Impair Consciousness

1

Coma or loss of consciousness may be defined as a loss of awareness of one's self or environment. Glasgow Coma Scale (GCS) is used to assess the level of consciousness:

Coma according to GCS is defined as inability to obey command, to speak words and to open the eyes. Therefore; none of patients with GCS of 9 or more are found to be in coma, but 90% of those with GCS of 8 or less could be found in coma.

The conscious state depends on the integrity of the reticular activating system, beginning in the medulla and extending to thalamus. The reticular nuclei seem to supply a baseline arousal level somewhat like the power control to a computer. The cerebral hemispheres may be thought of as analogous to the software and memory of a computer system. Therefore; the cause of impair consciousness can be classified into:

A- Cerebral hemispheres lesions:

like diffuse cortical lesions (hypoxia, hypoglycemia ...etc)

B- Brainstem lesions: includes;

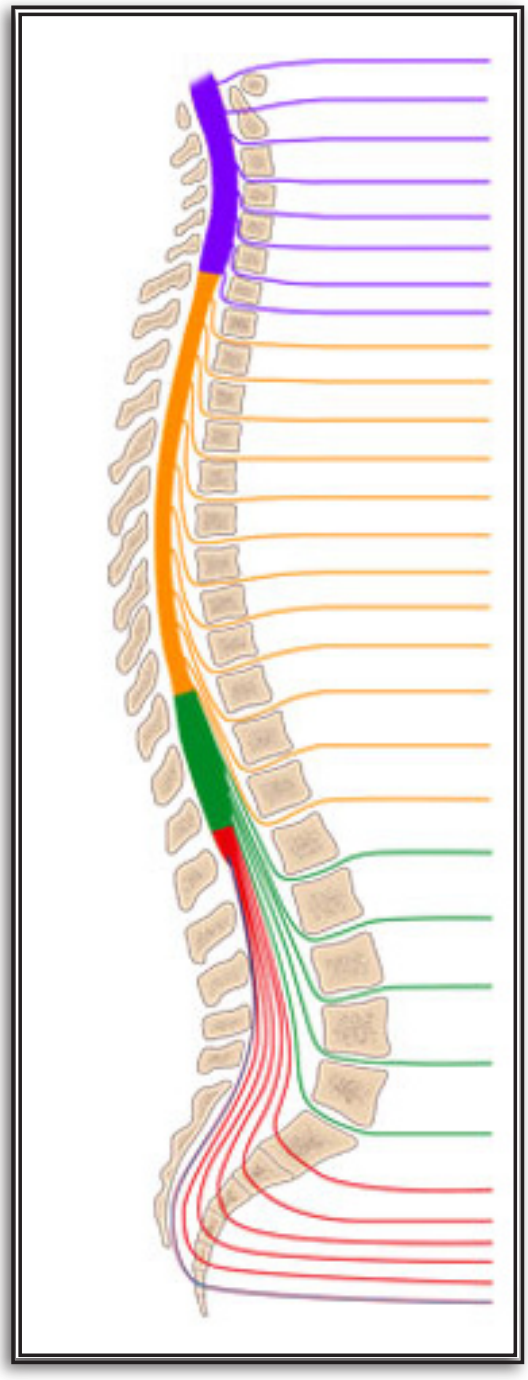
1- Supratentorial mass lesion through uncal herniation (cerebral tumors)
2- Direct lesion in the brainstem itself (Hemorrhages)
3- Infratentorial lesions with secondary compression on the brainstem (Cerebellar tumors).

The Spinal Cord

The length of the spinal cord is equal to the length of the vertebral column in foetus of **3 months** length. After 3 months, the vertebral column grows faster than the spinal cord. At **birth**, the spinal cord reaches only the level of the 3rd. lumbar vertebra. In **adult**, the spinal cord ends at the ***L1-2 disk***, therefore; the segments of the spinal cord are not in line with the vertebrae as appear in the following table:

C2	Scalp area behind imaginary line that connect both ears
C3	Neck area
C4	Shoulder area
C5	Lateral arm area
C6	Lateral hand area
C7	Middle hand area
C8	Medial hand area
T1	Medial Forearm area
T2	Angle of Lewis area
T10	Umbilical area
L1	Inguinal ligament area
L2	Lateral thigh area
L3	Medial thigh area
L4	Medial leg area
L5	Lateral leg area
S1	Lateral planter area
S2	Posterior thigh & leg area
S3-4-5	Buttock area

Spine	Spinal Cord Segments
C6	C8
D3	D6
D9	D12



2 Spinal Cord Injury

Spinal cord trauma is divided into;

1- Primary injuries: which are the damages that result at time of accidence, and include acute compression, impaction, distraction, laceration and shear.

2- Secondary injuries: which are defined as the damages that result within minutes, hours, or days after the events of primary injuries, and can lead to further damage of nervous tissue, prolonging and/or contributing to permanent neurological dysfunction. The end result of the secondary events is decreasing local blood supply at site of cord injury which results in further ischemic damage. If these changes are not corrected, the resulting biochemical changes from the infarcted area lead to ischemic changes in the adjacent areas and vicious circle will continue.

Lower limbs	Upper limbs
Hip flexors [L2]	Elbow flexors [C5]
Knee extensors [L3]	Wrist extensor [C6]
Ankle dorsiflexor [L4]	Elbow extensor [C7]
Long toe extensor [L5]	Finger flexors [C8]
Ankle planter flexor [S1]	Finger adductors [T1]

key muscle groups

Spinal Cord Injury is described in term of level and completeness.

Level of spinal cord injury:

is the most caudal segment with motor function that is at least **3** out of **5** and if the pain and temperature sensation is present. The motor part of the spinal segment can be assessed by examination of the ***key muscle groups***.

Completeness of the lesion

this include complete and incomplete lesions of the spinal cord.

