TUTORIAL 10

THE EXAMINATION OF CENTRAL NERVOUS SYSTEM

OVERALL OBJECTIVES

At the end of this module, students should to be able to:

- **Recognise** by appropriate history and examination:
  - The anatomical site of a neurological lesion i.e. to distinguish between upper and lower motor neuron lesion
  - The general pathology of the lesion i.e. to distinguish between vascular, inflammatory, metabolic, neoplastic or degenerative lesions
- **Investigate** appropriately and manage common and life threatening neurological problems in infants and children
- **Describe common indications** for lumber puncture, EEG, cranial ultrasonography, CT and MRI scans

Extra General Features for CNS examination

- Dysmorphology
- Neurocutaneous lesions – café au lait spots etc.
- Meningomyelocele, gibbus and kyphoscoliosis
- Head and abdomen for VP shunt scars
- Hypertrophied calf muscles, wasting, fasciculations and trophic signs

PARTS OF CENTRAL NERVOUS SYSTEM

Cerebral cortex: cortical functions

Frontal lobe (precentral gyrus is motor strip)

- Controls
  - Voluntary movements
  - Personality
  - Social behaviour
- Labile mood → possible frontal lobe involvement

Temporal lobe

- Responsible for memory and perception of smell
- Contains lower fibers of optic radiations

Parietal lobe (post central gyrus is sensory strip)

- Responsible for conscious sensations
- Contains upper fibers of optic radiations

Occipital lobe

- Responsible for analysis of visual information

Broca's area

- Responsible for speech production and articulation

Wernick's area

- Responsible for language comprehension

Motor system

Upper motor neuron (UMN)

1) UMNs pass down from cortex to corona radiata, internal capsule, midbrain, pons, medulla oblongata and spinal cord
2) Axons of UMN arborize around the LMN in the anterior horn
3) Axons of UMN while passing through the internal capsule occupy the genu and anterior 2/3rd of posterior limb
4) In midbrain these fibers occupy the middle 3/5th of the peduncles
5) In pons these fibers are split by the nuclei of the pons and pontine fibers
6) In medulla, these fibers are condensed anteriorly to form a bulge called pyramid
7) Majority of these fibers (90%) docusate in the lower part of medulla (and some uncrossed fibers) forming lateral corticospinal tract
8) While the rest (10%) pass uncrossed to the same side forming anterior corticospinal tract

Lower motor neurons (LMN) consist of:

1) Anterior horn cells & homologous cell in the brain stem
2) Fibers passing through anterior spinal nerve roots and peripheral nerves to muscles
3) Motor nuclei of cranial nerves

Extra-pyramidal system

This includes all the higher centres in the Nervous System

1) Basal ganglia
2) Substantia nigra
3) Red nucleus
4) Sub-thalamic nuclei
5) Reticular nuclei
6) Olivary nuclei

Five major extrapyramidal tracts

1) Reticulo-spinal tract
2) Rubrospinal tract
3) Olivo-spinal tract
4) Tectospinal tract
5) Vestibulo-spinal tract

All 5 tracts run in the anterior column of spinal cord & end around the anterior horn cells. Their functions include: movements, posture and tone in the voluntary muscles.

Lesions in these fibers produce three clinical features:

1) Change in the muscle tone
2) Involuntary movements
3) Slowing of the movements

SENSORY SYSTEM

There are 3 neurons:

1) **The cyton (cell body) of the first neurons** lies in the root ganglia of spinal cord and cranial nerve. It conveys 2 types of sensations:

   Superficial sensation (from the skin):
   1) Sensation of touch
   2) Temperature
   3) Pain

   Deep sensations include:
   1) Sense of position, movements and vibration
   2) Sense of size and shape of an object
   3) Sensations of muscle and joint movements

   Central axon of the first neurone arborises around the cells situated at the base of grey horn of the spinal cord. The fibers which convey deep sensations go up in posterior white column and end in the nucleus gracilis and cuneatus. From these nuclei second axons arise and cross to the opposite side.

2) **Cyton of the second neuron** lies in the nucleus gracilis and nucleus cuneatus in the medulla. They cross over to the opposite side and form two tracts:
   - Anterior spino-thalmic tract – crude touch and pressure signals
   - Lateral spino-thalmic tract – pain and temperature signals.

   Spinothalmic fibers go upward through the spinal cord, the brain stem and finally into thalamus

3) **Cyton of third neuron** lies in the thalamus. From here fibers arise and go to the sensory cortex (by way of the internal capsule) lying in the parietal lobes behind the fissures of Rolando. Sensory tracts passing through the brain stem receive fibers from sensory nucleus of trigeminal nerve.

SPINAL CORD

- Extends from foramen magnum down till upper border of the 2nd lumber vertebra
- Membranes of spinal cord end in the second sacral vertebra.
- Spinal cord terminates at L1 in adults and L3 in infants and children
- In upper cervical regions, spinal nerve roots are short and horizontal but lumber and sacral roots form vertical leash of nerves called as cauda equina
- There are 7 cervical vertebrae but 8 cervical roots
- There are 4 coccygeal vertebrae but one coccygeal spinal root
- Physiologically spinal cord is composed of supper-imposed segments:

Segments:	C 8	T 12	L 5	S 5	C 1-2 (coccygeal) = 31
Vertebrae:	C7	T 12	L 5	S 5	C4 (Three fused) = 30

Summary of Neurologic examination of a child

1) Mental state assessment: Assesses various zones of cerebral cortex
2) Cranial nerves assessment: Evaluate integrity of brain stem
3) Motor examination: Evaluates upper and lower motor neuron function
4) Sensory examination: Assesses peripheral sensory receptors & their central reflections
5) Deep tendon reflexes: Assess upper and lower motor neuron connections
6) Gait assessment: Puts motor system into a dynamic state for functional assessment.

CLINICAL EXAMINATION

Here the process differs from the other systems:

- First just look at the child for abnormal posture and movements
- Let the child stand, walk, sit & then get up from the floor, this will give you important information

1. LEVEL OF CONSCIOUSNESS

Consciousness has two components

- **State of consciousness:** *how awake the child is* – this determines the integrity of ascending reticular activating system
- **Content of consciousness:** *how aware the child is* – this determines integrity of cerebral cortex, thalamus and their connections

Mental state evaluation: consists of alertness and intellectual abilities

Alertness is assessed:

In infants by

- Observing spontaneous activities
- Feeding behaviour
- Fixing and following
- Response to tactile, visual and auditory stimuli

In older children

Orientation to time, place, space and person

Intellectual abilities are assessed by language skills

Receptive (*Wernicke) aphasia:* It is the inability to understand language i.e., fluent speech but nonsensical. For example, Tina had a stroke which involved Wernicke's area. She has difficulty understanding when spoken to.

Expressive (*Broca) aphasia:* It is the partial loss of the ability to produce language (spoken, manual or written) although comprehension generally remains intact. For example, Nandi had a stroke which involved Broca's area. She knows what she wants to say but has difficulty expressing herself. This should not be described as confused.

Receptive and expressive (*Global) aphasia:* It is severe form of non-fluent aphasia caused by damage to the left side of brain, that affects receptive and expressive language skills (needed for both written and oral language) as well as auditory and visual comprehension.

Glasgow coma scale (GCS)

EYE OPENING		VERBAL		MOTOR	
Spontaneous	4	Oriented	5	Obeys	6
Voice	3	Confused	4	Localises pain	5
Pain	2	Inappropriate words	3	Withdaws	4
None	1	Vocalises sounds – no words	2	Flexion	3
		No vocalization	1	Extension	2
				None	1

Severity of head injury:

- GCS <8: Severe
- GCS 9 to 12: Moderate
- GCS >13: Mild injury

AVPU score:

- **A**lert
- Responsive to **V**oice
- Responsive to **P**ain
- **U**nresponsive

P & U scores indicate coma and correspond to GCS of 8 or less

Signs of upper and lower motor neuron lesions

UMNL	LMNL
• Paralysis affects group of muscles	• Individual muscles are paralysed
• Muscle tone increases	• Muscle tone decreases
• Muscle wasting absent	• Muscle wasting present
• Babinski sign positive	• Babinski sign negative
• Deep reflexes exagerated	• Deep reflexes diminished
• Clonus present	• Clonus absent
• Trophic changes absent	• Trophic changes present
• Reaction of degeneration is not present	• Reaction of degeneration is present
• UMNL appears as stiffness	• LMNL is detected by weakness
• Involuntary movements (IM) absent	• IM present: fasciculations
Muscle fasciculations indicate denervation from disease of anterior horn cells or peripheral nerve	

Bulbar palsy

- Bulbar palsy is LMNL
- Bulbar refers to the lower brain stem (medulla oblongata) which is the control centre for cranial nerves 9 to 12
- Cranial nerves 9, 10 & 12 are involved in bulbar palsy; palsy means weakness

- If muscles supplied by these cranial nerves are weak then it referrs to as bulbar palsy
- There is no emotional lability.

Pseudobulbar palsy

- Pseudobulbar palsy is UMNL which affects the corticobulbar fibers
- Corticobulbar fibers are the neurons which connect cerebral cortex to cranial nerve nuclei in the medulla oblongata
- In pseudobulbar palsy these corticobulbar fibers get disrupted causing mess up of voluntary control but there is no intrinsic damage to the cranial nerves or brain stem
- Examples are
    - Stroke involving both hemispheres of brain
    - Degenerative disorders

2. SIGNS OF MENINGEAL IRRITATION

Neck stiffness: There is an increased resistance to passive flexion of neck due to inflammation of meninges. Patient feels discomfort or pain when trying to turn, move or flex the neck.

