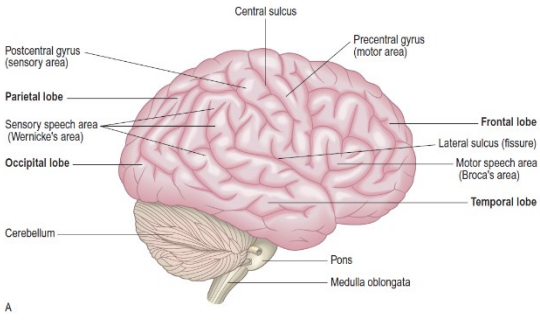
W05 – CNS & PNS

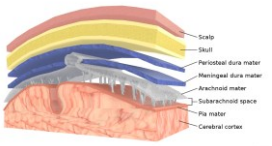
Collected from Al Thara'a, E-Learning Slides, Lectures Slides, Macleod 14th Ed

Anatomy

Intro

- The nervous system consists of CNS (Brain, Spinal Cord) and PNS (Sensory Nerves, Motor Nerves, Autonomic Nerves).
- The neuron is the functional unit of the nervous system. Each neuron has dendrites, a cell body and axon terminating at a synapse (terminals).
- Neurons are supported by astrocytes, microglial cells, oligodendrocytes & ependymal cells in the CNS, Schwann & satellite cells in the PNS.
    1. Astrocytes provide the structural framework for the neuron, control their biochemical environment, and form the blood– brain barrier.
    2. Microglial cells are blood-derived mononuclear macrophages with an immune function.
    3. In the CNS, myelin is produced by oligodendrocytes. In the PNS, myelin is produced by Schwann cells.





Meninges

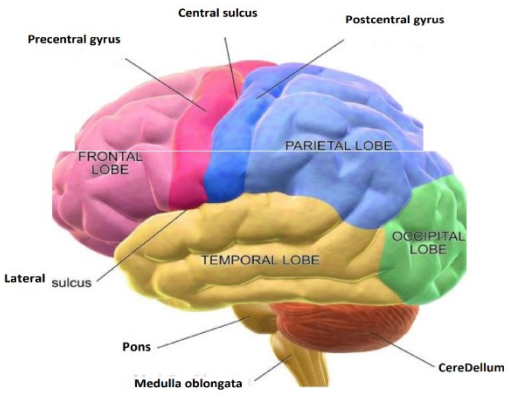
- Brain and spinal cord are covered with three membranous layers called the meninges: dura mater next to the bone, arachnoid and pia mater next to the nervous tissue.

CSF

- The subarachnoid space between the arachnoid and pia is filled with cerebrospinal fluid (CSF) produced by the choroid plexuses. The total volume of CSF is between 140 and 270 mL and there is a turnover of the entire volume 3–4 times a day. Rate of production is 700 mL per day.

Brain

- Two cerebral hemispheres, each with four lobes (frontal, parietal, temporal and occipital), the brainstem and the cerebellum.
    1. The brainstem comprises the midbrain, pons, and medulla.
    2. The cerebellum has two hemispheres and a central vermis attached to the brainstem by three pairs of cerebellar peduncles



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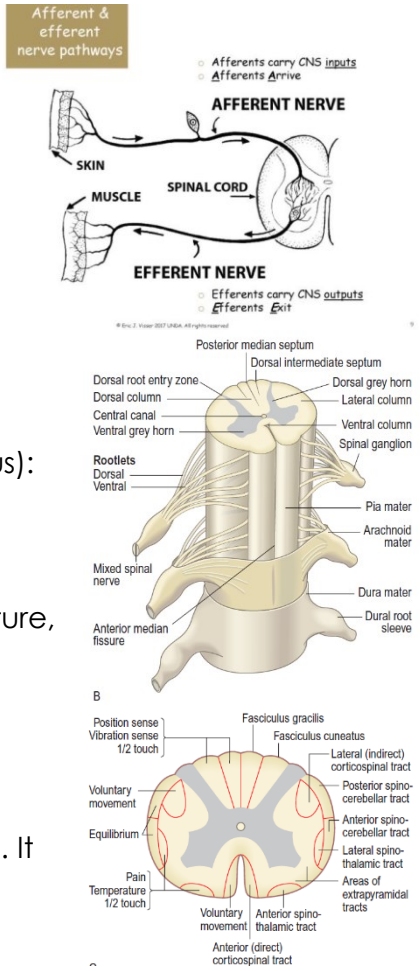
Spinal Cord



- The spinal cord is the main pathway for information connecting the brain and PNS. It contains the ventral grey horn and dorsal grey horn.
    1. Ventral (anterior) roots consist of efferent fibers that arise from motor neurons found in ventral grey horns.
    2. Dorsal (posterior) roots are afferent fibers, receiving sensory information from organs to be transmitted to brain through sensory neurons found in dorsal grey horn.
- The spinal cord contains multiple tracts:
    1. Dorsal column (fasciculus gracilis and fasciculus cuneatus): responsible for proprioception, vibration and half touch
    2. Anterior and lateral corticospinal tracts: voluntary movements
    3. Anterior and lateral spinothalamic tracts: pain, temperature, and half touch
    4. Spinocerebellar tract: equilibrium

PNS

- Peripheral nerves may have myelinated or unmyelinated axons. It contains somatic (sensory & motor) and autonomic nerves.

History

- The history is the key for diagnosis as physical exam may be normal or unhelpful.
- In cases of amnesia or loss of consciousness we need additional witness history.
- We should clarify exactly what the patient means by any neurological symptom.
- Ask the patient what they fear might be wrong.
- For any neurological symptom ask about onset, duration, pattern, exacerbating, relieving factors and associated symptoms.

Symptoms

1. Headache (causes can be primary or secondary)

- Isolated headache with a truly abrupt onset may represent a potentially serious cause such as subarachnoid haemorrhage or cerebral vein thrombosis, whereas recurrent headache is much more likely to be benign primary headache.
- Disturbances of consciousness: syncope, seizures, dizziness & vertigo.
- During migraines attack, the patient prefers to be in a dark room.
- During a cluster headache attack the patient keeps pacing around the room in an agitated state, or even head banging. It might wake him up during sleep.
- Cluster headaches from are part of trigeminal autonomic cephalalgias.
- Primary cough could also be due to cough or exertion, or it could be a primary thunderclap headache.

- Meningitis could be associated with neck stiffness (remember the meningeal irritation signs).

7.1 Clinical characteristics of headache syndromes

	Onset	Duration/periodicity	Pain location	Associated features
Primary syndromes				
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
Secondary syndromes				
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

Acute Headache		***Subacute Headache***	***Chronic Headache***
Single Episode	Recurrent Episodes	Infections (tuberculous meningitis, cerebral abscess)	Chronic daily headache syndrome
Angle-closure glaucoma	Angle-closure glaucoma	Raised ICP (tumor, hydrocephalus)	Depression
Acute meningitis	Sinusitis	Benign IC hypertension	Cervical spondylosis
Vasodilator drugs	Cluster headache	Temporal arteritis	Drugs (nitrates, overuse of analgesics)
Subarachnoid hemorrhage	Migraine		
	Neuralgias (trigeminal & post-herpetic)		

2. Disturbance of consciousness

- Could be caused by:
  1. Postural hypotension.
  2. Neurocardiogenic syncope (vasovagal).
  3. Hypersensitive carotid sinus syndrome (pressure over carotid sinus may lead to reflex bradycardia and syncope).
  4. Cardiac syncope due to arrhythmias or mechanical obstruction of cardiac output.