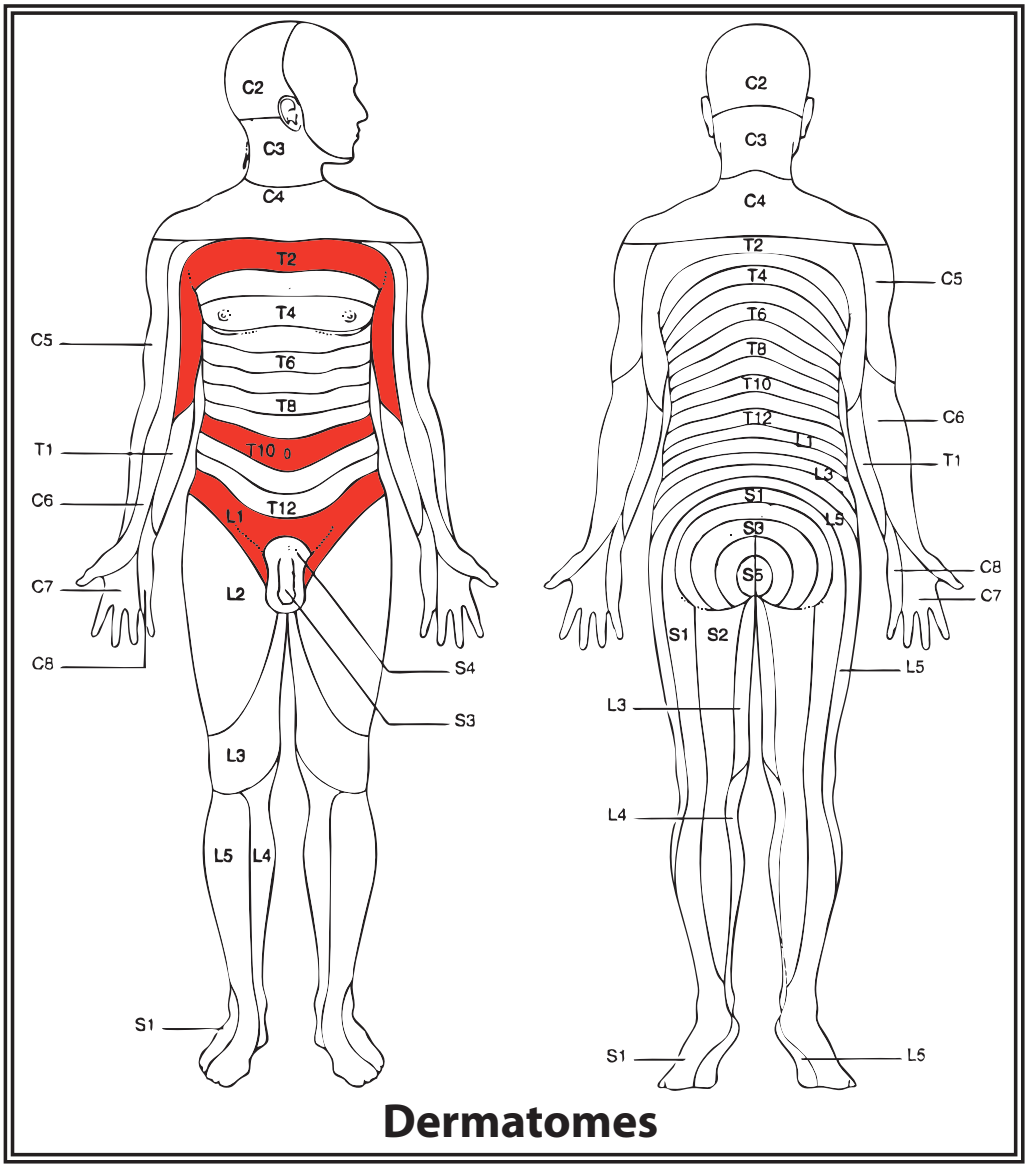
(A) Incomplete lesion: any residual motor or sensory function more than **3** segments below the level of the injury.

Signs of incomplete lesion include;

(1) Sensation (including position sense) or voluntary movement in the lower limbs.

(2) Sacral sparing [sensation around the anus, voluntary rectal sphincter contraction or voluntary toe flexion].

(3) An injury does not qualify as incomplete with preserved sacral reflexes alone.



Dermatomes

Types of incomplete lesion include;

(1) Central cord syndrome: there is a disproportionately greater motor deficit in the upper limbs than in the lower limbs. It is usually results from hyperextension injury in the presence of osteophytic spurs.

(2) Brown-Sequard syndrome (cord hemisection): there are **(a)** ipsilateral motor paralysis (due to corticospinal tract lesion) and loss of posterior column function (proprioception and vibratory sense) below lesion level. **(b)** contralateral loss of pain & temperature sensation (spinothalamic tract leasion) below lesion level. It is usually a result of penetrating trauma.

(3) Anterior cord syndrome: there are **(a)** paraplegia or (if the lesion is higher than C7) quadriplegia. **(b)** dissociated sensory loss below the lesion (loss of spinothalamic tract function and preservation of posterior column function). It may be results from occlusion of anterior spinal artery or from anterior cord compression (by dislocated bone fragment or by traumatic herniated disc).

(4) Posterior cord syndrome: associated with loss of position and vibration sensation (loss of posterior column function). It is relatively rare.

(5) Conus medullaris syndrome: the conus medullaris is found between the spinal levels of **T11** and **L2**. There are both upper and lower motor neuron signs in the lower limbs which tend to be symmetrical with flaccid rectal tone and urinary retention.

Cauda equine syndrome: is an injury to the nerve roots arising from the conus medullaris (fracture or acute disk herniation) extending from **L2** and below. Patient presented with asymmetrical lower motor neuron signs in the lower limbs with loss of bladder and bowel control.

(B) Complete lesion: there is no preservation of any motor and/or sensory function more than **3** segments below the level of the injury.

Spinal shock: is defined as a transient loss of all neurologic function below the level of spinal cord injury which result in flaccid paralysis and areflexia lasting for varying periods (uasually 1-2 weeks,occationally several months and sometimes permanently), the resolution of which results in hypertonia, exaggerated reflexes and in many cases spastisity below the level of the lesion.

