

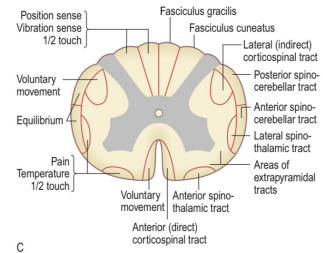
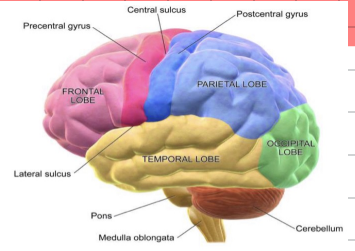
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CNS

# chapter 7: The nervous system.

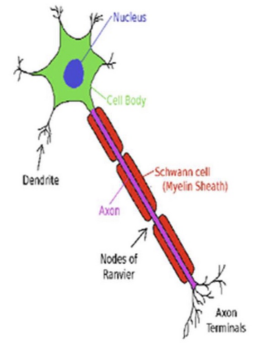
## 7.1: Anatomy & physiology

- CNS**
  - Brain**
    - 2 cerebral hemispheres (frontal, parietal, temporal, occipital).
    - Brainstem: midbrain, pons, medulla.
    - cerebellum: 2 hemispheres + vermis, 3 cerebellar peduncles
  - Spinal cord** - main pathway for information btw brain & PNS.
    - Horns**
      - gray ventral: efferent, motor cell bodies.
      - gray dorsal: afferent, sensory cell bodies.
    - tracts**
      - Dorsal column (fasciculus gracilis & cuneatus): proprioception, vibration, half-touch.
      - Ant & Lateral corticospinal tract: voluntary movement.
      - Ant & Lateral spinothalamic: Temp & half-touch.
      - spinocerebellar tract: equilibrium.

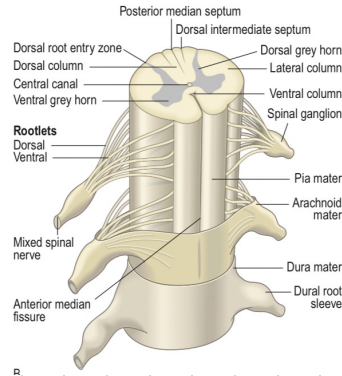


- PNS**
  - myelinated or unmyelinated axons.
  - somatic nerves: sensory & motor nerves.
  - autonomic nerves

- cells**
  - neuron: functional unit, cell body + axon (synapse).
  - Astrocytes: structural framework, biochemical environment control, from BBB.
  - Microglial: blood-derived macrophages, immune & scavenging functions.
  - oligodendrocyte: produce myelin in CNS.
  - Schwann cells: produce myelin in PNS.



- meninges**
  - Dura mater: next to bone.
  - arachnoid
  - pia mater: next to nervous tissue.



- CSF**
  - in subarachnoid space: between arachnoid & pia.
  - produced by: choroid plexuses.
  - total volume: 140-270 mL (x3-4 time)
  - Rate of production: 700 mL/day

## 7.2: The History

- The history is the key diagnosis, physical exam maybe normal or unhelpful.
- additional witness: amnesia or loss of consciousness
- clarify exactly what they mean, don't ask what they fear.
- Q**
  - onset.
  - duration.
  - pattern.
  - exacerbating & Relieving factor
    - what you was doing?
    - menstrual cycle? posture? medications?
  - associated symptoms (most important)
    - migrane: Headache + nausea, vomiting, photo & phnophopia
    - meningitis: Headache + neck stiffness, fever, Rash.
- Firstly (rare): multiple sclerosis, motor neurone disease, brain tumor.
- don't mention (common): carpal tunnel syndrome, functional disorders.
- Time-Relationship**
  - when it start?
  - persistent (torb) or intermittent.
  - onset → sudden or gradual?

# 7.2.1 :- Common presenting symptoms

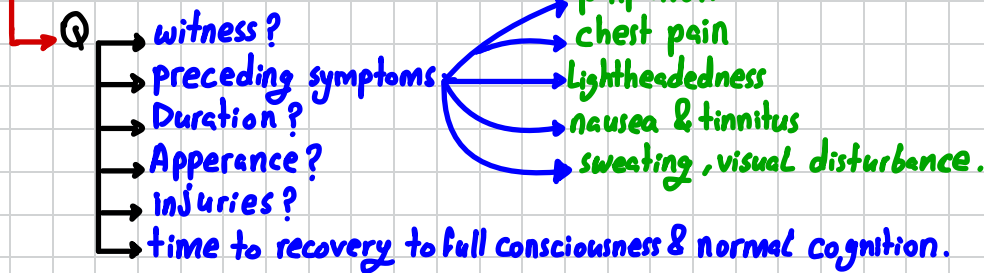
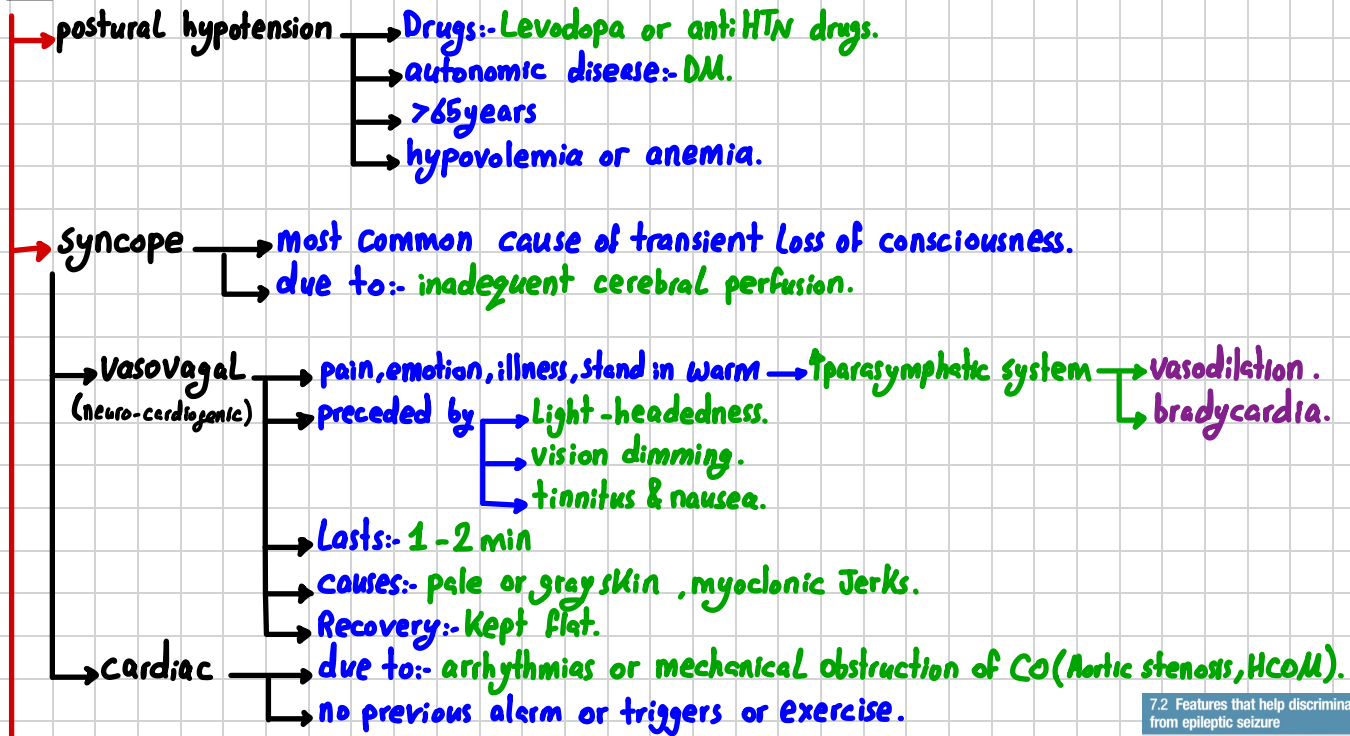
## 1 Headache (SOCRATES) :- most common symptom.



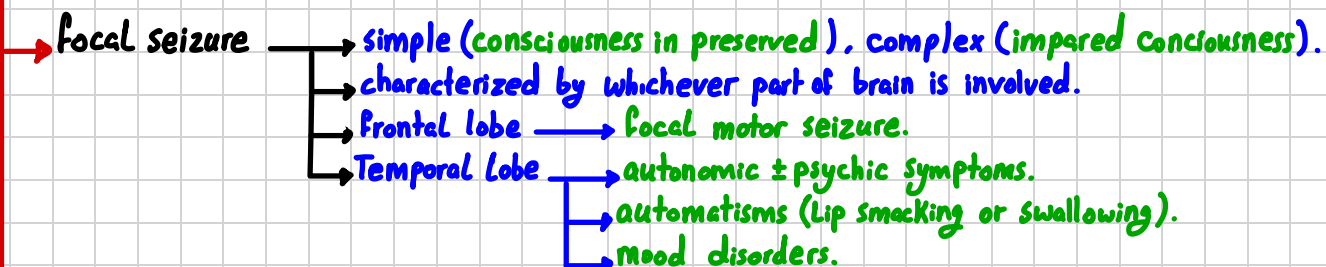
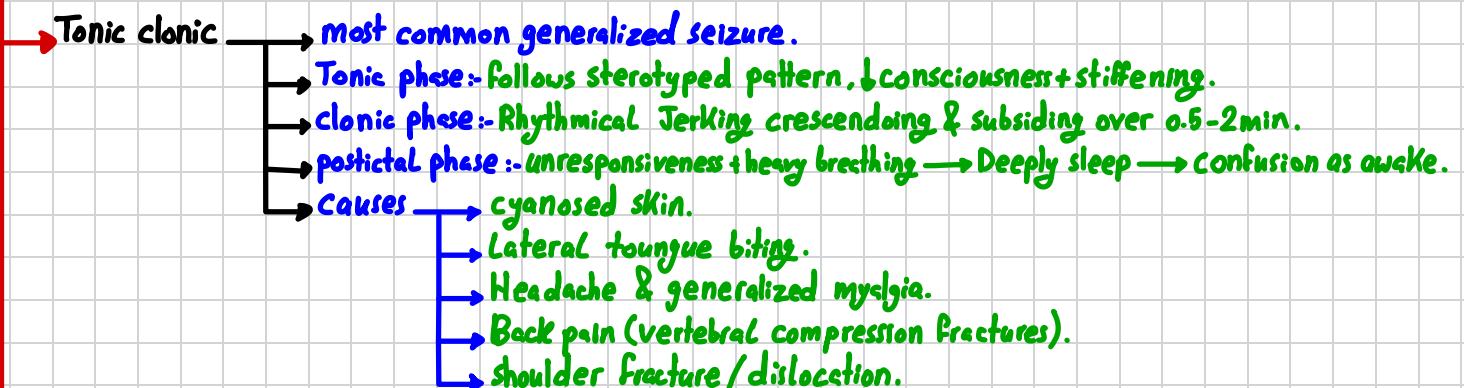
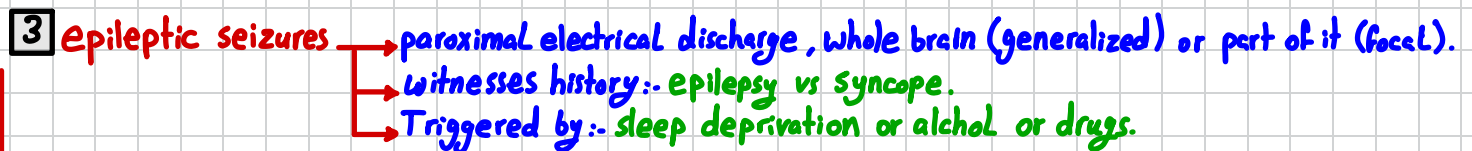
7.1 Clinical characteristics of headache syndromes

	Onset	Duration/periodicity	Pain location	Associated features
<b>Primary syndromes</b>				
Migraine	Evolves over 30-120 mins	Usually last <24 h, recurrent with weeks/months symptom-free	Classically unilateral but may be anywhere including face/neck	Aura (usually visual), nausea/vomiting, photophobia and phonophobia
Cluster headache	Rapid onset, often waking patient from sleep	30-120 mins, 1-4 attacks within 24 h, clusters usually last weeks to months, with months to years of remission	Orbital/retro-orbital; always same side during cluster, may switch sides between clusters	Autonomic features, including conjunctival injection, tearing, nasal stuffiness, ptosis, miosis, agitation
Stabbing headache	Abrupt, rarely from sleep	Very brief, seconds or less	Anywhere over head	Common in migraineurs
<b>Secondary syndromes</b>				
Meningitis	Usually evolves over a day or two, can be abrupt	Depends on cause and treatment, usually days to weeks	Global, including neck stiffness	Fever, meningism, rash, false localising signs, signs of raised intracranial pressure
Subarachnoid haemorrhage	Abrupt, immediately maximal, rare from sleep	May be fatal at onset, usually days to weeks	Anywhere, poor localising value	20% isolated headache only; nausea/vomiting, reduced consciousness, false localising signs, III nerve palsies
Temporal arteritis	Gradual onset of temple pain and scalp tenderness	Continuous	Temple and scalp	Usually in those >55 years; unwell, jaw pain on chewing, visual symptoms, tender temporal arteries, elevated erythrocyte sedimentation rate and C-reactive protein

## 2 Transient Loss of consciousness.



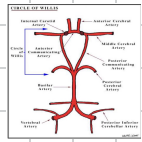
Feature	Vasovagal syncope	Seizure
Triggers	Typically pain, illness, emotion	Often none (sleep deprivation, alcohol, drugs)
Prodrome	Feeling faint/lightheaded, nausea, tinnitus, vision dimming	Focal onset (not always present)
Duration of unconsciousness	<60 s	1-2 mins
Convulsion	May occur but usually brief myoclonic jerks	Usual, tonic-clonic 1-2 mins
Colour	Pale/grey	Flushed/cyanosed, may be pale
Injuries	Uncommon, sometimes biting of tip of tongue	Lateral tongue biting, headache, generalised myalgia, back pain (sometimes vertebral compression fractures), shoulder fracture/dislocation (rare)
Recovery	Rapid, no confusion	Gradual, over 30 mins; patient is often confused, sometimes agitated/aggressive, amnesic



# Functional dissociative attack (non-epileptic, psychogenic, pseudoseizures)

- occurring multiple times in a day.
- may last considerably longer (>30min).
- symptoms waxing & waning.
- asynchronous movement.
- pelvic thrusts, side-to-side rather than flexion/extension
- abs of postictal confusion.
- no urine incontinence & normal eye.
- widespread videophones allows witnesses to capture such events.

