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- Primary CNS lymphoma is a relatively frequent occurrence in HIV patients.
- Metastatic tumors reach the brain via hematogenous dissemination through the arterial system

- Lung cancer is by far the most common solid tumor disseminating to the brain, followed by <u>breast</u>, melanoma, and <u>colon cancer</u>.

- Less common sources of metastasis are malignant melanoma, <u>testicular cancer</u>, and <u>renal cell cancer</u>.
- <u>- Prostate</u>, <u>uterine</u>, and <u>ovarian cancers</u> are unlikely sources of brain metastasis.

Clinical features

- <u>History.</u>
- Presenting complaints of patients with an intracranial neoplasm tend to be similar for primary brain tumors and intracranial metastases.
- Manifestations depend on the cause of the symptoms:-
 - An increase in ICP, direct compression of essential gray or white matter, shifting of intracranial contents, or secondary cerebral ischemia.

• Symptoms may be nonspecific and include headache, altered mental status, ataxia, nausea, vomiting, weakness, and gait disturbance.

• CNS neoplasms also may manifest as focal seizures, fixed visual changes, speech deficits, or focused sensory abnormalities.

 The onset of symptoms usually is insidious, but an acute episode may occur with bleeding into the tumor, or when an intraventricular tumor suddenly occludes the third ventricle.

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• Although headache is the symptom customarily associated with an intracranial neoplasm, it often is a late complaint.

• Mental status changes, especially memory loss and decreased alertness, may be subtle clues of a frontal lobe tumor.

• Complaints may be as mundane as sleeping longer, appearing preoccupied while awake, and apathy.

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 Temporal lobe neoplasms may lead to depersonalization, emotional changes, and behavioral disturbances.

• Vision, smell, and other sensory disturbances may be caused by a brain tumor.

• An acoustic neuroma may present as intermittent (then progressive) hearing loss, disequilibrium, and tinnitus.

• Symptoms of pediatric <u>posterior fossa tumors</u> include increased irritability, unsteadiness, ataxia, headache, vomiting. • Supratentorial tumors in children are more commonly associated with seizures, hemiparesis, visual field cuts, speech difficulties, and intellectual disturbance.

• Pituitary adenomas may be divided into 2 broad categories: nonfunctional and hypersecretory.

• Nonfunctional pituitary adenomas remain asymptomatic until they are large enough to encroach the optic chiasm and disturb normal vision.

• Most hypersecretory pituitary adenomas secrete prolactin, with affected women noting an amenorrhea-galactorrhea syndrome.

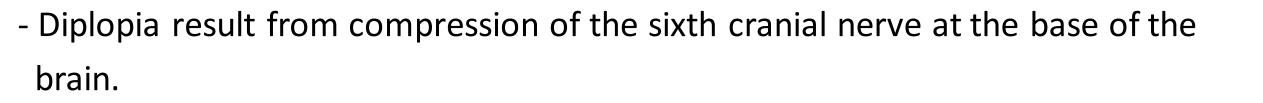
• Men with prolactin pituitary adenomas more commonly complain of headache, visual problems, and impotence.

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• Seizures, focal or generalized, may be the earliest expression of a brain tumor.



- No physical findings identifies a patient with a CNS neoplasm.
- Intracranial tumors produce a focal or generalized.
- Papilledema, which is more prevalent with pediatric brain tumors, reflects an increase in ICP of several days or longer.



- Impaired upward gaze, occur with pineal tumors.
- Tumors of occipital lobe produce homonymous hemianopia or partial visual field deficits.

- Anosmia may occur with frontal lobe tumors.

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- Brainstem and cerebellar tumors induce cranial nerve palsies, ataxia, incoordination, nystagmus, pyramidal signs, and sensory deficits on one or both sides of the body.

Treatment

- Surgery.
- Chemotherapy.
- Radiation therapy.
- Steroids for CSF pressure.
- Anti-seizure medication.

• VP shunt.

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- Spinal tap.
- Bone marrow transplantation.
- Antibiotics.
- Stereo tactic radio surgery.
- Gene therapy.

HYDROCEPHALUS

- Definition.
- Hydrocephalus is a disorder in which the cerebral ventricular system contains an excessive amount of cerebrospinal fluid (CSF) and is dilated because of increased pressure.
- The increased pressure distinguishes hydrocephalus from atrophy, in which dilatation is due to loss of brain tissue.

Epidemiology

• The prevalence of congenital and infantile hydrocephalus has been estimated as 0.48 to 0.81 per 1000 live and still births

Classification

- This accumulation results from an imbalance between production and absorption of CSF.
- Production is almost always normal, and the deficit is in the absorptive process.
- Mechanical or functional blockage of the flow of fluid along its usual pathway, thus interfering with normal absorptive mechanisms.

- There is increased pressure in the ventricular system of varying degree.
- Either transitory or persistent, during the process of ventricular dilation.
- Hydrocephalus is divided into:-
 - Communicating.

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- Non communicating.

Non-communicating hydrocephalus

- The ventricular fluid does not communicate with CSF in the spinal subarachnoid spaces or in the basal cisterns.
- This implies a block of CSF flow within the ventricular system i.e.,
 - Foramen of Monro,
 - Aqueduct of Sylvius,
 - Fourth ventricle and its outlets.