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- Syncope is an alteration (or loss) of consciousness resulting from inadequate cerebral blood flow. It is the most common cause of transient LOC. Could be due to a reduced cardiac output (Cardiac syncope) or a peripheral vasodilatation (Vasovagal syncope).
    1. Cardiac syncope is syncope with no previous alarm or trigger. It is provoked by exertion (severe aortic stenosis, HOCM) or sudden (arrhythmias). Recovery is usually rapid.
    2. Vasovagal syncope occurs due to stimulation of parasympathetic system due to pain, emotion, or illness or in people forced to stand in warm environment. Leads to vasodilation and bradycardia. Often preceded by light-headedness, vision dimming (darkening), tinnitus, and nausea. It lasts for 1-2 minutes and causes pale or grey skin. May be associated with myoclonic jerks & if kept flat, recovery is rapid.
- How to ask about syncope?
    1. Ask about a witness.
    2. Any preceding symptoms (palpitation, chest pain, light-headedness, nausea, tinnitus, sweating and visual disturbance).
    3. Duration of loss of consciousness.
    4. Appearance of the patient while unconscious.
    5. Any injuries sustained.
    6. Time to recovery to full consciousness and normal cognition.
- Postural hypotension could be due to:
    1. Drugs (levodopa or anti-hypertensive drugs).
    2. Autonomic diseases such as DM.
    3. People more than 65 years.
    4. Hypovolemia.
- Epileptic seizures are paroxysmal electrical discharges from either the whole brain (generalized; tonic-clonic seizures are the most common) or part of the brain (Focal: partial). The history from the patient and witnesses can help distinguish epilepsy from syncope. Usually triggered by sleep deprivation, alcohol, and drugs.
    1. Tonic clonic seizure:
        a. Prodromal phase: Change of mood or 'odd' feeling (aura)
        b. Tonic phase: typically follows a stereotyped pattern with early LOC associated with body stiffening, cyanosis, spasm of all muscles & falls.
        c. Clonic phase: rhythmical jerking of limbs & trunk subsiding over 0.5 – 2 minutes w/ tongue biting & incontinence of urine.
        d. Postictal phase: a period of unresponsiveness often with heavy breathing, the patient appears to be in deep sleep and finally confusion as the patient awakes. It could be associated with flaccidity, headache & amnesia.

7.2 Features that help discriminate vasovagal syncope from epileptic seizure

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

2. Focal seizures could be simple (consciousness is preserved) or complex (impaired consciousness). Complex partial seizures features are:
    a. Dream-like states.
    b. Disturbances of memory (déjà-vu, jamais vu).
    c. Hallucinations of smell, taste or auditory.
    d. Emotional disturbance.
    e. Abnormal behaviour.

   Focal seizures are characterized by whichever part of the brain is involved.
    a. Frontal lobe seizures: focal motor seizure.
    b. Temporal lobe seizures are characterized by autonomic and/or psychic symptoms, often associated with automatisms such as lip smacking or swallowing.

- Non-epileptic or psychogenic attacks or pseudo-seizures or functional dissociative attacks are difficult to distinguish from epileptic seizures, clues to differentiate psychogenic seizures: (The widespread availability of videophones allows witnesses to capture such events)

Item	Epileptic seizures	PNES
Eyes	Opened	Closed
Head	Fixed/unilateral	Side-to-side movements
Limbs	In phase/same direction	Out of phase
Body (axis)	Straight	Opisthotonus
Body (movement)	No rotation	Intense rotation in bed
Evolution of seizure	Continuous	Fluctuating

PNES, psychogenic non-epileptic seizures; ES, epileptic seizures.

1. occurring multiple times in a day
2. may last considerably longer
3. Symptoms waxing and waning
4. Asynchronous movements
5. Pelvic thrusts, side-to-side rather than flexion/extension movements
6. Absence of postictal confusion

3. Dizziness

- Recurrent dizzy spells affect approximately 30% of those over 65 years.
- Causes:
    1. Postural hypotension.
    2. Cerebrovascular disease or vertebrobasilar insufficiency.
    3. Cardiac arrhythmia.
    4. Hyperventilation induced by anxiety and panic.

4. Vertigo

- The illusion of movement. Specifically indicates a problem in the vestibular apparatus (peripheral) (most common) or the brain (central). (Check the table first then read points a-d).
    a. BPPV: recurrent episodes of vertigo lasting a few seconds, attacks increase when sleeping on the affected side or with movement.
    b. Meniere disease: vertigo lasting minutes or hours, associated with hearing loss, tinnitus, nausea, and vomiting.
    c. Migraine could occur with or without headache.
    d. TIAs do not cause isolated vertigo.

Causes of vertigo
Central
•Migraine •Brainstem ischemia or infarction •Multiple sclerosis
Peripheral
•Ménière's disease (hearing loss , tinnitus , nausea and vomiting •Benign paroxysmal positional vertigo •Vestibular neuritis •Trauma •Drugs, e.g. gentamicin, anticonvulsants

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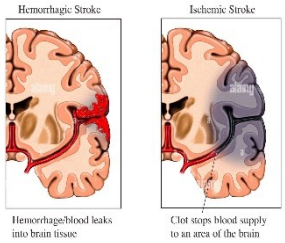
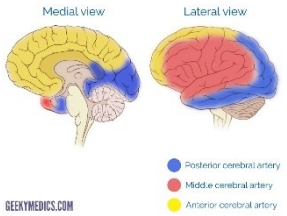
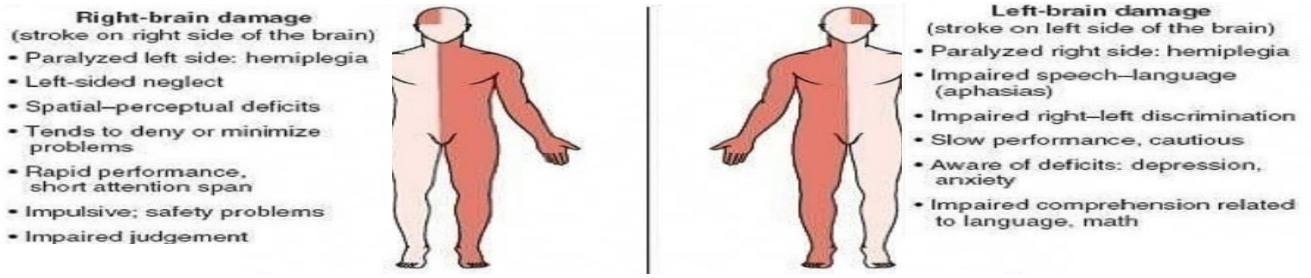
5. Stroke





- Stroke is a focal neurological deficit of rapid onset that is due to a vascular cause, maybe ischemic or haemorrhagic.
- A transient ischaemic attack (TIA) is the same, but symptoms resolve within 24 hours. TIAs are an important risk factor for impending stroke and demand urgent assessment and treatment.
- Symptoms are dictated by the vascular territory involved.
- Much of the cerebral hemispheres are supplied by the anterior circulation (ACA & MCA which are derived from the internal carotid artery) , while the occipital lobes and brainstem are supplied by the posterior circulation (PCA which is derived from the vertebrobasilar circulation).
- 80% of strokes are ischemic. Haemorrhagic stroke is much more frequent in Asian populations.
- Factors in the history or examination that increase the likelihood of haemorrhage include:
    1. The use of anticoagulation.
    2. Headache.
    3. Vomiting.
    4. Seizures.
    5. Early reduced consciousness.



- We must do brain CT without contrast to differentiate between them.
- Isolated vertigo, amnesia or TLOC are rarely, if ever, due to stroke.
- Spinal strokes are very rare; patients typically present with abrupt onset, depending on the level of spinal cord affected. The anterior spinal artery syndrome is most common and causes loss of motor function and pain/temperature sensation, with relative sparing of joint position and vibration sensation below the level of the lesion (sparing dorsal column).
- ACA occlusion occurs with weakness of foot and leg, sensory loss of foot and leg, ataxia & Incontinence
- MCA occlusion occurs with contralateral lower face weakness, hemiplegia & hemianesthesia. Also, ataxia, speech impairments (usually the left brain), perceptual deficits (usually the right brain) & visual deficits.
- PCA occlusion is midbrain syndrome (Weber's Syndrome) which is an occlusion of the paramedian branches of the posterior cerebral artery that occurs

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

with ipsilateral 3rd nerve palsy & contralateral hemiplegia. PCA occlusion is also associated with visual field deficits (macular sparing), visual hallucinations & memory problems.