## 4 stroke



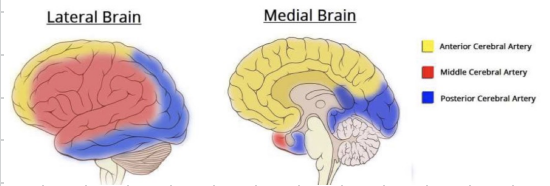
- focal neurological deficit of Rapid onset, due to vascular cause (ischemic or hemorrhagic).
- symptoms are dictated by the vascular territory involved.
- Anterior circulation (Ant & mid cerebral)
  - from internal carotid.
  - supplies most of cerebral hemispheres.
- posterior circulation (post. cerebral)
  - from verteobasilar circulation.
  - supplies occipital lobe & brainstem.
- ischemic :- 80% → CT scan.
- Hemorrhagic :- Asians
  - Anticoagulant.
  - Headache & vomiting.
  - seizures & early reduced consciousness.
- isolated vertigo, amnesia or TLoc are rarely, if ever due to stroke.

## Transient ischemic attack (TIA)

- time-based :- <24h resolved
- tissue-based :- MRI → no tissue loss.
- Risk factor for impending stroke.

## Total anterior circulation syndrome (TACS) (3H)

- Hemiparesis
- Hemianopia
- Higher cortical deficit :- dysphasia or visuospatial loss.



## partial anterior circulation syndrome (PACS).

- 2/3 of TACS
- or isolated higher cortical deficit.
- or motor/sensory deficit > LACS.

## posterior circulation syndrome (POCS)

- ipsilateral cranial nerve palsy + contralateral motor/sensory deficit.
- bilateral motor/sensory deficit.
- disorders of conjugate eye movement.
- cerebellar dysfunction without ipsilateral long-tract deficits.
- isolated homonymous visual field defect.

## Lacunar syndrome (LACS)

- pure motor :- >2/3 of face, arm, leg.
- pure sensory :- >2/3 of face, arm, leg.
- pure sensorimotor :- >2/3 of face, arm, leg.
- ataxic hemiparesis

## Spinal strokes

- Rare, abrupt, depending on the Lvl of spinal cord effected.
- Anterior spinal cord A syndrome :- most common

- Loss of motor function
- Loss of pain/Temp sensation.
- sparing of Joint position & vibration

Below the Lvl of Lesion

### 7.3 Clinical classification of stroke

#### Total anterior circulation syndrome (TACS)

- Hemiparesis, hemianopia and higher cortical deficit (e.g. dysphasia or visuospatial loss)

#### Partial anterior circulation syndrome (PACS)

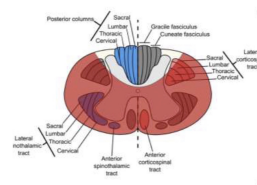
- Two of the three components of a TACS
- OR isolated higher cortical deficit
- OR motor/sensory deficit more restricted than LACS (see below)

#### Posterior circulation syndrome (POCS)

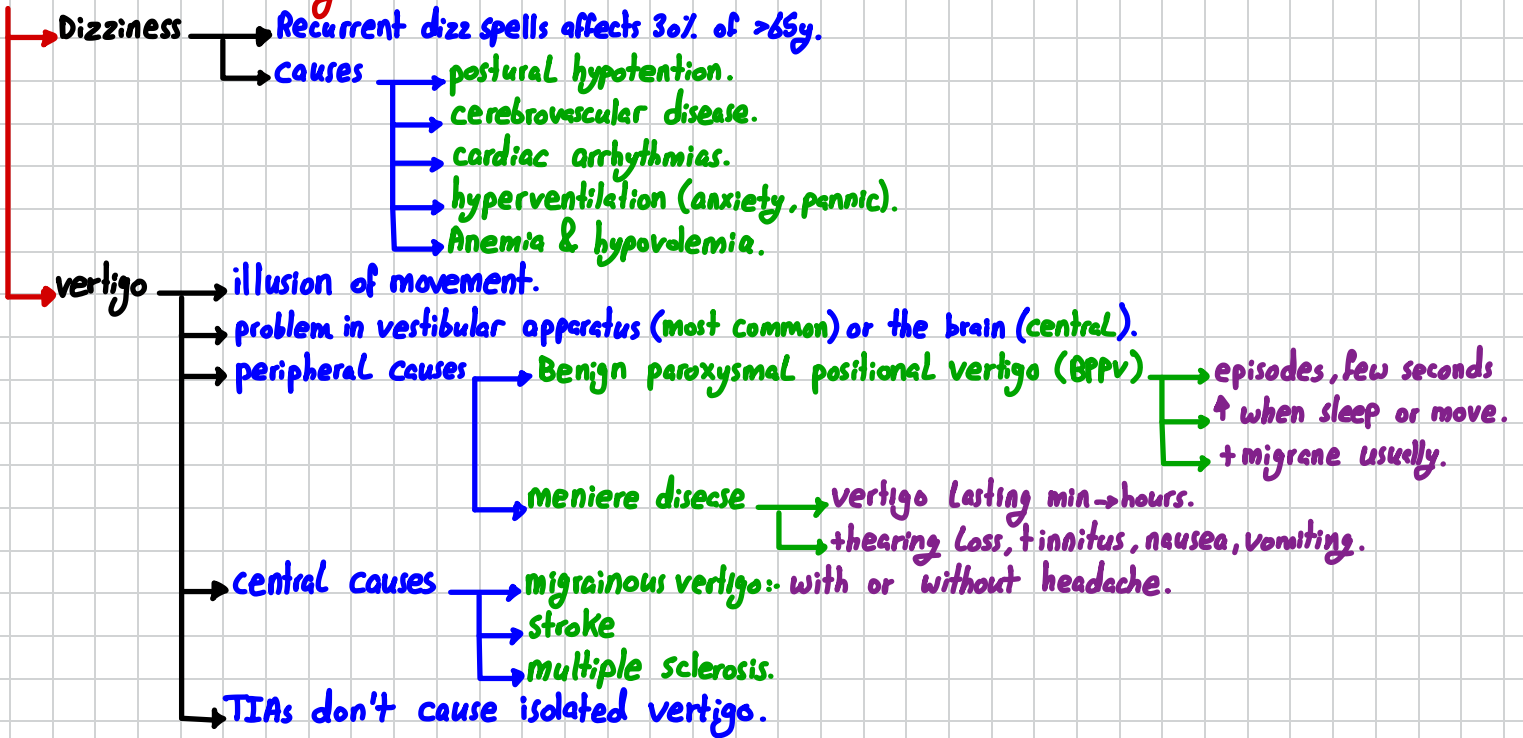
- Ipsilateral cranial nerve palsy with contralateral motor and/or sensory deficit
- OR bilateral motor and/or sensory deficit
- OR disorder of conjugate eye movement
- OR cerebellar dysfunction without ipsilateral long-tract deficits
- OR isolated homonymous visual field defect

#### Lacunar syndrome (LACS)

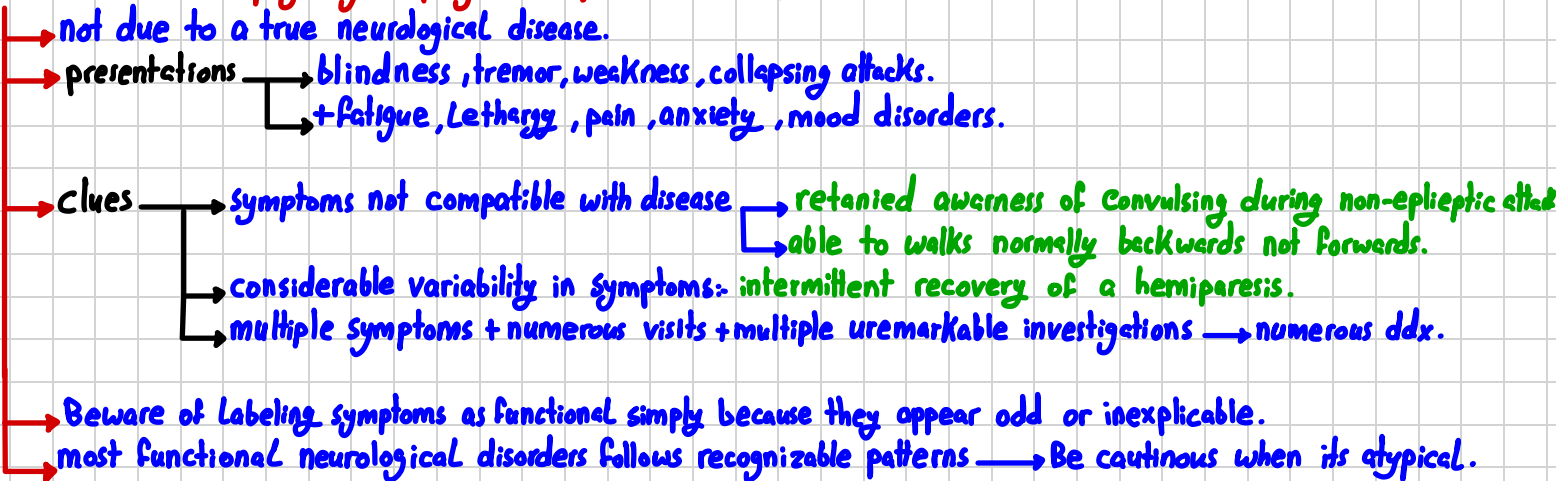
- Pure motor > 2 out of 3 of face, arm, leg
- OR pure sensory > 2 out of 3 of face, arm, leg
- OR pure sensorimotor > 2 out of 3 of face, arm, leg
- OR ataxic hemiparesis



## 5 Dizziness & vertigo



## 6 Functional / psychogenic / hysterical / somatisation / conversion disorders.



### • 7.2.2 :- past medical History

- multiple sclerosis :- Visual Loss (optic neuritis) + numbness.
- Epilepsy :- Birth history & development.
- Vascular cause :- HTN, smoking, family history, hyperlipidemia.

### • 7.2.3 :- Drug history.

- Always enquire about drugs (prescribed, over-the counter complementary & recreational / illegal drugs).
  - phenytoin toxicity → Ataxia.
  - ↑ simple analgesia → medication overuse headache.
  - Cocaine → convulsions.

### • 7.2.4 :- Family History.

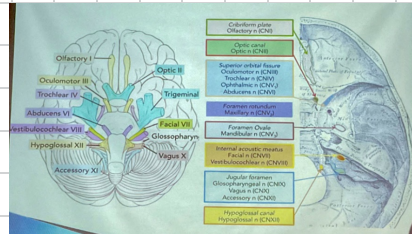
- parental consanguinity :- Common, ↑ risk of AR conditions.
- single-gene defects :- myotonic dystrophy or Huntington's disease.
- polygenic :- multiple sclerosis or migraine.
- charcot-marie-tooth disease :- AD or AR or X-linked.
- Mitochondria (own DNA) :- migraine or epilepsy.
- single-gene or sporadic :- parkinson's or motor neurone disease.

## 7.2.5: Social History

- How are patient coping with symptoms? work? drive?
- Support circumstance? adequate?
- Alcohol
  - CNS:- ataxia, seizures, dementia.
  - PNS:- neuropathy.
- Diet
  - vitamin deficiency:- alcoholism or dietary exculsion.
  - Vegetarians:- ↓B<sub>12</sub> → subacute combined degeneration of the spinal cord.
- recreational drugs
  - NO inhalers:- ↓B<sub>12</sub>
  - Smoking:- Vascular & malignant.
- travel
  - Lyme disease:- fascial palsy.
  - neurocysticercosis:- parasitic infection → Brain Lesion & epilepsy.
  - malaria:- Coma.
- sexually-transmitted or blood-borne infection
  - HIV
  - sypphilis ] → treatable neurological symptoms.
- Occupational History
  - Lead (heavy or organic metals):- motor neuropathy (toxic peripheral neuropathy).
  - manganase → parkinsonism.
  - effect on occupation:- epilepsy + driving/dangerous mechnery.
  - stop working:- dementia (cognitive disorder).

## 7.3: cranial nerves

- 12
  - cerebral cortex:- 1 & 2
  - midbrain:- 3 & 4
  - pons:- 5, 6, 7, 8
  - medulla:- 9, 10, 11, 12
  - Longest:- Vagus (X).
  - shortest:- olfactory (I).
  - Largest:- Trigeminal (V).
  - thinnest:- Trochlear (IV).
  - pure sensory:- 1, 2, 8
  - pure motor:- 3, 4, 6, 11, 12
  - mixed:- 5, 7, 9, 10
  - Autonomic:- 3, 7, 9, 10 (1973).



7.4 Summary of the 12 cranial nerves

Nerve	Examination	Abnormalities/symptoms
I	Sense of smell, each nostril	Anosmia/parosmia
II	Visual acuity Visual fields Pupil size and shape Pupil light reflex Fundoscopy	Partial sight/blindness Scotoma; hemianopia Anisocoria Impairment or loss Optic disc and retinal changes
III	Light and accommodation reflex	Impairment or loss
III, IV and VI	Eye position and movements	Strabismus, diplopia, nystagmus
V	Facial sensation Corneal reflex Muscles of mastication Jaw jerk	Impairment, distortion or loss Impairment or loss Weakness of chewing movements Increase in upper motor neurone lesions
VII	Muscles of facial expression Taste over anterior two-thirds of tongue	Facial weakness Ageusia (loss of taste)
VIII	Whisper and tuning fork tests Vestibular tests	Impaired hearing/deafness Nystagmus and vertigo
IX	Pharyngeal sensation	Not routinely tested
X	Palate movements	Unilateral or bilateral impairment
XI	Trapezius and sternomastoid	Weakness of scapular and neck movement
XII	Tongue appearance and movement	Dysarthria and chewing/swallowing difficulties

### 1 olfactory Nerve (I)

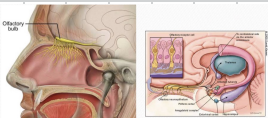
Anatomy:- Bipolar cells (olfactory bulb) → olfactory filaments + small receptors protecting cribriform plate → 2nd order neurons → olfactory tract → Temporal Lobe & amygdala.

purely sensory

Function → conveys the sense of smell.

