

# NEUROLOGY Hx

History of presenting complaint	<p><b>Headache</b>, <b>[SOCRATES]</b> → type/severity</p> <ul style="list-style-type: none"> <li>• <b>Time course</b> (esp. SAH vs IIH)           <ul style="list-style-type: none"> <li>a. <b>Acute (secs)</b> = SAH, stroke, focal/generalised seizure</li> <li>b. <b>Subacute (hrs-days)</b> = infection, inflammatory disorder (Guillain–Barré syndrome)</li> <li>c. <b>Insidious (wks – mths)</b> = IIH, tumour, neurodegenerative</li> </ul> </li> <li>• <b>Triggers?</b> → auras, hormone changes?</li> <li>• <b>Assoc. (photo/phonophobia, cold, mental clouding, rhinorrhoea, flushed head)</b></li> </ul> <p><b>Fits, faints or funny turns</b> <b>[Seizures/Strokes]</b></p> <ul style="list-style-type: none"> <li>• <b>Pre-event</b> <b>[LAD]</b> <ul style="list-style-type: none"> <li>a. <b>LOC</b></li> <li>b. <b>auras</b></li> <li>c. <b>DIZZY</b> (vertigo, lightheaded),</li> <li>d. <b>vision issue, speech,</b></li> <li>e. <b>trauma</b> → extra-dural, sub-dural haem.</li> </ul> </li> <li>• <b>During event (what happened?, duration – hrs, days?)</b></li> <li>• <b>After event</b> <b>[WILD]</b> (weakness incontinence, Lateral Tongue biting, drowsiness)</li> <li>• <b>Previous episodes / scans (MRI, CT)</b></li> <li>• <b>Underlying CV cause (i.e. palpitation, SOB)</b></li> </ul>	<ul style="list-style-type: none"> <li>• <b>Disturbed gait, sensation and weakness</b> <ul style="list-style-type: none"> <li>a. Dysesthesia (unpleasant feel)</li> <li>b. Paraesthesia (pins &amp; needles) &amp; numb</li> </ul> </li> <li>• <b>Disturbances of vision, hearing, smell, speech &amp; swallowing</b></li> </ul> <p><b>Consider pattern of symptoms:</b></p> <ul style="list-style-type: none"> <li>• <b>Unilateral vs bilateral</b></li> <li>• <b>Sensory vs motor vs sensori-motor</b></li> <li>• <b>Distal vs proximal weakness</b> <ul style="list-style-type: none"> <li>o Cranial vs Long tract (UL vs LL)</li> </ul> </li> <li>• <b>PNS vs ANS</b> (bladder, ED, irregular BP)</li> <li>• <b>Dermatomal/myotomal distribution or not</b></li> </ul>
Past MHx	<b>Current Conditions</b>	<ul style="list-style-type: none"> <li>• <b>Risk of Cerebrovascular disease:</b> epilepsy/convulsions   HT   DM   Dyslipidaemia           <ul style="list-style-type: none"> <li>o Previous strokes / STEMIs</li> <li>o Previous accidents (e.g. head/spinal injuries) or <b>Infection</b> (meningitis, STIs)</li> </ul> </li> </ul>
	<b>Medications</b>	<ul style="list-style-type: none"> <li>• Anticonvulsants, anti-Parkinsonian drugs, <b>COPC</b> [<math>\uparrow</math> stroke risk]</li> <li>• Steroids, stains &amp; Opiates</li> </ul>
	<b>Surgeries/ Treatments</b>	<ul style="list-style-type: none"> <li>• Chemotherapy for malignancy (leukaemia, myeloma or lymphoma)</li> <li>• <b>Splenectomy</b> (thrombocytopenia or lymphoma)</li> </ul>
	<b>Tests</b>	Results of CT or MRI brain scan
	Other	<b>Allergies?</b> + <b>Vaccinations</b> [ <i>Strep. Pneumoniae, FluVax</i> ]
Social Hx	<b>Occupation</b>	<ul style="list-style-type: none"> <li>• exposure to toxins (e.g. heavy metals)</li> </ul>
	<b>Smoking</b>	<ul style="list-style-type: none"> <li>• cerebrovascular disease [<math>\uparrow</math> vascular risk]</li> </ul>
	<b>Alcohol</b>	<ul style="list-style-type: none"> <li>• blackouts, alcoholic dementia, myopathy</li> </ul>
	<b>Drugs</b>	<ul style="list-style-type: none"> <li>• Marijuana + cocaine induced headache</li> </ul>
Family Hx	<ul style="list-style-type: none"> <li>• <b>Family Hx</b> of migraine, stroke, Alzheimer's, epilepsy</li> <li>• <b>X-LINKED:</b> Colour blindness, DMD   <b>Autosomal dominant (neurodegenerative disease):</b> Huntington's chorea, MS</li> </ul>	
SR	<ul style="list-style-type: none"> <li>• <b>CVS</b> – CAD SPIFE, <b>RESP</b> – SCSC FAWIF, <b>GIT</b> – BLIND CRAP, <b>GU</b> – FUNDWISE   PORN HAWC   <b>Menstrual Cycle</b></li> <li>• <b>General:</b> Fever   weight loss/gain   Speech   Smell   Hearing   Sight</li> </ul>	

## Red flags on history

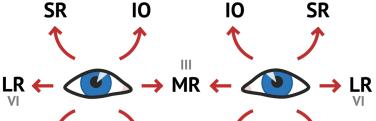
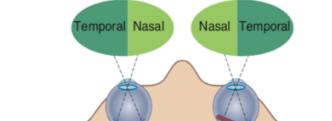
- Fever, photophobia or neck stiffness (**meningitis or encephalitis**)
- New neurological symptoms (**haemorrhage, malignancy or stroke**)
- Dizziness (**stroke**)
- Visual disturbance (**temporal arteritis or glaucoma**)
- Sudden onset occipital headache (**subarachnoid haemorrhage**)
- Worse on coughing or straining (**raised intracranial pressure**)
- Postural, worse on standing, lying or bending over or Pemberton's sign (**raised intracranial pressure**)
- Severe enough to wake the patient from sleep
- Vomiting (**raised intracranial pressure or carbon monoxide poisoning**)
- History of trauma (**intracranial haemorrhage**)
- Pregnancy (**pre-eclampsia**)
- **Recent head trauma within 3/12 = ?SDH**
- Headaches **with use of medications/illicit drugs** (e.g. **anticoagulants, sympathomimetic agents**)

## Red flags on Examination

- Abnormal vitals
- Neurological abnormalities
- Decreased LOC → other cause (**CV, metabolic, psychogenic**):
  - o **Arrhythmias** = assoc. with palpitations
  - o **Aortic stenosis** = **LOC** with heavy exercise
  - o **Transient ischaemic attacks** = 'drop attacks' means the patient falls but **NO LOC**.
  - o **Vasovagal syncope** = **LOC** due to abrupt drop in HR, BP (due to stress)
  - o **Micturition syncope** = **LOC** due to urination
  - o **Hypoglycaemia** (diabetics on insulin) = sweating, weakness and confusion **BEFORE** LOC.
  - o **Psychogenic non-epileptic seizures (PNES)** → **LOC** (no response to anticonvulsants) → need psychotherapy to Rx anxiety and depression
- Meningismus (similar to meningitis without inflammation of meninges → i.e. stiff neck, photophobia)
- Papilledema → Idiopathic intracranial hypertension (IIH). Raised ICP

Examination	Indication	Purpose
Mental state examination (MSE)	Mental health disorder	<ul style="list-style-type: none"> <li>• "A routine assessment to check your brain function that I do on all my patients of similar age"</li> <li>• provides snapshot of a patient's emotions, thoughts, and behaviour at the time of observation</li> <li>• helps identify the presence and severity of a variety of <b>mental</b> health conditions and the risk a patient poses to him- or herself, or to others</li> </ul>
Mini-Mental State Exam (MMSE)	Elderly	<ul style="list-style-type: none"> <li>• tests cognitive function among the <b>elderly</b> →</li> <li>• measures of orientation, registration (immediate memory), short-term memory (but not long-term memory) as well as language functioning</li> <li>• <b>Orientation</b> → <b>registration</b> → <b>registration</b> → <b>attention + calc</b> → <b>recall</b> → <b>language</b></li> <li>• Score out of 30 (&gt; 25 - normal, 21-24 = mild cognitive impairment, &lt;20 = dementia)</li> </ul>

## Cranial Nerve Examination (upper CN – I, II, III, IV, VI)

GI	<ul style="list-style-type: none"> <li>Today I have been asked to check your cranial nerves. These are the nerves that supply your face and neck</li> <li><b>Responsiveness + Orientated</b> I tell me how you got here today?"</li> <li><b>Ptosis</b> (drooping one/both eyelids = old age, 7<sup>th</sup> nerve palsy, Horner's (ptosis + anhidrosis, myosis, anophthalmos)</li> <li><b>Proptosis / strabismus</b> (misalignment/deviation of one/both eye)</li> <li><b>Facial asymmetry &amp; facial drooping</b> (salivation)</li> <li><b>Obvious muscle wasting</b> (Temporal)</li> </ul>															
CN I	<ul style="list-style-type: none"> <li><b>Alcohol wipe smell (test each nostril separately)</b> <ul style="list-style-type: none"> <li>"Close eyes + cover one nose" "Describe to me what you smell"</li> <li><b>DDx (anosmia):</b> Kallman URTI, smoking, ethmoid tumours, basal skull/frontal fracture, post pituitary surgery, congenital (eg. Kallmans syndrome), meningioma of olfactory groove, infectious (meningitis)</li> </ul> </li> </ul>															
CN 2 AFRO CAP	<div style="display: flex; justify-content: space-between;"> <div style="width: 45%;"> <p><b>Acuity [Snellen chart]</b></p> <ul style="list-style-type: none"> <li><b>ENSURE YOU POSITION PATIENT AT EYE LEVEL!!</b></li> <li>Cover one eye and read the smallest line you can</li> <li>If unable: → <b>"How many fingers"</b> [CF] → <b>Hand movement</b> [HM] → <b>Perception of Light</b> (PL) → <b>NPL</b></li> </ul> </div> <div style="width: 45%;"> <p><b>Visual Fields</b></p> <ul style="list-style-type: none"> <li><b>"Cover your own eye with one hand and then the other!"</b></li> <li>Cover your <b>left eye with your left hand</b> → "look into my eye and say 'yes' when you see <b>my finger moving</b>"</li> <li>Repeat with <b>coloured object</b> (NARROWER visual field – cones located centrally in macula, rods peripheral)</li> <li>Repeat for other eye</li> </ul> <div style="display: flex; justify-content: space-around; margin-top: 10px;">   </div> <div style="display: flex; justify-content: space-between; margin-top: 10px;"> <div style="width: 45%;">  <p>Visual field testing: 'Tell me when you first see the red pin come into view'</p> </div> <div style="width: 45%;"> <ol style="list-style-type: none"> <li><b>TUNNEL VISION</b> Concentric diminution, e.g. glaucoma, papilloedema, syphilis</li> <li><b>ENLARGED BLIND SPOT</b> Optic nerve head enlargement</li> <li><b>CENTRAL SCOTOMATA</b> Optic nerve head to chiasm lesion, e.g. demyelination, toxic, vascular, nutritional</li> <li><b>UNILATERAL FIELD LOSS</b> Optic nerve lesion, e.g. vascular, tumour</li> <li><b>BITEMPORAL HEMIANOPIA</b> Optic chiasm lesion, e.g. pituitary tumour, sella meningioma</li> <li><b>HOMONYMOUS HEMIANOPIA</b> Optic tract to occipital cortex, e.g. vascular, tumour (NB: incomplete lesion results in macular (central) vision sparing)</li> <li><b>UPPER QUADRANT HOMONYMOUS HEMIANOPIA</b> Temporal lobe lesion, e.g. vascular, tumour</li> <li><b>LOWER QUADRANT HOMONYMOUS HEMIANOPIA</b> Parietal lobe lesion</li> </ol> </div> </div> </div> </div>															
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CN 3	<p><b>Eye movements</b> [Draw large H]</p> <p><b>Reflex types:</b></p> <ol style="list-style-type: none"> <li>1) pursuit</li> <li>2) saccades</li> <li>3) convergence</li> <li>4) VOR</li> </ol> <p><b>LR6 = abduction</b> <b>SO4 = depressor</b> in eye adduction (head tilt away from lesion)</p> <p><b>Conjugate Gaze Palsy</b></p> <ul style="list-style-type: none"> <li><b>PSP = Loss of vertical → then horizontal gaze</b> → bilateral fixed unequal eyes but reflex eye movements intact</li> <li><b>Parinaud's syndrome</b> = Involuntary upward dev of the eyes + loss of vertical agaze = pinealoma, MS, vascular</li> <li><b>One and a half syndrome</b> = horizontal gaze palsy + impaired adduction</li> </ul>															
CN 4																
CN 6																

# Cranial Nerve Examination (Lower CN – 5, 7-12)

CN 5	<p><b>Sensory division of trigeminal</b> V1 = ophthalmic (sup. orbital) V2 = mandibular (foramen rotundum)</p>	<p>Examine facial sensation [close patients' eyes]</p>	<p><b>"This is what the cotton wool/pin feels like" [Both sides of Head → cheek → jaw]</b></p> <ul style="list-style-type: none"> <li>• Is it cold/hot or sharp/dull <b>AND</b></li> <li>• did it feel the same on both sides of the face?</li> </ul> <p>Dermatomes of the head and neck</p> <p>Facial sensation V, test all three divisions on each side</p> <p>Herpes zoster distribution of the maxillary nerve</p>		
			<p><b>*Corneal reflex [Not done]</b></p> <p>Lightly touch cornea (not the conjunctiva) with cottonwool brought to the eye from side</p> <ul style="list-style-type: none"> <li>• No sensation = corneal ulceration / ACOUSTIC NERUOMA ( <b>NO CN7 TO BLINK</b>)</li> </ul>		
	<p><b>Motor division of trigeminal</b> V2 = maxillary (foramen ovale)</p>	<p><b>Muscles of mastication</b></p>	<ul style="list-style-type: none"> <li>• (clench your teeth for me + relax): Feel for <b>temporalis</b> and <b>masseter</b> muscle wasting?</li> <li>• (ask patient to bite down on wooden tongue depressor with molars): <b>muscle strength</b></li> <li>• (open your mouth – don't let me close it): <b>pterygoid muscle</b></li> <li>• <b>Jaw deviates to affected side</b></li> </ul>		
		<p><b>Jaw Jerk Or Masseter Reflex</b></p>	<ul style="list-style-type: none"> <li>• "Relax your jaw down slightly for me → just going to tap tip of your chin/jaw lightly"           <ul style="list-style-type: none"> <li>◦ → exaggerated jaw jerk = <b>UMN lesion above pons</b> [pseudobulbar palsy]</li> </ul> </li> </ul>		

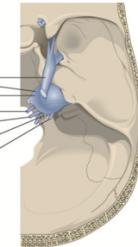


Cranial nerve V (motor): 'Clench your jaw'— feel the masseter muscles



Cranial nerve V: the jaw jerk

Cerebellopontine angle tumour.