Kerning's sign: When patient is in supine position with hips flexed at 90 degree, note pain and inability to fully extend the knees.

Brudzinski's sign: When patient is in supine position, do flexion of neck. There will be involuntary flexion at the knees and hips.

Jolt sign: Tell the patient to shake his head. Patient will experience severe headache or will not be able to shake his head due to inflamed meninges.

Picture: Examintion for signs of meningeal irritation: Neck stiffness, Brudzinski's sign, Kernig's sign

Meningism is not a sign of meningitis or meningeal irritation. It is neck stiffness due to non-meningitic causes; for example, neck muscle sprain, or soft tissue injury. It can also be due to an acute febrile illness in children which involves triad of nuchal rigidity, photophobia and headache. It therefore requires differentiation from other CNS problems with similar symptoms, including meningitis and some types of intracranial haemorrhage.

Signs of raised intracranial pressure

1) Irritability
2) Headache
3) Vomiting
4) High pitched cry
5) Full or buldging fontanelle in infants
6) Confusion
7) Somnolence
8) Coma
9) Cushing's triad (Bradycardia, Hypertension, Irregular breathing)

Important to note that:

- Raised ICP produces confusion which may mimic global encephalopathy
- Signs of compression of cranial nerve III: ptosis, anisocoria (unequal pupil size)
- Signs of compression of cranial nerve VI: lateral rectus weakness

Causes of raised ICP

CNS causes

1) CNS infections
2) Transverse myelitis
3) Guillain Bare Syndrome (GBS)
4) Tuberous sclerosis
5) Blood in subarachnoid space
6) Neurofibromatosis type 1

Other causes

1) Trauma / haemorrhage
2) Hyperaemia
3) Hydrocephalus
4) Tumors and abscesses
5) Metabolic abrasions
6) Hypoxic ischaemia

3. STANCE

Patient should stand upright with feet closed together and **eyes opened**

- Observe for swaying or lurching
- If present consider cerebellar ataxia

Patient should stand upright with feet closed together and **eyes closed**

- Observe for swaying/lurching or loss of balance.
- If present consider sensory ataxia. This can be due to proprioceptive (somatosensory) deficit due to dorsal column dysfunction and is not primarily a test for cerebellar dysfunction.
- This is also called as positive Romberg sign.

Romberg's Sign:
It is also called as Romberg's Test or Romberg's Manoeuvre which is used in an examination of neurological function for balance. This sign is also used to test for driving under the influence of an intoxicant.

The examination is based on the premise that a person requires at least two of the three following senses to maintain the balance while standing.

- Proprioception (the ability to know one's body position in space)
- Vestibular function (the ability to know one's head position in space)
- Vision (to monitor and adjust for changes in body position)

A patient who has a problem with proprioception can still maintain balance by using vestibular function and vision.

Procedure: patient should stand upright with slightly opened legs and you observe:

- If already ataxic – will get worse on closing eyes
- If not already ataxic – will become slightly ataxic on closing eyes

If there is sensory ataxia (dorsal column lesion), patient will need to use eyes to position feet and stand upright. These patients on closing eyes rely only on dorsal column to keep balance.

Picture: Method to elicit Romberg's sign

4. GAIT

Advise the opatient to:

- Walk normally for 10 meters & turn through 180^{o} – watch width of steps tendancy to veer away
- Toe walk/heel walk: if there is distal weakness, consider peripheral neuropathy

- Heel to toe walk (tendom gait) in straight line: If there is gait instability (broad based unsteady gait) consider gait ataxia that may be due to cerebellar dysfunction
- Lie down and get up quickly. Patient will role over prone position, get on knees and get up this is called as positive Gower's sign seen in proximal muscle weakness
- Stand and walk normally:
  - Scissors like stance and gait →consider bilateral UMNL
  - Hemiplegic gait → consider unilateral UMNL
  - Waddling gait may be due to myopathic proximal muscle weakness

NB! Access how motor & co-ordination systems are functioning:

- A 6 years old child can tendom walk and walk high on toes and heels
- An infant can creep, crawl or cruise or small child can walk or crawl

5. CRANIAL NERVES

Folowing is the list of Cranial Nerves and their place of origion

- 1, 2 out of the brain
- 3, 4 mid brain
- 5, 6, 7 ,8 pons
- 9, 10, 11, 12 medulla

Motor neuclei of Cranial Nerves are supplied by the pyramidal tract of both sides except the two:

- Hypoglossal and
- Part of the 7th cranial nerve which supplies the lower half of the face

EXAMINATION OF CRANIAL NERVES

1. Olfactory

It is difficult to examine this nerve in small children but we can test it in older & co-operative children.

Method: Close one nostril of the patient and bring some vinegar or mint closer to the other nostril and ask the patient to smell:

- If the patient has loss of smell, this is called as Anosmia
- If the patient has perversion of smell this is called as Parosmia
- When a person smells something which is no there, it's called as Phantosmia

Causes of Anosmia

- Severe head injury
- Upper respiratory tract infection
- Meningitis
- Invasion of cancer i.e., ethmoid tumors etc.

Causes of Parosmia

- Severe head injury
- Sinus infection

- Chemical exposure
- Side effects of certain drugs
- Certain conditions of Psychosis (especially elderly)
- Brain tumors

2. Optic nerve

This nerve is examined for: Visual acuity, visual field, colour vision and fundus

Visual acuity: This is the sharpness with which the details and contour of the object are perceived. Test it for near vision and far vision.

- For near vision: ask patient to read a book at a distance for about 10 inches
- For far vision: Use standard Snellen's Chart
- Visual acuity of the newborn is 20/200 and for an infant at 6 months is 20/20
- Best method of finding out if a child can see is to check if the child fixes and follows your smiling face. Babies should rapidly fix a large object and follow it
- The examiners face is the best visual target. In infants, the visual acuity can be assessed by the ability to follow rolling white balls of varying sizes
- From the age of above 3 years, a more accurate estimate of visual acuity can be made using the Sheridan-Gardiner Test.

Procedure:

- Mother holds a card with a number or letter on it
- The examiner then shows the child one of the letters on an identical card and asks the child to point to the same letter on his own card
- When the child has understood the test, the examiner moves to a distance of 6 meters and shows the child a series of letters of descending size until the child fails to make a match
- This test is also useful in assessing visual acuity in patients with whom the examiner has no common language.

Causes of blindness

- Sudden blindness in both eyes
  - Bilateral occipital lobe infarction
  - Bilateral occipital lobe trauma
  - Bilateral optic nerve damage
- Sudden blindness in one eye
  - Retinal artery or vein occlusion
  - Temporal arteritis
  - Optic neuritis
  - Migrain
- Gradual blindness bilateral
  - Cataract
  - Acute glaucoma
  - Diabetic retinopathy

- Bilateral optic nerve/optic chiasma compression
- Vitamin A deficiency

Visual fields

The full extent of this vision is called as the visual field. Forexample: when looking at an object, we not only see that object but also a number of other objects in the neighbourhood, more or less distinctly.

The simplest method of assessning the visual field is comparing the extent of the patient's visual field with your own; testing the field of confruntation. Both eyes must be tested together first and then each must be tested separately.

Confrontation test

- Sit opposite the patient, at a distance of about one meter from him.
- If his right eye is to be tested, ask him to cover his left eye with his hand and look stadily at your left eye.
- Cover your right eye with your right hand and gaze at the patient's right eye.
- Hold up your left hand in a plane midway betweeen the patient's face and your own, at almost a full arm's length to the side. Keep moving the fingers of the hand and bring it near until you can just perceive the movements of the fingers 'with the tail of your eye'.
- Ask the patient whether he/she can see the movements, telling him/her meanwhile to be sure not to take his own eye off yours. If he/she fails to see the fingers, keep bringing them closer untill he/she can see them.
- Test the field in this fasion in every direction; upwards, downwards, to the right and to the left; using the extent of your own visual field for the purpose of comparison.
- Followings may be noted:
  - **Central vision defects**: due to disease of macula
  - **Concentric contraction of the field:** due to optic arophy
  - **Hemianopias:** loss of sight in one half of the visual field
    - **Homonymous hemianopia**: same halves of both the visual fields are affected
      - Cerebral haemorrhage
      - Neurosurgical procedure
    - **Hetronymous hemianopia:** opposite halves of both the visual fields are affected
    - **Bitemporal hemianopia:** outer halves of both (temporal or lateral), the right and the left visual field affected
      - Pituitary adenoma
      - Craniopharyngioma
      - Meningioma

Colour vision

Colour vision is most easily tested by the use of pseudo-isochromatis plates, the best known being those of Ishihara. People with defective colour vison confuse certain colours. These plates are constructed in a way that a person with abnormal vision will read a different number to a normal person on the same plate.

Ophthalmoscopy

Examination of the fundus of the eye, i.e. the retina and its associated structures, with an ophthalmoscope is an essential part of every complete medical examination. Valuable infromation can be obtained about the state of the optic nerve head and of the arteries and veins of the retina, in addition to the detection of local ophthalmic disorders.