Anormal Findings → Hyposmia-ansomia

- upper Respiratory tract infection
- sinus disease
- Head injury → damage of olfactory filaments.
- Local compression:- olfactory groove meningioma.
- invasion of basal skull tumors.
- parkinson's & Alzheimer's.



→ hypogeusia / ageusia:- altered ability to taste when they lost the smell.



parosmia :- perception of pleasant odours as unpleasant

olfactory hallucinations → Alzheimer's disease.  
focal epilepsies.

Head trauma.  
sinus infection.  
Drugs.

examination → self-reporting can be surprising inaccurate.  
limited clinical value :- nasal clearance → close eyes & 1 nostril → scratch & sniff (UPSIT).

## 2 Trigeminal nerve (V).

function → sensory :- face, mouth, part of dura, Anterior 2/3 of the tongue.  
motor :- Jaw involved in chewing.

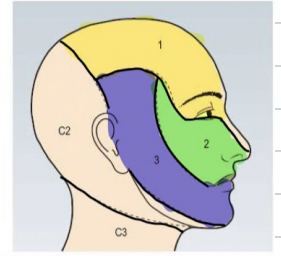


Fig. 11.5 The sensory distribution of the three divisions of the trigeminal nerve. (1) Ophthalmic division, (2) Maxillary division, (3) Mandibular division.

ophthalmic (V<sub>1</sub>) → course :- ganglion → superior orbital fissure → cavernous sinus.  
supplies → skin of upper nose, eyelid, forehead, scalp.  
eye (cornea & conjunctiva).  
mucus membrane of sphenoidal & ethmoid sinus.

Maxillary (V<sub>2</sub>) → course :- ganglion → cavernous sinus → foramen rotundum.  
supplies → mucus of upper mouth, roof of pharynx, gum, teeth & palate, maxilla.  
sphenoidal & ethmoid sinuses.

Mandibular (V<sub>3</sub>) → course :- exit the skull via foramen ovale.  
sensory :- Anterior 2/3 of tongue, gum, teeth, temporomandibular Joint.  
not the angle of Jaw (great auricular).  
motor :- muscles of mastication → temporals.  
masseter.  
medial & lateral pterygoids.

Examination → sensory → Ant. 2/3 of tongue.  
Light-touch :- cotton.  
superficial pain :- Neurotip.  
nasal tickle test.

motor → wasting of mastication muscles.  
fall the masseters.  
Jaw deviation.

Reflex → Corneal Reflex (Affrent :- V<sub>1</sub>, Effrent :- facial N).  
Jaw Jerk → Abs - Normal  
Brisk Jaw Jerk occurs in pseudobulbar palsy

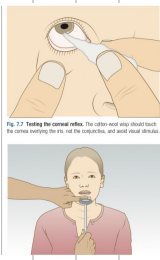


Fig. 7.7 Testing the corneal reflex. The afferent and efferent pathways are shown.

**Bulbar palsy** refers to impairment of function of the cranial nerves IX, X, XI and XII, which occurs due to a lower motor neuron lesion either at nuclear or fascicular level in the medulla oblongata or from lesions of the lower cranial nerves outside the brainstem.

**In contrast, pseudobulbar palsy** describes impairment of function of cranial nerves IX-XII due to upper motor neuron lesions of the corticobulbar tracts in the mid-pons. For clinically evident dysfunction to occur, such lesions must be bilateral as these cranial nerve nuclei receive bilateral innervation. Bulbar Palsy is an assortment of signs and symptoms, not the name of a precise disease.

7.5 Comparison of bulbar and pseudobulbar palsy		
	Bulbar palsy	Pseudobulbar palsy
Level of motor lesion	Lower motor neurone	Upper motor neurone
Speech	Dysarthria	Dysarthria and dysphonia
Swallowing	Dysphagia	Dysphagia
Tongue	Weak, wasted and fasciculating	Spastic, slow-moving
Jaw jerk	Absent	Present/brisk
Emotional lability	Absent	May be present
Causes	Motor neurone disease	Cerebrovascular disease, motor neurone disease, multiple sclerosis

Abnormalities → direct injury :- unilateral loss of sensation. (Sjögren's syndrome).  
Lesion of cavernous sinus :- Loss of corneal reflex & V<sub>1</sub> & V<sub>2</sub> cutaneous sensory loss.  
Herpes zoster ophthalmicus :- V<sub>1</sub> distribution (Hutchinson's sign).  
bilateral UMN lesion above pons :- Brisk Jaw Jerk.  
myasthenia gravis :- weakness of mastication M, fatigable chewing.

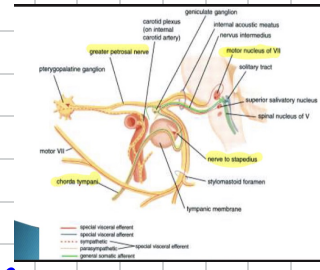


## 3 Facial nerve (VII)

functions → motor for facial expression → Frontalis orbicularis oculi.  
Buccinators.  
orbicularis oris.  
platysma.



parasympathetic :- via nervus intermedius → Lacrimal gland.  
 → submandibular & sublingual.  
 sensation from Anterior 2/3 of the tongue.

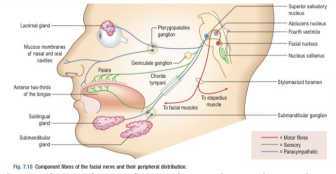


COURSE :- +VIII → internal acoustic meatus → facial canal → stylomastoid foramen  
 → parotid → Terminal branches

- Temporal ← "تلہر زلیہ بعب مرتہ کثیر"
- Zygomatic
- Buccal
- Mandibular
- cervical



give branches to → stapedius :- paralysis → ↑voice.  
 → parasympathetic + chorda tympani



**Examination**

- inspect for asymmetry, difrence in blinking or eye closure.
- watch any spontaneous or involuntary movements.
- Raise eyebrows → Asymetrical wrinkling of forehead (frontalis M).
- screw their eye tightly shut & Resist to open them (orbicularis oculi).
- Bare theeth (orbicularis oris).
- Blow out + closed mouth (buccinctors & orbicularis oris).



**Abnormal**

- unilateral LMN Lesion :- ipsilateral weakness of upper & Lower face.
- Bell's palsy :- idiopathic acute LMN (mastoid pain + imparment taste + hyperacusis (pitched sounds)).
- Bell's phenomenon :- closure is incomplete → globe rolls upward to avoid corneal exposure.
- Ramsay hunt syndrome :- Herpes zoster to geniculate ganglion → sever LMN palsy.
- other causes of LMN
  - trauma
  - parotid tumors
  - cerebellopontine angle tumors (acoustic neuroma)
- ipsilateral Loss of taste & buccal ulceration
- painful vesicular eruption in (EAM).

**Synkinesis**

deff :- involuntary contraction + voluntary movement (mouth corner + blinking).  
 sign of aberrant reinnervation, recovering LMN Lesion.

unilateral UMN Lesion :- contralateral paralysis of Lower face only (upper face got innervation bilaterally).

- nasolabial fold flattened.
- dropped mouth corner.
- eye closure preserved.
- Hemifacial spasm.
- synchronised twitching of ipsilateral eye & mouth.

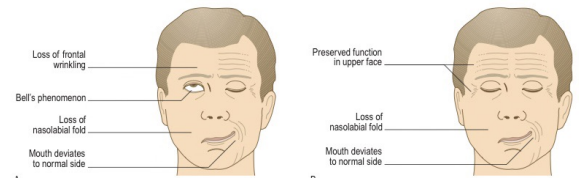


Fig. 7.12 Types of facial weakness. [A] Right-sided lower motor neurone lesion (within facial nerve or nucleus); Bell's phenomenon is also shown. [B] Right-sided upper motor neurone lesion.

**Bilateral facial palsies (rare)**

- Guillain-Barre syndrome.
- Sarcoidosis
- infection :- Lyme, HIV, Leprosy.

**DPx**

- congenital myopathias → facioscapulohumeral, myotonic dystrophies.
- parkinson's disease.

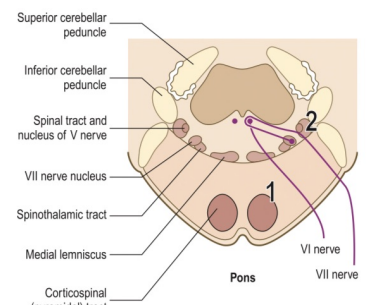
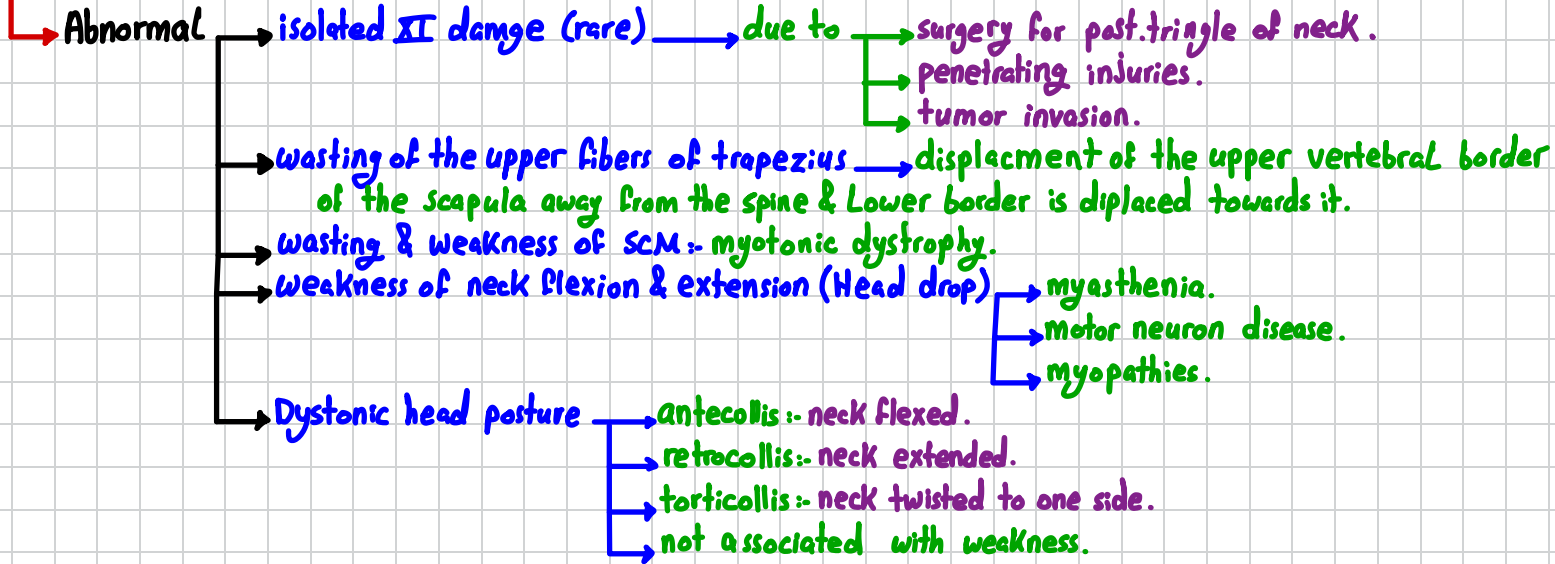
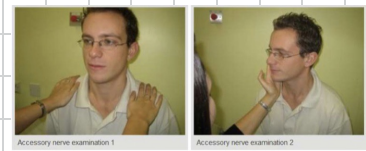
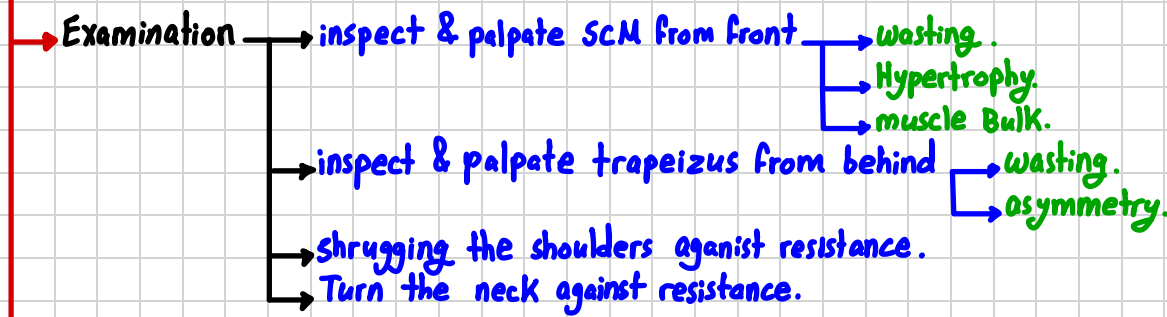


Fig. 7.11 Lesions of the pons. Lesions at (1) may result in ipsilateral VI and VII nerve palsies and contralateral hemiplegia. At (2) ipsilateral cerebellar signs and impaired sensation on the ipsilateral side of the face and on the contralateral side of the body may occur.