Communicating hydrocephalus

- The block is outside the ventricular system.
- Fluid within ventricles communicates with spinal subarachnoid space and basal cisterns.
- CSF is constantly being produced, and without absorption, the intracranial pressure would soon become elevated to such levels that continued neurologic function would be impossible.

Etiology

- Hydrocephalus can be **congenital** or **acquired**.

Congenital.

- Results from CNS malformations (which include nonsyndromic and syndromic disorders), infection, trauma, and teratogens.
- Congenital CNS tumor, especially if located near the midline

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- The majority of patients with myelomeningocele have hydrocephalus.
- The etiology is obstruction of fourth ventricular outflow or flow of CSF through the posterior fossa due to the malformation or an associated aqueductal stenosis.

- Isolated hydrocephalus is frequently caused by aqueductal stenosis.
- This can be due to congenital narrowing of the aqueduct, or result from inflammation due to intrauterine infection.

- Common genetic form of congenital hydrocephalus with stenosis of the aqueduct of Sylvius.
- 50 percent of affected boys have adducted thumbs, helps in making diagnosis.
- CNS abnormalities as agenesis or dysgenesis of the corpus callosum, small brainstem, or absence of the pyramidal tract

- Chiari malformation, which accompanies neural tube defect, portions of brain stem and cerebellum are displaced caudally into the cervical spinal canal.
- Portions of brainstem and cerebellum are displaced caudally into cervical spinal canal, and flow of CSF is impaired in the posterior fossa.
- This obstructs the flow of CSF in the posterior fossa, leading to hydrocephalus.

- Posterior fossa cyst that is continuous with the fourth ventricle and defective development of the cerebellum, including partial or complete absence of the vermis.
- In this condition, hydrocephalus results from secondary obstruction of the foramina of Luschka and Magendie.

- Obstruction results from compression of the aqueduct of Sylvius by the markedly dilated and distorted vein of Galen.
- Presentation in the neonatal period typically includes intractable heart failure

- Hydrocephalus can be part of syndromes associated with dysmorphic features and other congenital abnormalities.

- The most frequent cytogenetic disorders associated with hydrocephalus are trisomies 13, 18, 9 and 9p, and triploidy

- Intrauterine infections such as rubella, cytomegalovirus, toxoplasmosis, and syphilis can result in congenital hydrocephalus.
- The mechanism is inflammation of the ependymal lining of the ventricular system and the meninges in the subarachnoid space.
- This may lead to obstruction of CSF flow through the aqueduct or basal cisterns

Acquired hydrocephalus

- CNS infections such as bacterial meningitis, viral infections including mumps, and tumors, especially posterior fossa medulloblastomas, astrocytomas, and ependymomas.
- These conditions interfere with the flow of CSF through the ventricular system.

- Hemorrhage into subarachnoid space or ventricular system, by ruptured aneurysms, arteriovenous malformations, trauma, or systemic bleeding disorders.
- Hemorrhage induces inflammatory response followed by fibrosis, obstructing the flow and/or absorption of CSF.
- It can be obstructive, communicating, or both, and can be transient or sustained, with slow or rapid progression

Clinical features

- Symptoms and signs are caused by the basic process causing the hydrocephalus, such as tumor, infection, or bleeding.
- Increased intracranial pressure and dilation of the ventricles produce similar but nonspecific findings in all forms of hydrocephalus, independent of the primary cause.
- Nonspecific symptoms include headaches, produced by distortion of the meninges and blood vessels.

- Pain may vary in intensity and location and may be intermittent or persistent.
- Early morning headaches with nausea and vomiting are often caused by increased intracranial pressure.
- Personality, behavior changes include irritability, indifference, and loss of interest.
- Poor feed in children

- Lethargy and drowsiness occur as the disease progresses; these signs are related to midbrain and brainstem dysfunction.

- Nausea, vomiting, and decreased appetite are produced by increased intracranial pressure in the posterior fossa.

- Papilledema and extraocular muscle pareses leading to diplopia caused by compression of the third or sixth cranial nerve.
- Changes in vital signs: bradycardia, systemic hypertension, altered respiratory rates are produced by distortions of the brainstem.
- The anterior fontanelle may become full or distended.

- Infants and young children, excessive head growth may be noted on serial measurements of head circumference plotted on graphs of normal growth curve.
- Significant dilation of ventricles occur before abnormal head growth takes place.
- Infants, an abnormal skull contour develop in which forehead becomes prominent, and this is referred to as frontal bossing.

- The scalp veins become dilated and prominent.
- Brisk tendon reflexes, spasticity, clonus, babinsky.
- Macewen sign "cracked pot".

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- Prominent occiput (dandy-walker).

- Upward gaze is impaired because of pressure on the midbrain, and the sclera above the iris will be visible.
- This is known as the **setting-sun sign** because of appearance of sclera visible above the iris and is part of a larger constellation of neuro-ophthalmologic signs known as **Parinaud syndrome**

- Spasticity in the extremities, especially legs, develop as fibers from cortical motor areas are stretched around bodies of dilated ventricles in their course to cerebral peduncles.
- Disturbances in growth, accelerated pubertal development, fluid and electrolyte homeostasis develop from pressure exerted on hypothalamus by dilated third ventricle.

Diagnosis

- An infant whose head is growing excessively rapidly should be suspected of having hydrocephalus.
- The clinical impression can be confirmed by appropriate radiographic procedures.