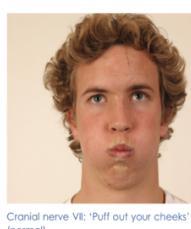


NB: schwannoma from CNVIII can compress adjacent CNV and CNVII nerves, brainstem and cerebellum

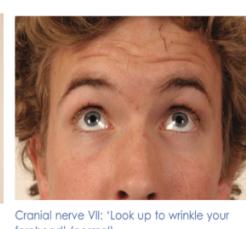
CN 7	<p><b>Facial movements</b></p> <ul style="list-style-type: none"> <li>• <b>Frontalis (temporal)</b> = "Raise your eyebrows and don't me push them down" → NO wrinkle (<b>UMN lesion – FOREHEAD SPARING</b>)</li> <li>• <b>OBICULARIS OCULI (ZYGOMATIC)</b> "Close eyes TIGHTLY as you can and don't let me open them" → <b>Bell's LMN palsy</b> upward movement of the eyeball and incomplete closure of the eyelid</li> <li>• <b>BUCCINATOR (BUCCAL)</b> "Puff cheeks and don't let me push them in" → asymmetry (<b>LMN lesion</b>)</li> <li>• <b>ZYGOMATIC MUSCLE (ZYG + BUCCAL)</b> "Smile and show me your teeth" → facial paralysis (<b>cortical lesion</b>)</li> <li>• <b>CERVICAL</b> Platysma + occipitalis</li> </ul>	
	<p><b>Q "Any change in taste"</b></p> <ul style="list-style-type: none"> <li>• <b>CN VII (chorda tympani)</b> has sensory fibres for <b>taste</b> from anterior 2/3 of tongue → fibres reach brain via <b>CN V</b></li> <li>• <b>Unilateral loss of taste:</b> middle-ear lesions involving the <b>chorda tympani (CN7)</b> or <b>lingual nerve (CNV)</b></li> </ul>	
	<p><b>Q "Any change in hearing"</b></p> <ul style="list-style-type: none"> <li>• <b>Stapedius</b> supplied by <b>VII</b> → controls stapes → hyperacusis when damaged</li> </ul>	



Bell's palsy on the right side



Cranial nerve VII: 'Puff out your cheeks' (normal)



Cranial nerve VII: 'Look up to wrinkle your forehead' (normal)



Cranial nerve VII: 'Shut your eyes tight and stop me opening them' (normal)

**Central causes** (pons, medulla, upper cervical cord) = **FOREHEAD SPARING**

- vascular lesion,
- tumour,
- syringobulbia.

**Peripheral causes**

- aneurysm, tumour,
- chronic meningitis.
- Trigeminal ganglion causes include trigeminal neuroma, meningioma fracture

Table 1. Distinguishing peripheral vs central vertigo using the HINTS examination

	Peripheral	Central
<b>Head impulse test</b>	Abnormal	Normal
<b>Nystagmus</b>	None or unidirectional	Bidirectional or vertical
<b>Test of skew</b>	No vertical skew	Vertical skew

<b>CN 8 (512Hz)</b>	<b>Inspect</b>	<ul style="list-style-type: none"> <li>Hearing aids</li> <li>Herpes zoster lesions (external acoustic meatus)</li> </ul>		
	<b>"whisper" test</b>	<ul style="list-style-type: none"> <li><b>Close your eyes and tell me if you hear a sound</b> → Scrunch fingers to test ear           <ul style="list-style-type: none"> <li>Mask opposite ear rubbing the tragus while testing other ear</li> </ul> </li> </ul>		
	<b>Vestibular Function</b>	<ul style="list-style-type: none"> <li><b>Stand up → march on spot [pathological = patient turns to side of lesion]</b></li> </ul>		
	   			
	<small>Cranial nerve VIII, Rinne's test: "Where does it sound louder?"</small> <small>Cranial nerve VIII, Weber's test: "Is the buzzing louder on one side?"</small>			
	<b>Special Tests (512Hz)</b>	<b>Normal</b>	<b>Sensorineural hearing deafness</b>	<b>Conductive deafness</b>
	<b>Rinne test</b> [place on mastoid process, behind the ear]	Air conduction > Bone conduction	Air conduction > Bone conduction (both conduction reduced equally)	Bone conduction > Air conduction
	<b>Weber's test</b> [test lateralisation]	Sound heard equally in both ears (no lateralisation)	Sound louder on side of <b>intact/unaffected ear</b>	Sound heard louder on side of <b>affected ear</b>
	<b>DDx</b>	-	<b>Unilateral</b> = schwannoma, trauma (# of petrous part of temporal bone), internal auditory artery rupture <b>Bilateral</b> = Infection (TORCH), Ménière's disease, Ab (aminoglycosides), diuretics	Wax, otitis media Paget's
	<b>Inspect</b> symmetry of soft palate			
<b>CN 9 "clean spatula"</b>	<b>Voice</b>	<ul style="list-style-type: none"> <li><b>Say "ahhhh"</b> → Uvula should be raised upright</li> <li><b>Pathology</b> = Uvula displaces <b>away</b> from side of lesion (<b>loss of innervation of levator palatini</b>)</li> </ul>		
	<b>Test cough (bovine)</b>	<b>"Cough for me"</b> <ol style="list-style-type: none"> <li>hoarseness OR bovine cough → <b>unilateral RLN lesion or vagal palsy</b></li> </ol>		
	<b>Swallow test</b>	"Skip this glass of water when I tell you" <ol style="list-style-type: none"> <li>Dysphagia of dry foods → <b>CN9 nerve lesion</b></li> <li>Dysphagia of solids and liquids + hoarseness → <b>Unilateral CNX paralysis</b></li> </ol>		
	<b>Distribution</b>	<b>Discuss how IX carries sensory fibres for taste from the posterior one-third of the tongue</b> <ol style="list-style-type: none"> <li>9<sup>th</sup> nerve → sensory fibres to <b>oropharynx</b>, the middle and inner ear and from the posterior 1/3 of the tongue (including taste fibres)           <ul style="list-style-type: none"> <li>secretory fibres to the parotid gland (PSNS)</li> </ul> </li> <li>10<sup>th</sup> nerve → sensory fibres to <b>laryngopharynx</b> <ul style="list-style-type: none"> <li>Motor innervation to muscles of the pharynx, the larynx and the palate</li> </ul> </li> </ol>		
	<b>CN 10 "wanderer"</b> <ul style="list-style-type: none"> <li><b>Gag reflex</b> (CN IX = sensory on soft palate and X = motor/vomiting component) → touch pharynx with spatula and observe reflex contraction of the soft palate</li> </ul>			
<b>CN 11</b>	<ul style="list-style-type: none"> <li><b>Trapezius:</b> "shrug your shoulders" → don't let me push them down</li> <li><b>SCM:</b> "turn your head left and push against my hand → and the other side (right SCM turns head left) → feel for SCM bulk</li> </ul> <small>(Check for winged scapula – serratus anterior)</small>		  	
<b>CN 12</b>	<p><b>"Stick your tongue out AND wiggle it around":</b></p> <ul style="list-style-type: none"> <li>Protrusion = <b>genioglossus muscle</b></li> <li><b>Deviation to affected side</b> → <b>unilateral LMN lesion</b></li> <li><b>cannot keep tongue protruded</b> → <b>Huntington's chorea</b></li> <li><b>coarse tremor of the tongue</b> → <b>Parkinson's disease</b></li> </ul> <p><b>"Press tongue into my fingers (on one side and the other)"</b></p> <ul style="list-style-type: none"> <li><b>immobile tongue</b> → <b>UMN lesion of CN XII</b></li> </ul> <p><b>Innervates ALL intrinsic muscles of tongue EXCEPT palatoglossus</b></p>		 <p>Fasciculations of the tongue in motor neurone disease</p>	

<b>Feature</b>	<b>Pseudobulbar</b> (bilateral <b>UMN</b> lesions of <b>IX, X and XII</b> )	<b>Bulbar</b> (bilateral <b>LMN</b> lesions of <b>IX, X, and XII</b> )
<b>Gag</b>	Increased or normal	Absent
<b>Tongue</b>	Spastic	Wasted, fasciculations
<b>Jaw jerk</b>	Increased	Absent or normal
<b>Speech</b>	Spastic dysarthria	Nasal
<b>Other</b>	Bilateral UMN (long tract) signs	Signs of underlying disease – eg. Limb fasciculations, normal emotions
<b>Causes</b>	Multiple sclerosis MND Brain trauma Raised ICP T2DM Vascular	MND GBS Poliomyleitis Brainstem infarction Myasthenia Gravis

### Practise point:

The tongue and jaw never lie → always point to side of lesion!

# UPPER LIMB NEURO EXAM

Today I examined Mr Smith, a 32-year-old male. On **general inspection**, the patient appeared comfortable at rest, with normal speech and no other stigmata of neurological disease. There were no objects or medical equipment around the bed of relevance."

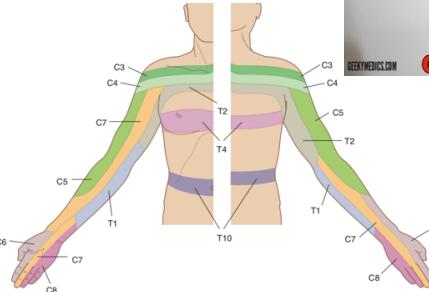
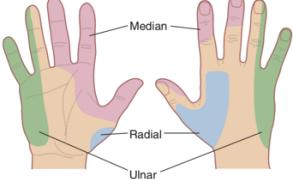
- “**Assessment of the upper limbs** revealed normal tone, power, reflexes, sensation and coordination.”
- “**In summary**, these findings are consistent with a normal upper limb neurological examination.”
- “Further assessment of the neck and cranial nerves as well as gait examination”

<b>DDx: small hand muscle wasting</b>	
➤	<b>SC lesion</b> = syringomyelia, tumour, trauma, cervical spondylosis
➤	<b>Ant horn cell</b> - MND, polio, SMA, C8 compression
➤	<b>Median / ulnar nerve palsy</b>
➤	<b>Lower branchial trunk lesion</b>
○	Thoracic outlet syndrome
○	Tumour, RT, infect
○	MS
➤	<b>Muscle</b> = DMD