## ⚠ Hypoglossal N (XII)

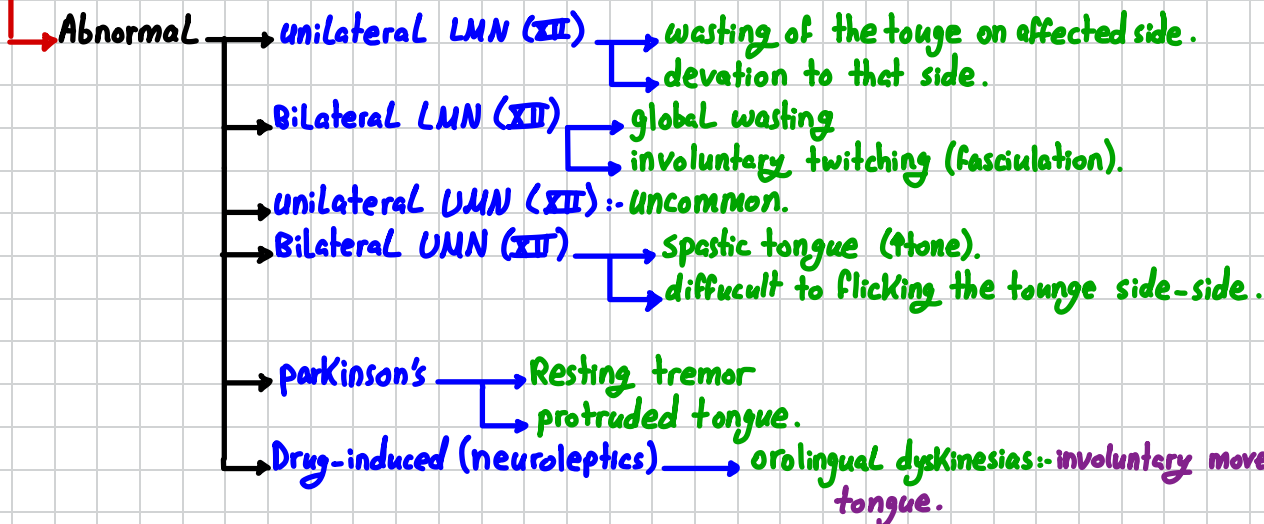
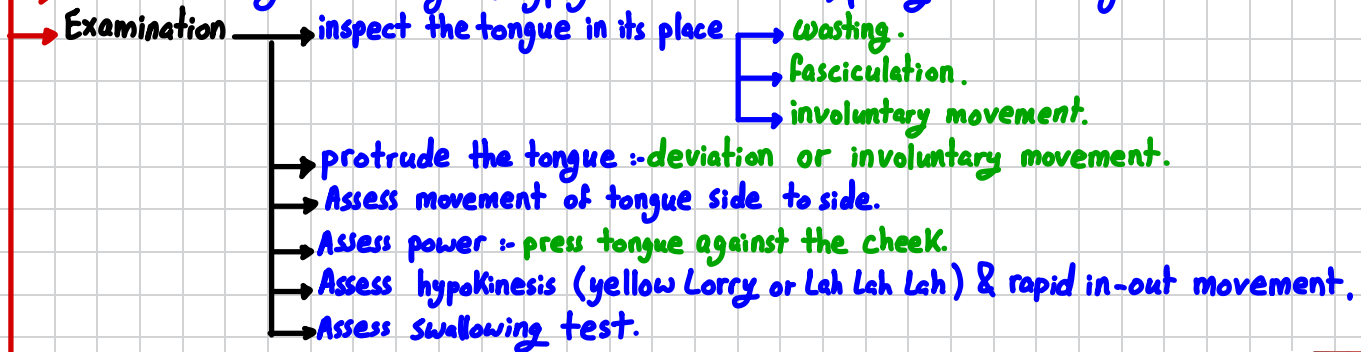
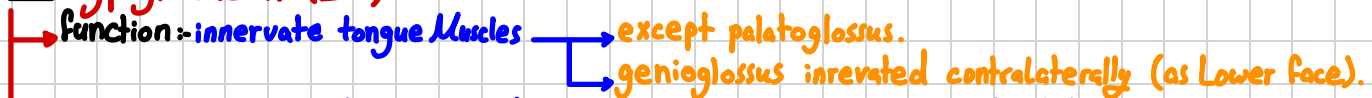


Fig. 7.14 Left hypoglossal nerve lesion. From Epstein O, Perkin GD, de Bono DP, et al. Clinical Examination, 2nd edn. London: Mosby; 1997.

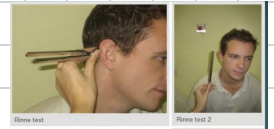
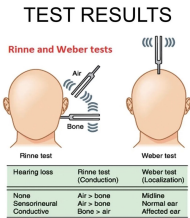
# 7.4: Vestibulocochlear N & vision.

## 1 Vestibulocochlear N (VIII)

**Function** → vestibular part: - Balance & proprioception.  
 → Cochlear part: - Hearing.

**Examination**

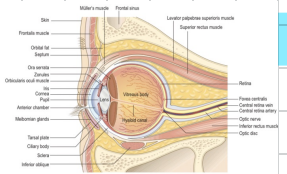
- Whispered voice test**
  - behind the pt, 15cm → repeat
  - start with normal speaking.
  - Normal: - repeat words whispered at 60cm.
- Weber's test**
  - place the vibrating tuning fork in the middle of forehead.
  - Normal: - noise heard in middle, equally in both ears
  - conductive: - Louder in affected ear.
  - sensorineural: - Louder in unaffected ear.
  - Symmetrical hearing Loss: - middle.
- Rinne's test**
  - place the vibrating tuning fork on mastoid process → voice disappears → place it at external auditory meatus.
  - Normal: - AC > BC → Rinne-positive.
  - conducting: - BC > AC → Rinne-negative.
  - sensorineural: - AC > BC → Rinne-positive (as normal).
  - false-negative: - in profound hearing loss on one side due to BC to good ear.



## • Vision

**Anatomy**

- The eye is located inside the orbit
- covered by eyelid: - protect the eye, maintain tear film.
- Elevation of eye by
  - Levator palpebrae superioris: - CN3
  - Muller muscle: - Sympathetics Nerves (Hurner syndrome).
- closure by: orbicularis oculi: - CN7.

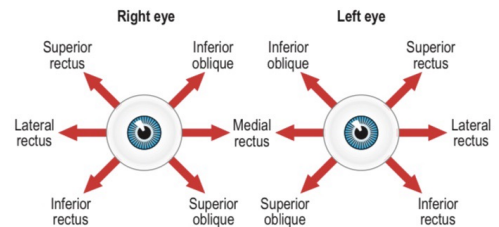


**conjunctiva**

- mucus membrane lining in posterior aspect of the eyelid.
- reflected at the superior & inferior fornices on globe surface.
- Coated in a tear film that protects & nourishes the ocular surface.

**orbit**

- 6 extraocular M
  - Superior rectus
  - Inferior rectus
  - Lateral rectus: CN6
  - medial rectus
  - superior oblique: - CN4.
  - inferior oblique
- All are supplied by CN3 except (SO4) & (LR6). from midbrain & pons → cavernous sinus
- others
  - Lacrimal glands
  - Blood vessels
  - autonomic & CN2, CN3, CN4, CN6
  - orbital fat



**Eye Ball: 25 mm in length & comprises 3 distinct layers.**

- outer fibrous: - sclera & cornea (2/3 of refractive power, focusing incident light on retina).
- middle vascular (uveal tract): - Ant. → ciliary body & iris, post. → choroid.
- inner neurosensory (retina): - converting light → neurological signals.

## → pupillary pathway

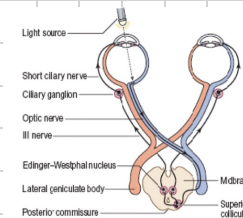
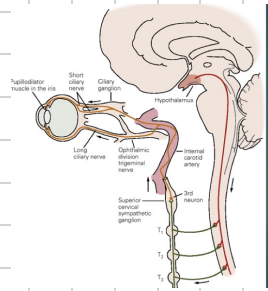


Fig. 12.5 Pathway of pupillary constriction and the light reflex (parasympathetic).

**pupils** → controls amount of light entering the eye.  
The intensity of light determines the pupillary aperture via autonomic reflexes.

**Constriction:- parasympathetic** → A:- optic N → LGB → pretectal Nucleus → Edinger-Westphal (III N).  
E:- short ciliary N → pupil.

**dilation:- sympathetic**:- Hypothalamus → ciliospinal centre of budge (T<sub>1</sub> LvL) → superior cervical G → nasociliary & long ciliary N → pupil.



## 2 optic nerve (2)

- purely sensory.
- similar to white matter rather than peripheral N (unable to regenerate).
- function → transmitting visual sensory info from retina → Brain.  
→ Affrent part of pupillary Reflex.
- visual pathway :- Retina → optic N → optic chiasm → optic tract → LGB → optic radiation → occipital cortex
- nasal fibers → temporal visual field (vise versa).
- Lesions → Before optic chiasm :- ipsilateral.  
→ After optic chiasm :- contralateral.

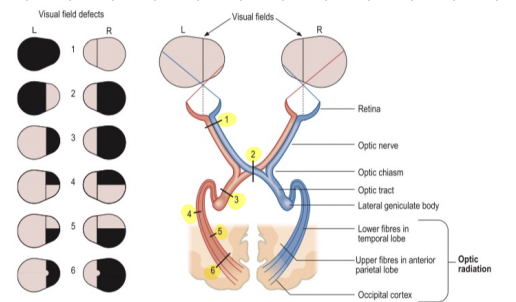
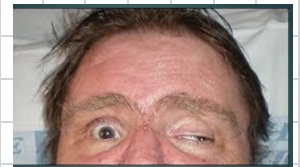


Fig. 8.5 Visual field defects. 1, Total loss of vision in one eye because of a lesion of the optic nerve. 2, Bitemporal hemianopia due to compression of the optic chiasm. 3, Right homonymous hemianopia from a lesion of the optic tract. 4, Upper right quadrantanopia from a lesion of the lower fibres of the optic radiation in the temporal lobe. 5, Lower quadrantanopia from a lesion of the upper fibres of the optic radiation in the anterior part of the parietal lobe. 6, Right homonymous hemianopia with sparing of the macula due to a lesion of the optic radiation in the occipital lobe.

## 3 Oculomotor nerve (3)

- motor & parasympathetic function.
- innervates → All extraocular except (SO<sub>4</sub>) & (LR<sub>6</sub>).  
→ Levator palpebrae superioris M.
- course :- Related to posterior communicating A & cavernous sinus.
- function → moves globe upward, downward, medially.  
→ elevate upper Lid.  
→ pupillary Reflex (constrict pupil).
- palsy → complete ptosis (Levator palpebrae M).  
→ Large pupil  
→ eye Looking inferiolateral. (medial Rectus paralyzed → Latent ructus overactive).  
→ Diplopia.



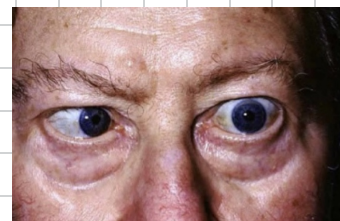
## 3 Trochlear nerve (4)

- supplies :- Superior oblique M (SO<sub>4</sub>).
- function :- inferiolateral movement.
- injury → Diplopia.  
→ Difficulty in turning the eye inferio-lateral.  
→ Difficulty in descending stairs.  
→ compensatory adjustment :- Head tilt to the opposite side of paralysed eye.



## 4 Abducent nerve (6)

- supplies :- Lateral Rectus M (LR<sub>6</sub>).
- function :- Abduct the eye (lateral gaze).
- course → Long around brainstem → pierces dura → cavernous sinus.  
→ direct relation to internal carotid A → superior orbital fissure → (LR<sub>6</sub>).
- injury → Diplopia  
→ eye turns medially (medial rectus overaction).



# • vision physical examination

## Examination of vision

- ▶ Inspection and palpation
- ▶ Pupils
- ▶ Visual fields:
  1. Homonymous defects
  2. Sensory inattention
  3. Peripheral visual fields
  4. Color desaturation
  5. Central visual field
  6. Blind spot
- ▶ Eye movements
- ▶ Visual acuity: snellen chart
- ▶ Ophthalmoscopy
- ▶ Color vision : ishihara test
- ▶ Macular function : amsler grid

### 1 General examination

- posture & gait
- Head position
- face asymmetry & dysmorphic features.
- eyelid position & periocular skin.
- position & symmetry of gaze.

### 2 Inspection & palpation (orbit & periorbital examination).

- Look for
  - swelling or erythema in periocular skin
  - symmetry of gaze.
  - pupil size & symmetry.
  - ptosis
  - proptosis:- eyeball protrusion.
  - Lid lag.
  - check eye movement.
- palpate → masses in orbital rim.
- ophthalmoscope:- optic disc swelling from compression.
- causes of ptosis
  - Neurological
    - 3rd nerve palsy.
    - Horner syndrome
  - myogenic
    - myotonic dystrophy.
    - chronic progressive external ophthalmoplegia.
  - Neuromuscular → myasthenia gravis.
  - Mechanical
    - infection.
    - trauma.
    - inflammation.
    - tumor.
  - Degeneration

Cause	Diagnosis	Associated distinguishing features
Neurogenic	Horner's syndrome	Ptosis, miosis, eye movement spared
	Cranial nerve III palsy	Dilated pupil, eye movements affected (see Fig. 8.10)
Myogenic	Myotonic dystrophy	Frontal balding, sustained handgrip
	Chronic progressive external ophthalmoplegia	Bilateral ptosis and impairment of eye movements, often without diplopia, sparing of pupil reflexes
	Oculopharyngeal dystrophy	History of swallowing abnormalities
Neuromuscular junction	Myasthenia gravis	History of variable muscular fatigue
Mechanical	Eyelid tumour	Evident on inspection
	Eyelid inflammation/infection	Evident on inspection
	Trauma	Scarring/history of trauma
Degenerative	Levator aponeurosis degeneration Long-term contact lens wear	Often unilateral, eye movement normal History of contact lens use

### 3 pupils

- anisocoria
  - unequal pupil size, 20% of population.
  - Examination
    - pt fixation at point of distance → ↑ & ↓ illumination.
    - ↑ in brighter light:- larger pupil is abnormal
    - ↑ in dim light:- small pupil is abnormal.
    - = in size in both light:- physiological.
  - causes
    - Dilated pupil
      - CN3 palsy.
      - Adie's pupils
      - post surgery.
      - physiological in 20%
      - Drugs (atropine, tropicamide).
    - constricted pupil
      - horner syndrome.
      - Late stage of Adie's pupils
      - mechanical (iritis, trauma).
      - physiological in 20%
      - Drugs (pilocarpine).

Dilated pupil	
• Cranial nerve III palsy	• Physiological
• Pharmacological treatment with a dilating agent (e.g. tropicamide or atropine)	• Post-surgical
	• Adie's tonic pupil
Constricted pupil	
• Horner's syndrome	• Late-stage Adie's tonic pupil
• Mechanical, e.g. secondary to posterior synechiae in iritis or trauma	• Pharmacological treatment with a constricting agent (e.g. pilocarpine)
• Physiological	

### → Direct & consensual Light Reflex

- direct:- pt. fixation on a point of distance + ambient light → constriction of ipsilateral pupil.
- consensual:- same as direct but look for constriction of contralateral pupil.

## Relative Afferent pupillary Reflex (RAPD)

- occurs when disease of retina & optic N reduces the response of eye to light stimulus
- Examination:- move light from one eye to the other, min. 3sec to each eye.
- Normal:- symmetrical constriction of both pupils.
- RAPD:- the affected eye cause weak constriction (apparent dilation).

## Accommodation

- Examination:- Ask the pt. to look at close fixation (no light) after fixation on a distance target.
- normally
  - constriction of pupil on near gaze.
  - convergence.