***Bulbocavernosus reflex* (S3-S4):** this reflex is elicited by pinching or pricking the foreskin of the penis or glans penis. The bulbocavernosus muscle contracts and this can be seen or felt in the perineum at the root of the penis. The associated involuntary contraction of the rectal sphincter can also be detected by performing rectal examination; therefore, this reflex can be elicited in male or female by tugging on the Foley catheter. During the early post-injury period, the bulbocavernosus reflex may be absent and suggests the continued spinal shock. This reflex tends to be one of the earliest reflexes to recover from spinal shock. The presence of this reflex implied the lack of supraspinal input to the sacral outflow and is suggestive of complete spinal cord injury**.**

Voluntary contraction of the sphincter during digital rectal examination or the presence of rectal sensation (but not the superficial anal reflexes (S3-S5)) or both supports the presence of a communication between the lower spinal cord and supraspinal centers (incomplete lesion); thus the potential for further motor or sensory recovery is favorable.

Neurogenic shock: is condition characterized by hypotension and bradycardia resulting from interruption of the sympathetic nervous system pathways within the spinal cord. The incidence of significant neurogenic shock increases with injuries above **D6** beacause unopposed vagal tone slows the heart and creates lower systemic vascular resistance, resulting in venous pooling.

Prognostic factors for recovery: **(1)** ***Completeness of injury:*** About **3%** of patients with complete injuries on initial exam will develop some recovery. In contrast, most patients who enter the hospital with an incomplete neurologic injury attain some degree of recovery (90% of Brown-Sequard syndrome, 50% of Central cord syndrome, and 10-20% of Anterior cord syndrome patients will regain ability to ambulate independently and control sphicter function).

(2) ***Level of injury:*** Cervical injuries have a higher potential for recovery than do thoracic or thoracolumbar injuries.

(3) ***Severity of injury:*** The less the severe the injury, the more likely it is that the patient will recover.

(4) ***Age:*** Younger patients fare much better than their older counterparts in terms of regaining neurologic function after spinal cord injury.

(5) MRI findings: Intramedullary hemorrhage is more commonly observed after neurologically complete injuries and signifies a worse neurologic and functional outcome.

Management The major causes of death in spinal cord injury are aspiration and shock.

Management proceeds in association with routine treatment of other injuries:

(A) Management in the field (at time of accident):

(1) Immobilization prior to and during extrication from vehicle and transport to prevent active or passive movements of the spine (place the patient on back-board with sandbags (or plastic intravenous bags) on both sides of the head and 3 inch strip of adhesive tape from one side of the back-board to the other across the forehead immobilizes the spine, a rigid cervical collar may be used as supplement).

(2) Maintain blood pressure (a) pressors (dopamine) (b) fluid as necessary to replace losses (c) Military anti-shock trousers (prevent venous pooling).

(3) Maintain oxygenation (by mask or if the patient need intubation; by nasotracheal method to avoid neck movement.

(4) Brief motor exam: ask patient to (a) move arms (b) move hands (fingers) (c) move legs (d) move toes.

(B) Management in the hospital:

(1) Immobilization: maintain backboard / head – strap to facilitate transfers to CT table..etc.

(2) Treatment of neurogenic shock (hypotension) by (a) dopamine (b) careful hydration (c) atropine for bradycardia.

(3) Maintaining oxygenation.

(4) NG tube for suction: prevent vomiting and aspiration, and decompresses abdomen which can interferes with respirations if distended (paralytic ileus)

(5) Indwelling (Foley) urinary catheter. During the period of spinal shock, the urinary bladder is atonic and flaccid. Over time it becomes an upper motor neuron bladder with small capacity. Initially, an indwelling Foley catheter is placed. After 3 to 4 days, the patient is switched to intermittent bladder catheterization to keep urinary volume at less than 500 ml..

(6) Temperature regulation. Disruption of the sympathetic nerve function at **D8** or above is frequently associated with hypothermia. Euthermia should be restored by external warming, warmed IV fluids & heated inspire air if the patient is intubated.

(7) Correction of electrolytes (hypokalemia due to increase aldosterone)

(8) More detailed neurological examination.

(9) Radiologic evaluation: **(I) Cervical spine:** AP, open –mouth and lateral X-ray was taken while in rigid collar (or the head strapped to the backboard); if all 7 vertebrae and C7-T1junction are adequately visualized and are normal, and if the patient has no neck pain and is neurologically intact, then we can remove the collar (or the strip of the adhesive tape) and no further radiological exam is needed. But, if the patient had neurological deficit and was complaining from neck pain or tenderness or if there are abnormal X-ray findings then further studied are needed (oblique for cervical spine X-ray, flexion-extension views, CT scan or MRI).

(II) Thoracic and lumbosacral spine AP and lateral X-ray view for all trauma patients with RTA, complaining of back pain or unconscious. If there are positive X-ray finding or if there is neurological deficit MRI of the spine is indicated.

(10) Medical treatment specific to spinal cord injury: using **methylprednisolone** within **first 8 hours** of injury to counteract secondary phenomena especially those related to lipid peroxidation and production of oxygen-free radicals. Methylprednisolone should be given as an IV bolus of 30 mg/kg administered over 15 minutes. This is followed 45 minutes later by 5.4 mg/kg/hour continuous infusion of methylprednisolone. If the treatment is initiated less than 3 hours after the injury, it continues for the next 23 hours. If the treatment started more than 3 hours but less than 8 hours after the injury, the infusion continues for 47 hours (for a total of 48 hours). If the treatment could not be started within 8 hours after the injury, steroids are of no benifit.

(11) Surgical treatment: surgery is indicated for (a) deformity correction (b) stabilization of the spine and (c) decompression of the neurological elements.

Neural Tube Defect

Neural tube defects are a group of common congenital malformations that believed to be caused by failure of the neural tube to close.