<b>General</b> <i>ISWIFT</i>	<b>Sit patient on bed</b>				
	<ul style="list-style-type: none"> <li>Scars/skin changes/Symmetry (herpes zoster, shingles with segmental distribution, NFT)</li> <li>Wasting</li> <li><b>Involuntary movements</b> (i.e. jerks, dystonia, myoclonus, chorea) <math>\beta\Sigma</math></li> <li><b>Fasciculations</b> (MND, thyrotoxicosis, primary neuropathy)</li> <li>Tremors (low freq = PD, high freq = thyrotoxicosis)</li> </ul>				
<b>Tone</b>	<p>Relax your arms → just going to check the tone:</p> <ul style="list-style-type: none"> <li><b>Hold onto elbow</b> (at epicondyles) + shake hands → pronate → supinate → flex/extend elbow (repeat faster movements)</li> <li><b>Cock patient's wrist repeatedly</b> → check for repetitive movement (clonus)</li> </ul>				
			<ul style="list-style-type: none"> <li><b>Paratonia</b> = increased tone = involuntary resistance against passive movement             <ul style="list-style-type: none"> <li>Assoc. Dementia</li> </ul> </li> <li><b>Hypotonia</b> = Slow Opening of fist or wrist drop</li> <li><b>Hypertonia #1: Spasticity</b> (unidirectional &amp; velocity dependent) → stroke (UMN lesion)             <ul style="list-style-type: none"> <li>Spastic/fast movement in one direction ONLY [catch it!]</li> </ul> </li> <li><b>Hypertonia #2: cog-wheel Rigidity</b> (bidirectional &amp; velocity INdependent) → Parkinson's disease             <ul style="list-style-type: none"> <li>Stiffness in BOTH directions</li> <li><b>Clonus</b> (&gt;5 abnormal repeated)</li> </ul> </li> </ul>		
<b>Power</b>	<b>Movement</b>	<b>Muscles</b>	<b>Nerve</b>	<b>Talk</b>	
	<b>Shoulder</b>	Abduction	Trapezius	CNXI	Shrug your shoulders for me
			Deltoid	Axillary (C5,C6)	Make a chicken wing for me and don't let me push down
		Adduction	Supraspinatus	Suprascapular	bring your arms up
	<b>Elbow</b>	Flexion	Pec. major & latissimus dorsi	Thoracodorsal (C6,8)	Brings elbows close to side and flap it like a wing
		Extension	Biceps brachii	MSC (C5,6)	Brings arms up like a boxer → don't let me pull you arm away from you [hold shoulder on same side!]
	<b>Wrist</b>	Flexion	Triceps brachii	Radial (C7, 8)	Brings arms up like a boxer → don't let me pull your arm towards you [hold shoulder on opp. side!]
		Extension	Flexor carpi ulnaris & radialis	Ulnar (ulnar half) Median (radial half)	“cock your wrists downwards + don't let me pull them up”
	<b>Finger</b>	Flexion	Extensor carpi group (EDC)	Radial (C7, C8)	“cock your wrists upwards + don't let me pull them down”
		Extension	FDP, FCU, FCR	Radial (C7, C8)	Bend your fingers 90° straight down and don't let me bring them up”
		Abduction	Extensor digitorum	Radial (C7, C8)	Hold your fingers out straight and don't let me push them down”
	<b>Thumb</b>	Abduction	Dorsal interossei	Ulnar (C8, T1)	“Splay your fingers outwards and don't let me push them together”
		Adduction	Palmar interossei	Ulnar (C8, T1)	“Use tissue paper”
		Abduction	Abductor pollicis brevis (APB)	Median nerve (T1)	“Point your thumbs to the ceiling and don't let me push them down”
	Extension	Extensor pollicis longus (EPL)	Radial	Stretch thumb	
<b>MRC MUSCLE POWER SCALE</b>					
<b>Score</b>	<b>Description</b>				
<b>0</b>	No contraction				
<b>1</b>	Flicker contraction				
<b>2</b>	Active movement (while lying down)				
<b>3</b>	Active movement (defy gravity)				
<b>4</b>	Active movement (against resistance)				
<b>5</b>	Normal				
<b>Reflexes</b>	<b>Lie patient on bed</b>				
	<p>“I'm now going to test your reflexes with a light tap using this hammer. Just relax for me as much as possible”</p>				
	<ol style="list-style-type: none"> <li>Biceps (C5, C6)</li> <li>Brachioradialis/Supinator jerk (C6)</li> <li>Finger jerk (C8) = hold weight of hand</li> <li>Triceps (C7)</li> <li>Hoffman's reflex = hold middle finger at DIP → flick nail of middle finger [+ve sign = index/thumb movement]</li> </ol>				
	<p><i>If reflex is not found → clench teeth “Jendrissek”</i></p>				
	<p><b>a</b> Testing power—shoulder (test each arm separately)</p>				
	<p><b>b</b> Testing power—elbow flexion: “Stop me straightening your elbow” (test each arm separately)</p>				
	<p><b>c</b> Testing power—wrist extension: “Stop me bending your wrist”</p>				
	<p><b>d</b> Testing power—finger abduction: “Stop me pushing your fingers together”</p>				
	<p><b>e</b> The biceps jerk examination</p>				
	<p><b>f</b> The triceps jerk examination</p>				
	<p><b>g</b> The finger jerk examination</p>				

<b>Coordination</b>	<b>1. Finger nose test "dysmetria"</b>	• Large H (touch my finger /your nose)	intention tremor, past pointing = <b>posterior lobe cerebellar disease</b>	  <p>Finger-nose test: 'Touch your nose with your forefinger and then reach out and touch my finger'</p> <p>Testing for dysdiadochokinesis in the upper limbs: 'Turn your hand over, backwards and forwards on the other one, as quickly and smoothly as you can'</p>
	<b>2. Dysdiadochokinesis</b> [fast repetitive movement]	• Tap hand on knee as fast as possible [Both sides] →	slow clumsy movement = <b>ipsilateral cerebellar disease</b>	
	<b>3. Rebound</b>	• Hold hands out palm facing up → and Lift arm up rapidly to my eye • Push up and down each arm → should return to normal position	hypotonia (delay in stopping arms) = <b>cerebellar disease</b>	
	<b>4. Pronator drift</b> [contralateral pyramidal tract lesion]	• "Close your eyes" → observe any DRIFT • Mild upper limb weakness and spasticity	• CST + internal capsule • UMN weakness (from stroke) = spasticity • Ipsilateral cerebellar disease	

<b>Sensation</b> [Close eyes!] Correlate with dermatome!	<ul style="list-style-type: none"> <li>C5 = shoulder tip &amp; outer part of upper arm</li> <li>C6 = lateral aspect of forearm and thumb</li> <li>C7 = middle finger</li> <li>C8= little finger</li> <li>T1 = medial aspect of upper arm and elbow</li> </ul>			
	<b>Spinothalamic pathway</b> <ul style="list-style-type: none"> <li>Pain (pinprick) → C7 = middle finger</li> <li>Cold (tuning fork) → distal → peripheral (compare both sides)</li> </ul>			
	<b>Dorsal column pathway</b> <ul style="list-style-type: none"> <li>Vibration (128 Hz tuning fork) → bony points (thumb → wrist → elbow)           <ul style="list-style-type: none"> <li>Tell me when the vibration stops (control with finger on fork)</li> </ul> </li> <li>Proprioception (play a game – this is down/up – close eyes → is this "up or down")           <ul style="list-style-type: none"> <li>Hold thumb on either side + close eyes → move thumb side-to-side</li> </ul> </li> </ul>			
	<b>Mixed</b> <ul style="list-style-type: none"> <li>Light touch (does NOT discern between spinothalamic and dorsal column loss)</li> </ul>			

#### TYPES OF MOTOR NEURONE LESIONS

	<b>Signs of UMN [Hyper – CENTRAL]</b>	<b>Signs of LMN lesions [Hypo – PERIPHERAL]</b>
<b>DDx:</b>	<ul style="list-style-type: none"> <li><b>Brain:</b> MS, Amyloidosis/Infiltrative, Trauma</li> <li><b>SC:</b> Transverse myelitis, Cauda Equina syndrome</li> <li><b>After 6 weeks post-surgery</b></li> </ul>	<ul style="list-style-type: none"> <li><b>Anterior horn:</b> MND, Poliomyositis</li> <li><b>Peripheral nerves:</b> Peripheral Nerve Injury, GBS</li> <li><b>NMJ:</b> Myasthenia Gravis</li> <li><b>Muscles:</b> DMD</li> </ul>
<b>Inspection</b>	<ul style="list-style-type: none"> <li>Absent fasciculations</li> <li>Quadriplegia, hemiplegia (contralateral), paraplegia</li> </ul>	<ul style="list-style-type: none"> <li><b>Fasciculations</b> (esp. tongue, triceps, calf, 1<sup>st</sup> dorsal interosseous)</li> <li>Denervation → loss of contraction → <b>muscle wasting</b></li> </ul>
<b>Pronator Drift</b>	<b>Present Pronator Drift</b>	Some drift if weak or de-afferented but not pronator
<b>Muscle Tone</b>	<b>Hypertonia+ clonus</b> (i.e. clasp-knife reflex +repetitive)	<b>Hypotonia</b>
<b>Contraction</b>	<b>Spasticity</b>	<b>Flaccidity</b>
<b>Reflexes</b>	Exaggerated or brisk ( <b>hyper-reflexia</b> ) Hoffman reflex	Absent (or hyporeflexia)
<b>Power</b>	<p><b>"pyramidal" pattern of weakness</b> (i.e. upper limb extensors <b>weaker</b> than flexors and lower limb flexors <b>weaker</b> than extensors)</p> <ul style="list-style-type: none"> <li>Leg affected: L1 or above</li> <li>Arm affected: C3 or above</li> <li>Face affected: pons or above</li> <li><b>Diplopia:</b> midbrain or above</li> </ul>	<p><b>Variable weakness</b></p> <ul style="list-style-type: none"> <li>proximal weakness in muscle disease</li> <li>distal weakness in peripheral neuropathy</li> <li><b>Focal pattern of weakness</b> = damaged nerves = correspond to weakness in innervated muscles</li> </ul>
<b>Plantar reflexes</b>	+ve Babinski response (toes point up)	-ve Babinski response (toes point DOWN - plantar)
<b>EMG</b>	NORMAL nerve conduction → decreased interference pattern and firing rate	Abnormal nerve conduction → large motor units → fasciculations

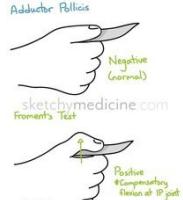
#### TYPES OF UPPER LIMB NERVE LESIONS

Radial Nerve (C5-C8)	Median Nerve Lesion (C6-T1)	Ulnar lesion (C8-T1)
<ul style="list-style-type: none"> <li>Triceps,</li> <li>Brachioradialis,</li> <li>Extensor Muscles Of Hand</li> <li><b>Lesion</b> = Wrist Drop [cannot straighten finger]</li> </ul>	<ul style="list-style-type: none"> <li>Ant. forearm muscles + LOAF exc. FCU and ulnar ½ of FDP</li> <li><b>Lesion at wrist</b> → <b>pen-touching test</b> for APB</li> <li><b>Lesion at cubital fossa</b> → ochsner's clasping test (loss of flexor digitorum)</li> <li>Cannot oppose thumb or grab objects</li> </ul>	<ul style="list-style-type: none"> <li>Supplies all muscles of small hand except LOAF</li> <li><b>Froment's signs</b> → Loss of <b>thumb adductor</b> → thumb flexes when holding card</li> <li><b>Clawing</b> = MCP hyperextension + IP flexion → <b>Diff dx</b> = brachial plexus lesion (C8-T1), RA, or neurological disease (e.g. polio, syringomyelia)</li> </ul>

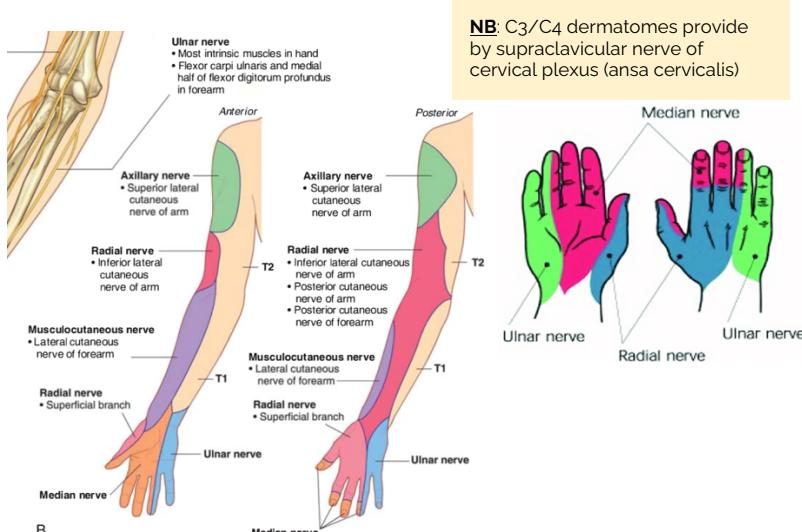
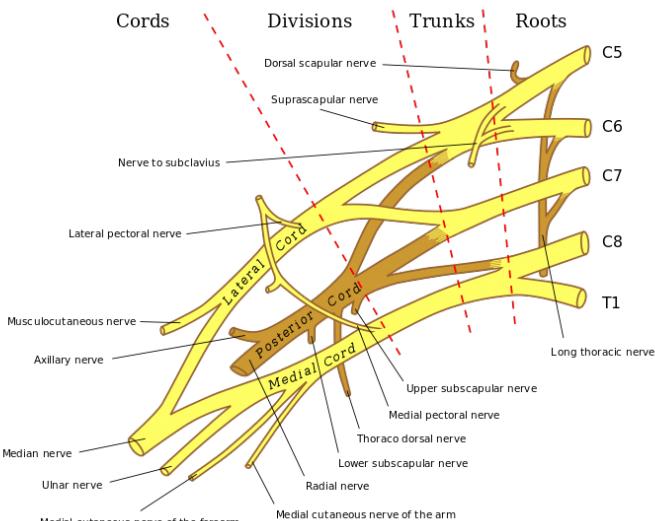


Brachial plexus cords, nerves and their supplied muscles		
Cords	Nerves formed	Muscles supplied
Lateral	Musculocutaneous, median	Biceps, pronator teres, flexor carpi radialis
Medial	Median and ulnar	Hand muscles
Posterior	Axillary and radial	Deltoid, triceps and forearm extensors

Nerve roots	Trunks	Muscles supplied
C5 and 6	Upper	Shoulder (especially biceps and deltoid)
C7	Middle	Triceps and some forearm muscles
C8 and T1	Lower	Hand and some forearm muscles