## Horner syndrome

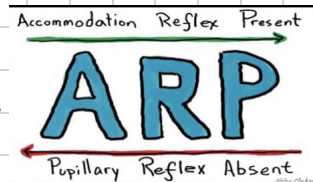
- causes
  - miosis (↓ sympathetic dilation).
  - ptosis (Muller's M).
  - anhidrosis on effected side
- due to
  - neck surgery or trauma
  - demyelination disease.
  - pneumothorax (apical lung tumor).
  - carotid A dissection
- Diagnosis:- cocaine eye drops - pupil dilation in unaffected pupil.

## Adie's pupil

- neurological disorder characterized by mid-dilated pupil that reacts poorly to light & accommodation
- with time the pupil constrict.
- more common in young women
- due to:- parasympathetic dysfunction.
- associated with ↓ ankle reflex → Holmes-Adie syndrome.

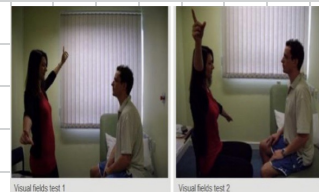
## Argyll Robertson pupil

- bilateral small irregular pupils, ⊕ Accommodation, ⊖ Light-Reflex
- causes
  - Syphilis
  - DM
  - severe optic disease.
  - midbrain lesion.



## 4 Visual field

- Normal:- 160° horizontally, 130° vertically.
- Blind spot:- 15° temporal to point of fixation (optic N).
- Compare pt. visual field with yours, face him/her 1m away.



## Homonymous defects

- Both eyes. 4 quadrants from periphery → centre.

sensory inattention, neglect:- can't see both sides moving.

peripheral visual fields:- each eye separately. (same as homonymous defects).

color desaturation:- dull or pale red → color desaturation (optic N dysfunction).

central vision:- each quadrant with red target, color desaturation

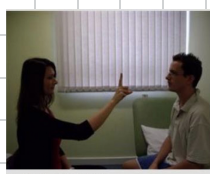
Blind spot:- move the target temporally → disappears → up-down, side-side.

early visual field loss:- Repeat the test with red neurotip, they should say when they first see that the target is red.

## 5 eye movement (3, 4, 6 CNS). H

### Examination

- Look for 0.5-1m away.
- Look for gaze palsy
- double vision or blurred (vertical/horizontal/combined).
- observe for nystagmus.



Oculomotor examination

## 6 Visual activity.

- each eye separately, mandatory in all ophthalmic pt.
- Snellen chart:- Lines are numbered according to distance in (m) that normal people can read them.
- wear their reading spectacles (Near/reading → Reading vision test).
- pt. Can't see:- 6m → 3m → 1m → count fingers → Hand movement → Light/dark.
- Results
  - distance (6m) / number of smallest font line read correctly on chart.
  - pt cannot read line 6 (6/6) :- place a pinhole directly in front of eye to correct any residual refraction error.
  - if its not improve with pinhole:- not related to refractive apparatus alone (optic N pathology).
  - Normal:- 6/6
  - near vision → text of ↓font → comfortable reading distance (>40y need spectacles for presbyopia).

## 7 ophthalmoscopy :- optic N pathology.



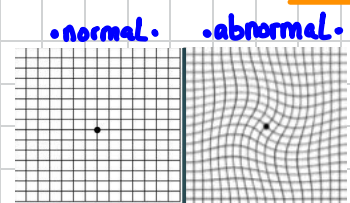
age-Related deterioration in near-vision

## 8 ishihara test:- color blindness.



## 9 Asmler grid for macular function (20x20)

→ macular pathology :- change of position in photoreceptors.

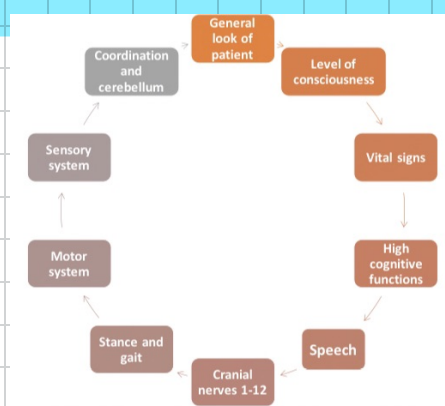


# 7.5:- Neuro examination

## physical examination

General Look :- Begins with your first contact with pt.

- facial expression
- General demeanor, dress.
- posture
- gait.
- speech.
- involuntary movement.
- mental state

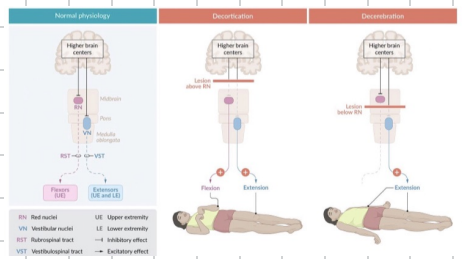


## 1 Assessment of conscious Lvl.

- 2 components
  - depends on integrity of (RAS), from Brain stem → thalamus.
  - How aware the person is & depends on cerebral cortex, thalamus, their connections.

## Glasgow coma scale

18.5 Glasgow Coma Scale (GCS)	
<b>Eye opening (E)</b>	
4	Spontaneously
3	To speech
2	To pain
1	No response
<b>Best verbal response (V)</b>	
5	Orientated
4	Confused
3	Inappropriate words
2	Incomprehensible sounds
1	No verbal response
<b>Best motor response (M)</b>	
6	Obeys commands
5	Localises painful stimulus
4	Normal flexion
3	Abnormal flexion (decortication)
2	Extends to painful stimulus (decerebration)
1	No response

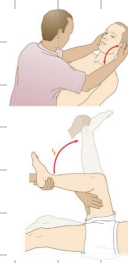


## 2 meningeal irritation signs.

- meningism
  - inflammation or irritation of meninges.
  - Lead to
    - neck stiffness :- Resistance to passive flexion of the neck → (Bruzinski's sign).
    - Kering's sign :- extended leg
      - ⊕ :- extension is resisted by spasm in hamstrings.
      - ⊖ :- Local causes of neck stiffness → cervical spine ↑intracranial pressure.
  - due to
    - meningitis
    - subarachnoid hemorrhage.
    - non-neurological infection
      - UTI
      - pneumonia.



→ meningitis → fever  
 → neck stiffness:- ↓sensitivity, ↑specificity.  
 → altered mental state



**3** Speech

→ Dysarthria → slurred or strangled speech caused by articulation problem due to motor deficit.  
 → due to → Localised lesion in tongue, Lips, mouth.  
 → ill fitting dentures  
 → neurological motor deficit

→ pseudobulbar palsy . Bilateral UMN (corticobulbar tract)  
 → slow, Harsh, strangled, ↓pronouncing consonants.  
 → +Brisk Jaw & emotional lability.  
 → tongue is contracted & stiff.

→ Bulbar palsy, Bilateral LMN Lesion (IX, X, XI, XII)  
 → weakness of the tongue results in difficulty with lingual sounds, while palatal weakness give a nasal quality to the speech.

→ cerebellar dysarthria:- slow & slurred, similar to alcohol intoxication.  
 → Myasthenia gravis:- fatiguing speech, becoming increasingly nasal & dissaper.  
 → parkinsonism:- dysarthria & dysphonia, ↓volume, monotonous voice, words running into each other (festination) & marked stuttering/heistation.

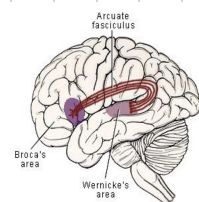
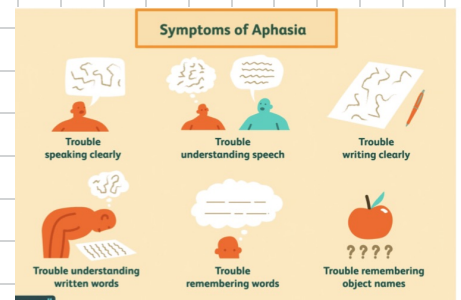
→ Dysphonia → due to → vocal cord pathology (Laryngitis).  
 → damage of vasaLN supply (recurrent laryngeal N).  
 → inability to abduct one vocal cord → Bovine cough. (ineffective).

→ examination → Listen to pt. spontaneous speech, noting volume, rhythm & clarity.  
 → special words → yellow lorry → Lingual (tongue).  
 → Baby hippopotamus → Labial (Lip).  
 → the Leith police dismisseth us → tongue twister.  
 → count steadily to 30 to assess fatigue → MG.  
 → pt. cough & say 'Ah' → soft palate raising bilaterally.

**4** Dysphasia → disturbance of language resulting in abnormalities of speech production and/or understanding.  
 → may involve other language symptoms (writing & reading).

→ Broca's Area → inferior frontal region  
 → word production & language expression.  
 → Expressive (motor) dysphasia → ↓verbal output, non-fluent.  
 → errors of grammar & syntax.  
 → comprehension is intact.

→ Wernicke's area → superior posterior temporal lobe.  
 → comprehension of spoken language.  
 → +understanding written language & numbers Areas.  
 → Receptive (sensory) dysphasia → fluent speech.  
 → meaningless.  
 → paraphrasias & neologism.  
 → comprehension is poor.



- Conduction dysphasia
- Global dysphasia:- Both (sensory + motor).
- Dyslexia:- difficulty comprehending written language.
- Dyscalculia:- problems with simple addition & subtraction.
- Dysgraphia:- impairment of writing.

Supramarginal <sup>awlogy</sup> gyrus.

- Gerstmann syndrome
  - dysgraphia + dyscalculia.
  - finger agnosia :- inability to recognise fingers.
  - inability to distinguish Lt from Rt.
  - Localises in Lt. parietal lobe (angular gyrus).

### GERSTMANN SYNDROME

**WHERE?**  
Dominant (usually left) inferior parietal lobe, angular gyrus & adjacent white matter

**WHY?**  
Stroke (main cause)  
Traumatic Brain Injury  
Tumor

- 1 Finger agnosia**  
Inability to name their own fingers, affecting both sides
- 2 Right-left confusion**  
Inability to differentiate right and left, affecting both sides
- 3 Acalculia**  
Inability to perform simple arithmetic calculations
- 4 Agraphia (without alexia)**  
Inability to write spontaneously, patient is still able to copy

## 5 Cortical Functions

- Thinking, emotion, language, behavior, planning, initiation of movement, perception of sensory info.
- Time-consuming.
- very important in pt. who display cognitive symptoms.
- tools
  - MMSE
  - MoCA → quick
  - Addenbrooke's :- more detailed.

## • Frontal Lobe

- posterior part (precentral gyrus) → motor strip (somatotopically)
- anterior :- personality, behaviour, emotions, cognition, expressive language & eye field, micturition.

- Damage
  - personality & behaviour change (apathy or disinhibition).
  - Loss of emotional responsiveness/stability.
  - cognitive impairment (memory, attention, concentration).
  - dysphasia
  - conjugate gaze deviation to side of lesion.
  - urinary incontinence.
  - primitive reflexes (grasp).
  - focal motor seizure (motor strip).

<b>2 Parietal lobe</b> <b>Dominant side</b> <b>FUNCTION</b> Calculation Language Planned movement Appreciation of size, shape, weight and texture. <b>LESIONS</b> Dyscalculia Dyspraxia Dyslexia Apraxia Agnosia Homonymous hemianopia	<b>Non-dominant side</b> <b>FUNCTION</b> Spatial orientation Constructional skills <b>LESIONS</b> Neglect of non-dominant side Spatial disorientation Constructional apraxia Dressing apraxia Homonymous hemianopia
<b>1 Frontal lobe</b> <b>FUNCTION</b> Personality Emotional response Social behaviour <b>LESIONS</b> Disinhibition Lack of initiative Antisocial behaviour Impaired memory Incontinence Grasp reflexes Anomia	<b>3 Occipital lobe</b> <b>FUNCTION</b> Analysis of vision <b>LESIONS</b> Homonymous hemianopia Homonymous scotomas Visual apraxia Impaired face recognition (prosopagnosia) Visual hallucinations (lights, lines and zig-zags)
<b>4 Temporal lobe</b> <b>Dominant side</b> <b>FUNCTION</b> Auditory perception Spoken language Verbal memory Smell <b>LESIONS</b> Dyspraxia Dyslexia Poor memory Complex hallucinations (taste, sound, vision) Homonymous hemianopia	<b>Non-dominant side</b> <b>FUNCTION</b> Auditory perception Music, tone sequences Non-verbal memory Faces, shapes, music Smell <b>LESIONS</b> Poor non-verbal memory Loss of musical skills Complex hallucinations Homonymous hemianopia

## • Temporal Lobe

- contains
  - primary auditory cortex.
  - wernicke's area.
  - part of limbic system :- memory, emotions, smell appreciation.
  - Lower fibers of optic radiation.
  - area of auditory perception.
- dysfunction
  - memory impairment
  - focal seizures with psychic symptoms.
  - contralateral upper quadrantanopia.
  - receptive dysphasia.

## • Parietal Lobe

- postcentral gyrus → sensory strip :- principal destination of conscious sensation.
- contains
  - optic radiation
  - aspect of language (dominant).
  - spatial awareness (non-dominant).

- dysfunction
  - cortical sensory impairment.
  - contralateral lower quadrantanopia.
  - dyslexia, dyscalculia, dysgraphia.
  - apraxia:- inability to carry complex tasks.
  - focal sensory seizures (postcentral gyrus).
  - visuospatial disturbance (non-dominant lobe).

• Occipital Lobe.

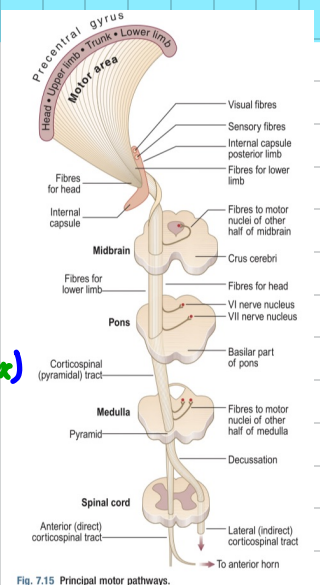
- Function:- analysis visual information.
- damage
  - visual field defect
    - Hemianopia:- Loss of part of visual field.
    - Scotoma:- Blind spot.
  - visual agnosia:- inability to recognize visual stimuli.
  - disturbances in visual perception
    - macropsia:- seeing things larger.
    - micropsia:- seeing things smaller.
  - visual hallucinations.