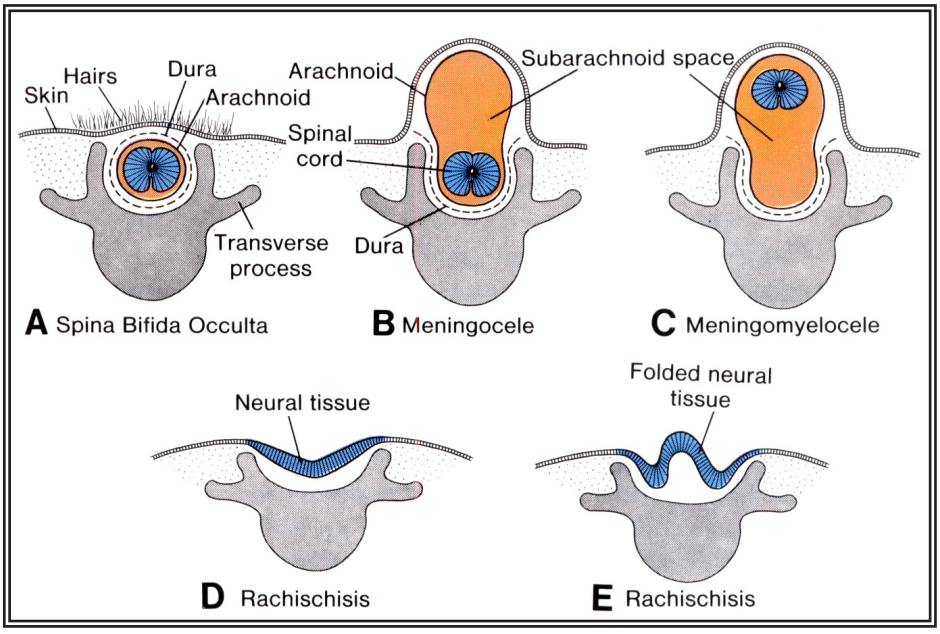
Classification

Neural tube defects can be classified, based on embryological considerations and the presence or absence of exposed neural tissue, as open or closed types. According to its location, the neural tube defect includes:

A- Spinal Neural Tube Defect

(1) ***Spinal meningocele*** is the simplest form of neural tube defect which consists of meninges only and contains cerebrospinal fluid (CSF), which is in continuity with that in the spinal canal but has no neural tissue within its confines. This entity is one-tenth as frequent as myelomeningocele and is rarely associated with ***hydrocephalus***.

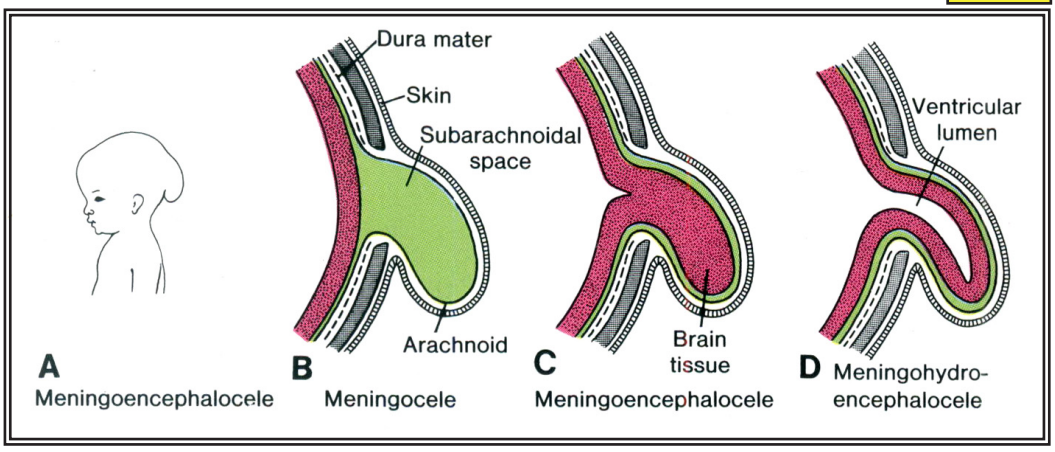
(2) ***Myelomeningocele (meningomyelocele):*** The far more common form of spinal neural tube defect is the myelomeningcele. Rudimentary dura and leptomeninges have developed around and are attached to the malformed neural tube. This type is frequently associated with ***hydrocephalus***.



(3) ***Myeloschisis,or rachischisis :*** is a term reserved for large piece of flattened neural tissue (known as placode) without any encasing meninges. Early hydrocephalus and a severe neurological deficit are present.

(4) A simple defect in the spinal laminae without herniation of tissue is known as ***spina bifida occulta***. A tuft of hair if present over the site of the defect may be the only external sign of this lesion.

B –Cranial Neural Tube Defect



Is malformation characterized by herniation of intracranial contents through a defect in the calvaria.

(1) If the cranial herniation contains only cerebrospinal fluid and meninges, it is by definition a ***Cranial meningocele.***

(2) When the herniation includes meninges and brain tissue (but not ventricles), the term ***encephalomeningocele*** or ***meningoencephalocele*** is applied.

(3) ***Encephalohydromeningocele*** or ***meningohydroencephalocele*** involves herniation of meninges, brain parenchyma, and ventricles.

(4) A simple skull defect without prolapse of brain or meninges is ***cranium bifidum occultum***.

Clinical assessment

The lesion site, type and size are determined and the cranium is assessed for any overt features of hydrocephalus. The infant should be examined neurologically and any neurological deficit should be evaluated. For Spinal Neural Tube Defect the neurological assessment of the trunk and lower limbs is based on the segmental innervations of the lower limb muscles. Bladder function should be assessed; frequently, dribbling of small volumes of urine which increases with crying or movements is indicative of future incontinence. Anal sphincter function should be evaluated by performing the ***superficial anal reflex (S3-S5)***

Imaging

Brain CT scan or MRI is useful for assessing the cranial neural tube defect and to exclude the associated hydrocephalus for neural tube defect in general. Spinal MRI is useful for evaluation of spinal neural tube defect in special circumstances.