6. Functional/psychogenic/hysterical/somatization/conversion disorder

- Not due to a true neurological disease.
- Presentations include blindness, tremor, weakness and collapsing attacks, and patients will often describe numerous other symptoms, with fatigue, lethargy, pain, anxiety, and other mood disorders commonly associated.
- Clues include:
    1. Symptoms aren't compatible with a disease (such as retained awareness of convulsing during non-epileptic attacks or being able to walk normally backwards but not forwards).
    2. considerable variability in symptoms (such as intermittent recovery of a hemiparesis).
    3. Multiple symptoms with numerous visits to other specialties and multiple unremarkable investigations, leading to numerous different diagnoses
- Most functional neurological disorders follow recognizable patterns, so be cautious when the pattern is atypical.

Past Medical History

- History of previous visual loss (optic neuritis) in someone presenting with numbness suggests multiple sclerosis.
- Birth history and development may be significant, as in epilepsy.
- If considering a vascular cause of neurological symptoms, ask about important risk factors, such as other vascular disease, hypertension, family history and smoking.

Drug History

- Always enquire about drugs, including prescribed, over-the counter, complementary and recreational/illegal ones.
- phenytoin toxicity causes ataxia.
- Excessive intake of simple analgesia causing medication overuse headache; use of cocaine provokes convulsions.

Family History

- Parental consanguinity is common, increasing the risk of autosomal recessive conditions.
- Single-gene defects: such as myotonic dystrophy or Huntington's disease.
- Polygenic influences, as in multiple sclerosis or migraine.
- Mitochondria uniquely have their own DNA, and abnormalities in this DNA can cause a range of disorders that manifest in many different systems (such as diabetes, short stature and deafness), and may cause common neurological syndromes such
- as migraine or epilepsy.
- Some diseases, such as Parkinson's or motor neuron disease, may be either due to single-gene disorders or sporadic.

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Social History

- How are patients coping with their symptoms? Are they able to work and drive?
- What are their support circumstances, and are these adequate?
- Ask about alcohol as it affects CNS (ataxia, seizures, dementia) and PNS (neuropathy).
- Ask about diet
    1. Vitamin deficiency may occur in alcoholism or dietary exclusion.
    2. Vegetarians may be susceptible to vitamin B12 deficiency (subacute combined degeneration of the spinal cord)
- Ask about recreational drugs
    1. nitrous oxide inhalation causes subacute combined degeneration of the cord due to dysfunction of the vitamin B12 pathway
    2. smoking contributes to vascular and malignant disease.
- A travel history may give clues to the underlying diagnosis such as:
    1. Lyme disease (facial palsy)
    2. Malaria (coma)
- Always consider sexually transmitted or blood-borne infection, such as human immunodeficiency virus (HIV) or syphilis, as both can cause a wide range of neurological symptoms and are treatable.

Occupational History

- Lead exposure: motor neuropathy.
- Manganese causes Parkinsonism.
- Some neurological diagnoses may adversely affect occupation, such as epilepsy in anyone who needs to drive or operate dangerous machinery.
- For patients with cognitive disorders, particularly dementias, it may be necessary to patients to stop working.

Physical Examination

General Approach

- General look / Vital signs / Level of consciousness / Speech / Stance & Gait / High cognitive function / Meningeal irritation / Cranial Nerves 1-12 / Motor & Sensory system / Coordination & cerebellum.

1. General Look

- Begins with your first contact with the patient and continues during history taking
- Note facial expression, general demeanor, dress, posture, gait, speech & Involuntary movements
- Mental state examination and general examination are integral parts of the neurological examination.

2. Assessment of conscious level

- Consciousness has two main components:

19.14 Glasgow Coma Scale

Eye opening	
Spontaneous	4
To speech	3
To pain	2
No response	1
Verbal response	
Orientated	5
Confused: talks in sentences but disorientated	4
Verbalises: words, not sentences	3
Vocalises: sounds (groans or grunts), not words	2
No vocalisation	1
Motor response	
Obeys commands	6
Localises to pain, e.g. brings hand up beyond chin to supraorbital pain	5
Flexion withdrawal to pain: no localisation to supraorbital pain but flexes elbow to nail bed pressure	4
Abnormal flexion to pain	3
Extension to pain: extends elbow to nail bed pressure	2
No response	1

Record the GCS as a total and its three separate components: e.g. GCS 9/15: E3, V2, M4

1. The state of consciousness depends largely on integrity of the ascending reticular activating system, which extends from the brainstem to the thalamus. It describes how awake a person is.
2. The content of consciousness refers to how aware the person is and depends on the cerebral cortex, the thalamus, and their connections.

3. Meningeal Irritation

- Meningism (inflammation or irritation of the meninges). Meningism suggests infection (meningitis) or (subarachnoid haemorrhage) but can occur with non-neurological infections, such as urinary tract infection or pneumonia.
- Conversely, absence of meningism does not exclude pathology within the subarachnoid space.
- The absence of all three signs of fever, neck stiffness and altered mental state virtually eliminates the diagnosis of meningitis in immunocompetent individuals.
- Meningeal irritation signs:
  1. Neck stiffness (increased resistance to passive flexion of the neck).
  2. Kernig's sign (increased resistance to passive extension of the leg)
  3. Brudzinski's sign (Flexion of the knees in response to neck flexion).

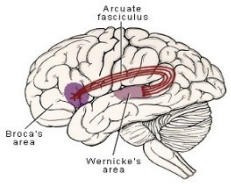
4. Speech Examination

- Listen to the patient's spontaneous speech, noting volume, rhythm, and clarity.
- Ask the patient to repeat phrases such as 'yellow lorry' to test lingual (tongue) sounds and 'baby hippopotamus' for labial (lip) sounds, then a tongue twister, e.g., 'the Leith police dismisseth us'.
- Ask the patient to count steadily to 30 to assess fatigue.
- Ask the patient to cough and to say 'Ah'; observe the soft palate rising bilaterally.
- Speech abnormalities:
  1. Dysarthria (slurred speech caused by articulation problems due to a motor deficit)
     a. Pseudobulbar palsy (contracted, spastic tongue and difficulty pronouncing consonants)
     b. Bulbar palsy (Weakness of the tongue results in difficulty with lingual sounds, while palatal weakness gives a nasal quality to the speech).
     c. Cerebellar dysarthria (slow and slurred, like alcohol intoxication.
     d. Myasthenia gravis (fatiguing speech).
     e. Parkinsonism (dysarthria and dysphonia, with a low-volume, monotonous voice)

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

  2. Dysphonia (loss of volume caused by laryngeal disorders, Results from either vocal cord pathology, or damage to the vagal (X) nerve supply to the vocal cords)
  3. Dysphasia (disturbance of language resulting in abnormalities of speech production and/or understanding). May also involve other language symptoms, e.g., writing and reading problems.
     - The language areas are in the dominant cerebral hemisphere, which is the left in almost all right-handed people and most left-handed people. Broca's area (inferior frontal region) is concerned with word production and language expression. Wernicke's area

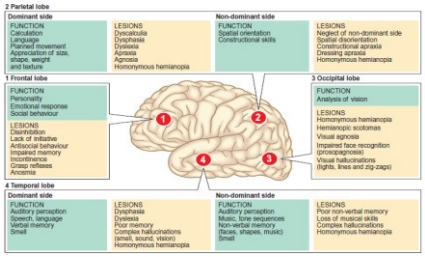


(superior posterior temporal lobe) is the principal area for comprehension of spoken language. Adjacent regions of the parietal lobe are involved in understanding written language and numbers. The arcuate fasciculus connects Broca's and Wernicke's areas.

a. Expressive (motor) dysphasia results from damage to Broca's area. It is characterised by reduced verbal output with non-fluent speech and errors of grammar and syntax. Comprehension is intact.
b. Receptive (sensory) dysphasia occurs due to dysfunction in Wernicke's area. There is poor comprehension, and although speech is fluent, it may be meaningless.
c. Global dysphasia is a combination of expressive and receptive difficulties caused by involvement of both areas.
d. Conduction dysphasia
e. Dyslexia
f. Dyscalculia
g. Dysgraphia

- listen to the fluency and appropriateness of the content during speech, ask the patient to name a common object, give a simple three-stage command, ask the patient to repeat a simple sentence, ask the patient to read a passage from a newspaper & ask the patient to write a sentence; examine his handwriting.