- Ultrasonography is preferred technique for initial examination because it is avoids ionizing radiation.
- CT or MRI shows brain tissue and CSF-filled spaces and has greatly facilitated the evaluation.

- Ventricular dilation suggest the site of obstruction to the flow of CSF.
- Dilated lateral and third ventricles with a normal-size fourth ventricle suggest stenosis of the aqueduct.
- Symmetrically dilated ventricles, including the fourth ventricle, suggest an extra ventricular obstruction.

- Neuroimaging studies will also show any associated malformations or causes of acquired hydrocephalus, such as tumors.
- CSF should be examined if an occult infection causing adhesive arachnoiditis or ependymitis is considered to be responsible for the hydrocephalus.
- LP is contraindicated if patient has evidence of a space-occupying lesion such as an intracranial tumor or a brain abscess, because of the risk of cerebral herniation.

Treatment

- Specific therapy for any associated conditions and measures directed toward the hydrocephalus.
- Surgical therapy (surgical drainage) is the most effective means of treating hydrocephalus.
- This does not cure the hydrocephalus but does treat the symptoms and stops its progression.

Shunt

- A mechanical shunt system is used to circumvent the normal CSF pathways and to drain the excessive accumulation of CSF.
- A catheter is placed into one of the lateral ventricles, usually the right, and is connected to a one-way valve system (usually placed beneath the scalp of the postauricular area) that opens when the pressure in the ventricle exceeds a certain baseline value.

- As fluid egresses from the ventricles, lowering the pressure, the valve closes and remains shut until the pressure again rises.
- Distal end of the apparatus is connected to a catheter that is placed:-
 - Right atrium of the heart (ventriculoatrial).
 - Peritoneal cavity (ventriculoperitoneal).
- Fluid flows directly from lateral ventricles back into the systemic circulation, by passing the site of mechanical or functional block in CSF absorption.

- CSF flows directly from the ventricles into the systemic circulation or to the peritoneum where it is absorbed.
- The surgical procedure is not curative, but it does effectively treat the symptoms, and it stops progression of the ventricular dilation

Complications of shunt

- In general, complications of treated hydrocephalus are due to malfunction of the shunt.
- If the hydrocephalus is still active, symptoms recur and another drainage procedure is required.
- Malfunction is due to infection or mechanical failure.
- Shunt infection is common, occurring at a rate of approx. 5 to 10 percent of procedures

- Ventriculitis may develop.

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- Infecting organisms are typically the patient's own skin flora, such as Staphylococcus epidermidis.
- Infection must be considered in a child with a shunt who develops persistent fever.

Mechanical failure:

- Mechanical failure is an important problem, especially in the first year after shunt placement.
- Majority of first shunt failures result from obstruction at ventricular catheter because shunts typically overdrain, greatly reducing the size of the ventricles.
- Fractured tubing is the cause of shunt failure.
- Migration of part or all of the shunt.

- Establishment of an opening in a ventricle, through the floor of third ventricle to subarachnoid space to relieve hydrocephalus.

- If the shunt system becomes disconnected or catheters become obstructed, symptoms will recur if the hydrocephalus is still active.

Medical therapy

- Nonsurgical treatment includes:-
 - Diuretics,
 - Fibrinolysis,
 - Serial lumbar punctures.

Diuretics:-

- Diuretics furosemide and acetazolamide decrease CSF production.
- Used for short periods in slowly progressive hydrocephalus in patients too unstable for surgery.

Fibrinolytic therapy:-

- Intraventricular administration of fibrinolytic agents in newborns with post hemorrhagic hydrocephalus to prevent permanent obstruction to CSF flow.

Serial lumbar punctures:-

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- Repeated lumbar punctures have been used as a temporizing measure in preterm infants with post hemorrhagic hydrocephalus, although they do not appear to be effective.

Complications

- Infection of the shunt system, accompanied by ventriculitis, often involves organisms that are not usually pathogens, such as *Staphylococcus epidermidis*, *S. aureus*, enteric bacteria, diphtheroids, and *Streptococcus* species.
- Infection must be suspected in any child with a shunt who develops an unusual or persistent febrile illness.
- Pulmonary hypertension owing to chronic micro embolism from thrombi formed on the atrial catheter.

ASSIGNMENT.

- 1. CSF Production.
- 2. CSF flow.
- 3. CSF absorption.
- 4. Pathogenesis of Hydrocephalus.
 - 1. Obstruction of CSF pathways.
 - 2. Impaired absorption.
 - 3. Over secretion of CSF.
- 5. Pathophysiology of hydrocephalus

HEAD INJURY

• Definition.

- Defined as any alteration in mental or physical functioning related to a blow to the head.
- Classified by the initial post resuscitation Glasgow Coma Scale (GCS) score, summed score for eye, motor, and verbal abilities.
 - Score of 13-15 mild injury,
 - Score of 9-12 moderate injury,
 - Score of 8 or less severe injury.

Aetiology

- Road accidents involving motor vehicle drivers and occupants, cyclists, and pedestrians are the main risk factor for head injuries.
- Assaults, Athletic participation.
- Falls in elderly patients and children, Blast injuries.
- Anticoagulants and antiplatelet medications.
- Alcohol use raises the risks of incurring a head injury.