Method of doing ophthalmoscopy:

- Check that the ophthalmoscope actually works: check batteries
- When switched on, the emitted light should be:
  - **Bright:** turn it to maximum
  - **White:** ignore all other colours
  - **Circular:** ignore all slits and crosses; turn the dial untill you get a round circle
- Ask the patient to remove his/her glasses but you can keep your or remove
- Switch the room lights off or make them dim
- Explain the procedeure and warn the patient that bright light can hurt a little
- Use 1% tropicamide in each eye and wait for 15 minutes to dilate the pupil
- Sit opposite the patient; confrontation position
- Tell patient to fixate on a precise area; corner of the room or any apecific object.
- Instruct the patient to look at this point no matter what; even if someone come in their way
- This spot should be located so that they are looking slightly away from you when they are examined i.e., to the left when you examine the right eye and to the right when you examin the left eye
- Its better that you examine the patient's left eye with your left eye and right eye with your right eye
- Now place your hand on the patient's forehead so that your fingers are splayed but your thumb is on the upper lid
- Look at the structures from front to back using the following ophthalmoscope settings:
  - Cornea +20
  - Iris +15
  - Lense +12
  - Vitreous +4
  - Fundus 0 (rotate the dial of the ophthalmoscope untill the vessels or the optic nerve is clearly seen.

Followings need to be looked for during Fundoscopy

1. **Red reflex:** media opacities obscure the red reflex like corneal scars, cataract, viterious haemorrhage
2. **Optic disc:** look for optic disc size, colour (pallor or congestion), cup disc ratio. Margins, haemorrhages, new vessels, collaterals. Pale and clearly demarcated disc indicates optic atrophy; yellow grey disc with blurred margins/haemorrhages indicates bilateral papilloedema and new vessels on the disc may indicate diabetic retinopathy etc.

3. **Vessels:** Follow the retinal vessels out from disc to periphery. Look for haemorrhages, excudates, abnormal vessels and pigmented lesions. Also look for microaneurysms, blot haemorrhages, hard exudates, cotton wool spots etc.
4. **Macula:** this is a structure temporal to the disc. To examine the macula, ask the patient to look at your ophthalmoscope's light and note the white foveal reflex in the middle of the macula. A circinate ring of hard excudate, haemorrhage or pigment deposition are the most common findings.
5. The ophthalmoscope can also be used for examining the anterior part of the eye by turning the lens dial to +10.
6. Record any abnormalities in a diagram using the disc diameter as a reference measure
7. Explain the patient that he/she may have blurred vision for 2-3 hrs after dilatation

Following may be noted during ophthalmoscopy

- Optic atrophy
- Papillooedema
- Chorioretinitis
- Retinitis pigmentosa

3. Occulomotor, 4. Trochlear, 6. Abduscens

Look for ptosis:

If ptosis is present, consider 3rd nerve involvement. Then see if the eyes move in all directions. If they do then these nerves are intact. A 6th nerve lesion means that the eye on the affected side cannot look laterally.

Check if the pupils are equal and reacting to light (PEARL)

Size of the pupils depends upon a balance of parasympathetic & sympathetic innervation

- Parasympathetic stimulation will cause pupillary constriction
- Sympathetic stimulation will cause pupillary dilatation

CHECK FOR NORMAL PUPILLARY REFLEXES

Direct light reflex:

- Examine each eye separately with the patient in an indirectly illuminated place
- Ask the patient to look at a distance (to relax his/her accomodation)
- Shine a bright light into the eye to be tested
- The pupil should contract immediately and then dilate again a little and after undergoing a few slight oscillations, settle down to a smaller size.
- When the light is switched off, the pupil should rapidly dilate to its previous diamater

Consensual light reflex:

When the bright light is shone into one eye, both pupils contract; and if the light is shut off from one eye both pupils dilate slightly. The former is the **consensual light reflex**.

Method:

- It is tested by keeping one eye in the shade while shining a bright light into the other one.
- The effect on the pupil of the unilluminated eye is then observed. This is due to the decussation of some of the fibers in the optic nerves at the optic chiasma.

Accommodation reaction:

The pupils become smaller on accomodation for a near object (miosis). Accomodation is only rarely lost in brain stem lesions, but may be impaired with lesions of the oculomotor nerve and in certain neuropathies in which there is autonomic involvement.

Method:

- Hold one finger close to the patient's nose.
- Ask him to look away at a distant object.
- Then ask him to look quickly at your finger.
- As the eyes converge to accomplish this pupils become smaller.
- If the patient is blind, the test may still be carried out by getting him to hold up his own finger about a foot in front of his face, and then asking him to 'look at the finger'.

CHECK FOR ABNORMAL PUPILLARY REFLEXES

Argyle Robertson pupils

This is a classical pupillary abnormality in neuro-syphilis. The pupil is small and irregular, reacts briskly to acommodation, but does not react to light. There is loss of direct and consensual light reflexes due to mid brain lesion which is usually due to neuro-syphilis. Near convergence reflex is preserved.

Marcus Gun Pupil (MGP):

When light is swung over the abnormal eye, both pupils dilate inappropriately. This is called as afferent pupillary defect or MGP. This may be due to optic nerve lesion. This is particularly useful sign in retrobulber neuritis and in ischemic or compressive lesions of optic nerve.

Constricted pupils

Sympathetic nerve activity causes pupillary dilatation and elevation of the upper eyelid; the later through the contraction of smooth muscle fibers in the levator palpebrae superioris.

Interruption of the cervical sympathetic nerve supply to the eye causes **Horner's synrome** (oculosympathetic paresis) which is characterised by the classic triad of:

- miosis (contricted pupil)
- partial ptosis and
- loss of hemifacial sweating (anhydrosis)
    - Anhydrosis is less commonly seen. It is the absence of sweating on the corresponding half of the face and neck both in front and behind, extending as low as the third rib and third cervical spine and over the whole of the upper limb on the same side.

- An apparent enophthalmos is often present, and is useful clue to the diagnosis when the patient is first seen.
- Congenital Horner's syndrome is associated with neuroblastoma and urine VMA should be measured.
- Drugs like opiates and cholinesterase inhibitors can also cause constricted pupils and should be considered in the differential dignosis of Horner's syndrome.

Ciliospinal reflex (pupillary-skin reflex)

It is the dilatation of the ipsilateral pupil in response to pain applied to the neck, face and upper trunk. If the right side of the neck is subjected to a painful stimulus, the right pupil dilates (increase in size 1-2 mm from base line). This is due to reflex excitation of the pupil-dilating cervical sympathetic fibers. This reflex is abolished by the lesions of these nerves and by some medullary, cervical and upper thoracic cord lesions.

Dilated pupils:

Causes

- Mydriatic drugs like atropine, tropicamide etc.
- Blindness due to damage to optic nerve

Unreactive pupils:

Causes

- Third nerve palsy
- Cataracts
- Corneal opacities
- Vitreous/retinal haemorrhage
- Prosthetic eye

Eye movements in different nerve lesions

- **3rd nerve lesion** may result in:
  - Ptosis, dilated pupils and loss of light and accomodation reflexes
- **4th nerve lesion** may result in:
  - Inability to look down and medially
- **6th nerve lesion** may result in:
  - Inability of eye ball to move laterally

Red reflex

Transparancy of the media of the eye may be observed by observing the red reflex .

Method to elicit red reflex:

- Set the ophthalmoscope at the zero and stand about 40 cm or an arm's length away from the patient.
- Whilst looking through the ophthalmoscope shine the light into each pupil.
- The vessels and the colour of choroid should elicit a red reflection.
- Red reflex may be dark or absent when cataract or viterous opacity is present

A **white reflex** may be seen in case of retinoblastoma.

Abnormalities of these three nerves may cause diplopia.

Since 6th cranial nerve within subarachnoid space has very long route, raised intracranial pressure (ICP) may cause failure of abduction of one or both eyes; a nonspecific sign of raised ICP.

5. Trigeminal

Major branches: ophthalmic, maxillary and mandibular divisions

Ophthalmic division

The lesions of ophthalmic division result in loss of cutaneous and corneal sensation. Trophic changes in the cornea may occur. Corneal reflex is abolished. Wisp of cotton or an esthesiometer can be used to measure sensation.

Method of testing corneal reflex

- Aske patient to gaze at the distance or onto the ceiling.
- Twist a light wisp of cotton into a fine hair and lightly touch the lateral edge of the cornea at its conjunctival margin with the wisp.
- If the reflex is present, the patient will blink.
- The two sides should be compared.
- Cornea should not be wiped with cotton and central part of cornea should not be touched to avoid the risk of corneal ulceration.