• Motor system

• Anatomy

- CNS:- corticospinal (UMN, pyramidal).
- PNS:- anterior horn cell (LMN)
- others:- Basal ganglia & cerebellum.

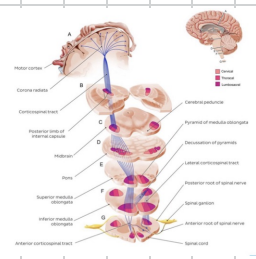
7.6 Features of motor neurone lesions		
	Upper motor neurone lesion	Lower motor neurone lesion
Inspection	Usually normal (may be disuse wasting in longstanding lesions)	Muscle wasting, fasciculations
Tone	Increased with clonus	Normal or decreased, no clonus
Weakness	Preferentially affects extensors in arms, flexors in leg	Usually more focal, in distribution of nerve root or peripheral nerve
Deep tendon reflexes	Increased	Decreased/absent
Plantar response	Extensor (Babinski sign)	Flexor



• Lesions

- UMN
  - affects CNS pathway (UMN under uninhibited influence of spinal reflex)
  - have an
    - exaggerated response to stretch + tone (spasticity).
    - clonus & brisk reflexes.
    - weakness but not wasting (atrophy when longstanding).
    - primitive reflex (Babinski sign).

- LMN
  - systems involved the control of movement
    - extrapyramidal
    - cerebellar
    - vestibular
    - proprioceptive afferents.
    - anterior horn of grey matter.
  - motor unit:- group of muscle fibers innervated by a single anterior horn cell.
  - causes
    - weakness & wasting
    - ↓ tone (flaccidity)
    - fasciculation
    - ↓ or abs reflexes.



Basal ganglia:- connected structure within the cerebral hemisphere & brainstem.

- include
  - striatum (caudate + putamen).
  - globus pallidus
  - thalamus
  - subthalamic nucleus
  - substantia nigra
- function
  - receive info from cortex → control movement.
  - eye movement.
  - behaviour & executive function control.

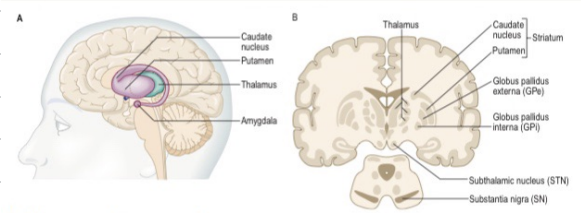
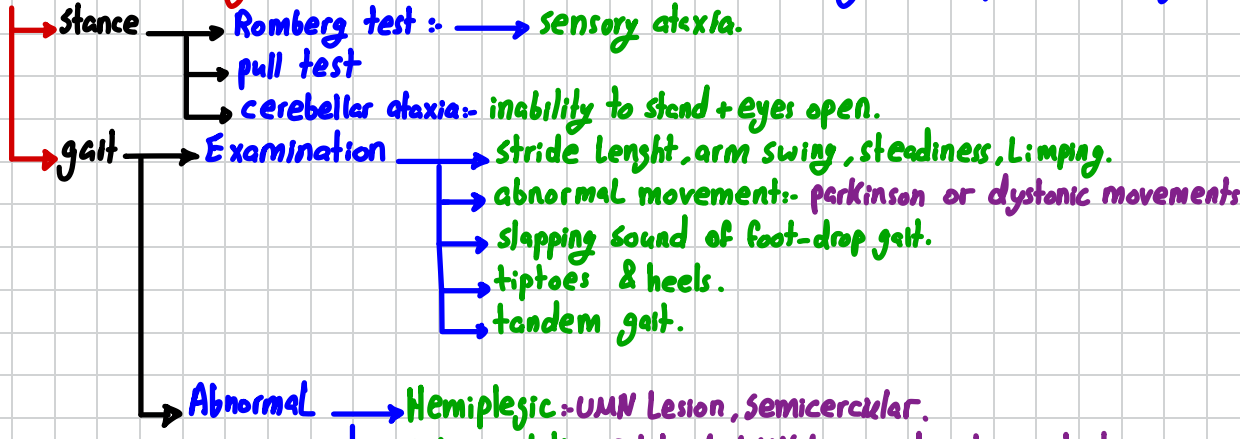


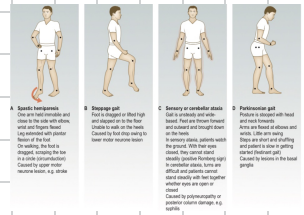
Fig. 7.16 Basal ganglia. [A] Anatomical location, [B] Coronal view.

Disorders → parkinsonism:- ↓ movement.  
 → Ballism or tics:- excessive movement.

**1 stance & gait** → depends on: visual, vestibular, sensory, corticospinal, extrapyramidal, cerebellar, LMN.

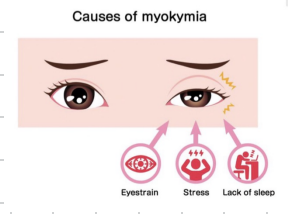
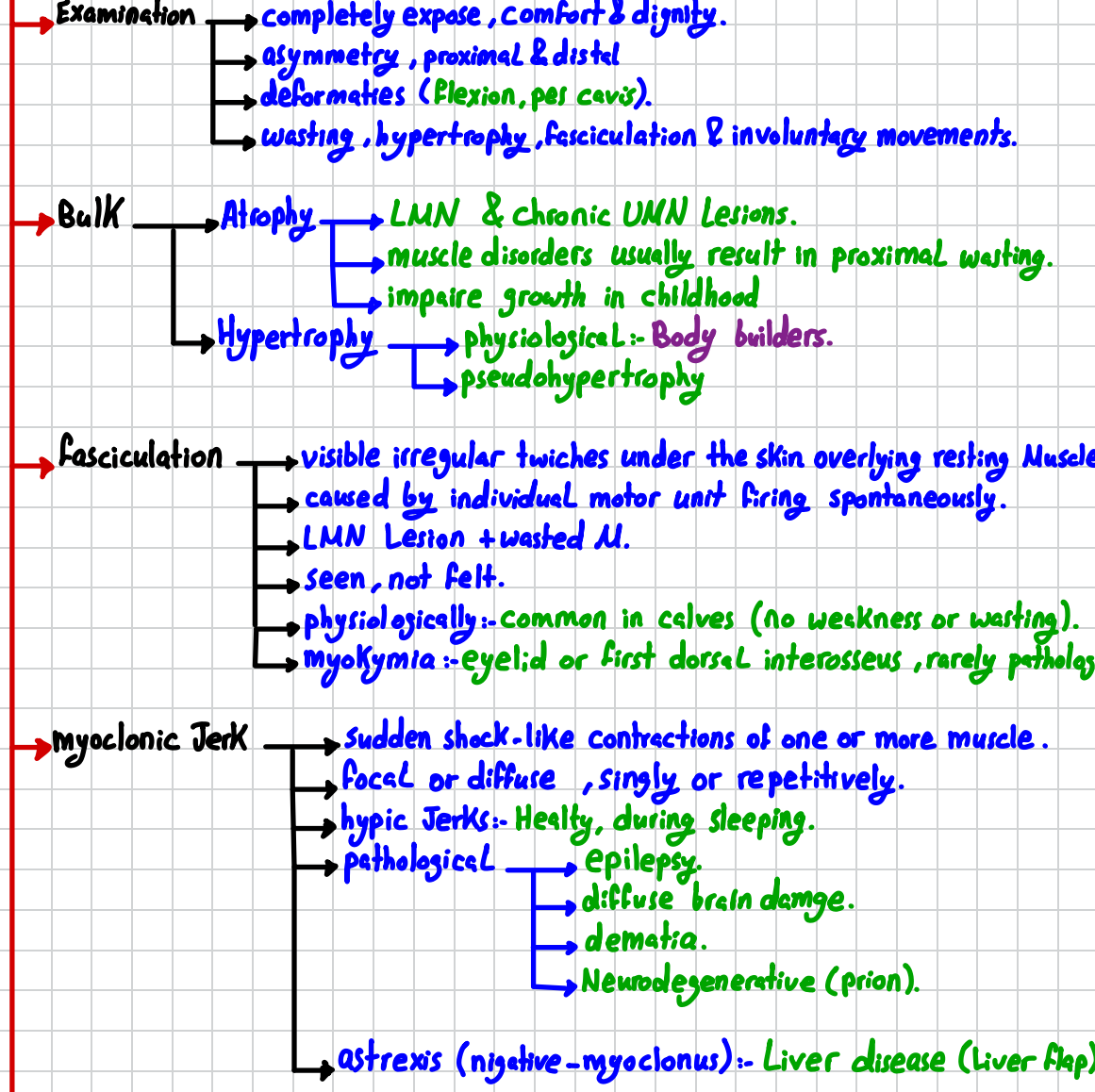


7.7 Common gait abnormalities		
Gait disturbance	Description	Causes
Parkinsonian	Stooped posture Shuffling (reduced stride length) Loss of arm swing Postural instability Freezing	Parkinson's disease and other Parkinsonian syndromes
Gait apraxia	Small, shuffling steps (march à petit pas) Difficulty in starting to walk/freezing Steer "cycling" on bed than walking	Cerebrovascular disease Hydrocephalus
Spastic	Stiff "walking-through-mud" or scissors gait	Spinal cord lesions
Myopathic	Waddling (proximal weakness) Bilateral Trendelenburg signs	Muscular dystrophies and acquired myopathies
Foot drop	Foot slapping	Neuropathies Common peroneal nerve palsy L5 radiculopathy
Central ataxia	Wide-based, "drunken" Tandem gait poor	Cerebellar disease
Sensory ataxia	Wide-based Positive Romberg sign	Neuropathies Spinal cord disorders
Functional	Variable, often bizarre, inconsistent Knees flexed, buckling Dragging immobile leg behind	Functional neurological disorders



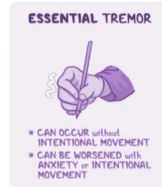
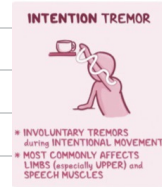
- scissors - Like :- Bilateral UMN Lesion, due to spasticity.
- Ataxic :- cerebellar, board-based, unsteady, impossible heel-to-toe.
- Foot drop
- parkinsonian :- delayed, short step, no arm swing, postural instability.
- waddling :- proximal muscle weakness, bilateral trendelenburg sign.
- Bizarre :- drag leg behind him, Huntington's.
- festinant :- impairment of postural reflex, rapid, short-step, hurrying.

**2 inspection & palpation of muscles**



→ **Tremor** → oscillatory movement about a Joint or a group of Joints from alternating contraction & Relaxation of M.  
 classified according to their :- frequency, amplitude, position & body part.

→ **physiological** → fine (amplitude), fast, postural  
 → causes → anxiety.  
 → Hyperthyroidism  
 → Intake of alcohol/caffeine.  
 → B-agonist (Bronchodilators)



→ **Essential** → most common pathological cause.  
 → AD  
 → upper Limb, Head, voice.  
 → with postural & action components.  
 → may be improved with alcohol.

→ **parkinson's** → slow, coarse, Resting, pill-Rolling.  
 → ↓ with voluntary movement.  
 → upper Limb (asymmetrical), Jaw, chin, Legs (not Head).

→ **cerebellar damage** → intention tremor is absent at rest but maximal during movement.  
 → finger-to-nose test

→ **functional** → inconsistent.  
 → varying frequencies & amplitudes.  
 → + another signs.

Isolated head tremor is usually dystonic and may be associated with abnormal neck postures such as torticollis, antecollis or retrocollis.

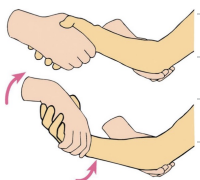
→ **others** → demyelinating neuropathies (Charcot-Marie-Tooth disease).  
 → neuropathic tumors  
 → Drugs :- sodium valproate, glucocorticoids & lithium.

→ **Dystonia** → caused by → sustained muscle contraction → twisting  
 → repetitive movements  
 → tremors.  
 → may be → focal :- torticollis  
 → segmental :- 2 or more adjacent body parts.  
 → generalised.

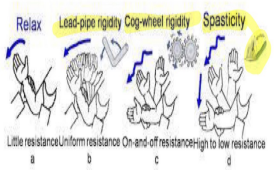
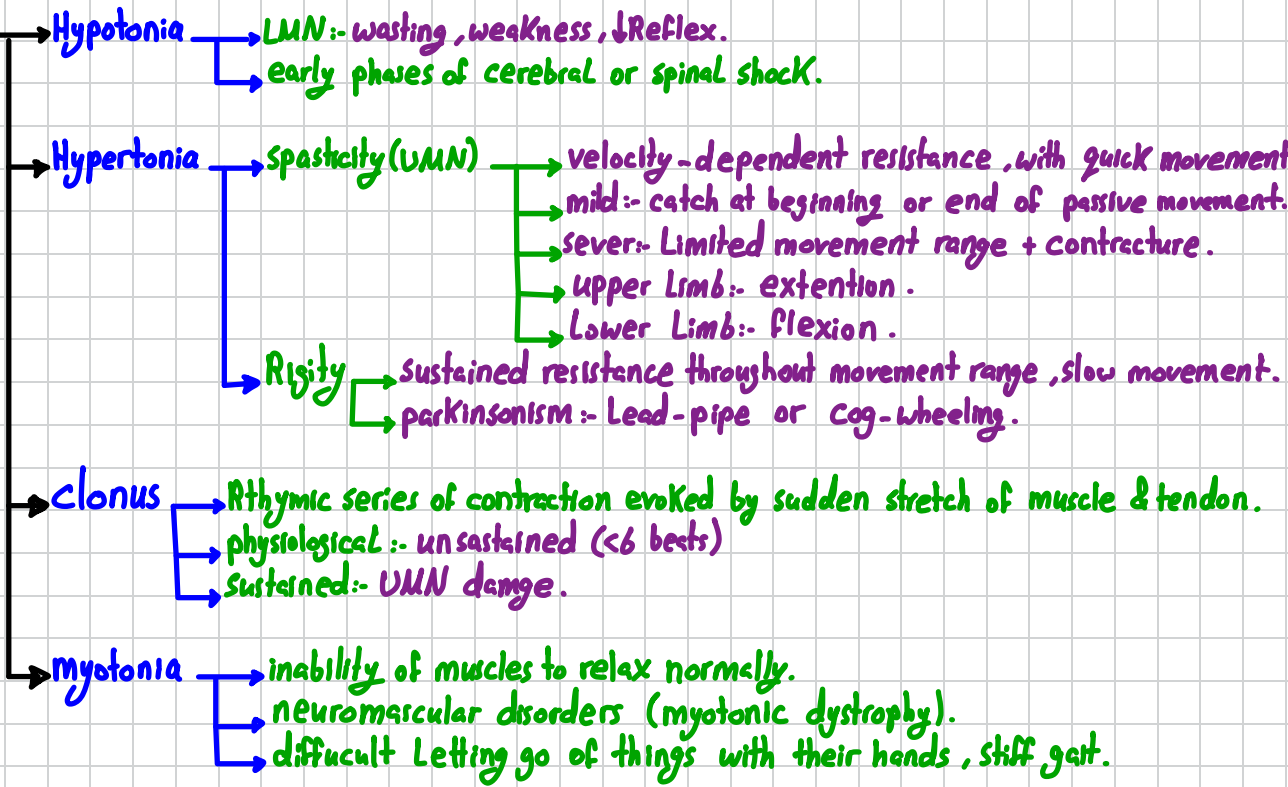
→ **Chorea** :- brief, random, purposeless movements, various body parts (arms).  
 → **Athetosis** :- slower, writhing movement, more similar to dystonia than chorea.  
 → **Ballism** :- violent flinging movement, one side (hemiballismus).  
 → **ticks** :- repetitive, stereotyped movement which can be briefly suppressed by the patient.