Classification

- TBI is divided into 2 broad categories:-
 - Closed head injury.
 - Penetrating head injury.
- Head injuries due to minor trauma without loss of consciousness, concussion, contusion, or fracture.

- Serious injuries associated with significant brain injury, results in diffuse axonal injury and varying types of hematomas (epidural, subdural, intraparenchymal, intraventricular).

- Skull fractures also occur often, with or without brain injury.

• A concussion:

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- Is a transient and rapidly reversible state of neuronal dysfunction associated with a loss of consciousness immediately after the head injury.

• A contusion:-

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- Is the focal bruising or tearing of cerebral tissue accompanied by parenchymatous haemorrhage and/or local oedema.
- The ventral surface of the frontal lobes and inferolateral aspects of the temporal lobes are the most common areas injured.

• Diffuse axonal injury:-

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- Results from acceleration-deceleration forces with shearing and tearing of axons (white matter) and disruption of myelin sheaths.
- Diffuse cerebral oedema results, often without obvious evidence of bleeding

Epidural hematoma

Definition:-

- It is a collection of blood between the Dura mater and the skull resulting from arterial or venous injury.

- EDH does not cross sutural margins, crosses dural attachments because it is located in the potential space between Dura and skull.

Investigation:

- CT, shows biconvex in shape because their outer border follows the inner table of the skull and their inner border is limited by locations at which the dura is firmly adherent to the skull.

• Treatment:

- Acute symptomatic EDH is a neurologic emergency.
- Requires surgical treatment to prevent irreversible brain injury and death caused by hematoma expansion, elevated intracranial pressure, and brain herniation.

- Craniotomy and hematoma evacuation is the mainstay of surgical treatment of symptomatic acute EDH.
- When indicated, identification and ligation of the bleeding vessel must be undertaken.

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subdural hematoma:

Definition:-

- Subdural hematomas form between the dura and the arachnoid membranes.

- Caused by tearing of the bridging veins that drain from the surface of the brain to the dural sinuses.

- Rupture of these vessels allows bleeding into the space

- Acute SDH presents one to two days after onset

- Sub acute SDH presents 3 to 14 days after onset
- Chronic SDH presents 15 or more days after onset

Investigations:

- CT, shows outer edge is convex, while their inner border is usually irregularly concave.
- Hematomas are not limited by the intracranial suture lines; this is an important feature that aids in their differentiation from epidural hematomas

Treatment:

- Acute symptomatic SDH is a neurologic emergency requires surgical treatment.

- Non-operative mx is appropriate for clinically stable pts with small hematomas.

- Surgery:-

- Surgical evacuation is performed by - burr hole trephination, craniotomy, and decompressive craniotomy

Intra-axial hematomas

Defined as haemorrhages within the brain parenchyma.

- Include intraparenchymal hematomas, intraventricular haemorrhages, and subarachnoid haemorrhages.
- Subarachnoid haemorrhages that occur because of trauma are typically located over gyri on the convexity of the brain.
- Subarachnoid haemorrhages that result from a ruptured cerebral aneurysm are usually located in the subarachnoid cisterns at the base of the brain.

Skull fractures

- Definition: Occur when the bony integrity of the skull is disrupted.
 - Fractures may be linear, depressed, or comminuted and can occur in occipital, temporoparietal, frontal, and basilar areas.
 - Impacts to the face may result in facial fractures of the nasal bones, sinuses, or orbital areas.
 - Scalp lacerations and depressed or compound depressed skull fractures should be treated surgically as appropriate.
 - Simple skull fractures require no specific treatment.

Clinical features of skull fractures

- Basilar skull fracture:-

- bruising about the orbit (raccoon sign),
- blood in external auditory meatus (Battle's sign),
- leakage of cerebrospinal fluid.
- Cranial nerve palsies (involving especially the first, second, third, fourth, fifth, seventh, and eighth nerves in any combination) may also occur.

Types of skull fractures:

Simple linear fractures:

- These require no specific neurosurgical management.

Depressed skull fracture:

- These fractures are a result of blunt trauma, usually to the left -frontal region.
- If Pericranium has been breached the fractures are technically compound.
- Dura and brain may be lacerated by the depressed fragment

- Surgery is undertaken to prevent risk of infection, alleviate mass effect and for cosmetic purposes.
- Contaminated wounds require extensive debridement, a duraplasty and irrigation before closure.
- A full course of intravenous antibiotics should be administered

- Base of skull fracture:
 - These are frequent fractures, usually diagnosed on clinical grounds.
 - Result in CSF fistula which persist but usually seal off after a few days.
 - Anterior fossa fractures present with sub conjunctival haematomas, anosmia, epistaxis and CSF rhinorrhoea and associated with caroticocavernous fistulae.

- Periorbital haematomas indicate subgaleal haemorrhage.
- Middle fossa fractures present with CSF otorrhoea or rhinorrhoea via the Eustachian tube, heamotympanum, ossicular disruption, or VII and VIII cranial nerve palsies

• *Ping-Pong fracture:* - This is a smooth depression of the cranial vault usually seen in children.

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- Also known as a opond' fracture.

 Blow-out fracture: - Fracturing of the orbital walls with herniation of orbital contents and subsequent tethering of the globe, resulting in pain and diplopia.

Clinical features

History.