# BRACHIAL PLEXUS



Nerves	Root	Location	Innervates	Damage Occurs	Pathology
Axillary	C5-C6	Posterior cord Quadrangular space	<ul style="list-style-type: none"> <li>Deltoid</li> <li>Teres minor</li> </ul>	Surgical neck of humerus fracture or dislocated shoulder "sergeant's patch"	<b>Erb's Palsy" =</b> IR, extended arm + arm length discrepancy <ul style="list-style-type: none"> <li>Loss of Flex/ext   Abd</li> <li>Loss of External rotation of arm</li> <li>Sensation loss = C5 dermatome (sup. lateral cutaneous nerve of arm)</li> </ul>
Musculo-cutaneous	C5-C7	<ul style="list-style-type: none"> <li>Terminal branch of lateral cord → from inferior pec. minor</li> <li>Pierces coracobrachialis</li> </ul>	<ul style="list-style-type: none"> <li>Biceps brachii</li> <li>Coracobrachialis</li> <li>Brachialis</li> </ul>	Rare (as between brachialis & biceps muscles → possible compartment syndrome)	<b>Forearm flexion and supination</b> <ul style="list-style-type: none"> <li>BUT can still flex elbow via brachioradialis with assistance from pronator teres</li> </ul>
Radial Runs with profunda brachii artery	C5- T1	<ul style="list-style-type: none"> <li>Posterior cord</li> <li>Triangular space</li> <li>Along radial groove → Between brachioradialis (medial) &amp; brachialis (lateral)</li> </ul>	Posterior compartment <ul style="list-style-type: none"> <li>Supinator</li> <li>Brachioradialis (flexion ONLY)</li> <li>Triceps (long and lateral)</li> </ul>	Spiral fracture of humerus	<b>"Saturday Night Palsy" = compressed nerve while asleep</b> <ul style="list-style-type: none"> <li>Forearm extension loss</li> <li>finger extension loss (PIN)</li> </ul>
				Radial groove injury "extension injury"	Entire triceps is <u>NOT</u> paralysed since innervated <u>BEFORE</u> radial groove → can still extend forearm <ul style="list-style-type: none"> <li>Wrist drop = reduced extension</li> </ul>
Median	C5- T1	<ul style="list-style-type: none"> <li>Medial &amp; lateral cord</li> <li>Middle of cubital fossa</li> <li>Medial to brachial artery</li> <li>Anterior interosseous nerve = thumb opposition</li> </ul>	Anterior compartment <ul style="list-style-type: none"> <li>Anterior forearm muscles esp. FCR (exc. FCU and ½ FDS)</li> <li>Pronator teres &amp; pronator quadratus (pronation)</li> <li>LOAF</li> </ul>	Injury at <u>elbow</u> = proximal nerve entrapment <ul style="list-style-type: none"> <li>Most common supracondylar syndrome</li> </ul>	Hand of benediction/waiter's tip <ul style="list-style-type: none"> <li>Loss of PIP flexion (1-5)</li> <li>Loss of DIP flexion (2-3) ONLY</li> <li>FDP (medial part) innervated by ulnar nerve → allows DIP flexion of (4-5)</li> </ul>
				Carpal tunnel syndrome = distal nerve entrapment	Loss of LOAF → CANNOT <ul style="list-style-type: none"> <li>flex at CMC of 1<sup>st</sup> - 3<sup>rd</sup> digits</li> <li>oppose thumb</li> </ul> BUT can still <b>flex at thumb at CMC</b> due to FPL innervation
Ulnar	C8, T1	Medial & lateral cord (under FCU)	Intrinsic hand muscles (exc. LOAF) <ul style="list-style-type: none"> <li>FCU and ½ FDS</li> </ul>	Elbow fracture <ul style="list-style-type: none"> <li>medial epicondyle of humerus</li> <li>flexion-type / post-op supracondylar #</li> </ul>	Claw hand → loss of intrinsic hand muscles (FDP loss) <ul style="list-style-type: none"> <li><b>Ulnar paradox = more proximal = less clawing</b></li> </ul>

\*compartment syndrome (pain = most sensitive symptom) = ↑↑ pressure within fascia → ischaemia & necrosis & 5 P's



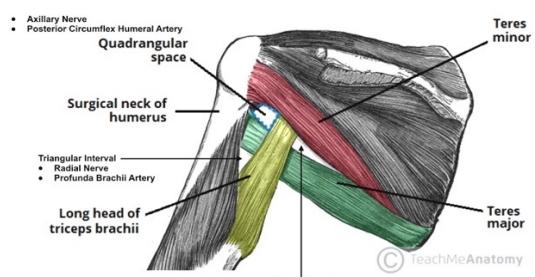
A) median nerve

B) ulnar nerve

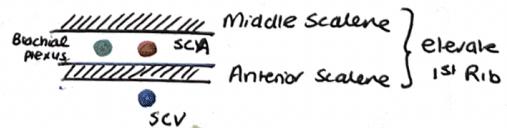
C) radial nerve



## The Quadrangular and Triangular Spaces; The Triangular Interval



Nb: Posterior scalene elevated 2<sup>nd</sup> rib AND all scalene muscles involved in lateral neck flexion

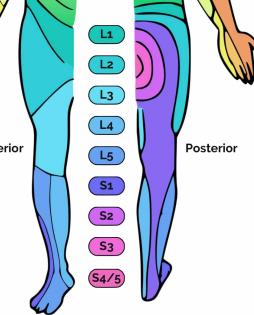


## LOWER LIMB NEURO EXAM

Today I performed a lower limb neurological examination on Mr Smith, a 32-year-old male. On general inspection, the patient appeared comfortable at rest, with a normal **gait** and no abnormal **posturing, muscle wasting** or **fasciculations**. **Tone** was normal throughout and **power** was 5/5 in all muscle groups bilaterally. **Reflexes** were normal and present at the knees, ankles and plantars bilaterally. **Coordination** was intact with **normal sensations** felt. This was a normal lower limb neurological examination. To complete the examination, I would like to examine the lumbar spine and review any available CT/MRI scans".

<b>Introduction</b>	<ol style="list-style-type: none"> <li>Do you mind if we examine your lower limbs. It's going to involve me moving your legs, watching you walk a bit"</li> <li>For this examination, I'll need you to <b>roll up your pants, remove socks and shoes</b></li> <li>Are you in any pain at all?</li> </ol>																																																													
<b>Gait</b>	<p><b>Test proximal leg weakness:</b></p> <ul style="list-style-type: none"> <li>Arms crossing chest</li> <li>Stand up from chair without using hands</li> </ul> <p><b>Romberg's test</b> [assess proprioception – ataxia = loss of balance?]</p> <ul style="list-style-type: none"> <li>Stand with your eyes closed"</li> </ul> <p><b>Conduct Gait</b></p> <ul style="list-style-type: none"> <li><b>Observe normal cadence:</b> posture, speed, symmetry, balance and arm swing, stride length, abnormal movements (e.g. writhing) <ul style="list-style-type: none"> <li>?POOR TO command</li> <li>on heels (dorsiflex - L5)</li> <li>on tip-toes (plantarflex - S1)</li> <li>heel-to-toe walk on tightrope (tandem gait) = cerebellum, PD</li> <li>Squat (L4)</li> </ul> </li> <li><b>High-stepping unsmooth gait</b> = foot drop</li> </ul>	<p><b>Romberg test.</b>  (a) 'Stand with your feet together.' (b) 'Now shut your eyes. I won't let you fall.'</p>																																																												
<b>General (SWIFTI)</b>	<p>Look from end of the bed: (raise bed to waist level)</p> <ul style="list-style-type: none"> <li><b>Scars/skin lesions</b> [hair from interdigts to arm = <b>SNS intact</b>] / <b>Symmetry</b> (shingles (zoster) with segmental distribution, NFT)</li> <li><b>Wasting</b> [vastus medialis, symmetry]</li> <li><b>Involuntary movements</b> (i.e. <i>jerks, dystonia/rigidity, myoclonus, chorea</i>)</li> <li><b>Fasciculations/twitching</b> [UMN lesions, MND, thyrotoxicosis, primary neuropathy]   <b>Feet arched or flat</b></li> <li><b>Tremors</b> (low freq = PD, high freq = thyrotoxicosis)</li> <li><b>Check Vitals:</b> what is the BP, pulse, RR, Temp, O<sub>2</sub> <ul style="list-style-type: none"> <li>AF = stroke (embolic)</li> <li>Febrile = Guillan Barre syndrome</li> </ul> </li> </ul>	<p><b>Differential Dx of Foot Drop</b></p> <ul style="list-style-type: none"> <li>Common peroneal nerve palsy</li> <li>Sciatic nerve palsy</li> <li>Lumbosacral plexus lesion</li> <li>L4, L5 root lesion</li> <li>MND</li> <li>Stroke (ACA or lacunar syndrome)</li> </ul>																																																												
<b>Tone</b>	<ul style="list-style-type: none"> <li><b>Palpate for tenderness</b></li> <li><b>Leg roll</b> – on knee cap → feel for looseness</li> <li><b>Leg lift</b> – under popliteal fossa (lift up at sufficient height and drop) <ul style="list-style-type: none"> <li>Any tense/rigid leg = ?UMN lesion</li> </ul> </li> <li><b>Ankle clonus with knee bent</b> [lift ankle and hold base of foot → single quick movement → &gt;5 abnormal]</li> </ul>	<p>(a) Foot drop: the patient lifts the affected leg high in the air to prevent the foot scraping on the ground. (b) Shoe supports to prevent foot drop</p>																																																												
<b>Power (MRC grade)</b>	<table border="1"> <thead> <tr> <th></th> <th>Movement</th> <th>Muscles</th> <th>Nerve</th> <th>Nerve</th> <th>Talk</th> </tr> </thead> <tbody> <tr> <td rowspan="4"><b>Hip</b></td> <td>Flexion</td> <td>Psoas &amp; Iliacus</td> <td><b>L2/L3</b></td> <td>Femoral</td> <td>Lift leg up and stop me from pushing it down</td> </tr> <tr> <td>Extension</td> <td>Gluteus Maximus</td> <td><b>L5-S2</b></td> <td>Inferior gluteal</td> <td>Push against the bed for me</td> </tr> <tr> <td>Adduction</td> <td>Adductor longus, brevis and magnus</td> <td><b>L2-4</b></td> <td>Obturator</td> <td>Push your legs apart [hold both <b>lateral</b> thighs]</td> </tr> <tr> <td>Abduction</td> <td>Gluteus medius &amp; minimus, sartorius and tensor fascia lata</td> <td><b>L4-S1</b></td> <td>Superior gluteal</td> <td>Push legs together for me [hold both <b>medial</b> thighs]</td> </tr> <tr> <td rowspan="2"><b>Knee</b></td> <td>Extension</td> <td>Quadricep femoris (3x stronger)</td> <td><b>L3, 4</b></td> <td>Femoral</td> <td>Kick out against me and straighten your leg [hold under knee to bend it and ask to straighten]</td> </tr> <tr> <td>Flexion</td> <td>Hamstring (biceps, semimembranosus, semitendinosus)</td> <td><b>L5, S1</b></td> <td>Tibial part of sciatic</td> <td>Bend your knee &amp; stop me from straightening your leg</td> </tr> <tr> <td rowspan="2"><b>Ankle</b></td> <td>Dorsiflexion</td> <td>Tibialis anterior, extensor digitorum longus/brevis</td> <td><b>L4/5</b></td> <td>Common fibular</td> <td>Bring your toes up towards your face and stop me from them down [<b>Hold down ankle</b>]</td> </tr> <tr> <td>Plantarflexion</td> <td>Gastrocnemius, soleus, plantaris</td> <td><b>S1/S2</b></td> <td>Tibia</td> <td>Push down against my hand [<b>palm on feet</b>]</td> </tr> <tr> <td rowspan="2"><b>Tarsal Joint</b></td> <td>Inversion</td> <td>Peroneus longus &amp; brevis &amp; extensor digitorum longus</td> <td><b>L5, S1</b></td> <td>Tibial</td> <td>Push your foot IN against my hand</td> </tr> <tr> <td>Eversion</td> <td>Tibialis posterior, gastrocnemius, hallucis longus</td> <td><b>L5, S1</b></td> <td>Superficial fibular</td> <td>Push your foot out against my hand</td> </tr> </tbody> </table>		Movement	Muscles	Nerve	Nerve	Talk	<b>Hip</b>	Flexion	Psoas & Iliacus	<b>L2/L3</b>	Femoral	Lift leg up and stop me from pushing it down	Extension	Gluteus Maximus	<b>L5-S2</b>	Inferior gluteal	Push against the bed for me	Adduction	Adductor longus, brevis and magnus	<b>L2-4</b>	Obturator	Push your legs apart [hold both <b>lateral</b> thighs]	Abduction	Gluteus medius & minimus, sartorius and tensor fascia lata	<b>L4-S1</b>	Superior gluteal	Push legs together for me [hold both <b>medial</b> thighs]	<b>Knee</b>	Extension	Quadricep femoris (3x stronger)	<b>L3, 4</b>	Femoral	Kick out against me and straighten your leg [hold under knee to bend it and ask to straighten]	Flexion	Hamstring (biceps, semimembranosus, semitendinosus)	<b>L5, S1</b>	Tibial part of sciatic	Bend your knee & stop me from straightening your leg	<b>Ankle</b>	Dorsiflexion	Tibialis anterior, extensor digitorum longus/brevis	<b>L4/5</b>	Common fibular	Bring your toes up towards your face and stop me from them down [ <b>Hold down ankle</b> ]	Plantarflexion	Gastrocnemius, soleus, plantaris	<b>S1/S2</b>	Tibia	Push down against my hand [ <b>palm on feet</b> ]	<b>Tarsal Joint</b>	Inversion	Peroneus longus & brevis & extensor digitorum longus	<b>L5, S1</b>	Tibial	Push your foot IN against my hand	Eversion	Tibialis posterior, gastrocnemius, hallucis longus	<b>L5, S1</b>	Superficial fibular	Push your foot out against my hand	
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**TEST POWER** - Provide resistance in middle thigh / leg / foot = avoid any mechanical advantage