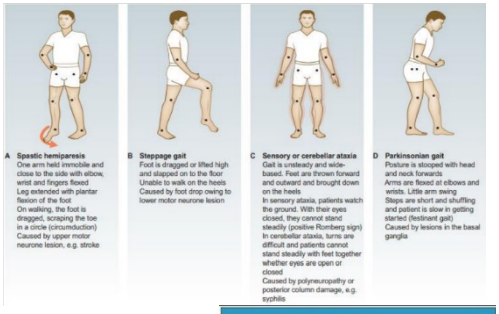
5. Cortical Function



• Thinking, emotions, language, behavior, planning and initiation of movements, and perception of sensory information are functions of the cerebral cortex and are central to awareness of, and interaction with, the environment.

• Certain cortical areas are associated with specific functions, so patterns of dysfunction can help localize the site of pathology.

6. Stance & Gait



- Stance and gait depend upon intact visual, sensory, corticospinal, extrapyramidal, and cerebellar pathways, together with functioning lower motor neurons and spinal reflexes.
- Certain abnormal gait patterns are recognizable, suggesting diagnoses.
- Stance (examine stance on narrow base while eyes are open (cerebellar ataxia) and closed (sensory ataxia, Romberg's sign)).
- Gait (note the gait, listen for the slapping sound of a foot drop gait, ask the patient to walk first on tip toes, then on the heels & don't forget about the tandem gait). Abnormal gaits are hemiplegic gait, scissors-like gait, ataxic gait, foot drop, parkinsonian gait, waddling gait & bizarre gaits.

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 (*marche à petits pas*) Difficulty in starting to walk/freezing Better '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

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Motor System

1. Inspection & Palpation of the muscles

- Completely expose the patient while keeping the patient's comfort and dignity.
- Look for asymmetry, inspecting both proximally and distally.
- Note deformities
- Examine for wasting or hypertrophy, fasciculation, and involuntary movement.

1. Muscle Bulk
    a. Muscle wasting: due to lower motor neurone lesions, not seen in acute upper motor neurone lesions, although disuse atrophy may develop with longstanding lesions. Muscle disorders usually result in proximal wasting.
    b. Muscle hypertrophy: certain occupations, e.g., professional sports players, may lead to physiological muscle hypertrophy. Pseudohypertrophy may occur in muscular dystrophy, but the muscles are weak. If you suspect wasting, ask the patient and whether he has also noticed this, as minor asymmetry in muscle bulk is often normal.
2. Abnormal Movement
    a. Fasciculations: visible irregular twitches under the skin overlying resting muscles caused by individual motor units firing spontaneously. Occurs in lower motor neuron diseases, usually in wasted muscles. It's seen not felt. Physiological fasciculation is common, especially in the calves. Myokymia is rapid bursts of repetitive motor unit activity often occurring in an eyelid or first dorsal interosseus and is rarely pathological.
    b. Myoclonic jerks: These are sudden, shock-like contractions of one or more muscles that may be focal or diffuse and occur singly or repetitively. Healthy people commonly experience these when falling asleep (hypnic jerks). They may also occur pathologically in association with epilepsy, diffuse brain damage and dementia.
    c. Dystonia: caused by sustained muscle contractions, leading to twisting, repetitive movements and sometimes tremor.
    d. Chorea: brief, random, purposeless movements which may affect various body parts, but commonly the arms.
    e. Athetosis: slower, writhing movement, more similar to dystonia than chorea.
    f. Ballism: refers to violent flinging movements sometimes affecting only one side of the body (hemiballismus).
    g. Tics: repetitive, stereotyped movements which can be briefly suppressed by the patient.
    h. Tremor: an oscillatory movement about a joint or a group of joints resulting from alternating contraction and relaxation of muscles. Classified according to their frequency, amplitude, position, and body part affected.
        - Fine, fast postural tremor: Physiological tremor seen with anxiety, hyperthyroidism, excess alcohol or caffeine intake, adverse effect of β-agonist.
        - Essential tremor is the most common pathological cause of an action tremor, often demonstrates an autosomal dominant pattern of inheritance, affecting the upper limbs (symmetrical) and head with postural and action components. It may be improved by alcohol
        - Action tremor: course and violent. Can be seen in CVA and MS.

- Resting tremor that is slow and coarse is typical of Parkinson, worse at rest but reduced with voluntary movement It is more common in the upper limbs, usually asymmetrical.
- Intention tremor: absent at rest and increases with movement. It is typical of cerebellum damage& is assessed with the finger-to-nose test.
- Functional tremors: inconsistent with varying frequencies and amplitudes. May be associated with other signs,

2. Assessment of the tone

- Tone is the resistance felt by the examiner when moving a joint passively.
- Ask the patient to lie supine and to relax. Enquire about any painful joints or limitations of movement before proceeding. Passively move each joint tested through as full a range as possible, both slowly and quickly in all anatomically possible directions. Be unpredictable with these movements, both in direction and speed, to prevent the patient actively moving with you; you want to assess passive tone.
    1. Upper limb: hold the patient's hand as if shaking hands, using your other hand to support his elbow. Assess tone at the wrist and elbow.
    2. Lower limb: roll the leg from side to side, then briskly lift the knee into a flexed position, observing the movement of the foot. Typically, the heel moves up the bed, but increased tone may cause it to lift off the bed due to failure of relaxation.
    3. Activation: a technique used to exaggerate subtle increase in tone and is useful for assessing extrapyramidal tone increase. Ask the patient to describe circles in the air with the contralateral limb while assessing tone. A transient increase in tone with this manoeuvre is normal.
    4. Ankle clonus: support the patient's leg, with both the knee and ankle resting in 90° flexion. Briskly dorsiflex and partially evert the foot, sustaining the pressure. Clonus is felt as repeated beats of dorsiflexion/plantar flexion.
- Abnormal findings:
    1. Hypotonia (flaccid): decreased muscle tone, lower motor neurone lesions and is usually associated with muscle wasting, weakness and hyporeflexia. Occurs In the early phases of cerebral or spinal shock.
    2. Hypertonia (spastic): decreased muscle tone
        a. Spasticity: velocity-dependent resistance to passive movement. it is detected with quick movements. In mild forms it is detected as a 'catch' at the beginning or end of passive movement. In severe cases it limits the range of movement and may be associated with contracture. In the upper limbs it may be more obvious on attempted extension; in the legs it is more evident on flexion.
        b. Rigidity: sustained resistance throughout the range of movement detected when the limb is moved slowly. In parkinsonism this is classically described as 'lead pipe rigidity' (it is constant through the movement, Parkinson without obvious tremor) . In the presence of a parkinsonian tremor there may be a regular interruption to the movement, giving it a

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

jerky feel ('cog wheeling'). Clasp-knife rigidity is seen on attempting flexion of the limp in strokes, MS or any other type of UMNL.

c. Clonus: rhythmic series of contractions evoked by sudden stretch of the muscle and tendon. Unsustained (<6 beats) clonus may be physiological. When sustained, it indicates upper motor neuron damage

3. Assessment of the power

- Test upper limb power with the patient sitting on the edge of the couch. Test lower limb power with the patient reclining. Ask about pain which may interfere with testing. Ask the patient to undertake a movement. First assess whether he can overcome gravity. Then apply resistance to this movement testing across a single joint.
- To test truncal strength, ask the patient to sit up from the lying position, or rise from a chair, without using the arms.
- In pronator drift: Observe the patient with his arms outstretched and supinated (palms up) and eyes closed for 'pronator drift', when one arm starts to pronate. It is an early feature of upper motor neurone lesions, and it has good sensitivity and specificity.