- Acute state, patient is comatose or confused, witnesses to accident or injury are of obvious and crucial importance.
- Elicit the type and mechanisms of the injury, as these may have prognostic value.
- Head injury from struck with a falling object have poor outcome because of greater axonal damage.

- Loss of consciousness is a marker of severe neurological injury.
- Prior head injuries, indicate the potential for more severe long-term outcomes.
- Drug or alcohol use raises the risk of intracranial bleeding and cloud the mental status assessment.
- Consider past psychiatric disease and a premorbid history of headaches.



- Glasgow Coma Scale (GCS) is the mainstay for rapid neurologic assessment in acute head injury.
- Following ascertainment of the GCS score, focus the examination on signs of external trauma.
- Bruising or bleeding on the head and scalp and blood in the ear

- Consider coexistent cervical spine and other systemic injuries.
- Anosmia is caused by the shearing of the olfactory nerves at the cribriform plate.
- A unilaterally dilated pupil with ipsilateral cranial nerve III paralysis (ptosis impaired ocular motility) indicate impending herniation.

- CN VI palsies indicate raised intracranial pressure.
- CN VII palsy, indicate a fracture of the temporal bone.
- Focal motor findings are of a localized contusion or, early herniation syndrome.

- Flexor or extensor posturing implies extensive intracranial pathology or raised intracranial pressure.
- Chronic phase, motoric manifestations include spasticity, akinesia and rigidity.
- Tremors and dystonia recede with time, still affect survivors of severe head injury 2 years after the initial trauma.

The Glasgow Coma Scale (GCS)

- This gives a reliable, objective way of recording the conscious state of a person.
- It can be used for initial and continuing assessment.
- It has value in predicting ultimate outcome.

- Three types of response are assessed:-
 - 1. Best motor response This has 6 grades:
 - 2. Best verbal response This has 5 grades
 - 3. Eye opening This has 4 grades.

Best motor response -This has 6 grades:

- Carrys out request (obeys command): Pt does simple things you ask = 6.
- Localizes response to pain: Pressure on pts nail, supraorbital, sternal: = 5.
- Withdraws to pain: Pulls limb away from painful stimulus. = 4.
- Flexor response to pain: Pressure on nail, abnormal flexion of limbs: = 3.
- Extensor posturing to pain: Stimulus causes limb extension (adduction, internal rotation of shoulder, pronation of forearm): = 2.
- No response to pain. = 1

Best verbal response This has 5 grades.

- Oriented: Patient knows who, where he is and why, year, season, month. = 5.
- **Confused conversation:** Patient responds to questions in a conversational manner but there is some disorientation and confusion. = 4.
- Inappropriate speech: Random or exclamatory articulated speech, but no conversational exchange. = 3.
- **Incomprehensible speech:** Moaning but no words. = 2.
- **None**. = 1

- Eye opening This has 4 grades.
- **Spontaneous eye opening**: = 4.
- **Eye opening in response to speech**: Any speech, or shout, not necessarily request to open eyes. = 3.

- **Eye opening to response to pain**: Pain to limbs as above. = 2.
- No eye opening. = 1

- Overall score is made by summing the score in the 3 areas assessed, e.g.: no response to pain + no verbalization + no eye opening = 3.
- Severe injury, GCS < 8; Moderate injury, GCS 9 12; Minor injury, GCS 13 15.
- Note: An abbreviated coma scale, AVPU, is sometimes used in the initial assessment (primary survey) of the critically ill.
- A = **a**lert
- V = responds to **v**ocal stimuli
- P = responds to **p**ain
- U = unresponsive

INVESTIGATIONS IN HEAD INJURIES

• 1 Plain skull x-ray.

- Indicated in loss of consciousness, localized contusion, swelling over the head.
- Skull x-ray shows skull fractures and intracranial air.
- Patients with skull fractures should be admitted to hospital for observation.
- AP ,lateral and Town.(Occipito-Frontal) Views.

2. CT scan

Indications.

- a. All moderate to Severe head injury GCS below 12.
- b. History of loss of consciousness or decreasing level of consciousness.
- d. Post-traumatic seizure.
- c. Lateralizing signs weakness of limb or un reactive pupil.
- f. Type of injury Penetrating injury Or Skull fractures.
- h. Otorrhea and rhinorrhea

- Useful to show long term effects of head injury.
- Depending on the availability it also could be used in investigating acute cases.

4. Beta transferrin.

- This is a test for an enzyme which is only found in CSF.
- It is the optimum test for CSF leak.

Other Important Baseline Tests

- 1. Urea and electrolytes
- 2. Arterial blood gases
- 3. Blood alcohol level
- 4. Random blood glucose
- 5. Full haemogram/esr
- 6. Grouping and cross match

MANAGEMENT.

• INITIAL MANAGEMENT - Primary Survey

A,B,C,D of resuscitation plus vital signs.

• 1. Airway and cervical spine.

- Maintain cervical spine immobilization in all unconscious, symptomatic patients.

- Inspect mouth remove debris by sweeping through.
- Chin lift/jaw thrust (tongue is attached to the jaw) avoids tongue falling back.
 - chin lift:-Two fingers under mandible, gently lift upward to bring chin anterior.
 Be careful not to hyperextend the neck.
 - jaw thrust:- Manually elevating angles of mandible to obtain same effect.