Treatment

It must be clear that surgery on the spinal neural tube defect is not designed to repair a faulty spinal cord, which at least in the exposed portion, has not matured beyond the fourth week of pregnancy. No one can improve the neurological disability related to that embryologic disorder: but operation limits the possibilities of retrograde ascending meningitis and preserves neurological function intact. The same is applied to the cranial neural tube defect. Therefore; the surgery involve reconstruction of abnormal anatomy, and it is known as ***Repair of neural tube defect*** (example; repair of myelomeningocele or repair of cranial meningocele ….etc).

Hydrocephalus

Hydrocephalus is the abnormal accumulation of CSF within the ventricles and subarachnoid spaces. It is often associated with dilatation of the ventricular system and increased intracranial pressure.

CSF pathway

CSF is produced at a rate of about 0.30 – 0.35 ml. /minute by two distinct processes;

(1) Energy- requiring process performed by the choroid plexuses in the lateral, third and forth ventricles. This process depends on the enzyme carbonic anhydrase and can be blocked by the carbonic anhydrase inhibitor; acetazolamide (Diamox).

(2) As by-product of cerebral and white matter metabolism. After its production, the ventricular CSF flows through a series of narrowings from one compartment to the next. The compartments begin with the lateral ventricles through the foramina of Monro to the third ventricle. From there it flows through the cerebral aqueduct of Sylvius into the forth ventricle. From there it flows into cisterna magna through 3 foramina [two Lateral foramina (Luschka) and one Median foramen (Magendie)], where it mixes with the CSF from the spinal subarachnoid space. Finally, the CSF flows through the cortical subarachnoid space to be absorbed through specialized organs; the arachnoid villi, into the sagittal sinus.

6 Aetiology and Classification of hydrocephalus

(A) Obstruction of CSF pathway:

this include;

(1) Non-communicating hydrocephalus: in which the obstruction localized at level of ventricular system (lateral, 3rd. and 4th. ventricle). [This mean that the ventricular system does not communicate with subarachnoid space].

	Non-Communicating	Communicating
Congenital	Aqueduct stenosis	Absence of arachnoid granulations
	Atresia of 4th. ventricle foramina	Arnold-Chiari malformation
	Benign intracranial cyst	Meningoencephalocele
Acquired	Aqueduct stenosis	Hemorrhage
	Infection	Infection
	Tumor	Tumor

(2) Communicating hydrocephalus: in which the obstruction localized at level outside the ventricular system (subarachnoid space and arachnoid villi). [this mean that the ventricular system does communicate with subarachnoid space].

(B) CSF oversecretion: (choroids plexus papilloma).

Clinical features

(A) Infantile hydrocephalus:

(1) Enlarged head (Craniofacial disproportion).

(2) Distended scalp veins.

(3) Tense and bulging of anterior fontanel.

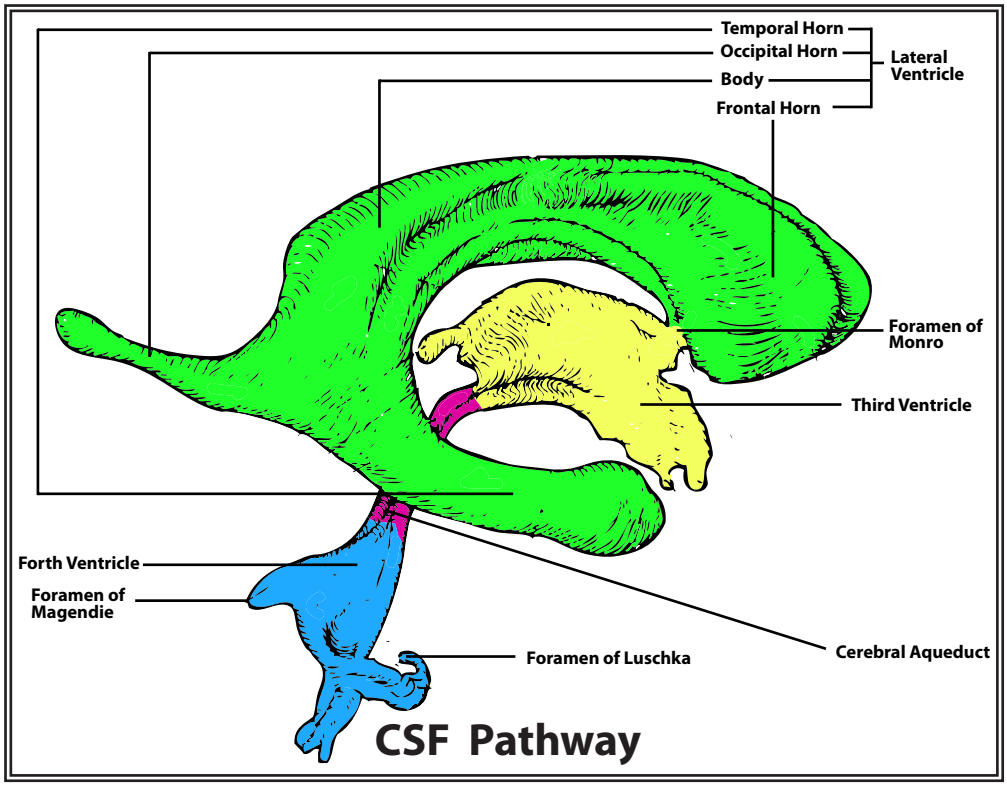
(4) Unilateral or bilateral abducent palsy.

(5) weakness of upward gaze (Sun-setting sign).