11.18 Medical Research Council scale for muscle power

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 examiner's resistance
4	Movement against resistance but weaker than normal
5	Normal power

Movement	Muscle	Nerve / Root
Shoulder abduction	deltoid	Axillary C5
Elbow flexion	Biceps Brachioradialis	Musculocutaneous C5,6 Radial C6
Elbow extention	Triceps	Radial C7
Wrist extension	Extensor carbi radialis longus	Posterior interosseous nerve (radial) C6
Finger flexion	Flexor pollicis longus Flexor digitorum profundus	Anterior interosseous (median) C8 Ulnar C8
Finger extension	Extensor digitorum communis	Posterior interosseous nerve (radial) C7
Finger abduction	First dorsal interosseous	Ulnar T1
Thumb abduction	Abductor pollicis previs	Median T1

Movement	Muscle	Nerve / Root
Hip flexion	iliopsoas	Iliofemoral L1,2
Hip extension	Gluteus maximus	Sciatic L5,S1
Knee flexion	Hamstrings	Sciatic S1
Knee extention	Quadriceps	Femoral L3,4
Ankle dorsiflexion	Tibialis anterior	Deep peroneal L4,5
Ankle plantar flexion	Gastrocnemius and soleus	Tibial S1,2
Great toe extention	Extensor hallucis longus	Deep peroneal L5
Ankle eversion	Peroneus	Superficial peroneal L5,S1
Ankle inversion	tibialis posterior	Tibial nerve L4,5

- Upper motor neurone lesions produce weakness of a relatively large group of muscles
- Lower motor neurone damage can cause paresis of an individual and specific muscle
- You need only show that the patient can achieve maximum power briefly
- Functional weakness is wildly fluctuating or sudden 'giveaway' weakness / Hoover's sign.

11.20 Definitions of paralysis

Term	Definition
Paresis	Partial paralysis
Plegia	Complete paralysis
Monoplegia	Involvement of a single limb
Hemiplegia	Involvement of one-half of the body
Paraplegia/diplegia	Paralysis of the legs
Tetraplegia	Paralysis of all four limbs

4. Assessment of the reflexes

- A tendon reflex is the involuntary contraction of a muscle in response to stretch.
- It is mediated by a reflex arc consisting of an afferent (sensory) and an efferent (motor) neuron with one synapse between (a monosynaptic reflex)
- These stretch reflex arcs are served by a particular spinal cord segment which is modified by descending upper motor neurons.

- Ask the patient to lie supine on the examination couch with the limbs exposed. He should be as relaxed and comfortable as possible, as anxiety and pain can cause an increased response. 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.
- Use reinforcement whenever a reflex appears absent. Never conclude a reflex is absent until you have used reinforcement. The patient should relax between repeated attempts. Strike the tendon immediately after your command to the patient.
    1. upper limb reflexes: ask the patient to clench the teeth or to make a fist with the contralateral hand.
    2. Lower limb reflexes: ask the patient to interlock the fingers and pull one hand against the other
- Record the response as:
    1. Increased (+++)
    2. Normal (++)
    3. Diminished (+)
    4. present only with reinforcement (+/-)
    5. Absent (0)
- Abnormal Findings:
    1. Hyperreflexia: a sign of upper motor neuron damage.
    2. Diminished or absent jerks: due to lower motor neuron lesions. In healthy elderly people the ankle jerks may be reduced or lost. Isolated loss of a reflex suggests a mononeuropathy or radiculopathy.
    3. An 'inverted' biceps reflex is caused by combined spinal cord and root pathology localizing 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 the spinal cord increasing reflexes below this level (including the finger jerks). It is most seen in cervical spondylitis myeloradiculopathy.
    4. In cerebellar disease, the reflexes may be pendular. Muscle contraction and relaxation tend to be slow; these are not sensitive or specific cerebellar signs.
    5. Positive Hoffmann's and finger jerks suggest hypertonia. It can occur in healthy individuals and are not useful signs in isolation

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

- Superficial reflexes: This group of reflexes is polysynaptic and elicited by cutaneous stimulation rather than stretch. Except for the plantar response, they are not part of the routine examination, and have poor sensitivity and specificity. The cremasteric reflex applies only in males.
- Abdominal reflex T8 – T12: The patient should be supine and relaxed. Use an orange stick and briskly, but lightly, stroke the upper and lower quadrants of the abdomen in a medial direction The normal response is contraction of the underlying muscle, with the umbilicus moving laterally and up or down depending upon the quadrant tested.

    Abnormal findings:

1. Superficial abdominal reflexes (T8–12) are lost in upper motor neuron lesions but are also affected by lower motor neuron damage affecting T8–12. They are usually absent in the obese, the elderly or after abdominal surgery.

- Cremasteric reflex (L1–2): Explain what you are going to do and why it is necessary. Abduct and externally rotate the patient's thigh. Use an orange stick to stroke the upper medial aspect of the thigh. Normally the testis on the side stimulated will rise briskly.

  Abnormal findings:

  1. The cremasteric reflex in males (L1 and L2) is rarely elicited. Typically, is lost in spinal cord or root lesions.

- Plantar response (S1–2): Run a blunt object along the lateral border of the sole of the foot towards the little toe. Watch both the first movement of the great toe and the other leg flexor muscles. The normal response is flexion of the great toe with flexion of the other toes.

  Abnormal findings:

  1. A true Babinski sign involves activation of the extensor hallucis longus tendon (not movement of the entire foot, a common 'withdrawal' response to an unpleasant stimulus. Coincides with contraction of other leg flexor muscles. It is reproducible. This is a sign of upper motor neuron lesion. Fanning of the toes is normal and not pathological.

- Primitive reflexes: These are present in normal neonates and young infants but disappear as the nervous system matures. Their return after early childhood is often associated with brain damage or degeneration.

  Abnormal findings:

  1. The primitive reflexes have little localizing value and in isolation are of little significance, but in combination suggest diffuse or frontal cerebral damage. Unilateral grasp and palmomental reflexes may occur with contralateral frontal lobe pathology. The glabellar tap is an unreliable sign of Parkinson's disease

11.25 Primitive reflexes

Snout reflex
• Lightly tap the lips. An abnormal response is lip pouting
Grasp reflex
• Firmly stroke the palm from the radial side. In an abnormal response, your finger is gripped by the patient's hand
Palmomental reflex
• Apply firm pressure to the palm next to the thenar eminence with a tongue depressor. An abnormal response is ipsilateral puckering of the chin
Glabellar tap
• 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

5. Assessment of the coordination

- Performing complex movements smoothly and efficiently. Depends upon intact sensory and motor function and an intact cerebellum. In general, cerebellar midline structures, e.g., vermis, influence body equilibrium, while each hemisphere controls ipsilateral coordination.

- What to examine:
  1. Stance & Gait: In disorders predominantly affecting midline cerebellar structures, truncal ataxia may be the only finding. In the most severe cases, this may mean the patient cannot sit unsupported. Tandem gait (heel–toe walking) may be impaired in less severe cases.
  2. Speech (Dysarthria)

3. Nystagmus
4. Limb coordination (Upper & Lower)
    a. Upper limbs:
        1. Finger-to-nose test
            - Ask the patient to touch his nose with the tip of his index finger and then touch your fingertip. Hold your finger just within the patient's arm's reach. Ask him to repeat the movement between nose and target finger as quickly as possible. Make the test more sensitive by changing the position of your target finger. Move your finger just as the patient's finger is about to leave his nose, otherwise you will induce a false-positive finger-to-nose ataxia. Some patients are so ataxic that they may injure their eye/face with this test. If so, use your two hands as the targets.
            - Abnormal findings
                1. Weakness may produce false-positive finger-to-nose test, so demonstrate that power is normal first.
                2. Dysmetria or past pointing: tendency to fall short or overshoot the examiner's finger.
                3. Intention tremor: In more severe cases there may be a tremor of the finger as it approaches the target finger and the patient's own nose
                4. Dyssynergia: The movement may be slow, disjointed, and clumsy.
        2. Rapid alternating movements
            - First method: demonstrate repeatedly patting the palm of your hand with the palm and back of your opposite hand as quickly and regularly as possible. Ask the patient to copy your actions. Repeat with the opposite hand.
            - Second method: ask the patient to tap a steady rhythm rapidly with his hand on the other hand or table, 'listen to the cerebellum' ataxia makes this task difficult, with a slower, irregular rhythm than normal.
            - Abnormal findings
                1. Dysdiadochokinesis: impairment of rapid alternating movements is evident as slowness, disorganization, and irregularity of movement
        3. Rebound phenomenon (rarely useful)
            - Ask the patient to stretch his arms out and maintain this position. Push the patient's wrist quickly downward and observe the returning movement.
            - Abnormal findings
                1. The normal response is to return to the original position. The rebound phenomenon occurs when the displaced outstretched arm flies up past the original position.
    b. Lower limbs:
        1. Heel-to-shin test
            - With the patient lying supine, ask him to place his heel on his opposite knee, and then slide his heel up and down the shin between knee and ankle.
            - Abnormal findings

1. Same as finger-to-nose test. It is abnormal if the heel wavers away from the line of the shin. Weakness may produce false-positive heel-to-shin test, so demonstrate that power is normal first.