- Guided airway/nasopharyngeal airway to secure airway.
- Insert the oral airway into the mouth behind the tongue.
- Usually inserted upside down until the palate is encountered and is then rotated 180 degrees.

- Intubations; keep the neck immobilized in neutral position.
- Intubate all unconscious patients (GCS < 9) to secure airway.
- Use sedation and short acting neuromuscular blockade if necessary.
- Tracheostomy.

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- Cricothyrotomy.

Breathing: Oxygenation and ventilation

- LOOK OUT for six major problems that impair breathing:-
- Tension pneumothorax put through a needle
- Massive pneumothorax chest tubes insertion
- Sucking wounds strap the open wound
- Flail chest positive pressure ventilation
- Cardiac tamponade
- Airway obstruction

- Inspect (LOOK):-
- Inspection of respiratory rate is essential. Are any of the following present?
- - Cyanosis.
- -Any signs of respiratory distress Use of accessory muscles, flaring of alae nasae, subcostal recession.
- Inspect Chest movements, Penetrating injury, Presence of flail chest, Sucking chest wounds

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- -Tracheal shift
- -Broken ribs
- -Subcutaneous emphysema.
- -Chest expansion

Percussion.

• For dullness is useful for diagnosis of haemothorax and pneumothorax.

Auscultate (LISTEN)

- - Pneumothorax (decreased breath sounds on site of injury).
- - Detection of abnormal sounds in the chest.
- - Maintain patient on oxygen until complete stabilization is achieved

- Tension pneumothorax, introduce a large-bore needle into the pleural cavity through second intercostal space, mid clavicular line, to decompress the tension and allow time for the placement of an intercostal tube.
- If intubation in one or two attempts is not possible, a cricothyroidotomy should be considered a priority

3. Circulation and arrest of bleeding.

- "Shock" in trauma patient is most due to haemorrhage and hypovolaemia.
- The diagnosis of shock is based on clinical findings:-
- Hypotension
- Hypothermia
- Tachycardia
- Tachypnoea
- Cool extremities
- Decreased capillary refill
- Pallor
- Decreased urine production

Resuscitation

- Stop bleeding by gauze pack and Manual compression on the proximal artery.
- Carefully applied compressive dressing of entire injured limb can be done.
- Vascular access 2 large bore size 16 on the 2 basilic veins.
- Fluids:-
 - Infuse 0.9% NaCl initially 2L to run as fact as possible through 2 large bore IV lines in the ante cubital fossa then re-assess

Resuscitation action

- Insert an intercostal drainage tube before chest X-ray if respiratory distress exists, to drain the chest pleura of air and blood.

- When indications for intubation exist but trachea cannot be intubated, consider using a laryngeal mask airway or direct access via a cricothyroidotomy.

- Resuscitate to attain:- Mean arterial pressure (MAP)>90 mmHg. Cerebral perfusion pressure (CPP)>70-80mmHg.
- Urinary catheter insertion, monitor input-output chart at least 30-50 ml/hour or 0.5/kg/hour of urine flow.
- Asses by vital signs, pallor, sweating, anxiety ,skin warmth clammy, input and output

- Blood transfusion must be considered when the patient has persistent haemodynamic instability despite fluid (colloid/crystalloid) infusion.
- If type specific or cross matched blood is not available, use group O negative packed red blood cells.
- Transfusion should be seriously considered if the haemoglobin level is less than 7 g/dl and the patient is still bleeding.

- Neurological dysfunctions.
 - Establish preliminary level of consciousness by AVPU
 - A Awake, V Verbal response, P- Painful response
 - U Unresponsive and any focal neurologic deficits.
- Exposure and environmental modification.
 - Cover patient in case of shock and shivering.

SECONDARY SURVEY

Head to toe exam with emphasis on the evaluation of head injury

- HEAD EXAMINATION.
 - Scalp and ocular abnormalities Raccoon eye and battle signs, wounds ,bleeding around the head.
 - External ear and tympanic membrane.
 - Periorbital soft tissue injuries.

NEUROLOGICAL EXAMINATION

- Glasgow coma scale - Gold standard for evaluation of the severity of head injury. - Monitoring improvement, deterioration of head injury.

- Mild head injury GCS-14-15- loss of consciousness for less than 5 minutes.
- Moderate head injury 9-13 loss of consciousness more than 5 minutes
- Severe head injury GSC 5-8
- critical head injury GSC 3-5.
- All cranial Nerve examination.
- Peripheral sensory and motor examination

NECK EXAMINATION

- _ Penetrating wounds and bleeds.
- _ Subcutaneous emphysema.
- _ Tracheal deviation.
- _ Neck vein appearance.

• CHEST EXAMINATION.

- <u>-</u>Clavicles and all ribs.
- -Breath sounds and heart tones.
- -ECG monitoring (if available).

• ABDOMINAL EXAMINATION.

- -Penetrating abdominal wound requiring surgical exploration.
- -Blunt trauma: insert a nasogastric tube (not in the presence of facial trauma).
- -Rectal examination.
- -Insert urinary catheter (check for meatal blood before insertion)

PELVIS AND LIMBS

- -Fractures
- -Peripheral pulses
- -Cuts, bruises and other minor injuries.
- <u>X-RAYS</u>
- -Chest, C-spine and pelvis X-rays may be needed during primary survey
- NB-Cervical spine films (must see all 7 vertebrae)
- -Pelvic and long bone X-rays

INTRACRANIAL PRESSURE (ICP) and CEREBRAL PERFUSION PRESSURE (CPP) MONITORING

- Symptoms of increased ICP.
- -Severe bursting headache
- -Projectile vomiting
- -Blurring of vision
- -Convulsions/seizures
- -Drowsiness

• Signs of increased ICP.