<b>Reflexes</b>	<p>"I'm going to test your reflexes on with a tendon hammer. Let your legs go floppy for me"</p> <ul style="list-style-type: none"> <li>• <b>knee jerk</b> (L3/4 – Femoral) → you have to flex knee with left hand (hold underneath)</li> <li>• <b>ankle jerk</b> (L5/S1 – sciatic) – bend your knee and bring it to the side</li> <li>• <b>plantar reflex</b> - Babinski (S1) → use fingernail or tip of tendon hammer → upgoing toes = ? UMN lesion</li> </ul> <p><b>Jendrassik maneuver</b> = clench teeth to reinforce reflex</p>	  <p>The knee jerk with reinforcement: 'Grip your fingers and pull your hands apart'</p> <p>The ankle jerk (first method, see also p 456): the examiner reflexes the foot slightly to stretch the tendon</p>																								
<b>Coordination</b>	<ol style="list-style-type: none"> <li>1. <b>Heel-shin test</b> <ul style="list-style-type: none"> <li>o Put your ankle on the opposite knee</li> <li>o Lift it off and go back to your knee and keep going in a circle</li> <li>o Repeat on other leg</li> <li>o <b>heel wobbles (cerebellar disease) or lower limb weakness</b></li> </ul> </li> <li>2. <b>Toe-finger test</b> → intention tremor (when foot lifted)</li> <li>3. <b>Foot-tapping test</b> <b>[each side separately]</b> <ul style="list-style-type: none"> <li>o Tap your feet against my hand as quickly as possible</li> <li>o loss of rhythmicity (when tapping foot on your hand)</li> </ul> </li> </ol>	  <p>The heel-shin test: 'Run your heel down your shin smoothly and quickly'</p> <p>Above knee = L2, Knee = L3, Great toe = L4, Lateral foot = L5, Heel S1, Up calf = S2</p>																								
<b>Sensation</b> <b>[Close eyes!]</b> Correlate with dermatome!	<p><b>Close your eyes [distal → proximal] → Complete both sides</b></p> <table border="1" data-bbox="287 743 1505 916"> <tr> <td data-bbox="287 743 462 833"> <b>Spinothalamic</b> </td><td data-bbox="462 743 1505 833"> <ol style="list-style-type: none"> <li>1. <b>Pin prick</b> (show pin 1<sup>st</sup>) = check symmetry and from distal to proximal <ul style="list-style-type: none"> <li>a. TELL me when it becomes normal/sharp</li> </ul> </li> <li>2. <b>Temp</b> = check symmetry from distal to proximal [small gradient normal since distal areas colder – smaller temp. diff] → 3-4 locations</li> </ol> </td></tr> <tr> <td data-bbox="287 833 462 916"> <b>Dorsal columns</b> </td><td data-bbox="462 833 1505 916"> <ol style="list-style-type: none"> <li>3. <b>Vibration (128Hz)</b> → 1<sup>st</sup> on wrist/sternum → bony prominence on <b>big toe</b> → <b>patella</b> → <b>ASIS</b></li> <li>4. <b>Proprioception (joint sensation)</b> → "Play a game" → hold distal phalanx by its side "tell me if your toe is moving or down"</li> </ol> </td></tr> </table> <p><b>5. Why not do fine touch?</b> Light touch activates the 2 sensory pathways and hence does not allow me to discriminate which pathway could be the problem</p>		<b>Spinothalamic</b>	<ol style="list-style-type: none"> <li>1. <b>Pin prick</b> (show pin 1<sup>st</sup>) = check symmetry and from distal to proximal <ul style="list-style-type: none"> <li>a. TELL me when it becomes normal/sharp</li> </ul> </li> <li>2. <b>Temp</b> = check symmetry from distal to proximal [small gradient normal since distal areas colder – smaller temp. diff] → 3-4 locations</li> </ol>	<b>Dorsal columns</b>	<ol style="list-style-type: none"> <li>3. <b>Vibration (128Hz)</b> → 1<sup>st</sup> on wrist/sternum → bony prominence on <b>big toe</b> → <b>patella</b> → <b>ASIS</b></li> <li>4. <b>Proprioception (joint sensation)</b> → "Play a game" → hold distal phalanx by its side "tell me if your toe is moving or down"</li> </ol>																				
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 <p>Sensory distribution of the common peroneal nerve (compression at the fibular neck)</p>	<p><b>a</b> Position sense: 'Shut your eyes and tell me whether I have moved your toe up or down'</p> <p><b>c</b> <b>d</b></p>	<p><b>Testing for Nerve Root Compromise</b></p> <table border="1" data-bbox="954 1012 1505 1522"> <thead> <tr> <th data-bbox="954 1012 1049 1035">Nerve Root</th> <th data-bbox="1049 1012 1208 1035">L4</th> <th data-bbox="1208 1012 1367 1035">L5</th> <th data-bbox="1367 1012 1505 1035">S1</th> </tr> </thead> <tbody> <tr> <td data-bbox="954 1035 1049 1215">Pain</td> <td data-bbox="1049 1035 1208 1215"></td> <td data-bbox="1208 1035 1367 1215"></td> <td data-bbox="1367 1035 1505 1215"></td> </tr> <tr> <td data-bbox="954 1215 1049 1394">Numbness</td> <td data-bbox="1049 1215 1208 1394"></td> <td data-bbox="1208 1215 1367 1394"></td> <td data-bbox="1367 1215 1505 1394"></td> </tr> <tr> <td data-bbox="954 1394 1049 1439">Motor weakness</td> <td data-bbox="1049 1394 1208 1439">Extension of quadriceps</td> <td data-bbox="1208 1394 1367 1439">Dorsiflexion of great toe and foot</td> <td data-bbox="1367 1394 1505 1439">Plantar flexion of great toe and foot</td> </tr> <tr> <td data-bbox="954 1439 1049 1462">Screening exam</td> <td data-bbox="1049 1439 1208 1462">Squat and rise</td> <td data-bbox="1208 1439 1367 1462">Heel walking</td> <td data-bbox="1367 1439 1505 1462">Walking on toes</td> </tr> <tr> <td data-bbox="954 1462 1049 1484">Reflexes</td> <td data-bbox="1049 1462 1208 1484">Knee jerk diminished</td> <td data-bbox="1208 1462 1367 1484">None reliable</td> <td data-bbox="1367 1462 1505 1484">Ankle jerk diminished</td> </tr> </tbody> </table>	Nerve Root	L4	L5	S1	Pain				Numbness				Motor weakness	Extension of quadriceps	Dorsiflexion of great toe and foot	Plantar flexion of great toe and foot	Screening exam	Squat and rise	Heel walking	Walking on toes	Reflexes	Knee jerk diminished	None reliable	Ankle jerk diminished
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 <p>Distribution of the lateral cutaneous nerve of the thigh</p> <p>Sensory distribution of the femoral nerve</p>	 <p>Sensory distribution of the common peroneal nerve (compression at the fibular neck)</p> <p>Sensory distribution of the sciatic nerve</p>	<p><b>Lateral cutaneous nerve (L2, L3)</b></p> <p>likely entrapment between inguinal ligament and ASIS → <b>meralgia paraesthesia</b></p> <p>➤ <b>Anterior thigh</b></p> <p>➤ Assoc. w/ long periods of sitting, obese, tight belts</p> <p>➤ Assoc. w/ DM, pregnancy, trauma</p> <p><b>Femoral nerve (L2, L3, L4)</b></p> <p><b>Weakness:</b></p> <ul style="list-style-type: none"> <li>• knee extension</li> </ul> <p><b>Reflexes:</b></p> <ul style="list-style-type: none"> <li>• absent knee jerk reflex</li> </ul> <p><b>Common peroneal (L4-S1)</b></p> <p><b>weakness of:</b></p> <ul style="list-style-type: none"> <li>• ankle dorsiflexion</li> <li>• ankle eversion</li> <li>• toe extension</li> </ul> <p><b>Reflexes:</b></p> <p>➤ present</p> <p><b>Sciatic nerve (L4-S2)</b></p> <ul style="list-style-type: none"> <li>• <b>Loss of power below knee</b></li> <li>• <b>Weak knee flexion</b> → foot drop (plantar-flexed foot)</li> <li>• <b>Absent ankle jerk</b> and plantar response (but knee jerk intact)</li> </ul>																								

## Contrast peroneal nerve lesion vs. L5 radiculopathy

	Common peroneal nerve (L4, L5, S1)	L5 radiculopathy
Location of weakness	weakness of: <ul style="list-style-type: none"> <li>• ankle dorsiflexion</li> <li>• ankle eversion</li> <li>• toe extension</li> </ul>	weakness of: <ul style="list-style-type: none"> <li>• ankle dorsiflexion</li> <li>• ankle eversion</li> <li>• toe extension</li> </ul>
Sensory loss	Lateral aspect of dorsum of foot	L5 distribution
Reflexes	Yes	Lost
Key differential		<ul style="list-style-type: none"> <li>• <b>weakness of ankle inversion</b></li> <li>• <b>weakness of knee flexion</b></li> </ul> <p>[since tibialis posterior – ankle inverter supplied by tibial nerve - supplied by L5]</p>

## DIFFERENT TYPES OF DIZZINESS (NB: Vertigo is a subtype of dizziness)

	Vertigo	Presyncope	Disequilibrium	Light-headed						
System affected	Vestibular	Cardiovascular	Neurological	Psychological						
Sensation type	Spinning or motion sensation Head spinning → worse on turning head	Near fainting	Unsteady/ imbalance in lower limbs	Non-specific → woozy disconnected from environment						
Assoc.	<ul style="list-style-type: none"> <li>• No LOC + headache</li> <li>• N + V</li> <li>• pallor, sweating</li> </ul>	Tunnel vision Still standing	Unsteady gait	<b>postural hypertension</b>						
Timing	Episode / continuous	Episodic (few secs) → relief on lying down	Continuous							
DDx	<table border="1"> <tr> <td>Intermittent vertigo</td> <td>Constant vertigo</td> </tr> <tr> <td>Hearing normal</td> <td>BPPV = (Lying in bed)</td> </tr> <tr> <td>Hearing loss</td> <td>Ménière's disease (&gt;50 y.o.) + tinnitus</td> </tr> </table> <p>DDx:</p> <ul style="list-style-type: none"> <li>• <b>Stroke</b> (&gt; 70 y.o.)</li> <li>• <b>Vestibular migraine</b></li> </ul>	Intermittent vertigo	Constant vertigo	Hearing normal	BPPV = (Lying in bed)	Hearing loss	Ménière's disease (>50 y.o.) + tinnitus	<ul style="list-style-type: none"> <li>• Dehydration</li> <li>• Anaemia</li> <li>• Arrhythmia</li> <li>• Hypoglycemia</li> </ul>	<ul style="list-style-type: none"> <li>• Peripheral neuropathy</li> <li>• Vision loss</li> </ul>	<ul style="list-style-type: none"> <li>• Anti-hypertensives</li> <li>• Anxiety</li> <li>• Hyperventilation</li> </ul>
Intermittent vertigo	Constant vertigo									
Hearing normal	BPPV = (Lying in bed)									
Hearing loss	Ménière's disease (>50 y.o.) + tinnitus									

## Medical terms for Involuntary Movements:

Akathisia	Motor restlessness; constant semipurposeful movements of the arms and legs → <b>INNER RESTLESSNESS (CLUSTER HEADACHE)</b>	
Asterixis	Sudden loss of muscle tone in tonic contraction of an outstretched limb → CO <sub>2</sub> retention, hepatic disease	
Athetosis	Wriggling, slow sinuous movements, especially of the hands and wrists	
Chorea	Jerky small rapid movements → transformed into voluntary movement to scratch head [ABNORMAL POSTURE] → <b>HUNTINGTONS</b>	
Dyskinesia	Purposeless / continuous movements, → face and mouth (due to Rx with major tranquillisers for psychotic illness)	
Dystonia	Sustained contractions of agonist / antagonist muscles → <b>bizarre postures</b>	
Hemiballismus	Exaggerated form of chorea → unilateral →: wild flinging movements that can injure patient (or bystanders) → <b>LESION TO SUBTHALAMIC NUCLEUS</b>	
Myoclonic jerk	Involuntary/purposeless jerking → affects <b>ALL LIMBS → encephalitis, dementia, tumours</b>	
Myokymia	repeated contraction of a small muscle group; often involves the orbicularis oculi muscles	
Tic	repetitive irresistible movement that is <b>purposeful or semipurposeful [preceded by action to do specific task]</b>	
Tremor (Essential)	rhythmic alternating movement at <b>REST</b>	