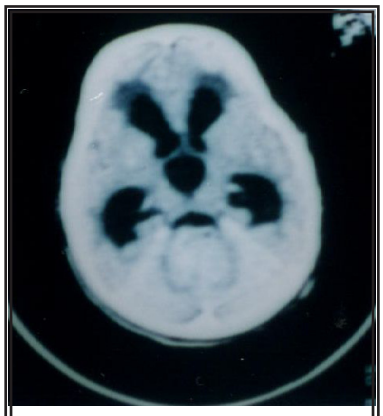
(B) Adult-type hydrocephalus:

(1) clinical Features of raised intracranial pressure and/or clinical features of brain herniation.

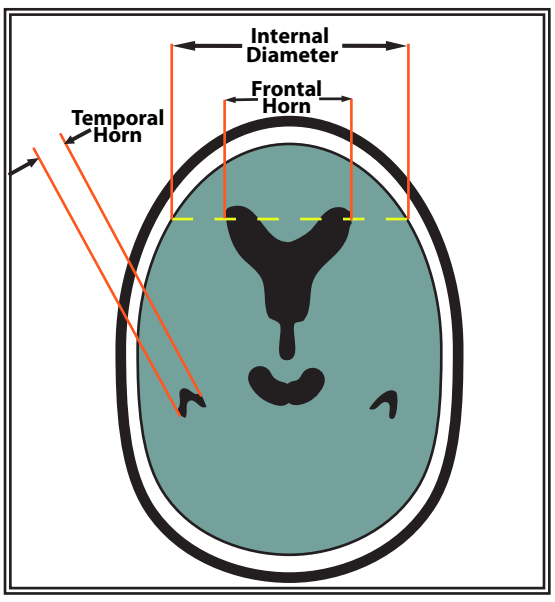
(2) weakness of upward gaze (Parinaud's syndrome)

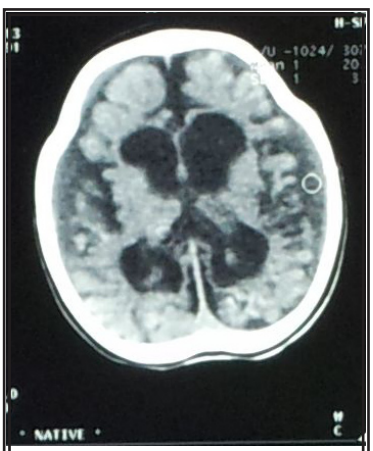


Imaging



Brain CT scan shows dilated ventricular system with effacement of cerebral gyri; the TH > 2 mm. width and the ratio of FH to ID > 0.5 (sign of hydrocephalus)





Brain CT scan shows dilatation of ventricular system and prominent cerebral gyri (sign of brain atrophy)

By CT scan or MRI, Hydrocephalus is suggested when **either**:

A- The size of both temporal horns is more or equal to 2 mm. in width (in the absence of hydrocephalus the temporal horns should be barely visible), and the sylvian & interhemispheric fissures and cerebral sulci are not visible (this is known as effacement) ***OR***

B- Both TH are more or equal to 2 mm. in width and the ratio of the largest width of Frontal horns to the Internal diameter from inner-table to inner-table of skull at this level, is more than 0.5 .

These finding may or may not associated with interstitial edema which present as periventricular low density on CT scan or periventricular high intensity signal on T2 MRI. Hydrocephalus should be differentiated from cerebral atrophy in which there is enlargement of the ventricles due to loss of cerebral tissue. In this case the sylvian & interhemispheric fissures and cerebral sulci are visible and enlarged to a proportion of almost equal to that of ventricular system enlargement.

Treatment of hydrocephalus

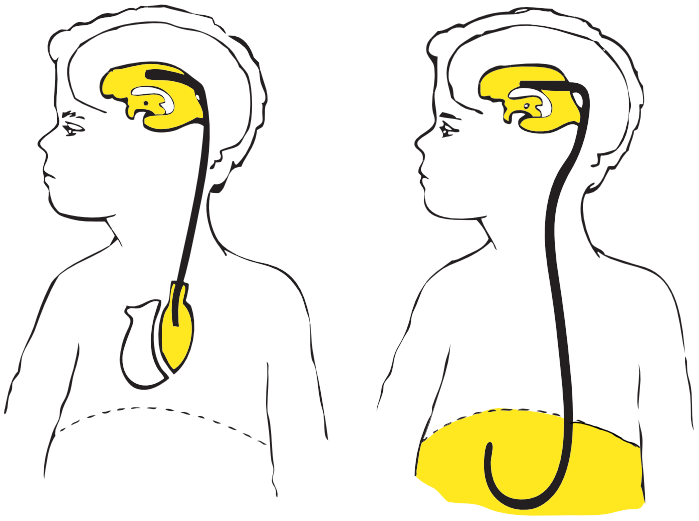
Hydrocephalus remains a surgically treated condition. Acetazolamide may be helpful in temporizing. Normal sized ventricles are not the goal of therapy. Goals are optimum neurologic function and good cosmetic result.

Surgical options include;

(1) Eliminating the cause of hydrocephalus (tumor removal).

(2) Third ventriculostomy (opening of the 3rd. ventricle to the basal cisterna that communicate with the subarachnoid space to by-pass the obstruction by using flexible or rigid endoscope introduced through a frontal Burr-hole). Suitable for patients with tri-ventricular hydrocephalus.

(3) Shunting: this is a method for CSF diversion to a place where it could be absorbed. This is usually done using special tube system containing valve.



Ventriculo-Atrial shunt **Ventriculo-Peritoneal shunt**

Shunt systems are classified according to the sites of CSF diversion into;

(a) Ventriculo-Atrial shunt (divert CSF from ventricular system to the Rt. atrium)

(b) Ventriculo-Peritoneal shunt (divert CSF from ventricular system to the peritoneal cavity).

Shunt Complications:

(1) Infection: results in meningitis, peritonitis or inflammation extending along the subcutaneous channel. In patient with ventriculo-atrial shunt, bacteraemia may lead to shunt nephritis. Staphylococcus epidermidis or aureus are usually involved, with infant at particular risk. Prophylactic antibiotics may minimized the risk of infection, but, when established, eradication usually requires shunt removal.