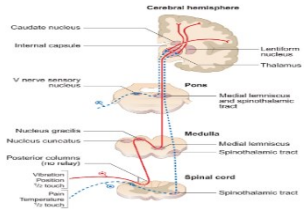
5. Apraxia: Dyspraxia or apraxia is difficulty or inability to perform a task, despite no impairment of the necessary individual functions. It is a sign of higher cortical dysfunction, usually localizing to the non-dominant frontal or parietal lobes.
    - Ask the patient to perform an imaginary act. Ask the patient to copy movements you make with your fingers. Ask the patient to copy a geometrical figure. Ask the patient to put on a pajama top or dressing gown, one sleeve of which has been pulled inside out.
    - Abnormal findings
        1. Constructional apraxia:
            a. Difficulty drawing a figure.
            b. Is a feature of parietal disturbance.
        2. Dressing apraxia:
            a. Often associated with spatial disorientation and neglect
            b. is usually due to non-dominant hemisphere parietal lesions.

Sensory System

- Detailed examination of sensation is time-consuming and unnecessary unless the patient volunteers sensory symptoms or you suspect a specific pathology

1. Anatomy

- Proprioception and vibration sensation: conveyed in large, myelinated fast-conducting fibers in the peripheral nerves and in the posterior columns of the spinal cord. The posterior column remains ipsilateral from the point of entry up to the medulla.
- Pain and temperature sensation: Carried by small, slow-conducting fibers of the peripheral nerves and the spinothalamic tract of the spinal cord. Most pain and



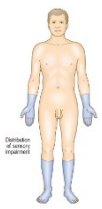
temperature fibers cross to the contralateral spinothalamic tract within one or two segments of entry to the spinal cord.
- All sensory fibers relay in the thalamus before sending information to the sensory cortex in the parietal lobe.

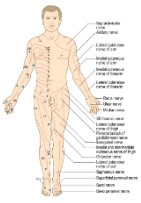
2. Symptoms & Definitions

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

3. Sensory Modalities

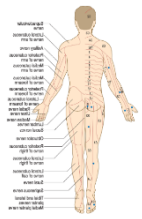
- Peripheral nerve and dorsal root
    1. Many diseases affect peripheral nerves, generally resulting in peripheral neuropathies or polyneuropathies. Peripheral neuropathies tend to affect the lower limbs first (length-dependent). Symptoms affecting the



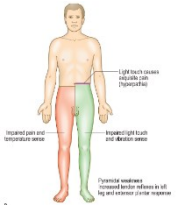


upper limbs first suggest a demyelinating rather than axonal neuropathy or a disease process in the spinal cord.

2. When joint position sense is affected in the arms, pseudo athetosis may be demonstrated by asking the patient to close his eyes and hold his hands outstretched: the fingers will make involuntary, slow wandering movements, mimicking athetosis.
3. Interpretation of sensory signs requires knowledge of the relevant anatomy of sensory nerves and dermatomes



- Spinal cord
    1. Traumatic and compressive spinal cord lesions cause loss or impairment of sensation in a dermatomal distribution below the level of the lesion. A zone of hyperesthesia may be found immediately above the level of sensory loss.
    2. Anterior spinal artery syndrome results in loss of spinothalamic sensation and motor function, with sparing of dorsal column sensation.
    3. A similar dissociated pattern of pain and temperature loss and sparing of dorsal column sensation occurs in syringomyelia.
    4. Brown-Séquard syndrome: when one-half of the spinal cord is damaged. This is characterized by ipsilateral motor weakness and loss of vibration and joint position sense, with contralateral loss of pain and temperature.



- Intracranial
    1. Thalamic lesions may cause a patchy sensory impairment on the opposite side with unpleasant, poorly localized pain, often of a burning quality. Cortical parietal lobe lesions typically cause sensory inattention but may also affect joint position sense, two-point discrimination, stereognosis (tactile recognition) and localization of point touch.
    2. Lower brainstem lesions may cause ipsilateral numbness on one side of the face (V nerve nucleus) and contralateral body numbness (spinothalamic tract).

4. Examination Steps

a. Light Touch

- While the patient looks away or closes his eyes, use a wisp of cotton wool (or lightly apply your finger) and ask the patient to say yes to each touch. Time the stimuli irregularly and make a dabbing rather than a stroking or tickling stimulus. Compare each side for symmetry.

b. Superficial Pain

- Use a fresh neurological pin. Explain and demonstrate that the ability to feel a sharp pinprick is being tested. Map out the boundaries of any area of reduced, absent, or increased sensation and compare with. Move from reduced to higher sensibility: i.e., from hypoesthesia to normal, or normal to hyperesthesia.

c. Temperature

- Touch the patient with a cold metallic object, e.g., tuning fork, and ask if it feels cold. More sensitive assessment requires tubes of hot and cold water at controlled temperatures but is seldom performed.

d. Vibration

- Teach your patient what to fell, place a vibrating 128 Hz tuning fork over the sternum. Ask the patient, 'Do you feel it buzzing?'
- The upper limb: Start at the distal interphalangeal joint of the forefinger, and if sensation is impaired, proceed proximally.
- The lower limb: place it on the tip of the great toe. If sensation is impaired, place the fork on the interphalangeal joint and progress proximally, to the medial malleolus, tibial tuberosity, and anterior iliac spine, depending upon the response.
- If in doubt as to the accuracy of the response, ask the patient to close his eyes and to report when you stop the fork vibrating with your fingers.

e. Joint Position

- With the patient's eyes open, demonstrate the procedure. Hold the distal phalanx of the patient's great toe at the sides. Tell the patient you are going to move his toe up or down, demonstrating as you do so. Ask the patient to close his eyes and to identify the direction of small movements in random order.
- Test both great toes (or middle fingers). If impaired, move to more proximal joints in each limb.

f. Stereognosis & Graphesthesia

- Ask the patient to close his eyes.
- Stereognosis: Place a familiar object, e.g., coin or key, in his hand and ask him to identify it.
- Graphesthesia: use the blunt end of a pencil or orange stick and trace letters or digits on the patient's palm. Ask the patient to identify the figure.

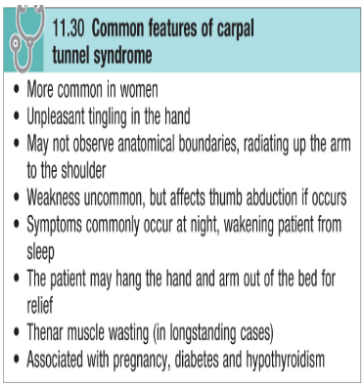
g. Sensory Inattention (if sensory pathways are otherwise intact)

- Ask the patient to close his eyes. Touch his arms/legs in turn and ask which side has been touched. Now touch both sides simultaneously and ask whether the left, right or both sides were touched.

Peripheral Nerves

1. Sensory Examination of the hand

- Test for altered sensation over the hand involving:
  a. the thumb (Radial)
  b. index and middle fingers (Median)
  c. the lateral half of the ring (Ulnar)



2. Motor Examination of the hand (Functions)

- Median Nerve
  a. Look for wasting of the thenar eminence.
  b. Test thumb abduction with the patient's handheld palm up on a flat surface. Ask the patient to move the thumb vertically against your resistance (abductor pollicis brevis).
  c. Test opposition by asking the patient to touch the thumb and ring finger together while you attempt to pull them apart (opponens pollicis).
- Carpal tunnel syndrome is the most common entrapment neuropathy. The nerve may be compressed as it passes between the flexor retinaculum and the carpal bones at the wrist. Initially produces sensory symptoms.