Visual disturbances and deafness	• Cataracts	• Polyopia (many images)
	• Amaurosis Fugax (TIA in eye)	• <b>Curtain closing down vision loss</b> – painless temporary vision loss due to obstructed BV
	• Migraine, Vascular Lesions	<ul style="list-style-type: none"> <li>• <b>Double vision</b> (diplopia), blurred vision (amblyopia),</li> <li>• light intolerance (photophobia) and visual loss</li> </ul>
Disturbances of gait	• Spastic (Scissoring)	• UMN disorder (stroke, MS)
	• High Stepping (Foot Drop)	• L4-L5 deep fibular nerve compression ( <b>MND, sciatic nerve palsy, CP palsy</b> )
	• Slapping Gait	• B12 deficiency → <b>subacute degeneration of spinal cord</b>
	• Shuffling	• Parkinson's disease → <b>more prefrontal lobe "glued to floor" → TRAPS</b>
	• Waddling	• Myopathic → <b>DMD, proximal myopathy</b>
	• Wide-Based Ataxic	• Anterior cerebellar lobe lesion ( <b>fall to side of lesion</b> )
Disturbed sensation or weakness in the limbs	• Paraesthesia In Hands Or Feet	• nerve entrapment or a peripheral neuropathy (e.g. carpal tunnel syndrome) → nocturnal paresthesia
	• UMN (Pyramidal) Weakness	• <b>LESION</b> at level above anterior horn cell → NO muscle wasting
	• LMN Weakness	• lesion at reflex arc between the anterior horn + and muscle → reduced tone + reflexes + fasculation → <b>MUSCLE WASTING</b>
	• Muscle Disease (E.g. DMD)	• wasting and decreased tone and absent reflexes (proximal weakness)
	• Disease At NMJ	• myasthenia gravis [general weakness BUT normal reflexes/tone]
	• Non-Organic Weakness	• Hysteria with normal tone and power
Tremor and involuntary movements [become worse with fatigue, active SNS, beta-agonist drugs or caffeine]	• Resting Tremors [3-5 Hz]	• Parkinson's disease - when muscles relaxed
	• Benign Essential Tremor [4-7Hz]	Autosomal dominant = postural tremor disappears when muscles at rest ( <b>esp. upper limb</b> )
	• Physiological Tremor [8-13 Hz]	Thyroiditis - fine tremor assoc. with maintaining posture or performing a movement slowly
	• Postural Tremors	When limbs active (e.g. writing)
	• Action ('Intention') Tremors	Cerebellar disease - voluntary actions (tremor worse at end of action)
	• Shivering	• type of tremor caused by cold

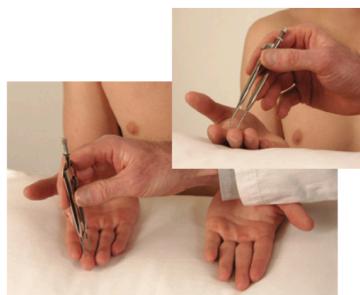
## Higher Centres & Mental State:

Cerebral hemisphere	Dysfunction	Testing
<b>Frontal lobe defect</b> [frontal lobe dementia or space occupying lesion]	<ul style="list-style-type: none"> <li>Disinhibition + Impaired cognition, judgement</li> <li>Volatile and unkept appearance</li> <li>'Jacksonian march' = progressive jerks peripheral to central (distal to proximal)</li> </ul>	<ul style="list-style-type: none"> <li>Primitive reflexes – grasp, palmomental, pout and snout (normal in children/elderly, abnormal in adults)</li> <li>Interpret a proverb</li> <li>Gait apraxia</li> <li>Smell (anosmia - CNI)</li> </ul>
<b>Temporal lobe defect</b>	<ul style="list-style-type: none"> <li><b>Korsakoff's psychosis</b> (confabulation, short term memory)</li> <li><b>TBI / concussion</b> (Retrograde amnesia)</li> </ul>	<ul style="list-style-type: none"> <li><b>Memory loss</b> <ul style="list-style-type: none"> <li><b>Short term</b> - name 5 items, ask patient to repeat this 5 minutes later</li> <li><b>Medium</b> – who is married to your son?</li> <li><b>Long term</b> – when did WW2 end?</li> </ul> </li> <li><b>Hearing loss and inability to lateralise</b> (Weber's test)</li> <li><b>Hallucinations</b></li> <li><b>Epigastric rising</b></li> <li><b>Automatisms</b></li> <li><b>Déjà vu feeling</b></li> </ul>
<b>Dominant Parietal lobe defect</b>	<ul style="list-style-type: none"> <li><b>Gerstmann's syndrome</b> (impaired in dementia)</li> <li><b>NB: contralateral defects (make sure to ask about dominant hand!)</b></li> </ul>	<ul style="list-style-type: none"> <li><b>Acalculia</b> (<math>7 + 7 + 7 + 7 =</math> series of 7's)</li> <li><b>Agraphia</b> (Cannot Write)</li> <li><b>L/R disorientation</b> → place tissue paper with left hand and put it on right foot</li> <li><b>Finger agnosia</b> (Cannot Name fingers)</li> </ul>
<b>non-dominant Parietal lobe defect</b>	<ul style="list-style-type: none"> <li>Issues with cortical sensation</li> </ul>	<ul style="list-style-type: none"> <li><b>Agraphaesthesia</b> → Draw numbers/letters in skin and ask patient to tell you what number/letter was drawn</li> <li><b>Sensory/visual inattention</b></li> <li><b>Two point discrimination</b> → use a compass pin to measure distance between two points</li> <li><b>Spatial neglect</b> → draw a clockface with the time</li> <li><b>Dressing/constructional apraxia</b> → cannot dress properly</li> <li><b>Tactile agnosia</b></li> <li><b>Visual field defects</b> → check visual fields</li> <li><b>Dysprosody</b> = loss of the normal ups and downs in speech, without a change in the content from a non-dominant hemisphere lesion [not dysphasia]</li> </ul>
<b>Occipital lobe</b>	<ul style="list-style-type: none"> <li>Visual mainly</li> </ul>	<ul style="list-style-type: none"> <li><b>Vision loss</b></li> <li><b>Floater and flashes</b></li> </ul>

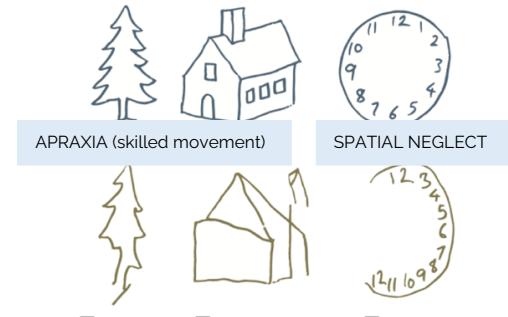
Agraphaesthesia: 'What number have I drawn?' Patient's reply: 'One'.



Avoid the use of an indelible pencil



Two-point discrimination: 'Can you feel one point or two?'



Lower figures show (a and b) constructional apraxia and (c) spatial neglect

### Speech / Language Issues

Always examine speech in a post-stroke setting → all paraphasic errors

	Brain area affected	Name object [speech]	Comprehend	Impaired Repetition	Reading	Writing
<b>Receptive (sensory) dysphasia</b>	Wernicke's area	Fluent, good grammar but meaningless	Poor [auditory dysphasia]	<u>Yes</u>	dyslexia	
<b>Expressive (motor) dysphasia</b>	Broca's area	<b>Slow, non-fluent</b> poor grammar → often frustrated	Good	Ok [With effort]		dysgraphia
<b>Conduction dysphasia</b>	Lesion to arcuate fasciculus	Poor naming Fluent, good grammar	Good	<u>Yes</u>	dyslexia	dysgraphia
<b>Nominal dysphasia</b>	Lesion to dominant posterior temporoparietal area	<b>Cannot name</b> objects [Fluent Speech and use of long sentences]	Good	Good		

What is dysarthria? = difficulty articulating

**Dysarthria "slurred, slow speech"** = when speech muscles become weak or lost control of them.

- Causes include: **bell's palsy (CNVII lesion)**, **cerebellar lesion**, **drunkenness**, **opiate usage**

**Dysphonia (or hoarseness/abnormal sounds)**

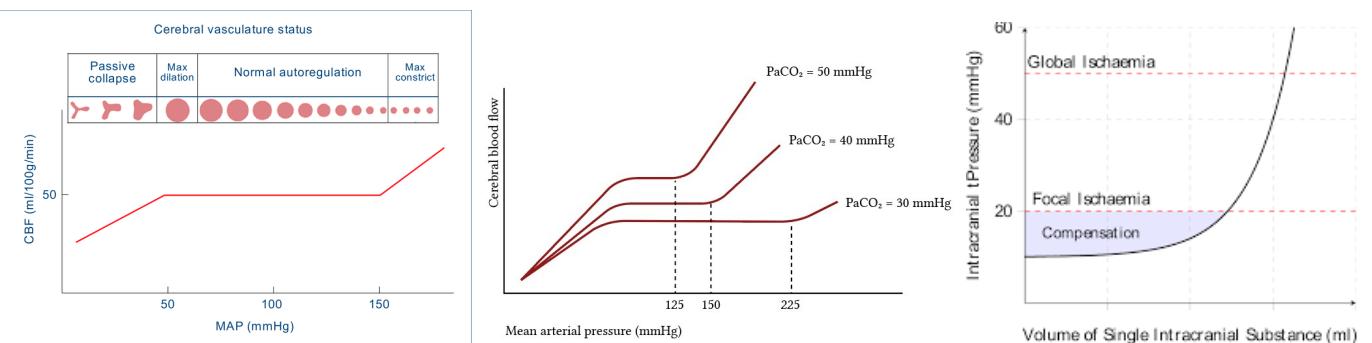
- Causes include **vocal cord injury** → **RLN damage/mass compression OR CNX lesion**

### DDx causes of aphasia:

- Infection
- Brain tumour (temporal lobe)
- Stroke (CNX)
- Psychogenic
- Vagal nerve paralysis
- Vocal cord damage

# RAISED ICP & BRAIN ISCHAEMIA

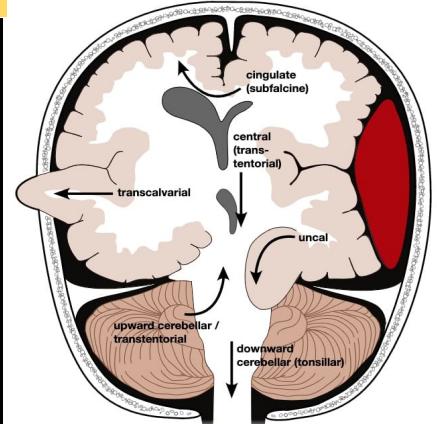
ICP causes & Pathophysiology	Clinical Px	Rx
<ul style="list-style-type: none"> <li>Trauma → Bleeding <ul style="list-style-type: none"> <li>Contusion? Shear injury?</li> <li><b>Raccoon Eyes</b> (anterior skull bone fracture) → risk of meningitis = <b>open fracture in paranasal sinuses</b></li> <li><b>Battle sign</b> (petrous temporal bone fracture) – otorrhea (CSF leakage) = <b>basal skull Fracture</b></li> </ul> </li> <li>Tumour</li> <li>Infection (Abscess, mastoiditis)</li> <li>Metabolic failure (causing lipolysis, proteolysis etc.)</li> <li>Late cause: hydrocephalus, epilepsy</li> </ul> <p><b>Monro-Kellie Doctrine:</b> <math>V_{CSF} + V_{Blood} + V_{Brain} + V_{Intracranial Space} = V_{Other} = KI</math></p> <ul style="list-style-type: none"> <li>incompressible rigid skull (<math>\approx 1600\text{mL}</math>)</li> <li>ICP <math>\approx 7\text{--}15\text{mmHg}</math> (supine) affected by cardiac and resp. cycles <ul style="list-style-type: none"> <li>Pressure-volume index (PVI) = vol. added to intracranial vol. to increased ICP by factor of 10</li> </ul> </li> <li><b>Changes in vol. up to 100-120mL can be compensated if CSF shunted down SC</b> <ul style="list-style-type: none"> <li>CSF = shunted down spinal cord</li> <li>Increased venous blood in dural venous sinuses</li> </ul> </li> <li><b>Loss of autoregulation:</b> → Non-compliant brain = <b>lower PVI</b> = small vol. changes causes greater changes in Pressure</li> </ul>	<ul style="list-style-type: none"> <li>Headache (Herniation &amp; midline shift)</li> <li>Vomiting</li> <li>Stupor/Coma</li> <li>Bulging fontanelles (infants)</li> <li><b>Cushing's triad</b> (irregular RR, widened PP, and bradycardia)</li> <li>Cheyn stokes breathing (shallow rapid breaths)</li> <li><u>Mass effect:</u> <ul style="list-style-type: none"> <li><b>CN3 palsy</b></li> <li><b>CN6 palsy</b> (most likely affected by raised ICP due to long course)</li> <li><b>FND</b></li> </ul> </li> <li><b>Extensor postutre</b></li> </ul>	<ul style="list-style-type: none"> <li>Head of bed elevation</li> <li>Mannitol</li> <li><b>Decompressive craniectomy</b> (remove fontanelle)</li> <li><b>Burr-hole washout</b> (subdural)</li> <li><b>Clot evacuation + ligate bleed</b> (epidural)</li> <li><b>Intubate + hyperventilate</b> (raise CPP and prevent herniation e.g. CNV3 – Foramen ovale)</li> </ul>