Maxillary division

The lesions of maxillary division, lead to loss of palatal reflex and loss of sensation at:

- the cheek
- the front of the temple
- the lower eye lid and its conjunctival surface

Mandibular division

The lesions of mandibular divison, lead to loss of sensations at:

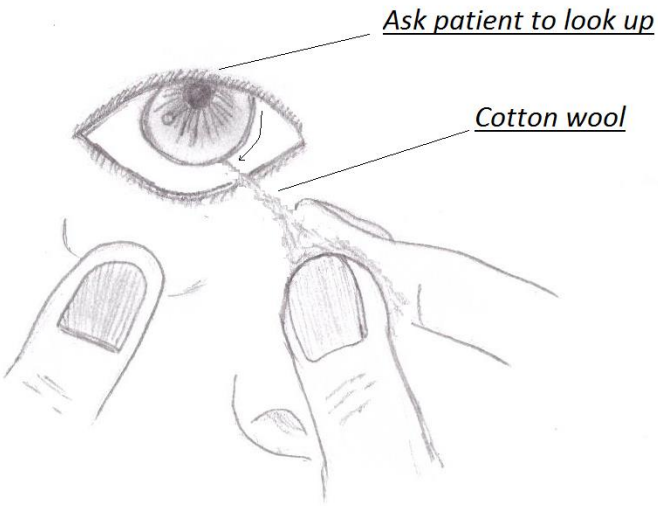
- lower part of the face
- lower lip
- ear
- tongue
- lower teeth

Whole trigeminal nerve

Lesions of the whole trigeminal nerve:

- will lead to loss of sensations in skin & mucous membrane of the face & nasopharynx.
- may develop diminished, salivary, buccal and lacrimal secretations leading to the development of trophic ulcers in the mouth, nose and cornea.

- may spare the taste but lack of oral secretions may result in its subjective impairment.
- will result in weakness of the muscles of mastication.

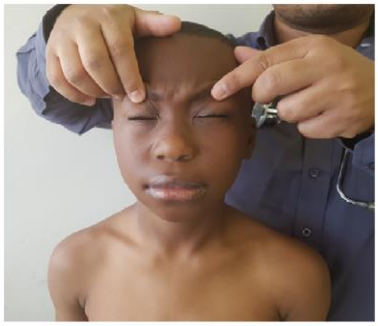


Corneal reflex

7. Facial

Examine the child and look if the face is symmetrical when crying or smiling?

- Can the child wrinkle the forehead and close eyes tightly?
    - A LMN lesion will affect both upper and lower halves of the face whereas an UMN lesion will affect only the lower half.
- Check blood pressure in a child with facial palsy; this may be the first presentation of systemic hypertension.
- Check the sense of taste



Picture: Examination to check if patient can wrinkle the forehead and close eyes tightly

Motor function: facial nerve supplies all the muscles of facial expression

- Any asymetry of the face
    - Opens eyes → Occulomotor - sympathetic nerves

    - Closes eyes → facial nerve
- Spontaneous movements
- Involuntary movements
- Wrinkle forehead
- Screw your eyes
- Show me your teeth
- Blow out cheeks

Common lesions of facial nerve

- Bells palsy and Bells phenomenon
- Ramsay Hunt Syndrome
- Unilateral LMNL: weakness of both upper and lower facial muscles
- Unilateral UMNL: facial paresis is marked in the lower facial muscles – sparing of upper face – because there is bilateral cortical innervation of the upper facial muscles

D/d of bilateral facial weakness, palsy or paralysis

Lower motor neuron lesions of facial nerve

Unilarteral or bilateral

- **Bell's palsy:** idiopathic acute paralysis of facial nerve (most commonly due to herpes simplex infection) affecting all the muscles on ipsilateral side of the lesion
- **Ramsay Hunt syndrome:** acute facial paralysis (most commonly) due to vericella zoster infection of lateral geniculate body
- Vascular lesions
- Bilateral parotid disease
- Myasthenia gravis
- Miller- Fisher varient of GBS

Upper motor neuron lesions of facial nerve

- Stroke, haemorrhagic/thrombotic vascular accidents
- **Brain tumours** causing raised ICP may present with buldging fontanelle & increased OFC
- **Infections:** herpes/ vericella
- **Moebius syndrome:** Palsy of 6 and 7^{th} cranial nerve usually bilateral + TEV (Talipes Equinovarus) & micrognathia. It is a sporadic condition and is the result of a number of different etiologies including developmental abnormalities of brain, peripheral nerve or myopathy
- **Metabolic** (seizure, myopathy):
    - MELAS (Mitochondrial Encephalopathy, Lactic acidosis, and Stroke like episodes)
    - MERF (Myoclonic Epilepsy with Ragged-red Fibers)
- **Di-George syndrome**: associated with facial palsy and congenital heart disease versus cardiofacial syndrome

8. Vestibulocochlear Nerve

Vestibulocochlear division comprises of:

- Auditory fibers which arise from cochlea
- Vestibular fibers which arise from otolith organs & semicircular cannals

Vestibulocochlear nerve runs with facial nerve in internal auditary meatus and both enter the brain stem at the cerebellopontine angle. Both may be affected by posterior fossa tumors or an accoustic neuroma

- Lesions of cochlear component of 8th cranial nerve will produce deafness &
- Lesions of vestibular component of 8th cranial nerve will produce symptoms of
  - Vertigo
  - Nausea
  - Vomiting
  - Diaphoresis
  - Nystagmus

For examination ask mom if the child can hear?

- Jangle the keys away from the vision of the child – see if he/she turns to the sound
- Clap hands and observe if he/she stares or blinks
- Alert neonates blink in response to bell
- Four months old infant turns his head and eyes to lacalise a sound

Cochlear division: to test the hearing loss

The simplest method of testing the hearing is a conversational voice that should be heard at 3.6 m in each ear separately and if this facility extends to 6 m it can be presumed that the patient has normal hearing.

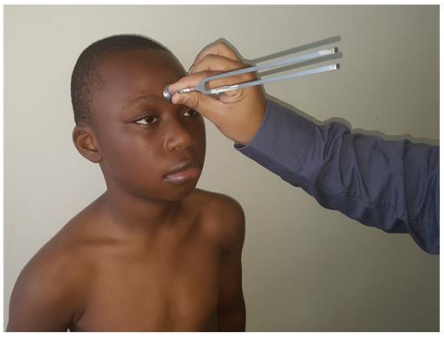
Tuning fork tests: Tuning forks 256 or 512Hz are usually used

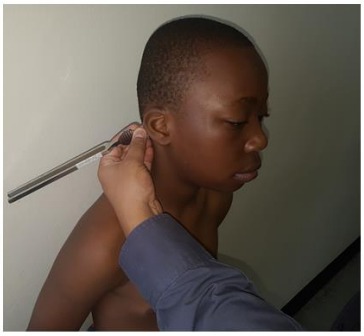
Rinne test:

- It compares the hearing through air conduction with the bone conduction (with base of the fork on the mastoid process).
- In the normal ear, air conduction is better than bone conduction (Rinne positive).
- This also applies to patients who have a sensorineural deafness, where as patients with a conductive deafness will show the opposite response i.e., bone conduction better than air conduction (Rinne negative).

Webber's test:

- The base of tuning fork is placed anywhere on the midline of the skull or mandible.
- In conductive deafness patient will perceive in the deafer ear whilst, in the cases of sensorineural loss, it is referred to the better ear.





Picture: Above - Webber's Test , Below - Rinne Test

CAUSES OF DEAFNESS

CONDUCTIVE DEAFNESS

1. Wax in the ears
2. Chronic secretory otitis media
   - Glue ear
   - Grommets
   - Damage to ear drums

PERCEPTIVE DEAFNESS

1**. Congenital**
   - Inherited – may be isolated or part of a syndrome
   - Waardenburg's syndrome (AD)
   - Congenital malformation of ears
   - Intrauterine infection: TORCHES

- **Perinatal causes**
   - Perinatal hypoxia
   - Kernicterus
   - Use of aminoglycosides

- **Postnatal causes**

1. Meningitis
2. Encephalitis
3. Trauma
4. Ototoxic drugs

Vestibular division

To assess for sense of balance and spacial orientation for the purpose of coordinationg movement with balance i.e., vertigo & nystagmus

- Abnormality of posture or gait
- Induction of positional nystagmus
- Doll's eye reflex
- Oculovestibular reflex
- Dix-Hallpike maneuver

Nystagmus is caused by the lesion of cerebellum, brainstem, cervical cord or inner ear - always offer to examine hearing, cerebellar functions and the occular fundi

Nystagmus

Involuntary & repetitive oscillations of the eye or eyes is called as nystagmus. It is defind by the direction of the fast phase but it is the slow phase which is pathological. Nystagmus may suggest end-organ or intrinsic disease of the nervous system.

The most common cause of nystagmus in children is caused by the centrally acting medications like anticonvulsants

Central nystagmus:
Nystagmus in any direction results from:

- Brain stem lesions
- Vertebrobasilar ischaemia
- Phenytoin toxicity

Pendular nystagmus
This refers to the waveform of involuntary eye movement in which eye moves in sinusoidal trajectory, similar to that of a pendulum. Rhythmic abnormalities that are equal in all planes of gaze; associated with either cerebellar or retinal disease

Jerk nystagmus
Consists of a slow and fast phase and refers to abnormalities of vestibular system

Vertical nystagmus
Primarily seen in abnormalities of the brain stem

Ocular nystagmus
Due to poor macular vision which impairs retinal fixation

9. Glassopharyngeal Nerve

Ask simple questions to the care giver and observe like:

- Can the child swallow?
- Is the child drooling?

Then:

Assess patient's speech for dysarthria or dysphonia

Check for:

- Palatal reflex: tickle the back of the pharynx, and note if contraction occurs. This is also a test for vagus nerve.
- Ask the child to puff out cheeks with lips closed: air leak from nose will indicate lesion
- Examine the taste in the posterior 1/3rd of tongue: Loss of taste at this part of the tongue may occur with a lesion of the trunk of the glossopharyngeal nerve.