(2) Subdural hematoma: ventricular collapse pull the cortical surface from the dura and leaves a subdural CSF collection or tears bridging veins causing subdural hemorrhage. The risk may be reduced with a variable pressure or programmable valve.

(3) Shunt obstruction: blockage of the shunt system with choroid plexus, debris, omentum or blood clot results in intermittent or persistent recurrence of symptoms. Demonstration of an increase in the ventricular size compared to a previous baseline CT scan confirm shunt malfunction.

(4) Low pressure state: following shunting, some patient developed headache and vomiting on sitting or standing. This low pressure state usually resolve with high fluid intake and gradual mobilization. If not, insertion of an antisyphon device or conversion to high pressure valve is required.

(5) Seizures: Prophylactic seizure medications are rarely indicated in the management of shunted patient.

(6) Spontaneous pneumocephalus: Thinning of skull base and enlargement of congenital bone defects as result of raised intracranial pressure, or during initial episodes of trauma that results in skull base fractures; all these lead to ingress of air once the intracranial pressure has been lowered by shunt.

(7) Ascitis.

(8) Bowel perforation: this complication can occur either at time of shunt placement or in another time later.

8 Questions:

(**01**) What are the differences between:
(a) Two patients with GCS = 7 ; One of them had **E**2**V**2**M**3 and the other **E**1**V**2**M**4
(b) Vasogenic and Cytotoxic brain edema.
(c) Spinal and Neurogenic shock
(d) Brown-Sequard syndrome and Central cord syndrome in regarding to the prognosis.
(e) Using Methyl-Prednisolone before and after 8 hours from the time of spinal cord injury.
(f) Meningocele and Myelomeningocele.
(g) Infentile and adult-type of hydrocephalus.
(h) Shunting and third ventriculostomy in treatment of hydrocephalus.
(i) Hydrocephalus and cerebral atrophy.

(**02**) The following statements are **True**:
(a) Steroid is effective in treatment of brain edema that associated with head injury.
(b) In right uncal brain herniation it is possible to find right sided body weakness.
(c) Right frontal intraparenchymal hematoma could cause loss of conscousness if not associated with brain herniation.
(d) Brainstem intraparenchymal hematoma could cause loss of conscousness if not associated with brain herniation.
(e) In patient with brain tumor , a late evening frontal headache is characteristic.
(f) Tonsillar herniation is type of supratentorial brain herniation.
(g) Bradycardia and hypotension is characteristic signs of raised intracranial pressure.
(h) Tachycardia and hypotension is characteristic signs of spinal cord injury.
(i) Absence of bulbocavernosus reflex suggest complete spinal cord injury.
(j) Positive superficial anal reflex indicate incomplete spinal cord injury.
(k) Significant neurogenic shock increases with cord injuries blow D6.
(l) Myelomeningocele is rarely associated with hydrocephalus.
(m) Repair of myelomeningocele will result in improvement to the neurological state.
(n) Diagnosis of hydrocephalus is suggested if the size of both temporal horns is more than 2 cm in brain CT scan.
(o) Staphylococcus epidermidis or aureus are usually the commonest microorganisms involve in shunt infection.

(**03**) At Emergency Unit you received a 38 years male who was a victim of Road traffic accident 2 days ago. There was no loss of consciousness, vomiting or fit. He was unable to move his lower limbs and only flexion of the elbow was possible.
(a) What is the level of spinal cord injury? What further clinical examination do you need to confirm it ?.
(b) Name single test to define the completeness of the injury and how ?.
(c) Name the imaging of choice (just one) for this patient ?.
(d) What do you expect the possible findings regarding his pulse, blood pressure ?.
(e) After 2 weeks, the patient developed severe early morning headache. What is your next step and what is the possible diagnosis ?.

(**04**) A 18 years male patient with history of stabbing of his back by a knife 5 hours ago. There was parasthesia in the left lower limb and unable to move his right lower limb.
(a) What do you expect to find in sensory examination of the right lower limb ?.
(b) Is there is possible role of using steroid in this patient ? (yes or No)
(c) If your answer in (b) is "Yes"; what type of steroid do you want to use and how ?. If your answer in (b) is "No" then describe why ?.
(d) What do you expect the prognosis of his injury ?.

(**05**) At Emergency Unit you received a 22 years female who was a victim of Road traffic accident 3 hours ago. There was no loss of consciousness, vomiting or fit. She was quadriplegic.
(a) Describe your lines of management regarding her injury.
(b) After 3 hours, her GCS became 9 and there was dilatation of left pupil;
(I) What you would like to do ?
(II) What are the possible causes of her condition ?.

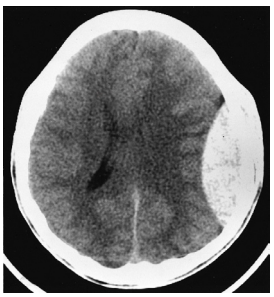
(**06**) Name the possible diagnosis in the following situations:

(a) pulse = 50 beats/min. and Blood pressure = 180/120 mmHg in patient with head injury.
(b) pulse = 50 beats/min. and Blood pressure = 85/40 mmHg in patient with head injury.
(c) Right dilated pupil and left hemiparesis in patient with head injury.
(d) Crescentic hyperdense lesion in brain CT scan in patient with head injury.
(e) Loss of consciousness for 2 hours and negative brain CT scan in patient with head injury.
(f) Paraplegia with intact position and vibration sense in patient with spinal cord injury.
(g) Absence of bulbocavernosus reflex in patient with spinal cord injury.
(h) Voluntary contraction of the sphincter during rectal examination in patient with spinal cord injury.
(i) There is a disproportionately greater motor deficit in the upper limbs than in the lower limbs in patient with spinal cord injury.
(j) There are both upper and lower motor neuron signs in the lower limbs which tend to be symmetrical in patient with spinal cord injury.