Cerebral perfusion pressure	Cerebral blood flow	Oedema										
$\boxed{\text{CPP} = \text{MAP} - \text{ICP}}$ <p><math>\text{MAP} = [1/3 (\text{SP}-\text{DP})] + \text{DP}</math></p> <ul style="list-style-type: none"> <li>CPP maintained via <b>autoregulation</b> over MAP range (60-150mmHg)</li> <li><b>CPP &lt; 70mmHg OR ICP &gt; 50 mmHg</b> <ul style="list-style-type: none"> <li><b>ischaemic brain damage</b></li> <li>Hypereamic response</li> </ul> </li> <li>Chemo-regulation (<b>mainly CO<sub>2</sub></b>, also O<sub>2</sub>, pH, calcium, NO) <ul style="list-style-type: none"> <li><b>hypercapnia</b> = vasodilatation = <math>\uparrow \text{CBF}</math></li> <li><b>hypocapnia</b> = vasoconstriction = <math>\downarrow \text{CBF}</math></li> </ul> </li> </ul>	$\text{CBF} = \frac{\text{CPP}}{\text{CVR}} = \frac{\Delta P}{\frac{8\mu L}{\pi r^4}} = \frac{\Delta P \pi r^4}{8\mu L}$ <p>CVR = cerebrovascular resistance</p> <ul style="list-style-type: none"> <li><math>\approx 50\%</math> of total CVR arises from <b>small pial arteries (150-200μm in diameter)</b> and <b>arteries of circle of Willis</b> <ul style="list-style-type: none"> <li><b>R</b> = vessel radius</li> <li><b>L</b> = vessel length</li> <li><math>\mu</math> = coefficient of fluid viscosity</li> </ul> </li> </ul>	<table border="1"> <thead> <tr> <th>Type</th><th>Description</th></tr> </thead> <tbody> <tr> <td><b>Cytotoxic</b></td><td>cellular swelling 2° to cell injury (hypoxia, reduced substrate)</td></tr> <tr> <td><b>Vasogenic</b></td><td>vascular leakage via disrupted BBB = <math>\uparrow</math> fluid → cancer, TBI, inflamed</td></tr> <tr> <td><b>Interstitial</b></td><td>transepndymal flow of CSF → <b>normal pressure hydrocephalus</b> (dementia, gait disturbed, incontinence)</td></tr> <tr> <td><b>Osmotic</b></td><td>Normally hyperosmolar brain → <math>\uparrow</math> osmosis from blood plasma into brain via gradient (intact BBB)</td></tr> </tbody> </table>	Type	Description	<b>Cytotoxic</b>	cellular swelling 2° to cell injury (hypoxia, reduced substrate)	<b>Vasogenic</b>	vascular leakage via disrupted BBB = $\uparrow$ fluid → cancer, TBI, inflamed	<b>Interstitial</b>	transepndymal flow of CSF → <b>normal pressure hydrocephalus</b> (dementia, gait disturbed, incontinence)	<b>Osmotic</b>	Normally hyperosmolar brain → $\uparrow$ osmosis from blood plasma into brain via gradient (intact BBB)
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## Herniation Types

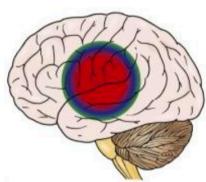
	Symptoms
<b>Subfalcine</b>	<ul style="list-style-type: none"> <li>Midline shift of septum pellucidum</li> <li>Infarction ACA → contralateral hemiparesis (legs &gt; arms)</li> <li>cognitive/emotional impairment</li> </ul>
<b>Central / transtentorial</b>	<ul style="list-style-type: none"> <li><b>Cushing triad</b> (HTN, bradycardia, irregular RR)</li> <li>decorticate → decerebrate posture <i>if involvement of rubrospinal and the vestibulospinal tract</i></li> <li>Vegetative state OR death</li> </ul>
<b>Uncal / Temporal transtentorial</b>	<ul style="list-style-type: none"> <li>CN3 Palsy (ipsilateral mydriasis, anisocoria) <ul style="list-style-type: none"> <li><b>contralateral hemiplegia</b> (motor weakness)</li> </ul> </li> <li><b>Kernohan notch phenomenon</b> → ipsilateral weakness due to excess midline shift causing compression of opposite cerebral peduncle containing CST fibres</li> </ul>
<b>Cerebellar tonsillar</b>	<ul style="list-style-type: none"> <li>Medulla compression → respiratory dysfunction → death</li> <li>May lead to <b>chiari malformation</b> (i.e. tonsils compress SC)</li> </ul>



# Discuss the metabolic requirements of neurons.

## Ischaemic core:

- Perfusion below a threshold needed to maintain certain biochemical functions
- Neurons – terminal loss of membrane potential (permanent depolarisation) – cannot be revived - infarction

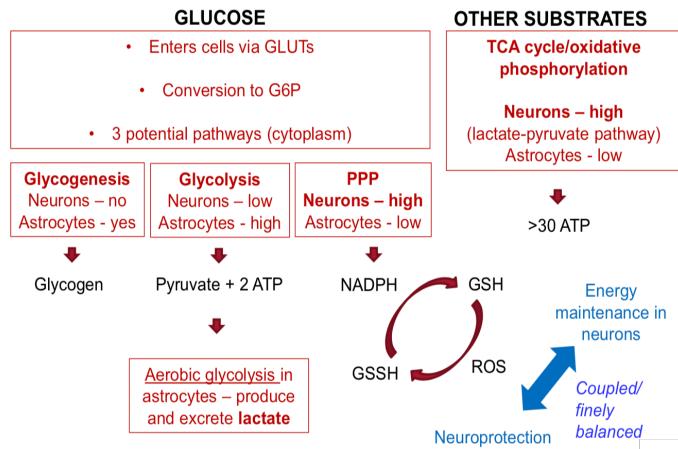


## TIME COURSE

- 10–20 min: few scattered dead neurons in core.
- 60 min: infarct observed in core.
- 6-24h: infarct encompasses core & penumbra.

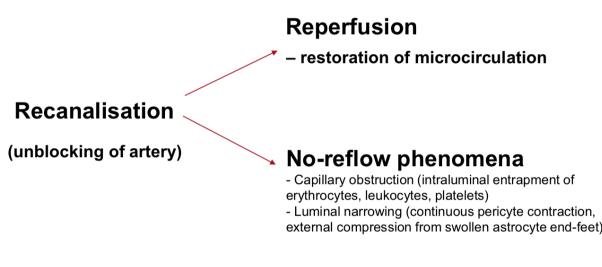
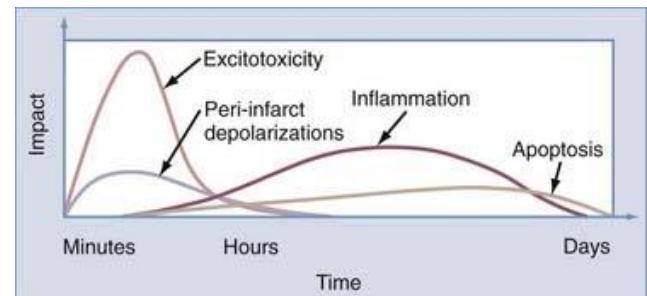
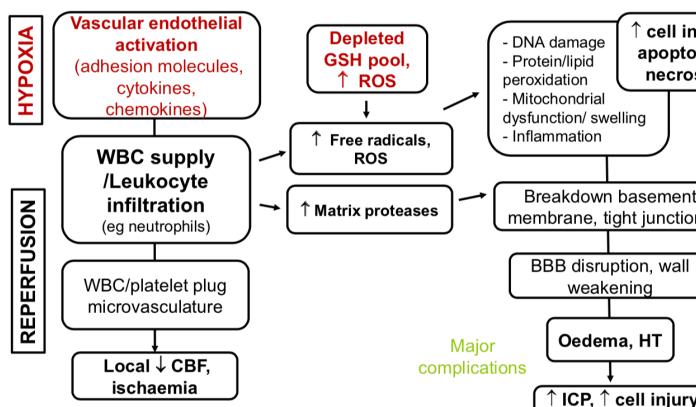
## Penumbra:

- Surrounding region of intermediate perfusion (may be collateral supply)
- Neurons depolarise intermittently
- Potentially salvageable if re-perfused (blood flow restored)



## Key points:

- Neurons have **high ATP/energy needs** and rely on oxygen and glucose for oxidative phosphorylation (TCA cycle)
  - ATP powers  **$Na^+/K^+$  ATPase and transporters to generate APs and release neurotransmitters, respectively**. Excess glutamate at pre-synaptic terminal is toxic, hence needs to be removed
  - However, pathway requires substrate (i.e. lactate) + oxygen availability** → **Ischaemia (no  $O_2$ ) stops this process or no glucose will cause neuronal death**
  - Neurons **CANNOT** store glucose as glycogen
- Astrocyte-neuron coupling** plays an important role in metabolism and antioxidant defence.
  - Astrocytes use aerobic glycolysis → produce lactate** (even in presence of  $O_2$ ) → lactate released via MCT → received by neurons via monocarboxylate transporters (MCTs) → converted into pyruvate → enters mitochondria and TCA cycle
  - NB: primary source of pyruvate for neurons comes from lactate NOT glucose**
- Mature neurons **down-regulate** glycolysis = switch off glycogenesis → divert glucose into PPP (to generate GSH antioxidant and replenish reserves) – to stop neurotoxicity caused by ROS/oxidative species.
  - GSH = antioxidant that needs to be constantly replenished to **prevent** endogenous stress (from ROS) + neuronal death
  - Astrocyte-neuron coupling** → couple glucose metabolism to antioxidant defence



- Ishaemia**
  - $no ATP = inactivated Na^+/K^+$  ATPase → influx and accumulation of  $Na^+$  in cell = cellular oedema
  - reduced GSH (antioxidant) production
- Loss of neuronal selective membrane permeability and ion gradient** = anoxic depolarisation
- Over-stimulated post-synaptic glutamate receptors → Excess influx  $Ca^{2+}$  → **Glutamate excitotoxicity & free radical production (proteases, lipases etc.)**
- Neuronal nitric oxide synthase (NOS)** → increase free radicals (NO) → stimulate cell damage and inflammation – leukocyte infiltration, microglial activation

## What happens if we reperfuse?

Rapid Reperfusion injury can **worsen outcomes** after ischaemic episode

- Enlarge infarcted area and increase neurological damage
- Cerebral oedema and/or haemorrhagic transformation → increasing ICP → FND or global effects

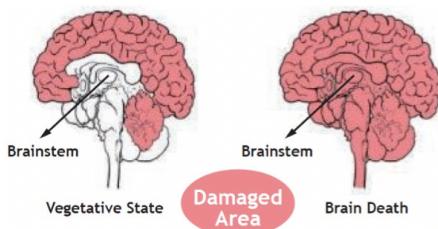
Solution:

- Pre-conditioning** (expose/adapt to short periods of hypoxia)
- Post-conditioning** – transient reperfusion/re-occlusion cycles to **induce protective** mechanisms → reduce ROS
- Therapeutic Hypothermia** (reduces enzymatic activity and cellular damage) – during or after ischaemia

# Define Brain Death

An individual is dead if they sustain either:

- Irreversible cessation of circulatory and resp. functions
- Irreversible cessation of all BRAIN functions  
PLUS brain stem



	GCS	PTA	LOC
Mild	13–15	<1 day	0–30 minutes
Moderate	9–12	>1 to <7 days	>30 min to <24 hours
Severe	3–8	>7 days	>24 hours

## Longer the amnesia OR lower the GCS

↑ risk of death/ disability

### Vital signs for brain death

- Core temp. >35°C** [anything below = hypothermia = stunned state]
- SBP >100mmHg** [anything below = hypoperfusion = shock]
- No drugs** simulating brain death (e.g. baclofen, anti-cholinergic, barbiturates)
- Blood alcohol content <0.08%**
- Absent brainstem reflex (corneal, VOR, RAPR)**
- No response to deep central pain** (*cerebration/decortication are not compatible with brain death!*) → trap squeeze, press supratrochlear
- Failed apnoea challenge** (no resp. with  $pCO_2 > 60 \text{ mmHg}$ )
- Brain death in children** → 2 exams inc. apnoea testing separated by an observation period
  - 12–24 hrs according to age, with cut off of 30 days of age for term newborns (i.e. 37 wks gestational age)
- No guidelines for infants < 37 weeks gestational age

### Most common etiologies of brain death

- TBI
- SAH (most common = 2° to TBI, other = aneurysm)
- ICH (2° to TBI)
- Stroke with cerebral edema & herniation (↑ ICP)
- Hypoxic-ischemic encephalopathy
- Fulminant hepatic necrosis + cerebral edema + ↑ ICP

### DDX:

- Locked-in syndrome
- Guillain-Barre' syndrome [antibodies to gangliosides - SC]
- Severe hypothermia (<28 C)
- Anaesthetic or sedative medications