**3** → **tone** :- resistance felt by examiner when moving a Joint passively.

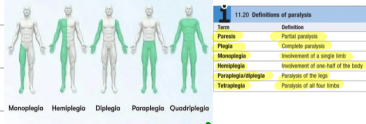
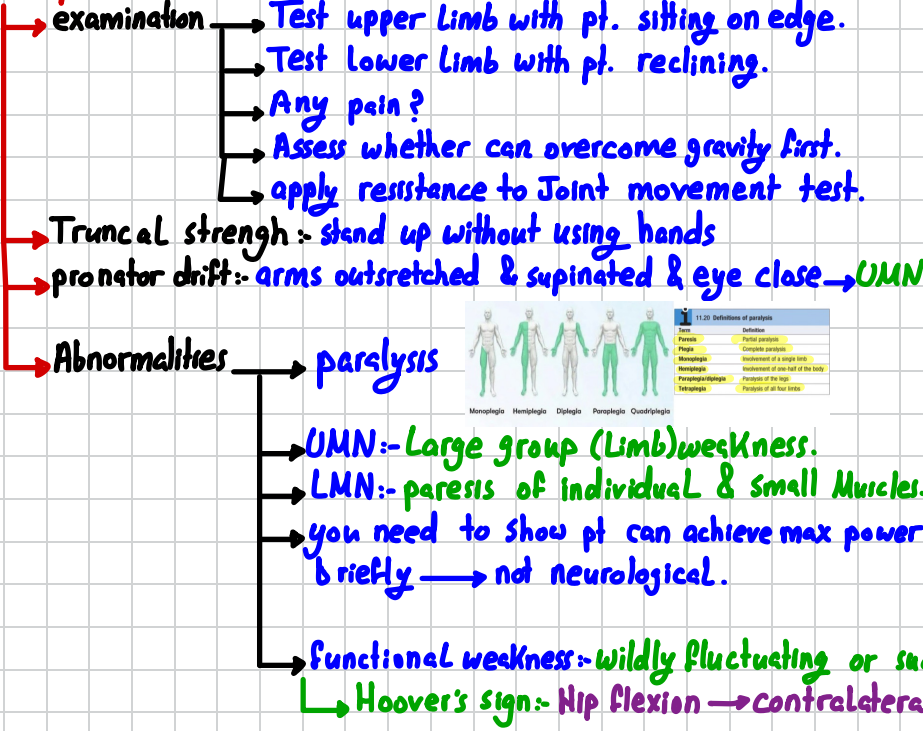
→ **Examination** → supine, Relax, floppy.  
 → pain or Limitation?  
 → move each Joint as full range as possible.  
 → Be unpredictable (direction & speed) :- to prevent active movement.  
 → upper Limb :- wrist & elbow  
 → Activation (synkines) :- ↑ tone (extrapyramidal) \* slides \*  
 → Lower Limb  
 → Ankle clonus



# Abnormalities



# 4 power

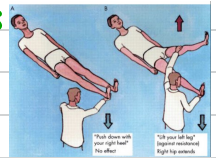


## 7.8 Medical Research Council grading of muscle power

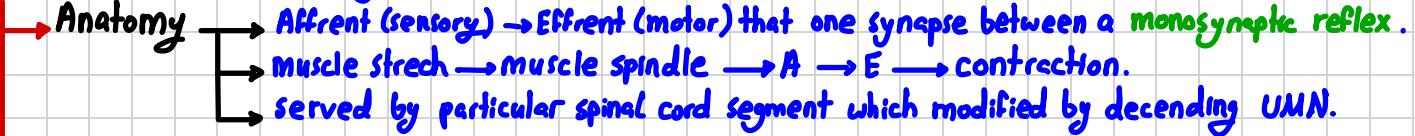
Grade	Description
0	No muscle contraction visible
1	Flicker of contraction but no movement
2	Joint movement when effect of gravity eliminated
3	Movement against gravity but not against resistance
4 <sup>a</sup>	Movement against resistance but weaker than normal
5	Normal power

## 7.9 Nerve and muscle supplies of commonly tested movements

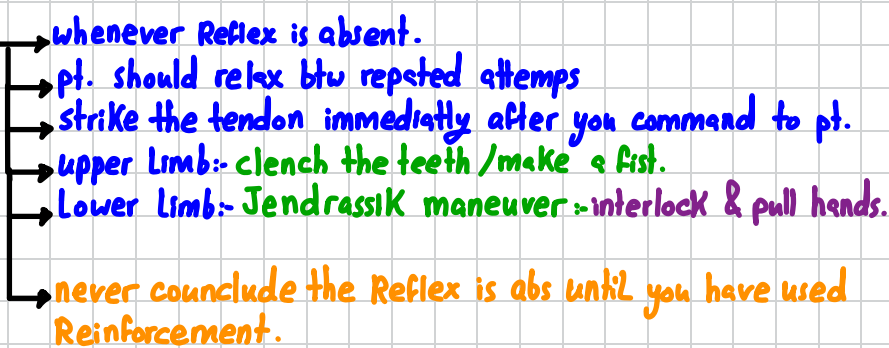
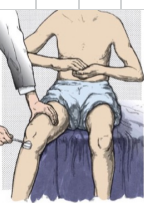
Movement	Muscle	Nerve and root
Shoulder abduction	Deltoid	Axillary C5
Elbow flexion	Biceps Brachioradialis (supinator reflex) <sup>a</sup>	Musculocutaneous C5/6 Radial C5 <sup>b</sup>
Elbow extension	Triceps <sup>a</sup>	Radial C7
Wrist extension	Extensor carpi radialis longus	Posterior interosseous C6
Finger extension	Extensor digitorum communis	Posterior interosseous C7
Finger flexion	Flexor pollicis longus (thumb) Flexor digitorum profundus (index and middle fingers) Flexor digitorum profundus (ring and little fingers)	Anterior interosseous C8 Ulnar C8
Finger abduction	First dorsal interosseous	Ulnar T1
Thumb abduction	Abductor pollicis brevis	Median T1
Hip flexion	Iliopsoas	Iliofemoral nerve L1/2
Hip extension	Gluteus maximus	Sciatic L5/S1
Knee flexion	Hamstrings	Sciatic S1
Knee extension	Quadriceps <sup>a</sup>	Femoral L3/4
Ankle dorsiflexion	Tibialis anterior	Deep peroneal L4/5
Ankle plantar flexion	Gastrocnemius and soleus <sup>a</sup>	Tibial S1/2
Great toe extension (dorsiflexion)	Extensor hallucis longus	Deep peroneal L5
Ankle eversion	Peronei	Superficial peroneal L5/S1
Ankle inversion	Tibialis posterior	Tibial nerve L4/5



# 5 Reflexes



# Reinforcement



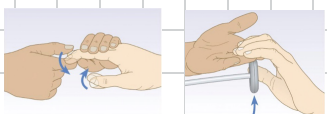
- Position: supine on the examination couch with the limbs exposed.
- Setting: relaxed and comfortable as possible, as anxiety and pain can cause an increased response.
- Techniques: Flex your wrist and allow the weight of the tendon hammer head to determine the strength of the blow.
- Strike the tendon, not the muscle or bone
- Compare each reflex with the other side; check for symmetry of response

11.24 Monosynaptic (deep tendon) reflexes and root innervation	
Reflex (muscle)	Nerve root
Biceps	C5
Supinator (brachioradialis)	C6
Triceps	C7
Knee (quadriceps)	L3, 4
Ankle (gastrocnemius, soleus)	S1

- Recording response
  - increased (+++)
  - Normal (++)
  - Diminished (+)
  - only with reinforcement (+/-)
  - Absent (0)

- Abnormalities
  - ↑: UMN
  - ↓ or abs
    - LMN
    - Healthy elderly the ankle Jerk may be ↓ or abs & Holmes-Adie syndrome.
    - myotonic pupils associated with loss of some reflexes.
    - isolated loss of Reflex: mononeuropathy or radiculopathy.
  - cerebellar
    - pendular Reflex
    - slow muscle contraction/Relax.
    - not sensitive or specific.

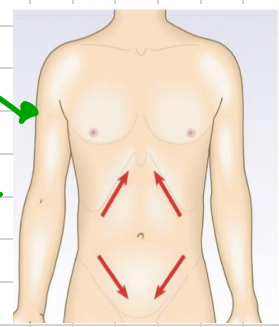
An 'inverted' biceps reflex is caused by combined spinal cord and root pathology localising to a specific spinal level. It is most common at the C5/6 level. When elicited, the biceps reflex is absent or reduced but finger flexion occurs. This is because the lesion at the C5/6 level affects the efferent arc of the biceps jerk (C5 nerve root), causing it to be reduced or lost, and also the spinal cord, increasing reflexes below this level (including the finger jerks, C8). It is most commonly seen in cervical spondylotic myeloradiculopathy.



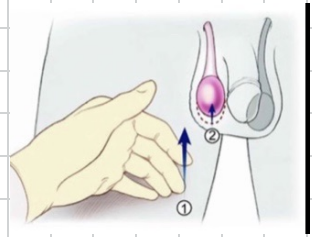
- Hoffmann's Reflex: flick middle finger → Reflex Flexion of pt thumb. → hypertonia
- Finger Jerk: tap on fingers with hammer → Flexion → not useful signs in isolation (Healthy).

superficial Reflexes: polysynaptic & elicited by cutaneous stimulation rather than stretch, ↓ sensitivity & specificity.

- Abdominal (T8-T12)
  - position: supine & Relaxed
  - Technique: stroke lightly
  - Normal
    - contraction of underlying muscles
    - umbilicus moving laterally, up/down.
  - Abnormal
    - UMN or LMN (T8-T12)
    - Abs in
      - obese
      - elderly.
      - after abdominal surgery.



- cremasteric (L1-L2)
  - Technique: Abduct & rotate thigh, stroke medial aspect.
  - Normally: testis will rise briskly.
  - Rarely elicited, abs in: spinal cord or root lesions.



- planter response (S1-S2)
  - Technique: Lateral → toe
  - Normally: Flexion of toe.
  - Babinski
    - extensor hallucis longus tendon (not withdrawal of foot).
    - coincides with contraction of other leg flexor muscles.
    - Reproducible
    - sign of UMN Lesion: + spasticity, clonus, ↑ Reflex.
    - fanning of toes is normal & not pathological.



11.25 Primitive reflexes	
<b>Snout reflex</b>	Lightly tap the lips. An abnormal response is lip pouting
<b>Grasp reflex</b>	Firmly stroke the palm from the radial side. In an abnormal response, your finger is gripped by the patient's hand
<b>Palmomental reflex</b>	Apply firm pressure to the palm next to the thenar eminence with a tongue depressor. An abnormal response is ipsilateral puckering of the chin
<b>Glabellar tap</b>	Stand behind the patient and tap repeatedly between his eyebrows with the tip of your index finger. Normally the blink response stops after three or four taps

- primitive Reflexes
  - in neonates, disappears as NS mature.
  - Brain damage or degeneration → Return.
  - Abnormal
    - ↓ Localizing value, ↓ significance, combination → diffuse or frontal cerebral damage.
    - contralateral frontal lobe pathology: unilateral grasp, palmomental reflexes.
    - parkinson's: glabellar tap (myerson sign).



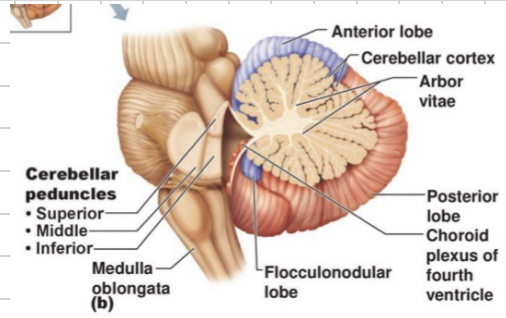
## 6 Coordination

- performing complex movements smoothly & efficiently.
- Depends upon intact sensory/motor/cerebellum functions.

**Anatomy** → **vermis**:- equilibrium.  
**Hemispheres**:- ipsilateral coordination.

**What to examine**

- Stance & gait :- ataxia
- Limb coordination
- Dysarthria
- Nystagmus (H)
- power
- tone
- Reflex
- Rebound phenomenon.



**stance & gait**

- affecting vermis → truncal ataxia.
- ↑sever:- pt. cannot sit unsupported.
- ↓sever:- impaired tandem gait.

### Limb coordination

**upper limb**

- finger-to-nose (slides)** → demonstrate power (weakness → false).  
 → Dysmetria or past-pointing - fall short or overshoot.  
 → intention (Hunting) tremor - tremor.  
 → Dys-synergia :- slow, disjointed & clumsy movement.



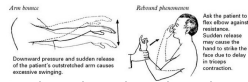
**Rapid alternating movement (slides)** → **Dysdiadochokinesis**

- slowness movement
- disorganization movement
- irregularity movement.



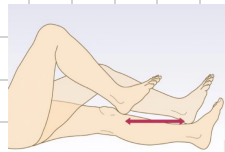
**Rebound phenomenon**

- Normal:- Arm return to original position
- Abnormal:- arm may fly up.



**Lower limb:- Heel-to-shin**

- Same as finger-to-nose.
- Abnormal:- Heel wavers away from line of the skin.