(**07**) A 40 years male patient with history of fall from height and watery discharge from right ear since yesterday. There was history of loss of consciousness for 1 hour. Examination reveals right temporal scalp wound, paraplegia and D6 sensory level. Dorsolumbar Spine X-ray (AP & Lateral) showed D3 wedge fracture.

(a) Enumerate the additional necessary imaging investigation for this patient.
(b) How do you observe such patient regarding his brain injury at Neurosurgical ward?
(c) Is there any benefit to treat his paraplegia with steroid? (Yes or No).
(d) What type of brain injury does he have if his brain CT scan was negative?
(e) After 1 week, the bulbocavernosus reflex is positive but he still paraplegic. What does this mean ?
(f) From all the above data, what do you expect the prognosis regarding his paraplegia?

(**08**) At Emergency Unit, You received 40 years female patient who was a victim of Road traffic accident. Her Glasgow Coma Scale (GCS) was 10, Pulse was 140 beat/min., Blood pressure was 80/40 mmHg., Brain CT scan was as in the following image:

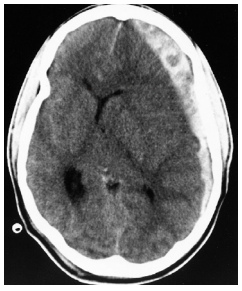


(a) What do you expect to find in the examination of the motor system ?.
(b) Is it possible to explain her vital signs to the CT findings, and why ?.
(c) Is it possible to see right dilated pupil after 2 hours of observation, and why ?.
(d) What type of fluid would you preferred to give in this situation ?, and why ?.

(**09**) At Intensive Care Unit (ICU). A 30 years male patient, with history of trauma to the head by iron pipe, had been referred to you from Neurosurgical ward after deterioration in his neurological condition. His brain CT scan showed generalized brain edema:

(a) Enumerate your lines of management.
(b) What you would do if he developed left dilated pupil after 3 hours ?.
(c) What you would do if there is improvement in his condition after 10 days of ICU monitoring ?.

(**10**) A patient with history of trauma to the head was admitted to neurosurgical ward. Examination revealed that the patient was able to open his eye spontaneously and obey commands but not able to speak although he was able to understand the speech. Motor system examination revealed right sided weakness. He had right parietal scalp wound. His Brain CT scan showed the following image:



(a) Calculate the GCS.
(b) Describe his brain CT scan image.
(c) What is the possible cause of his speech abnormality?
(d) Name the mechanism of head injury that cause his right sided weakness.
(e) What is the possible diagnosis according to his brain CT scan image ?.
(f) According to the above information give the possible duration of his head trauma.

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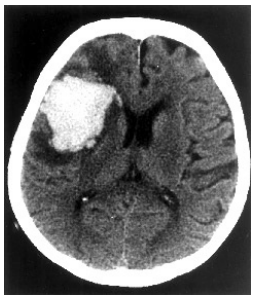
(**11**) A patient had been referred from ICU (Intensive Care Unit) to Neurosurgical ward after stabilizing of his condition. Examination revealed GCS=9 and he had left sided weakness. He had right temporal scalp wound. His brain CT Scan showed the following image:



(a) How do you observe this patient?.

(b) Name the mechanism of head injury that causes his left sided weakness ?.

(c) Describe his brain CT scan image & what is the possible diagnosis?.

(d) After 5 days the GCS became 6 and he developed left dilated pupil.

(I) What is your next step ?.

(II) What is the possible diagnosis?

(III) Name the mechanism of head injury that causes his left dilated pupil.

(**12**) 20 days infant with history of progressive enlargement of the head for 2 weeks duration. Examination reveal lower back mass and normal movement of his lower limbs since birth.

(a) What is the possible diagnosis of his lower back mass ?.

(b) How would you evaluate this patient regarding his lower back mass clinically ?.

(c) What would you expect to find in the examination of the head ?.

(d) What type of imaging would you preferred to use for evaluation of his head ?.

(e) How would you confirm the diagnosis in (a) ?.

(f) What are the possible types of this mass based on the investigation in (e) ?.

(g) What is your diagnosis if the above imaging [in (d)] showed enlargement of the forth, third and both lateral ventricles with absence of cerebral sulci, sylvian and interhemispheric fissure ?.

(h) What is the name of the treatment for this lower back mass ?.

(i) What type of treatment would you preferred to use for his head enlargement ?.

(j) Does the treatment in (i) result in normalization of his head ? (Yes or No).

(k) 3 months after the treatment [in (i)], the new imaging still shows the enlargement of the ventricular system but with the presence of cerebral sulci, sylvian and interhemispheric fissure. What is your diagnosis ?

(l) 6 months after the treatment [in (i)], the patient presented with attacks of vomiting and examination revealed tense anterior fontanel and impairment of upward gaze, and the new imaging still shows the enlargement of the ventricular system but with the absence of cerebral sulci, sylvian & interhemispheric fissure. What is your diagnosis ?

(**13**) 2 days infant was born with large head and occipital mass. Brain CT scan showed enlargement of the third and both lateral ventricles with absence of cerebral sulci, sylvian and interhemispheric fissure. The CT scan also showed herniation of small part of the right lateral ventricle with some of brain tissue into the occipital mass.

(a) What is the possible diagnosis of the occipital mass ?.

(b) What are the clinical signs that you could find in the head examination ?.

(c) Name the associated pathology that was present in his brain CT scan beside his occipital mass and what are the options to treat this pathology ?.

(d) Name the surgical treatment of the occipital mass.

(e) After 2 months from the treatment in (c), a new brain CT scan was performed and the result was brain atrophy. Describe the CT findings.

(f) 3 months after the evaluation in (e), his mother gave history of progressive enlargement of his head; Describe the possible clinical findings in the head examination.

(g) What is the possible diagnosis in (f).

(**14**) Enumerate the possible complications that associated with insertion of shunts that used in treatment of hydrocephalus.

(**15**) What are the criteria of diagnosing hydrocephalus in brain CT scan ?. Name the type of the associated edema and describe its treatment if caused by brain mass.