10. Vagus Nerve

Following test can be performed:

- Ask the patient to say Aah and look at the palate and note position of uvula. If it is in the center and moving in either side than that's normal and if it is deviating on one side that side is considered normal because of pull by the stronger muscles
- Note nasal twang
- Note regurgitation of food
- Note hoarsenes of voice; due to paralysis of laryngeal muscles

Common causes of bilateral vagus nerve injury

- Progressive bulber palsy
- Bilateral supranuclear lesions
- Cerebrovascular disease
- Multiple sclerosis

9th through 12 cranial nerves are called bulbar cranial nerves. Gag reflex is caused by touching the posterior pharyngeal mucosa. Both 9th and 10th cranial nerves are responsible for gag reflex.

11. Accessary Nerve

It is purely a motor in function. Check bulk and test power of sternocleidomastoid and trapezius muscles.

Method

- **Testing the trapizius muscle**
  - Tell the patient to shrug shoulders while the examiner presses downward on them. Paralysis of the upper part of trapizius muscle is demonstrated in this way.
- **Testing the right sternocleidomastoid muscle**
  - Face the patient and place your right palm laterally on the patient's left cheek. Tell the patient to turn head to the left, resisting the pressure you are excerting in the opposite direction. Paralysis of the sternomastoid causes weakness of rotation towards the opposite side.
- **Testing the left sternocleidomastoid muscle**
  - Face the patient and place your left palm laterally on the patient's right cheek. Tell the patient to turn head to the right, resisting the pressure you are excerting in the

opposite direction. Paralysis of the sternomastoid causes weakness of rotation towards the opposite side.

12. Hypoglossal Nerve

It is purely a motor nerve. It supplies the tongue and the depressors of the hyoid bone.

It is tested by examining the tongue and its movements as follows:

- If the nerve is injured, at rest the tongue may appear to have the appearance of a "bag of worms" (fasciculations) or wasting (atrophy). Fasciculations must be assessed with the tongue relaxed in the mouth, not when protruded.
- Ask the patient to stick the tongue out as far as possible. If the hypoglossal nerve is paralysed, the tongue, instead of being protruded straight, will be pushed over to the paralysed side.
- Ask the patient to move the tongue from side to side and lick each cheek with it; observe whether patient can do so freely.
- Strength may aslo be assessed by pressing against the tongue with a finger as the patient protrudes it into each cheek in turn.
- Patients with hypoglossal nerve disorder have difficulty speaking, chewing and swallowing.
- Assess speech by asking the patient to say "red lorry and yellow lorry" etc.

SUMMARY: IF ALL CRANIAL NERVES ARE NORMAL - SAY

1. Pupils equal and reactive to light
2. Fixes and follows
3. No ophthamoplegia
4. No facial asymetry
5. Hearing appears intact
6. Tongue central
7. Positive gag reflex
8. No drooling

NB! Cavernous sinus thrombosis causes problems in 3 cranial nerves**;** Occulomotor, Trochlear and Abducent (OTA) nerves.

6. EXAMINATION OF MOTOR SYSTEM

Achronym: BPTR: Bulk, Power, Tone, Reflexes

EXAMINE THE MUSCLE BULK

Inspection

- Examine after full exposure and look for deformity
    - Bony or soft tissue
    - Pes cavis/Pes planus or claw of hands
        - Pes mean foot
        - Pes cavis: foot with abnormally high longitudinal arch

        - Pes planus or flat foot: when entire sole of the foot touches the floor on standing
    - Ankle valgus: an insidious deformity that results in pronation of the foot and medial malleolar prominence
- Look for asymmetry: Proximal/distal
- Look for muscle wasting or hypertrophy
    - Loss of muscle bulk may be due to a lower motor neuron lesion, disuse atrophy or generalised wasting
    - Excessive muscle bulk may be due to Duchenne Muscular Dystrophy or Myotonia Congenita, may look like baby Hercules

Examine the posture:

- Shoulder adducted, elbow flexed, hand clinched may be due to pyramidal tract problem
- Erb's palsy (policeman tip hand deformity)
- Claw hand (atrophy and distortion of hand)
- Arthrogryposis (persistant flexion of a joint, tetnoid spasm)

Palpate the muscles to assess bulk

Common abnormalities

- Athetosis: repetitive involuntary, slow gross movements which involve distal parts of the extremities
- Peripheral neuropathy: distal wasting (inverted champaign bottle)
- Check thenar/hypothenar for muscle wasting

EXAMINE THE POWER

Grade the power according to Medical Research Council Scale

- Grade 0: No muscle movement (complete paralysis)
- Grade 1: Muscle flicker, no joint movement
- Grade 2: Full movement is possible when gravity is illiminated
- Grade 3: Movement against gravity but not against resistance
- Grade 4: Power against resistance but can be overcome by resistance
- Grade 5: Normal power against gravity and full resistance is present

Check proximal/distal power

- **Upper limbs**
    - Check thumb for flexion/extension
    - Check abduction/adduction of fingers
    - Squeez hands and check grip/strength
    - Examine wrists – stablise at elbow – extend/flex
    - Examine elbows – stabilize upper arm – extend/flex
    - Check shoulders for abduction/adduction – shrug shoulders

- Elicit pronator drift: when child extends his or her arms out in front with palms upword and eyes closed. Hands on weak side cup and pronate slowly. This is scalled as apronator drift.

- **Examine neck:** patient to lie flat, check for flexion/extension of neck
- **Examine trunk for muscle weekness:** note for pot belly/lumber lordosis
- **Check for proximal weakness:** elicit Gower's sign
- **Lower limbs**
  - Big toe: extend & check for muscle wasting at the dorsum of the foot
  - Foot: invert/evert, dorsiflex/planter flex
  - Hip, knee – extend and flex
- **Compare**
  - UL/LL
  - Right to left
  - Proximal to distal

Picture: Examination - Power

EXAMINE THE TONE

Muscular tone is a state of tension or contraction found in healthy muscles. It represents the dynamic resistance of muscles to movement across a joint (stretch or to gravity). An increase in the tone is called as hypertonia and a diminution as hypotonia. The degree of tone is estimated by handling the limbs and moving them passively at their various joints.

- **Passive tone** is the resistance to stretch felt when limb is flexed and extended manually
    - In LMNL, passive tone is reduced
    - In chronic UMNL, passive tone is increased
- **Active tone** is the posture that an infant adopts when placed in a particular position

Hypertonia

Causes of hypertonia may include upper motor neuron disease, lesion in basal ganglia, and anxiety. Hypertonia following lesions of the corticospinal system (UMNL) is termed as spasticity. The term spasticity is used to describe a state of increased tone which is of claps knife type.

- **Claps knife type hypertonia** is when the limb is fairly rapidly flexed or extended i.e. when limb is flexed, initially it is very rigid but after certain degree of flexion the resistance suddenly disappears & same thing happens when extending it. Clasped knife response or spasticity is seen in upper motor neuron lesions.
- **Cogwheel rigidity** hypertonia results from disease of the basal ganglia also termed as extrapyramidal rigidity**.** In this type, the resistance to passive movements is fluctuant i.e. if you try to flex the elbow, it will flex in a jerky manner described as like a lever rubbing on the teeth of a cogwheel (cogwheel rigidity). This cogwheel rigidity is seen in extrapyramidal disease.
- **Lead pipe rigidity**, the rigidity is uniformly present throughout the passive movements (flexion or extension). This is present in cases in which patient's state of consciousness is impaired.

Hypotonia

In hypotonia, there is little or no resistance to passive movement of the limb and when handled or shaken the unsupported part flops about inertly. Hypotonic muscles are abnormally soft to palpation. Causes of hypotonia may include lower motor neuron disease, unconsciousness, and neural shock i.e. in early stages of UMNL.

Do passive movements of flexion and extension in all the four limbs, slowly and then quickly and note abnormalities of tone:

- Hypotonia
- Spasticity
- Rigidity

EXAMINE THE REFLEXES

There are superficial & deep tendon reflexes.

Superficial reflexes

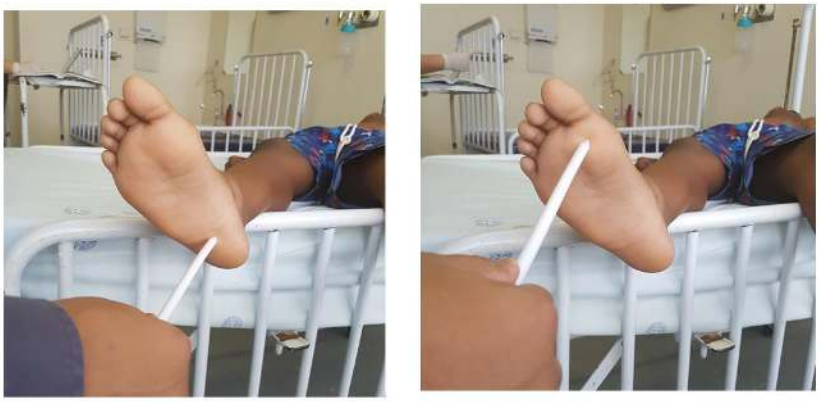
Reflex	How elicited	Result	Level of cord concerned
Anal	Stroking or scratching the skin near the anus	Contraction of anal sphincter	S3, S4
Planter	Stroking sole of foot	Flexion of toes, foot or leg	L5, S1
Cremasteric	Stroking skin at upper and inner part of thigh*	Upward movement of testicle	L1, L2 Lost in UMNL
Abdominal	Stroking abdominal wall below costal margin, at the level of umbilicus and in iliac fossa	Contraction of abdominal muscles	T7 to T12 Lost in UMNL & LMNL
Scapular	Stroking skin in the intrascapular region	Contraction of scapular muscles	C5 to T1
*the cremasteric reflex can often be more easily elicited by pressing over the sartorius in the lower third of **Hunter's canal** (an aponeurotic tunnel in the middle third of the thigh, extending from the apex of the femoral triangle to the opening in the adductor magnus, the adductor hiatus)			

Babinski's sign:

Scratch the outer border of the sole of foot with edge of a key or the stick of a patella hammer. Normally the greater toe goes up and there is flexion and adduction of all other toes and foot is dorsiflexed and inverted. This is called as negative Babinski sign or planter reflex (planters are flexors). In positive Babinski the big toe bends up and back to the top of the foot and other toes fan out. Nerve roots involved are L5, S1.