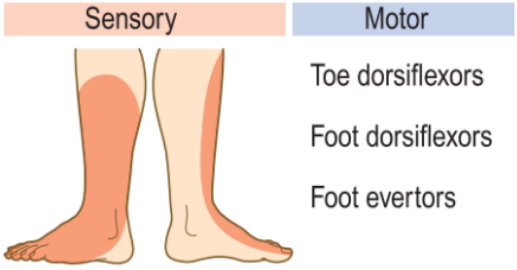
- Ulnar Nerve

a. Look for wasting of interossei (dorsal guttering).
b. Test for weakness of finger abduction with the patient's fingers on a flat surface and ask him to spread the fingers against resistance from your fingers.
c. Test adduction by placing a card between the patient's fingers and pulling it out using your own fingers.
d. Examine the elbow (the commonest place of entrapment). Note any scars or other signs of trauma.
e. Feel for the nerve in the ulnar groove.

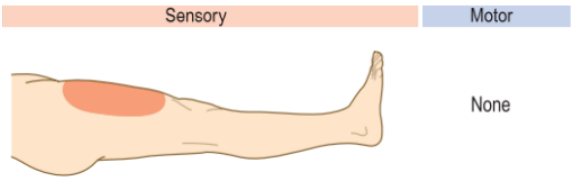
- Radial Nerve
  a. Test for weakness of arm and forearm extensors (triceps and the wrist and fingers).
  b. Look for sensory loss over the dorsum of the hand.
  c. loss of triceps tendon jerk.

3. Specific Nerve Lesions

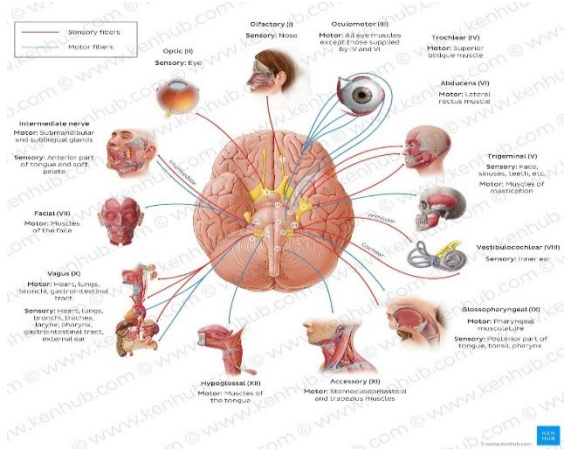
- Common Peroneal Nerve
  a. This typically presents with foot drop.
  b. It may be damaged in fibular head fractures, or compressed particularly in immobile patients, or because of repetitive kneeling or squatting.
  - Test for weakness of ankle dorsiflexion and eversion. Inversion will be preserved.
  - Test for sensory loss over the dorsum of the foot & lateral aspect of the leg.



- Lateral cutaneous nerve of the thigh
  a. This purely sensory nerve may be compressed as it passes under the inguinal ligament, producing paraesthesia in the lateral thigh (meralgia paraesthetica).
  b. It is related to obesity & pregnancy.



Cranial Nerves



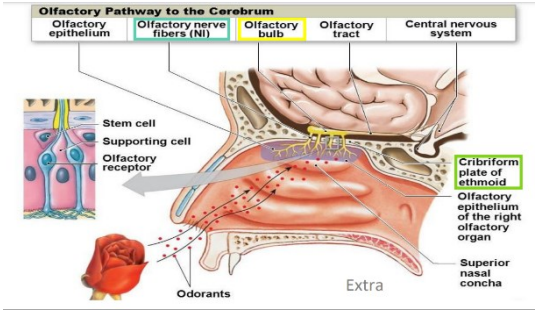
	Pseudobulbar (UMN CN IX, CN X, CN XII)	Bulbar (LMN CN IX, CN X, CN XII)
Gag	Increased	Absent
Tongue	Spastic	Wasted, Fasciculations
Jaw Jerk	Increased	Absent / Normal
Speach	Spastic Dysarthria	Nasal
Limbs	UMN signs	LMN signs
Emotions	Labile	Normal
Causes	Bilateral CVA Multiple Sclerosis Motor Neuron Disease	Motor Neuron Disease GB Syndrome Poliomyelitis Brainstem infarction

W05 – CNS & PNS

Collected from Al Thara'a, E-Learning Slides, Lectures Slides, Macleod 14th Ed

- UMN: above the nerve nucleus, or anterior horn cell of spinal cord.
- The 12 pairs of cranial nerves arise from the brain.
  1. CNs 1 and 2 arise from the cerebral cortex.
  2. CNs 3 and 4 arise from the midbrain.
  3. CNS 5,6,7 and 8 arise from pons.
  4. CNs 9,10,11 and 12 arise from medulla.

1. CN1 – Olfactory



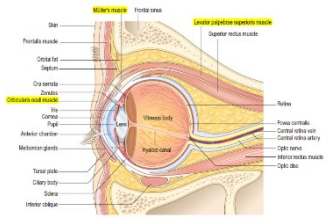
- Bipolar cells in the olfactory bulb form olfactory filaments, with small receptors projecting through the cribriform plate high in the nasal cavity. These cells synapse with second-order neurons, which project centrally via the olfactory tract to the medial temporal lobe and amygdala.
- Purely sensory & conveys the sense of smell
- Abnormalities:

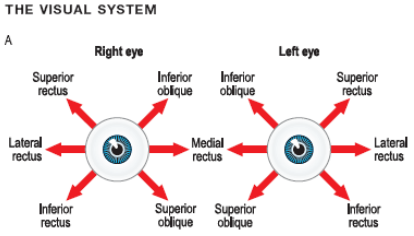
1. Hyposmia or anosmia (reduction or loss of the sense of smell): URTI, head injury, compression on invasion by tumors (basal skull tumors).
2. Parosmia (perception of pleasant odors as unpleasant): head trauma, infections, drugs (ACEI, ARBs, antibiotics)
3. Phantosmia/Olfactory hallucination; Alzheimer's disease and focal epilepsies.
4. Disturbance of smell may also occur very early in Parkinson's and Alzheimer's diseases. Patients often note hypogeusia/ageusia (altered taste) with anosmia too, as taste is crucially influenced by the sense of smell.

- The exam is of limited clinical value, and rarely performed
- Check the nasal passages for clearance. Ask the patient to close his eyes. Close one nostril at a time. Use 'scratch and sniff' test cards (the University of Pennsylvania Smell Identification Test (UPSIT)).

2. CN2 – Optic

- The eye is a complex structure situated in the bony orbit. It is protected by the eyelid, that helps to maintain the tear film.
- The upper lid is elevated by:
  1. The levator palpebrae superioris (CN III).
  2. Müller's muscle (sympathetic autonomic system).
- Eyelid closure: orbicularis oculi muscle (CN VII).
- The conjunctiva is a thin mucous membrane lining the posterior aspects of the eyelids. The conjunctiva is coated in a tear film that protects and nourishes the ocular surface.
- There are 6 extraocular muscles; all are supplied by CN 3, except superior oblique by CN 4 (SO4) and lateral rectus by CN 6 (LR6). 3rd, 4th CN originate form midbrain and 6th CN from pons then all of them pass through cavernous sinus

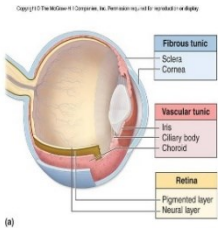


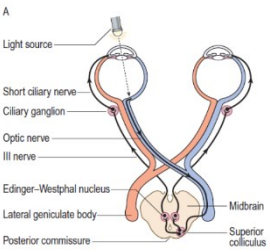


W05 – CNS & PNS

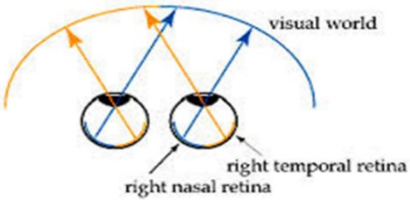
Collected from Al Thara'a, E-Learning Slides, Lectures Slides, Macleod 14th Ed

- The eyeball comprises three distinct layers:
    1. Outer fibrous layer: this includes the sclera and the clear cornea.
    2. Middle vascular layer (uveal tract): anteriorly this consists of the ciliary body and the iris, and posteriorly the choroid.
    3. Inner neurosensory layer (retina): responsible for converting light to neurological signals.