- -Vital signs increased BP and decreased pulse rate.
- -Anisocoria unequal pupils
- -Papiloedema on fundoscopy
- -Nerve palsy e.g. 3rd and 6th cranial nerves
- -Tense fontanels.
- -Irregular breathing.
- Parameters: Normal ICP = 0-10 mmHg.

TREATMENT OF INCREASED ICP

1. Elevation of head

- promotes venous drainage from the head.

2. Ventilation O2 by mask

- Prevention of hypoxia and hypercapnia which increase ICP.

3. Mannitol.

-doses 0.25-1 gm/kg, by intermittent bolus infusion every 4-6 hrs.

4. Hyperventilation

- blows out co2, reduces hypercapnia.

5. Anticonvulsant therapy

- Phenytoin used to prevent seizure activity.
- used for 1 week following injury, then discontinued if seizures are not recurrent.

6. Nimodipine

- calcium channel blocker reduces death, severe disability.

7. Relieve and prevent pyrexia

- which increases intracranial pressure. e.g. NSAIDS Provision.

8. Sedatives

- High dose diazepam is considered for hemodynamically stable, with intracranial hypertension refractory to maximal medical and surgical therapy.
- Other narcotics may depress respiration.

9. Steroids

- Dexamethasone use is controversial in head injury

Early post-traumatic seizure prophylaxis (7 days):

- Phenytoin(Dilantin) is indicated for:-
- a. Glasgow coma scale score < 10.
- b. Cortical contusion.
- c. Depressed skull fracture.
- d. Subdural hematoma.
- e. Epidural hematoma.
- f. Penetrating head wound.
- g. Seizure within 24 hrs. of injury.

- Therapy should be instituted for 7 days.

HEAD INJURY OBSERVATION CHART

- Monitoring in half, hourly or 2 hourly
- 1. Continuous monitor of level of consciousness
- Best eye opening score
- Best verbal response score
- Best motor response
- 2. Vital signs
- Pulse
- Temperature
- BP
- Respiratory rate

3. Pupillary reflexes

- Reaction to light
- Size of the pupil

4. Motor examination of limbs

- Spontaneous movement of all the limbs
- Paralysis

5. Monitor danger signs

- -Severe headache
- -Vomiting
- -Convulsions/seizures.
- -Drainage of fluids ear or nose

6. Presence of other injuries

- Chest
- Abdomen
- Neck
- Spine
- Arm or leg

<u>Scalp wound.</u>

- -Analgesia plus tetanus toxoid
- -Clean the wound
- -Debride
- -Stitch

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-Antibiotics

- <u>Scalp wound plus fracture.</u>
- -Surgical toilet under GA
- -Clean

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- -Stitch
- -Antibiotics

• <u>Depressed Fracture</u>.

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- Conservatively managed .
- Surgical elevation if depression is twice the table width or clinically lateralizing sings.

Nutritional support.

- Enteral feeds should be instituted within 72 hours of injury.

- Nonparalyzed patient – replace 140% of estimated energy expenditure. (normal person at rest need about 3000kcal per day) - Paralyzed patient – replace 100% of estimated energy expenditure.

a. Start via NGT, rate 10 ml/hr every 4 hrs. Until goal is reached.

b. Hold for residual > 100 ml, if abdominal injury present or surgery required.

-Parenteral nutrition - Use only if enteral feeds contraindicated or not tolerated.

OTHER GENERAL CARE UNCONSCIOUS PATIENTS

- 1. Bladder care
- 2. Bowel care
- 3. Physiotherapy chest and limbs
- 4. Skin care
- 5. Analgesics

Diagnosis of brain death

- Brain death is a diagnosis of what is, not what might be, and must be proven rather than insinuated.

- Accurate diagnosis of brain death, there must be clear evidence of an acute, catastrophic, irreversible brain injury.

- Physical examination must show complete unresponsiveness, absent motor responses, absent brainstem reflexes, and apnoea.

- Confirmatory studies, such as EEG or cerebral blood flow studies, may be ordered if there is any ambiguity in the clinical evaluation.

- A typical brain death protocol may be summarized as follows:
- Confirm patient is in a coma.
- Evaluate for seizure activity and decerebrate or decorticate movements.
- Test for motor response to painful stimulation.
- Test for pupillary response to light.
- Test for corneal reflex.
- Test for oculocephalogyric reflex (doll's head reflex).
- Test for vestibulo-ocular reflex (caloric test).
- Test for upper and lower airway stimulation (eg, pharyngeal and endotracheal suction).
- Test for gag reflex.

COMPLICATIONS OF HEAD INJURY

- 1. CN palsies and Focal neurological signs
- 3. Infections
- 4. Hydrocephalus
- 5. Convulsive disorder/epilepsy
- 6. Psychiatric disorders
- 7. Cerebrospinal fluid fistulae, either in the form of rhinorrhea or otorrhea

- 8. Posttraumatic movement disorders Tremor, dystonia, Parkinsonism, myoclonus,etc.
- 9. Vascular injuries
 - include arterial transactions, thromboembolic phenomena, posttraumatic aneurysms, dissections, and carotid-cavernous fistulae (CCF).