Positive Babinski is present in:

- Upper motor neuron lesion
- During sleep
- In infants – untill they start walking (about 1 yr)
- Post ictal or coma



Picture: Babinski's sign

Deep tendon reflexes

If the tendon of a slightly stretched muscle is struck (a single, sharp blow) with a soft rubber hammer, the muscle contracts breifly. This is the monosynaptic stretch reflex. It is the test of integrity of the afferent and efferent pathways and of the excitability of the anterior horn cells in the spinal segment of the stretched muscle. When eliciting the tendone reflexes grade responses as follows:

- Grade 0: Absent reflex
- Grade 1: Present normal
- Grade 2: Brisk (as normal knee jerk)
- Grade 3: Very brisk
- Grade 4: Sustained clonus

When eliciting a tendon reflex following need to be kept in mind:

- There should be no joint disease of the limb on which reflex is going to be elicited
- Patient should be fully relaxed and the part of the body to be tested should be fully exposed.
- Concentration should be on the movements of the muscles whose tendon has been struck by the hammer

BICEPS JERK (C5, C6)

Method 1:

- Flex the arm at the elbow and place forearm on the abdomen.
- Put your index finger over the tendon of biceps muscle and press it lightly.
- Now strike the hammer on your finger and note the Biceps jerk.

Method 2:

- Hold the patient's elbow in your left hand in a manner that your thumb should be on the tendon of biceps muscle.
- The patient's upper limb should be fixed at elbow and it should rest on your left forearm relaxed.

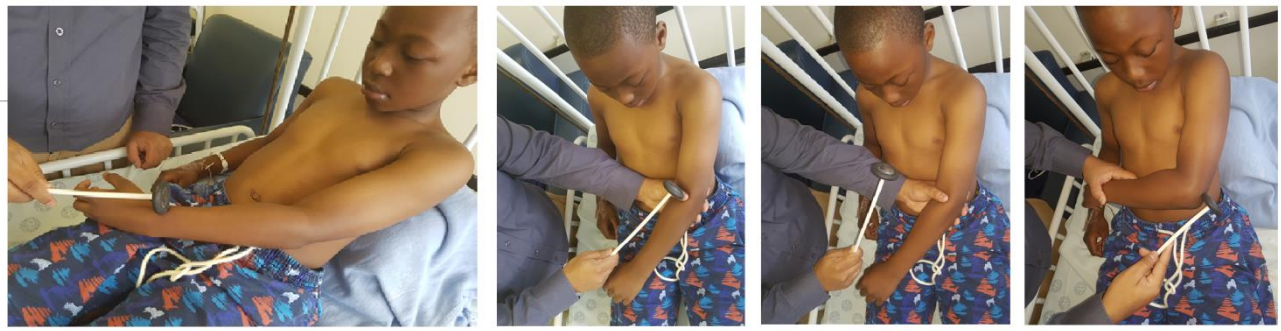
- Now press the tendon of biceps muscle lightly with your thumb and strike the hammer over your thumb.
- Note intensity of reflex by seeing the contraction of the muscle.

TRICEPS JERK (C6, C7)

- Keep the forearm on the abdomen with elbow flexed.
- Strike the hammer on the tendon of triceps muscle just above the olecranon process.
- Note intensity of reflexion by seeing the contraction of the triceps muscle.

SUPPINATOR JERK (C5, C6)

- Keep the forearm placed on the abdomen in semi pronated position with flexed elbow.
- The hand should slightly deviate towards the ulnar side.
- Now strike with patella hammer on the styloid process of the radius (the tendon of brachio-radialis muscle).
- Note intensity of reflex by seeing the contraction of Brachio-radialis muscle.



Picture: Deep tendon reflexes of the upper limbs

KNEE JERK (L3, L4)

There are many methods to elicit the knee jerk. Following three are commonly used:

Method 1:

- Make the patient to sit at the edge of the bed with his legs hanging down.
- Remove trousers from the thigh's and locate the tendon of the quadriceps femoris
- Give a stroke on the tendon with patella hammer and note the grade of reflex.

Method 2:

- Make the patient to lie on the bed with both knee joints flexed.
- Pass your hand under the knee to be tested and bring it to rest upon the opposite knee.
- In this way the knee to be tested will hang on the dorsum of your wrist.
- Now divert the attention of the patient in some way and strike the hammer on the tendon of the quadriceps midway between the tibial tuberosity and the patella.

Method 3:

- Make the patient to lie supine on the bed with lower limbs slightly flexed at hip and knee joints with femur externally rotated in a manner that the heels of both sides are near to each other.
- Now strike the tendon of quadriceps femoris muscle and note intensity of reflex by seeing the contraction of the muscle.

JENDRASSIK MANEUVER

Jendrassik manoeuvre is used to elicit the deep tendon reflexes. In this manoeuvre the patient clenches the teeth, flexes both sets of fingers into a hook-like form, and interlocks those sets of fingers together. The tendon below the patient's knee is then hit with the patella hammer. The elicited response is compared with the reflex result of the same action when the manoeuvre is not in use.

The amplitude of the response is increased because a larger number of motor unit have been activated. Clinically some brain stem lesions are manifested by exaggerated spinal cord reflexes. Isometric contractions in another muscle group enhance the response of the reflex. Often this is used to distract the patient and relax the muscles in the quadriceps.

ANKLE JERK (S1, S2):

Method:

- Ask the patient to lie on the bed with legs everted and slightly flexed at the knee.
- Now, with one hand slightly dorsiflex the foot and strike the tendon Achilles.
- There will be contraction of the calf muscles and the foot will undergo planter flexion.

Picture: Deep tendon reflexes of the lower limbs

CLONUS

This is elicited when the tendon reflexes are exagerated.

Clonus ordinarily occurs when the stretch reflex is highly sensitised by facilitatory impulses from the brain. For instance in a decerabrate animal in which stretch reflexes are highly facilitated, clonus develops readily. It is done to determine the degree of facilitation of spinal cord.

Clonus may be sustained or unsustained. Sustained clonus is indicative of hyper-reflexia (UMNL), while unsustaiend clonus is present in nervous and anxious persons. This is of two types: Patellar and Ankle clonus

Patellar clonus

Method:

- Ask the patient to lie down supine on the bed and you hold the tendon of quadriceps by upper border of the patella.
- Now give 2 to 3 tendon jerks downwards. In positive cases, there will be series of clonic contractions of the quadriceps and the patella will move with it.

Ankle clonus

Method:

- Ask the patient to lie down supine on the bed.
- Flex the knee along with slight flexion of hip joint and support the leg with your hand.

- Now grasp the forepart of the foot with other hand and give 2 to 3 sudden dorsiflexion movement to the foot.
- The pressure on the sole of the foot is sustained by keeping it in dorsiflexed position. There will be series of contractions of calf muscles in positive cases. So there will be repeated vibrations at the ankle joint.

Picture: Examination to elicit ankle clonus

Hoffman's reflex

The test involves tapping the nail or flickering the terminal phalanx of the middle finger. Hoffman's test is positive if you see adduction of thumb and flexion of index finger which may indicate an upper motor neuron lesion or a pyramidal sign. Hoffman's sign may be seen in multiple sclerosis and conditions which cause spinal cord compression such as cervical myelopathy, cervical spondylitis, tumors or degenerative arthritis.

Wartenburg's sign

It is a neurological sign consisting of involuntary abduction of the little finger, caused by injury of the ulner nerve which supplies intrinsic hand muscles (particularly palmer interosseous muscle) which results in unopposed action of the extensor digiti minimi. This is commonly seen in ulner nerve neuropathy and cervical neuropathy.

Pendular knee reflex

This is one of the cerebelalr sign where leg keeps swinging after knee jerk more than 4 times (4 or less is normal), which ma be due to cerebellar damage.

Crossed adductor reflex

Contraction of both hip adductors when either knee jerk is elicited. The cross adductor response usually disappears by 7 to 8 months. Its persistance beyond 8 months is a sign of pyramidal tract dysfunction.

Method

- Ask the patient to lie down suppine on the bed with hips and knees partially flexed and abducted with soles of the feet facing each other in the centre.
- Now strike the hammer on the medial aspect of the knee. If both the legs are adducted the test is considered as positive which indicates pyramidal tract dysfunction.