## 7 Apraxia (Dyspraxia)

- Difficulty or inability to perform a task, despite no impairment of the necessary individual functions.
- Sign of:- higher cortical dysfunction (non-dominant frontal & parietal lobe).

**Examination**

- perform an imaginary act.
- copy movements you make.
- copy a geometrical figure.
- ask him to put on a pajama top.

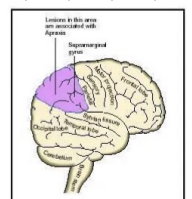
**Abnormal**

- constructional apraxia** → Difficulty drawing a figure.  
 → parietal dysfunction.

**Dressing apraxia** → spatial disorientation & neglect.  
 → non-dominant parietal lesion.

pt. with gait apraxia have difficulty walking but are able to perform cycling movements on the bed surprisingly well.

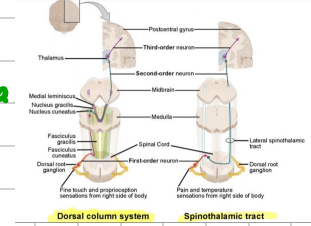
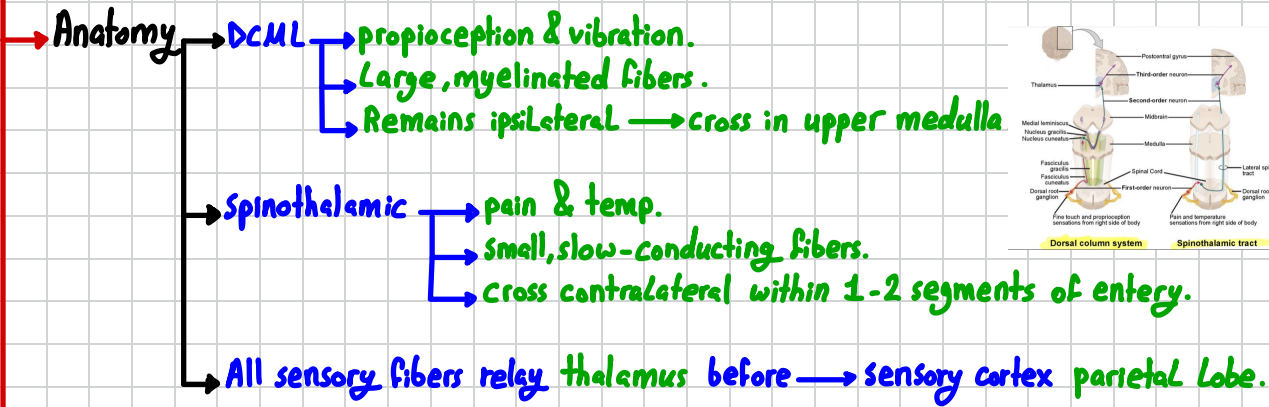
Apraxia	Score
Drawing of the pentagon	1 point
Drawing of the spiral	1 point
Drawing the house	1 point
Drawing a clock	3 points
Putting a sheet of paper in an envelope	3 points
"Goodbye" - correct movement	1 point
Scissors - correct movement	1 point
Brushing teeth - correct movement	1 point
<b>Total</b>	<b>12 points</b>



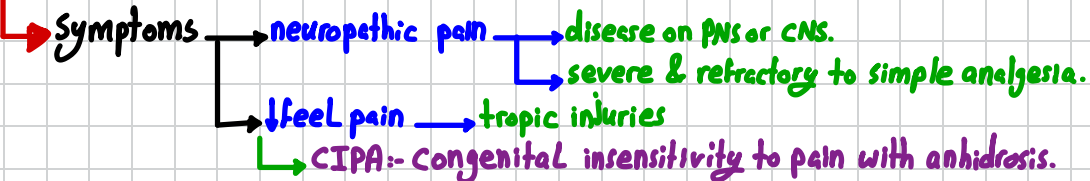


# Sensory System

- Detailed examination of sensation is time-consuming & unnecessary unless the pt. demonstrate sensory symptoms or you suspect a specific pathology (spinal cord compression or mononeuropathy).
- in pt. without sensory symptoms, assessing light touch of all 4 limbs as screening process may suffice

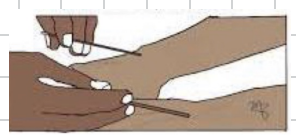


- 1 Light Touch
- 2 Superficial pain
- 3 Temperature
- 4 Vibration
- 5 Proprioception
- 6 Stereognosis and graphaesthesia



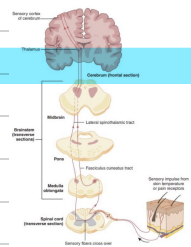
Paraesthesia	Tingling, or pins and needles Spontaneous or provoked Not unduly unpleasant or painful
Dysaesthesia	Unpleasant paraesthesia
Hypoaesthesia	Reduced sensation to a normal stimulus
Analgesia	Numbness or loss of sensation
Hyperaesthesia	Increased sensitivity to a stimulus
Allodynia	Painful sensation resulting from a non-painful stimulus
Hyperalgesia	Increased sensitivity to a painful stimulus

- 1 Light touch**
  - close eye → cotton
  - Time the stimuli irregularly & make dapping rather than stroking or tickling stimulus.
  - Compare each side for symmetry.
- 2 Superficial pain**
  - neurological pin
  - map out boundaries of ↓ or abs or ↑ sensation.
  - move from ↓ to ↑ sensibility.
- 3 Temp**
  - cold metallic object (tuning fork)
  - ↑ sensitive assessment:- tubes of cold/hot water as controlled temp.
- 4 Vibration**
  - note any oedema.
  - what to feel
    - 128Hz over sternum
    - feel buzzing?
  - Lower Limb → toe → interphalangeal Joint → medial malleolus → tibial tuberosity → anterior iliac spine.
  - upper Limb → distal interphalangeal Joint of forefinger → proximally.
  - if in doubt as to the accuracy of the response, ask the pt. to close his eyes & report when you stop the fork vibrating with your fingers.
- 5 proprioception**
  - pt eyes open:- demonstrate to learn him/her the procedure.
  - close eyes:- identify the direction of small movements in random order
  - Test both great toes → proximal Joints (if impaired).
- 6 cortical sense**
  - stereognosis:- object on his hand → identify.
  - Graphaesthesia:- trace Letters or digits → identify.
- sensory inattention**
  - if sensory pathways are otherwise intact.
  - close eyes → touch his arm/leg in turn → which?
  - touch both sides simultaneously → Lt/Rt/Both?



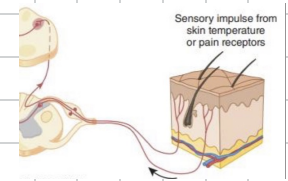
# The sensory modalities (Identifying Lesion Lvl)

- sensory cortex:- only if the main pathway sensation are intact.
- peripheral nerve & dorsal root.
- spinal cord
- intracranial

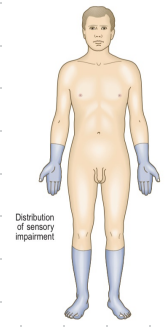


## 1 peripheral nerve & dorsal Root.

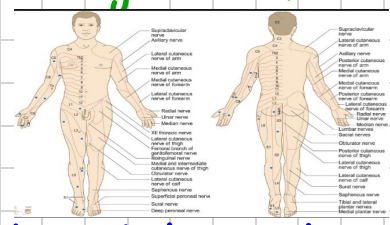
- many diseases affect peripheral nerves → peripheral neuropathies  
→ polyneuropathies.
- Length-dependent:- peripheral neuropathies tend to affect Lower Limb first (toe).
- upper Limb first:- demyelinating rather than axonal neuropathy or spinal cord disease.



- Gloves & stocking → in diabetic neuropathy.  
→ + autonomic symptoms (sweating & cvs).
- Mononeuritis multiplex:- different nerves affected in a stepwise fashion.



- Large-fibre neuropathy → Such as:- Guillian-Barre' syndrome.  
→ vibration & Joint position affected.  
→ Romberg's syn:- staggering when they close their eyes or dark.
- Small fibre → ↓ pinprick pain & temp.  
→ autonomic involment.  
→ due to:- DM & HIV infection.



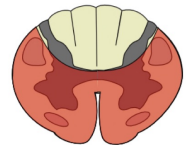
- pseudoathetosis → Loss of proprioception  
→ close eyes → outstretched hand → slow, involuntary wandering movement  
→ mimic athetosis.

## 2 Spinal cord

- Trauma or compression:- Loss or impairment of sensation in a dermatomal distribution below the Lvl of Lesion.
- Zone of Hyperaesthesia:- immediately above the Lvl of sensory Loss.

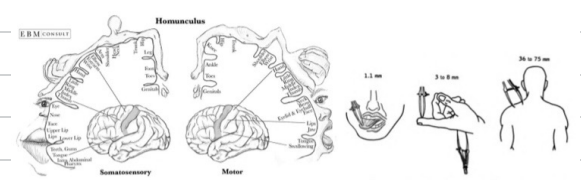
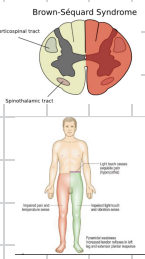
- Lesions → Anterior spinal Artery syndrome → Loss of spinothalamic sensation.  
→ Loss of motor function  
→ sparing of dorsal column.
- Syringomelia:- fluid-cavity within spinal cord (same pattern of Ant.spinal.A)
- Brown-segward syndrome:- Half spinal cord → ipsilateral vibration & Joint position Loss.  
→ contralateral Loss of pain & temp.

Anterior Cord Syndrome



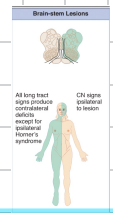
## 3 intracranial

- thalamic → patchy sensory imparment contralateral  
→ + unpleasant, poorly localized pain with burning quality.
- parital lobe → sensory inattention  
→ Joint position sense  
→ two point discrimination  
→ stereognosis:- tactile recognition  
→ Localization of pain touch



# 4 Brain stem

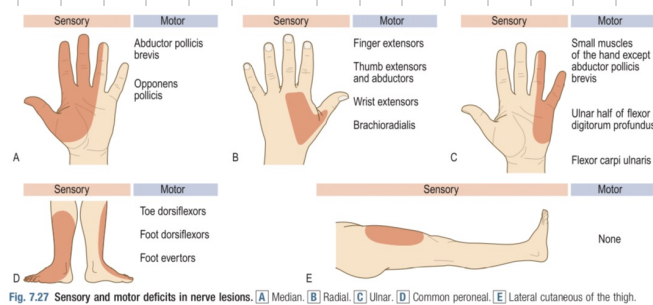
Lower Brainstem Lesion → ipsilateral numbness on one side of face (V nerve nucleus).  
 → contralateral body numbness (spinothalamic).



## Peripheral nerves examination

sensory examination → thumb  
 → index & middle fingers  
 → Lateral Half of rings

mononeuropathy:- individual peripheral N damage.  
 peripheral neuropathy or mononeuritis multiplex:- multiply.



## 1 Median nerve

compressed in:- B/w the flexor retinaculum & carpal bones at wrist.

Examination → wasting of thenar eminence.  
 → thumb abduction (abductor pollicis brevis):- move vertically against your resistance.  
 → opposition (opponens pollicis):- Ring finger against resistance.  
 → Tinel's sign:- tapping distal wrist crease (↓sensitivity & specificity).  
 → phalen's test:- forced flexion of wrist upto 60sec (↓sensitivity & specificity).



carpal tunnel syndrome → most common entrapment neuropathy.  
 → produces sensory symptoms & pain in hands.  
 → radiating up to arm, typically at night.  
 → common during pregnancy.

**11.30 Common features of carpal tunnel syndrome**

- More common in women
- Unpleasant tingling in the hand
- May not observe anatomical boundaries, radiating up the arm to the shoulder
- Weakness uncommon, but affects thumb abduction if occurs
- Symptoms commonly occur at night, waking patient from sleep
- The patient may hang the hand and arm out of the bed for relief
- Thenar muscle wasting (in longstanding cases)
- Associated with pregnancy, diabetes and hypothyroidism

## 2 ulnar nerve

affected → external compression or injury of elbow.  
 → in condylar groove behind medial epicondyle of humerus.

Examination → wasting of interossei (dorsal guttering).  
 → finger abduction:- spread fingers against resistance.  
 → finger adduction:- card b/w fingers & pull it.  
 → Elbow:- commonest place of entrapment.  
 → note any scars or trauma.  
 → feel for the nerve in the ulnar groove.



## 3 Radial nerve.

compressed - through axilla, in spiral groove of the humerus → wrist drop (saturday night palsy).

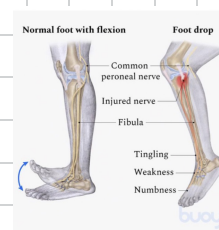
Examination → weakness of arm & forearm (triceps, wrist, fingers).  
 → sensory loss over the dorsum of hand.  
 → Loss of triceps tendon Jerk



## 4 Common peroneal Nerve.

damaged in → fibular Head fractures  
 → compressed particularly in immobile pt.  
 → repetitive kneeling or squatting.

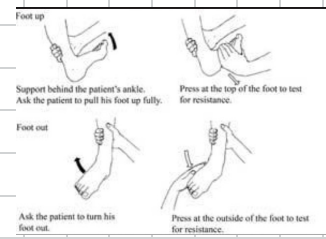
typically presents:- foot drop.



**Symptoms of Radial Nerve Injury**

At the axilla:	To the spiral groove:	To the posterior interosseous nerve:
<ul style="list-style-type: none"> <li>• weakness</li> <li>• tingling and numbness from the neck of the arm to the hand</li> </ul>	<ul style="list-style-type: none"> <li>• weakness of the brachioradialis muscle of the forearm</li> <li>• inflex with a person's ability to bend the arm back and straighten the forearm</li> </ul>	<ul style="list-style-type: none"> <li>• muscle weakness</li> <li>• inability to extend one's fingers</li> </ul>

- examination
- weakness of ankle dorsiflexion & eversion
  - extension of Big toe (extensor hallucis Longus).
  - inversion & ankle Reflex will be preserved.
  - Test for sensory Loss over the dorsum of the foot.



**B** Lateral cutaneous nerve of the thigh

- purely sensory
- compressed as:- passes under the inguinal Ligament.
- producing:- paraesthesiae in lateral thigh → meralgia paraesthetica :- burning numbness