- 10. Post-concussional symptoms e.g. Bradycardia, Hypertension.
- 11. Cumulative brain damage ('Punch-drunk syndrome').
- 12. Neurological & neuropsychological deficits e.g. Parkinsonism, Dementia.
- 13. Neuroendocrine & metabolic disturbances e.g. Diabetes insipidus.

CLEFT LIP AND PALATE

- The aetiology of cleft lip and palate have a genetic predisposition and environmental component.
- A family history of cleft lip and palate increases the risk to 1:25 live births.
- Genetic influence is more significant in cleft lip/palate than cleft palate alone, in which environmental factors exert a greater influence.

Incidence of cleft lip and palate

- The typical distribution of cleft types is:-
 - Cleft lip alone: 15%.
 - Cleft lip and palate: 45%.
 - Isolated cleft palate: 40%.

Cleft lip

- The abnormalities in cleft lip is disruption of the muscles of the upper lip and nasolabial region.
- The facial muscles can be divided into three muscular rings of Delaire:-
 - 1. Nasolabial muscle ring surrounds the nasal aperture;
 - 2. Bilabial muscle ring surrounds the oral aperture;
 - 3. Labiomental muscle ring envelops the lower lip and chin regions.

Cleft palate

- The *primary* palate consists of all anatomical structures anterior to the incisive foramen, namely the alveolus and upper lip.

- The *secondary* palate is defined as the remainder of the palate behind the incisive foramen, divided into the hard palate and the soft palate.

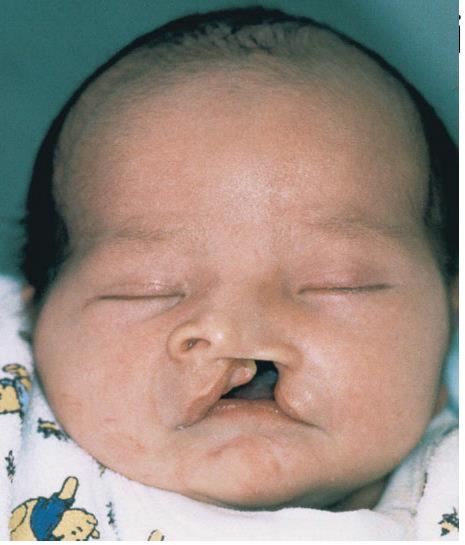
- Cleft palate results in failure of fusion of the two palatine shelves.

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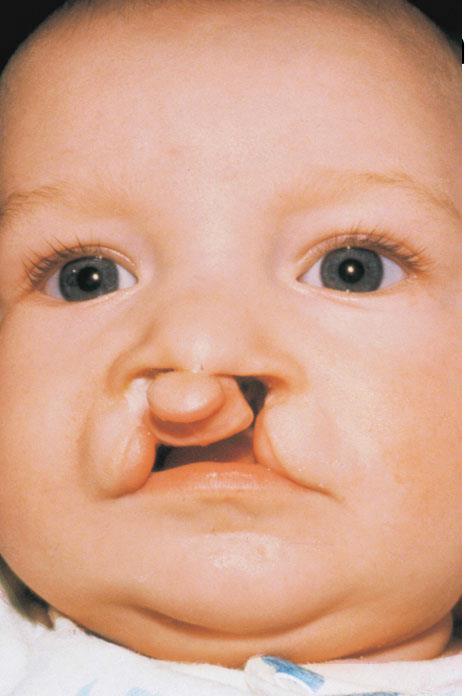
- This failure may be confined to the soft palate alone or involve both hard and soft palate.

- When the cleft of the hard palate remains attached to the nasal septum and vomer, the cleft is termed *incomplete*.

- When the nasal septum and vomer are completely separated from the palatine processes, the cleft palate is termed *complete*.



lateral cleft lip.



teral cleft lip.

MANAGEMENT

- Cleft lip, palate surgery
- Cleft lip alone
- Unilateral (one side) One operation at = 5–6 months.
- Bilateral (both sides) One operation at = 4–5 months

- Cleft palate alone
- Soft palate only One operation at = 6 months.
- Soft and hard palate Two operations. Soft palate at = 6 months
 Hard palate at = 15–18 months

• Cleft lip and palate.

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- Unilateral Two operations Cleft lip and soft palate at = 5–6 months.
- Hard palate and gum pad with or without lip revision at = 15–18 months

• Cleft lip and palate.

• Bilateral Two operations Cleft lip and soft palate at = 4–5 months.

• Hard palate and gum pad with or without lip revision at 15–18 months

Principles of surgery

■ Cleft lip surgery attaches and reconnects the muscles around the oral sphincter.

- Cleft palate surgery aims to bring together mucosa and muscles with minimal scarring.
- Two-stage procedures attempt to minimise dissection

Cleft lip revision

- Indications for revision include:
 - Lip deformity:
- – Malaligned vermilion.
- – Asymmetrical Cupid's bow.
- – Muscle discontinuity or malalignment.

- Nasal deformity:
- – Lateral drift of alar base.
- – Poor nasal tip projection.
- – Deviation of cartilaginous nasal septum into the non-cleft nostril.