7. EXAMINATION OF SENSORY SYSTEM

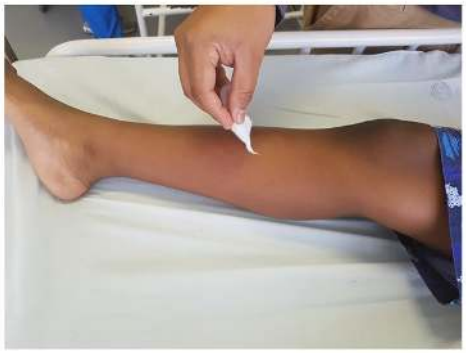
Loss of sensation means there is no feeling of pain, heat, or cold. This can happen in one or more parts of the body. And because of this, one is more likely to hurt himself so look for sores especially on feet and legs of the patient. Main conditions that may cause loss of sensations include problems with the peripheral nerves (neuropathy), stroke, spinal cord injury, tumours and chronic infections.

Following various sensations can be tested:

Sense of light touch

- Expose the part of the skin to be examined.
- Ask the patient to close his eyes.
- Now take a wisp of cotton and touch the identical points of the body of the patient; the upper limbs, then trunk, then back and finally lower limbs.
- Two questions are asked when examining sensation of touch.
    - **Where the wisp of cotton is touching?** This will give you an idea of sense of localisation of touch (tactile localisation) of the patient. Ask the patient to say "yes" every time he feels the touch.
    - **How many wisps of cotton are touching him?** Before asking the question, touch the patient with two wisps of cotton close to each other. By this we can make out the sense of discrimination of touch (tactile discrimination) of the patient.

If there is lesion in the anterior spinothalamic tract, the sense of localisation and discrimination of the patient will be affected.



Picture: Sensory examination - Light touch

Sense of course touch

Method

- Press the limb with finger or blunt probe on identical points.
- Now ask the patient whether he appreciates or not.

Sense of pain

Pain is carried by lateral spinothalamic tract; therefore, its lesion will impair the pain sensation.

Method

- Take a steel pin or sterile needle (rarely done in children).
- The pin is pricked on the identical points and the patient is asked whether he feels pain or not.

Sense of deep pain: Two tests are done

- Squeeze the calf muscles and apply pressure in between the thumb and the index finger. Note if the patient feels pain.
- Deep pain is absent in tabes dorsalis (a form of neurosyphilis in which there is loss of both axons and the myelin in the dorsal roots, with pallor and atrophy in the dorsal columns of the spinal cord).

Sense of temperature

- This is tested by hot and cold water contained in two copper test tubes.
- The naked area under the examination is touched with each, in turn, and the patient is asked whether he feels it hot or cold.
- Lesion in the lateral spinothalamic tract will impair or even abolish this sensation.

Sense of position and passive movements (posterior column integrity):

Method 1

Here the movements of thumb and big toe are tested.

- The patient is made to understand the movements of big toe or thumb which is moved up, down and sideways.
- Ask the patient to close his eyes.
- Now hold the big toe with your thumb and index finger.
- Flex it at the distal interphalangeal joint, slightly without touching the other toes and ask the patient whether it is directed upward or downward.
- If unable to make final assessment test can be repeated number of times.
- Same procedure can be used for upper limb by the movement of thumb.

Method 2

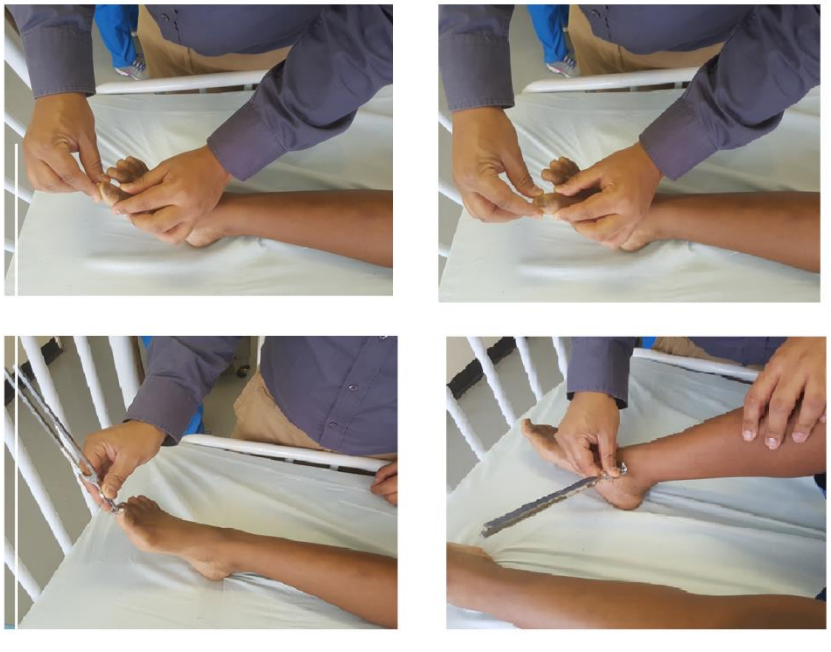
- Ask the patient to close his eyes.
- Hold the thumb of the patient and move it in various directions through the air.
- Now place the thumb of the patient in an abnormal position and ask the patient to place his other thumb in the same position.
- In the case of hand, move the fingers in various directions and ask the patient to imitate with other hand while eyes are closed.

- Testing limb should not touch any other skin surface; otherwise patient will be able to appreciate its position in spite of the lesion.

Sense of vibration

Tuning fork (frequancy 128/sec) is used to test sense of vibration.

- Strike the tuning fork on the side of your knee and then place its base over the bony prominances, i.e. medial and lateral maleoli, tubercles of tibia, anterior superior iliac spines and olecranon processes.
- Normal individual will feel the buzzing sensation.
- If unable to feel the vibration sense, consider polyneuropathy or disease of posterior column.



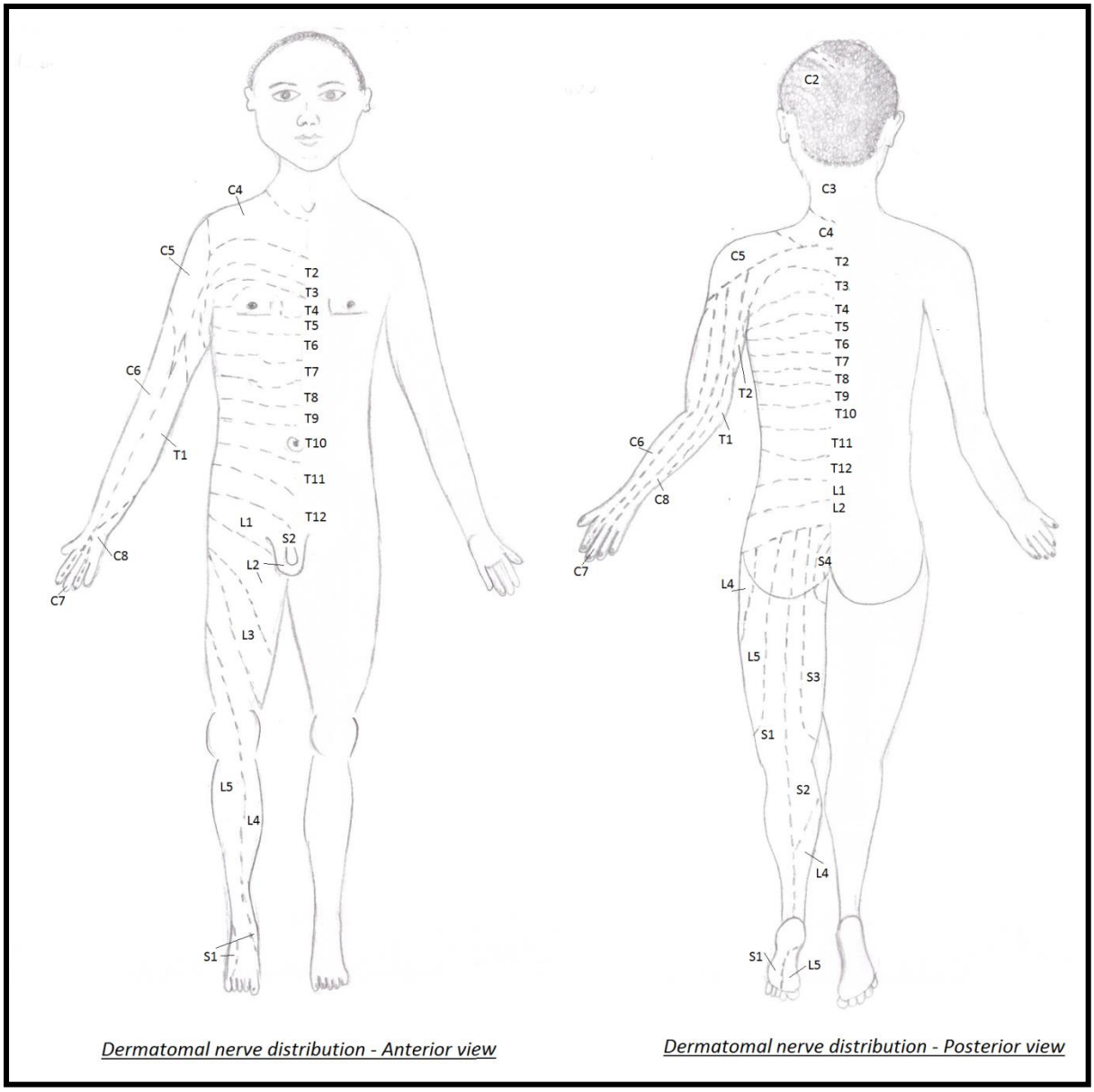
Picture: Sense of position and passive movements

Brown-Sequard Syndrome

It is a rare neurological condition cheracterised by an incomplete lesion in the spinal cord (often in the cervical cord region) which results in weakness or paralysis (hemiparaplegia), spascity and ataxia on one side of the body (upper motor neuron paralysis) and loss of pain and temperature sensation (hemianaesthesia) on the opposite side. There may also be loss of bladder and bowel control.