- Pupillary pathway: the pupil controls the amount of light entering the eye. Pupillary dilatation is controlled by sympathetic nerves (The sympathetic pathway originates in the hypothalamus, passing down to the ciliospinal centre of Budge at the level of T1. Fibers then synapse in the superior cervical ganglion before joining the surface of the internal carotid artery and passing to the pupil along the nasociliary and the long ciliary nerves). Pupillary constriction is controlled by parasympathetic nerves (Afferent nerve CN 2, Efferent nerve CN 3).
    a. The afferent limb involves the optic nerve, chiasm (where some fibres decussate) and the optic tract, bypassing the lateral geniculate nucleus, synapsing in the pretectal nucleus of the midbrain then terminate in the III nerve (Edinger–Westphal) nucleus.
    b. The efferent limb involves the inferior division of the III nerve, passing through the ciliary ganglion in the orbit to the constrictor muscle of the iris via ciliary nerves.
- The optic nerve is purely sensory & unable to regenerate.
- Responsible for transmitting visual sensory information from the retina to the brain & the afferent part of the pupillary reflex
- The visual pathway consists of the: retina > optic nerve > optic chiasm > optic tracts > lateral geniculate bodies > optic radiations > and visual cortex.
- Deficits in the visual pathway lead to specific field defects.
- The nasal fibers of the optic nerve are responsible for the temporal visual field and vice versa.



- Before the Optic chiasm - The visual field loss is seen on the same (ipsilateral) side as the lesion.
- After the optic chiasm - The visual loss is seen on the opposite (contralateral) side of the lesion because the optic nerves have already crossed over at the optic chiasm

W05 – CNS & PNS

Collected from Al Thara'a, E-Learning Slides, Lectures Slides, Macleod 14th Ed

The visual field loss is seen on the same (ipsilateral) side as the lesion

Bitemporal hemianopia at optic chiasm

The visual loss is seen on the opposite (contralateral) side of the lesion because the optic nerves have already crossed over at the optic chiasm

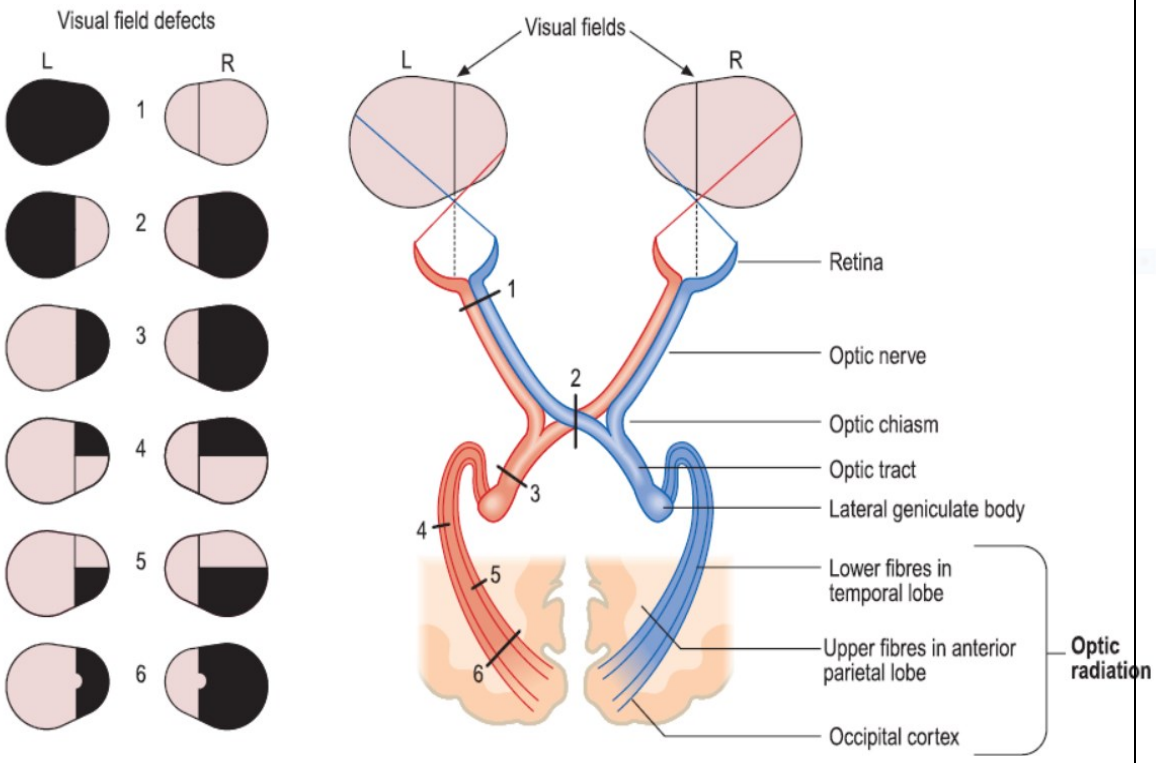


Fig. 8.5 Visual field defects. 1, Total loss of vision in one eye because of a lesion of the optic nerve. **2,** Bitemporal hemianopia due to compression of the optic chiasm. **3,** Right homonymous hemianopia from a lesion of the optic tract. **4,** Upper right quadrantanopia from a lesion of the lower 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. CN3 – Oculomotor

- Motor and parasympathetic function. It's course is related to posterior communicating artery and cavernous sinus. Innervates the superior, medial, and inferior recti, the inferior oblique and levator palpebrae superioris muscles.
- Function:
    1. It Moves the globe upwards, downwards, and medially (the superior, medial, and inferior recti, the inferior oblique).
    2. It elevates the upper lid (levator palpebrae superioris muscles).
    3. Pupillary reflex (Efferent, constrict pupil).
- Abnormalities (unilateral oculomotor nerve palsy)
    1. Unilateral ptosis that is often complete.
    2. Dilated pupil.
    3. The eye is looking inferolaterally.

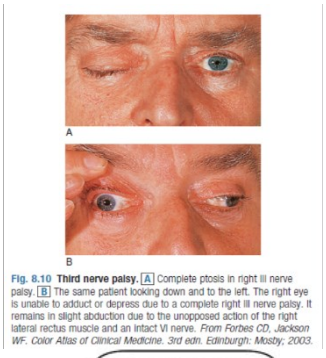


Fig. 8.10 Third nerve palsy. A Complete ptosis in right III nerve palsy. B The same patient looking down and to the left. The right eye is unable to adduct or depress due to a complete right III nerve palsy. It remains in slight abduction due to the unopposed action of the right lateral rectus muscle and an intact VI nerve. *From Forbes CD, Jackson WF. Color Atlas of Clinical Medicine. 3rd edn. Edinburgh: Mosby; 2003.*

4. CN4 – Trochlear

- Supplies the superior oblique muscle
- Function: downward movement of the globe when the eye is adducted (inferiomedially).

5. CN6 – Abducens

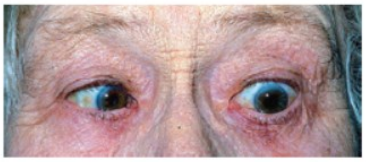


Fig. 8.9 Sixth nerve palsy causing weakness of the lateral rectus muscle. The patient is attempting to look to the left.

- Supplies the lateral rectus muscle.
- Abducts the eye (lateral gaze).
- Has a long course around the brainstem before it pierces the dura to enter the cavernous sinus.
- In direct relation to the internal carotid artery before it passes through the superior orbital fissure to the lateral rectus muscle.

Due to time constraint, finish what's left of the Cranial Nerves from Dr Ibtihal Slides (Slides 10-56) & Slides (77-103) + Check out the collected tables (some of them were already mentioned in this file).