Papilloedema is observed in few patients usually in association with markedly elevated CSF protein levels that presumably interfere with normal CSF flow dynamics.

Focus mainly need to be on the underlying cause of the disorder. Early treatment with high dose steroids may be beneficial in many cases. Physical, occupational and recreational therapy is important aspects of patient rehabilitation.



Dermatomal nerve distribution - Anterior view

Dermatomal nerve distribution - Posterior view

8. CO-ORDINATION: CEREBELLAR SIGNS

The cerebellum receives information from the sensory system, the spinal cord and other parts of the brain and then regulates motor movements such as posture, balance, coordination, speech and rate, range, rhythm and force of muscle contraction. Following signs should be lookd for in the lesions of cerebellum:

Ataxia

Ataxia is impaired balance or coordination of movement. Patients with cerebellar lesion will not be able to walk in straight line with the eyes open. Cerebellum and associated pathways modulate volitional movement.

If either afferent cerebellar connections (joint position sense) or efferent cerebellar connections (cerebellum through thalamus through cerebral cortex) are disturbed, the patient experiences ataxia.

Adiadochokinesia (inability to conduct rapid alternating hand movements)

Method 1

To elicit loss of rapid alternate movements.

- Ask the patient to flex his elbows at the right angle and to pronate and supinate his forearm by placing one hand over the other.
- The patient will not be able to do so and his movements will be slow and incomplete.

Method 2

- Ask the patient to tap your palm with his finger tips as fast as possible.
- Patient will not be able to do so.

Asynergia: This means inco-ordination. This is tested in both limbs:

UPPER LIMB

Finger-Nose test (finger nose pointing or dysmetria)

- Ask the patient to touch tip of your index finger which is at adistance of about ½ meter from the patient) with his index finger and then to touch his nose.
- This should be done rapidly. Patient will fail to do so.

Picture: Finger-nose test

Finger-Finger test

- Ask the patient to touch the tips of his both index fingers with extended arms bringing them from a distance rapidly.
- Patient will not be able to do so.

Rebound phenomenon (loss of rebound control):

- Ask the patient to flex his elbow slightly and flex it further firmly against resistance (i.e. you hold his hand) then release it suddenly.
- The patient will slap his chest due to rebound phenomenon but normal individual will be able to prevent it to happen.
- If the results of above tests are doubtful, then patient should repeat the above tests while his eyes closed - irregularity in the movements will become more marked

LOWER LIMB

Heel-Knee Test

- Ask the patient to lie in the bed in supine position

- Then to put the heel of his foot on the knee of other leg and rub it down along the shin
- Then raise the leg in the air and again put the heel on the knee.
- He will fail to do so.

Picture: Heel knee test

Cerebellar gait

- It is a staggering, wide-based, unsteady gait which has lurching movements with a breakdown of normal synergy of walking.
- In the older child one can test ataxic gait by asking the patient to walk by heel to toe (like walking on a tight rope).

Pendulous knee jerk

- Ask the patient to sit in the bed with legs hanging down and elicit the knee jerk.
- The leg after extension will fall down in a pendulous fashion

Charcot's triad [SIN]

- Scanning speech
  - Ask the patient, to speak few words
  - Note slow, slurring or explosive dysarthria, poor or no cordination
- Intension tremor
  - Ask the patient to reach out and touch your finger
  - Note the fine tremor increasing as it approaches the finger
- Nystagmus
  - Ask the patient to look straight ahead at you
  - Note the abnormal movement of eye from slow to rapid phase - nystagmus

9. SPINAL CORD (CERVICAL, THORACIC AND LUMBER SPINE)

Do full examination of the spine

- Look for hairy patch, lipoma or spina bifida
- Palpate the spinous processes and paraspinal tissues
- Note overall alignment and focal tenderness

- Lightly percuss the spine with your closed fist – note any tenderness
- Move and observe flexion, extension, and lateral flexion

Note clinical signs in the case of "Hemisection of Spinal Cord"

- Below the level of lesion
  - Features ipsilateral to the lesion
    - Impaired light touch
    - Impaired vibration touch
    - Corticospinal tract signs
  - Features contralateral to the lesion
    - Impaired pain sensation
    - Impaired temperature sensation
- At the level of lesion
  - Features ipsilateral to the lesion
    - Segmental zone of hyperpathia (pain)
    - Signs of lower motor neuron at this level

Lesion in the anterior horn cells	**Lesion in the nerve**
• Disease process is gradual in onset	• Disease process is sudden in onset
• Fibrillation and fasciculations are present	• Fibrillation and fasciculations are not present
• Only motor system is involved	• Both motor and sensory systems are involved
• There is no sphinteric disturbance	• Sphinteric loss is present

10.AUTONOMIC NERVOUS SYSTEM

Sympathetic Nervous System

Horner's syndrome:

This is due to the lesions in the cervical sympathetic trunk which leads to:

- Ptosis of the eye lid
- Enophthalmos (eye ball moves inward)
- Miosis (constriction of the pupil)
- Loss of sweating (anhydrosis)

Loss of cilio-spinal reflex:

On pinching the lateral side of the neck, sympathetic trunk gets stimulated which results in dilatation of the pupil. This reflex is lost in the diseases involving cervical sympathetic trunk.

Parasympathetic nervous system

- Cranial nerve III (ciliary ganglion) supply cilliary muscles and the sphincter pupillae
- Cranial nerve VIII (Pteropalatine ganglion + submandibular ganglion) axons projet to the lacrimal glands, mucous membrane and nasal mucosa
- Cranial nerve IX (otic ganglion) scretomotor fibers then pass to parotid gland
- Cranial nerve X (ganglions close to organ) supplies the:
  - Heart
  - Larynx/trachea/bronchi
  - Oesophagus, stomach
  - Abdominal blood vessels
  - Liver & bile duct
  - Pancrease
  - Adrenal gland
  - Small intestine
  - Large intestine

S1 to S4 → Colon, rectum, kidney, bladder, penis, clitoris

11.EXTRAPYRAMIDAL SYSTEM

Lesions of the basal ganglia cause involuntary movements

Chorea (of hands and fingers)

Cheracterised by involuntary, irregular and purposeless movements of the hands and fingers like piano-playing movements

Huntington's chorea: absent caudate shadow on CT brain

Signs

- Protruded toungue
- Milkmaid sign
- Shelving sign

Sydenham chorea

- Post streptococcal
- Increased ASOT
- Cardiac murmur

Athetosis

Slower and writhing movements

Dystonia

Abnormal posture due to periodic sustained contraction of opposing muscle group

Ballism
Sudden massive jerky flinging movements

Basal nuclei
Lesions of basal nuclei do cause cog wheel or lead pipe regidity

12.PERIPHERAL NERVE DAMAGE

Lesions in bracheal plexus

Upper lesion – Erb-Douchenne's paralysis

- Deformity:
  - upper limb hangs by the side
  - internally rotated
  - and forearm pronated
  - also called as policeman's receiving tip hand
- Lesion C5 and C6
- Lesion C7 in 50% cases
- Paralysis of biceps brachialis, brachioradialis and supinator muscles

Lower lesion – Klumpke's paralysis: here lower nerve roots are injured

- This deformity is also called as "Claw Hand" in which there is:
  - Weakness of the flexor muscles of the wrist
  - Weakness of the small muscles of the hand
  - Lesion C8, T1
  - Up to 1/3rd of these patients have Horner's syndrome.

Median nerve lesion: carple tunnel syndrome
Radial nerve lesion: wrist drop
Ulner nerve lesion: claw hand
Common peronial nerve lesion: foot drop
Lateral cutaneous nerve of thigh lesion: loss of sensation over lateral aspect of thigh

GENERAL PRINCIPLE

When hemiplegia is crossed i.e. hemiplegia on the one side and involvement of cranial nerves on the opposite side, the lesion is in the brainstem which may be in the:

- Midbrain
- Pons
- Medulla oblongata

But if the hemiplegia is uncrossed i.e. hemiplegia and the involvement of cranial nerves are on the same side, the lesion is above the brainstem which may be in:

1) Internal capsule
2) Corona radiata
3) Cerebral cortex

AT THE END OF THE EMANIBATION MAKE A REASONABLE ASSESSMENT

What is the dysfunction → like spastic quadriplegia with profound developmental delay
Where is the lesion → like in cerebral cortex
What is the aetiology → like infarct due to hypoxic ischaemic encephalopathy

WHAT IS THE DIAGNOSTIC EVALUATION LIKE

1) Normal or abnormal
2) UMNL or LMNL
3) Site of lesion
4) Nature of lesion
5) Complications
6) Developmentally normal or abnormal
7) Static or progressive
8) Acute or chronic

If it is lower motor neuron lesion, then describe as:

It is lower motor neuron lesion due to

- Hypotonia
- Weakness
- Absent reflexes
- Wasting

And conclude with summary of your findings and make a provisional diagnosis on the basis of your clinical examination.