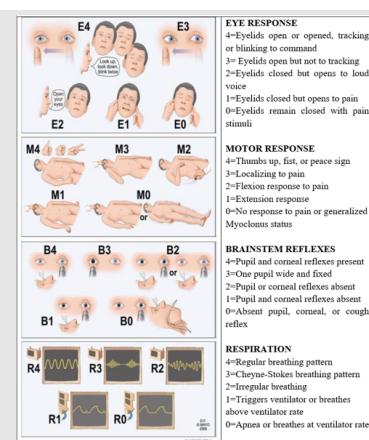
TABLE 38-2

### Glasgow Coma Scale

BEHAVIOR	RESPONSE	SCORE
Eye opening response	Spontaneously	4
	To speech	3
	To pain	2
	No response	1
Best verbal response	Oriented to time, place, and person	5
	Confused	4
	Inappropriate words	3
	Incomprehensible sounds	2
Best motor response	No response	1
	Obey commands	6
	Moves to localized pain	5
	Flexion withdrawal from pain	4
	Abnormal flexion (decorticate)	3
	Abnormal extension (decerebrate)	2
	No response	1
Total score:	Best response	15
	Comatose client	8 or less
	Totally unresponsive	3

### Limitations:

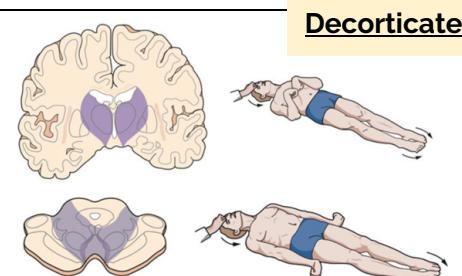
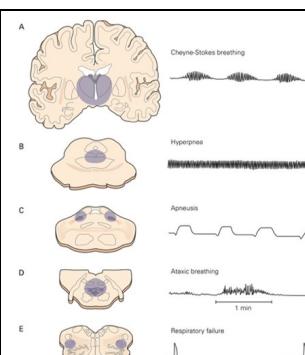
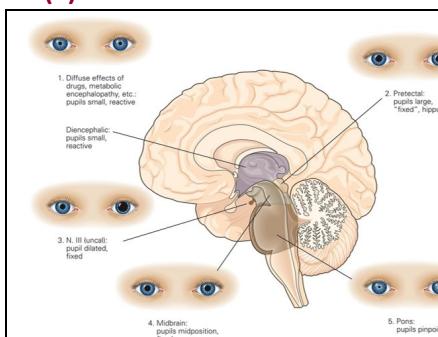
- Everything will score 3** (even the kitchen table!)
- Requires a **verbal response** – can't be used on patients that are intubated, mutes, paraplegics?
- Does **NOT** assess brainstem reflexes.
- Biased** towards a motor response



### Benefits of FOUR score:

- Can be used on intubated patients
- Can detect early changes in consciousness (particularly useful for metabolic disturbances etc).
- NO bias**

## (3) LOCALISING SIGNS to assess UNCONSCIOUS PATIENT



### #1: Eye Movements

The **reticular activating system** is anatomically close to the midbrain structures that control pupillary function and breathing.

- upper brainstem lesion** = Enlarged pupils + loss of light reaction and vertical and adduction movement of the eye
- intact brainstem [Vegetative state]** = normal pupillary light reactivity and eye movement [likely widespread structural lesions or metabolic suppression of cerebral hemispheres causing LOC]

### #2: Breathing changes

- Bilateral cortical or diencephalic dysfunction** → **Cheyne-Stokes breathing** (waxing and waning breathing pattern interspersed with apnoea)
- Hyperpnea** or **hyperventilation** → **brainstem tumour**.
- Lesions of the parabrachial region of the rostral pons** causes **apneusis** (pattern of deep, prolonged inspiration)
- Lesions of the pontomedullary junction** → **ataxic breathing** (irregular frequency, duration and depth of breathing). → LEADS to respiratory failure
- Lesions at level of the rostral ventrolateral medulla** → **respiratory failure**. Occasional gasps interspersed with long apnoea may occur.

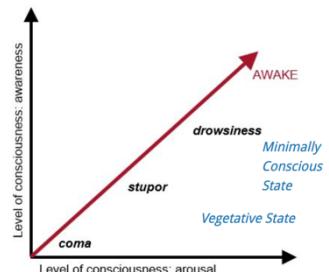
### #3: Changes in posturing

<b>Decorticate</b> Posturing (M3)	<b>Bilateral damage to diencephalon</b>	Upper limb flex Lower limb extend
<b>Decerebrate</b> Posturing (M2)	<b>Severe midbrain damage</b>	All limbs extend

\*NB: Progression from decorticate → decerebrate posturing indicates **rostrocaudal deterioration of brainstem** (→ respiratory arrest if medulla affected)

# Collapse Consciousness

Term	Definition
<b>Awareness</b>	content of consciousness (i.e. you know what is happening)
<b>Arousal</b>	level of consciousness (e.g. asleep vs. awake)
<b>Coma</b>	most severe state of reduced alertness (cannot be aroused) <ul style="list-style-type: none"> <li>ONLY result from <b>BILATERAL</b> cortical damage OR damage to <b>reticular activating pathway</b></li> </ul>
<b>Stupor</b>	higher degree of arousability than a coma (aroused with vigorous stimuli)
<b>Drowsiness</b>	simulates light sleep (easy arousal & alertness for brief periods)
<b>Vegetative State</b> "awake coma"	<ul style="list-style-type: none"> <li>awake but unresponsive state (i.e. eyes open, CV regulation maintained but not responsive to environ. cues)</li> <li><b>widespread cortical damage</b> → BUT reticular activating is intact because they have a sleep-wake cycle</li> <li><i>Due to head injury or cardiac arrest</i></li> </ul>
<b>Minimally conscious state</b>	<ul style="list-style-type: none"> <li>Less severe vegetative state → shows some awareness of self and surroundings</li> <li><i>Due to head injury or cardiac arrest</i></li> </ul>
<b>Akinetic Mutism</b>	virtually immobile and mute BUT can think and form impressions
<b>Catatonia</b>	hypomobile and mute syndrome + few voluntary or responsive movements
<b>Locked-In Syndrome</b>	Patient is awake and can move eyes BUT cannot speak <b>[PONTINE ARTERY STROKE]</b>



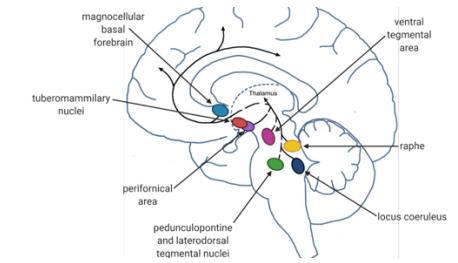
Explain the role of the reticular activating system in consciousness – how is consciousness processed in the NS?

Trigger/stimuli		2 pathways	Origin of pathway	Structures involved
<ul style="list-style-type: none"> <li>Somatic</li> <li>Visual</li> <li>vestibular</li> </ul>	<b>ASCENDING RETICULAR ACTIVATING SYSTEM</b>	AWARENESS	Thalamus (Dorsal)	cortex
		AROUSAL	reticular formation (ventral)	hypothalamus, thalamus, basal forebrain projections and cortex

\*Without arousal = there is NO awareness

Identify drug classes affecting reduced LOC & Describe how to reverse this

	Pathway	Function
<b>Reticular structures</b>	<b>monoaminergic pathways</b> from locus ceruleus and raphe =	regulate sleep and wakefulness
	<b>glutamatergic projections</b> from the rostral pons and caudal medulla	regulate sleep and wakefulness
<b>Non-reticular structures</b>	<b>lateral hypothalamus</b> (orexin and melanin concentrating hormone containing neurons)	regulate sleep and wakefulness
	<b>cholinergic and GABAergic neurons</b> in the basal forebrain	arousal



	MoA	Overdose	Examples
Sedatives-Hypnotic Agents	facilitating actions of GABA <ul style="list-style-type: none"> <li>Sedatives portion provide calming anxiolytic</li> <li>hypnotics produce sleep-inducing</li> </ul>	No reversal agent – supportive care only	<ul style="list-style-type: none"> <li><b>short acting GABA agonists</b> (e.g. triazolam),</li> <li><b>benzodiazepines</b> (e.g. temazepam, diazepam),</li> <li><b>barbiturates</b> (e.g. thiopental, pentobarbital),</li> </ul>
<b>Opioids</b>	Widespread reduced neuronal activity → Resp depression	reversed with $\mu$ -opioid receptor antagonists <b>e.g. naloxone</b>	heroin, morphine, oxycodone.

Others: antihistamines, centrally acting sympatholytic antihypertensives (clonidine), antidepressants, antiemetics (metoclopramide)

Occlusion of which blood vessel would result in ipsilateral CN VII palsy, decreased consciousness, hemiparesis and hemiataxia, oculomotor defects and arm posturing?

Which type of herniation occurs when there is symmetrical downward movement of the diencephalon through the opening of the tentorium cerebelli?

- ✓ Mid Basilar Artery
- Extracranial Vertebral Artery
- Intracranial Vertebral Artery
- Distal Basilar Artery

Foraminal herniation.

Uncal herniation.

- ✓ Central herniation.

Subfalcine herniation.

1/1

Which of the following would not result in a loss of consciousness?

- Increasing intracranial pressure.
- ✓ Increasing blood supply to the brain.
- Disrupting the activity of the sodium potassium pump.
- Decreasing the neuronal membrane potential.

1/1

Which brain region would antihistamines affect to produce drowsiness?

- ✓ Tuberomamillary Nucleus.
- Perifornical Area
- Ventral Tegmental Area
- Raphe.

# STROKE: TYPES

Box 5. The recognition of stroke in the emergency room (ROSIER) score

	Score	Yes	No
Has there been loss of consciousness or syncope?	-1	0	
Has there been seizure activity?	-1	0	
Has there been new acute onset (including on waking from sleep) of:			
Asymmetric facial weakness	+1	0	
Asymmetric arm weakness	+1	0	
Asymmetric leg weakness	+1	0	
Speech disturbance	+1	0	
Visual field deficit	+1	0	

## FAST Symptoms (Face/arm Weakness, speech/dysphasia, or vision)

### Stroke (ROSIER score >1)

#### Haemorrhagic (15%)

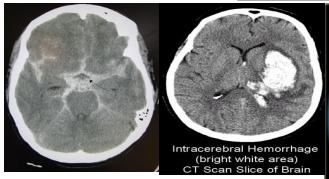
**Risk factors:**

Haemorrhagic (15%)

#### 3 H's of stroke

SAH

Cause: aneurysm, TBI, AVM  
Xanthochromasia (in CSF)  
Hydrocephalus  
Vasospasm (released blood products)



ICH

Cause: HTN, infection, tumour, TBI, AVM, amyloid deposits  
Raised ICP = MASS effect (haematoma)  
Bleeding = releases haemolysis products causing toxicity --> LOC

#### Global Ischaemia (LARGE VESSEL)

Assoc. atherosclerosis and embolism

Total stroke (3/3 H's)  
Partial stroke (2/3 H's)

#### Ischaemic (85%)

**Mod. Risk factors:**

**Non-mod. Risk factors:**

#### Lacunar "silent" infarct (SMALL VESSEL)

Small infarct < 20mm  
Assoc. HC, HTN, T2DM

Lacunar stroke (1/3 H's)  
NO cortical signs  
NO preference (arm = leg = face)

**Types include:**  
1) pure motor (50%)  
2) pure sensory  
3) mixed  
4) dysarthria  
5) Ataxic hemiparesis

#### Thromboembolic

##### Thrombotic (50%)

Local clot  
Slow onset

##### Embolic (30%)

Distal clot  
rapid onset  
large vessel  
Fat embolic, AF, infection, tumour

## STROKE: Work up

### Stroke (time-based)

Non-contrast CT head (exc. intracranial bleed)  
can also use DWI and CTA +/- perfusion scan after

Thrombolysis (alteplase) if ≤ 4.5 hours

Or if not

Aspirin 300mg PO/PR for 2 weeks  
Clopidogrel 300mg PO STAT then 75mg OD

Consider endovascular clot retrieval (if within 6 hours AND pt has large vessel occlusion with minimal brain damage)  
Transfer to acute stroke unit (ASU)

**MDT management:**  
1) Speech pathologist, ophthalmologist, psychologist  
2) neurologist + neurosurgeon  
3) stroke rehabilitation nurse coordinator  
4) dietitian = nutritional optimisation  
5) social worker = coordinate stroke rehab

EARLY immobilisation (physio)  
TREAT INFECTIONS early + Protect pressure areas  
**Acute:** Give Anti-HTN + Warfarin 24 hrs after clot removal  
**Long-term drugs** (Anti-HTN, 75mg PO Clopidogrel, statins - wait 24 hrs after stroke)  
Consider carotid endarterectomy (carotid USS doppler)

### Stroke in young

Functional fake → "drop hand over face"

- > dissociative disorder (stress),
- > factitious
- Organic:
- > APS, homocysteineuria
- > Fabry's
- > AVM
- > 'berry' aneurysm / cardiac embolism

### CI for thrombolysis

- > Concurrent warfarin
- > SAH or previous ICH
- > Aneurysm or AVM
- > Brain cancer
- > Acute pericarditis
- > Recent surgery / LP
- > Active GI or UT bleed
- > Uncontrolled HTN (>185/110)

### TIA (SUSPECTED) (not time-based)

Higher risk of developing lacunar stroke within 24 hrs

Aspirin 300mg PO/PR for 2 weeks  
Clopidogrel 300mg PO STAT then 75mg OD

Specialist review within 24 hours  
(and CTA +/- perfusion scan)

### DRIVING CONSIDERATIONS

- > **If normal** = wait 2 weeks
- > **If recovering** = NO DRIVING for ≥ 4 weeks (Service NSW report if Heavy vehicle driver)
- > **If seizure** = cannot drive for 6/12

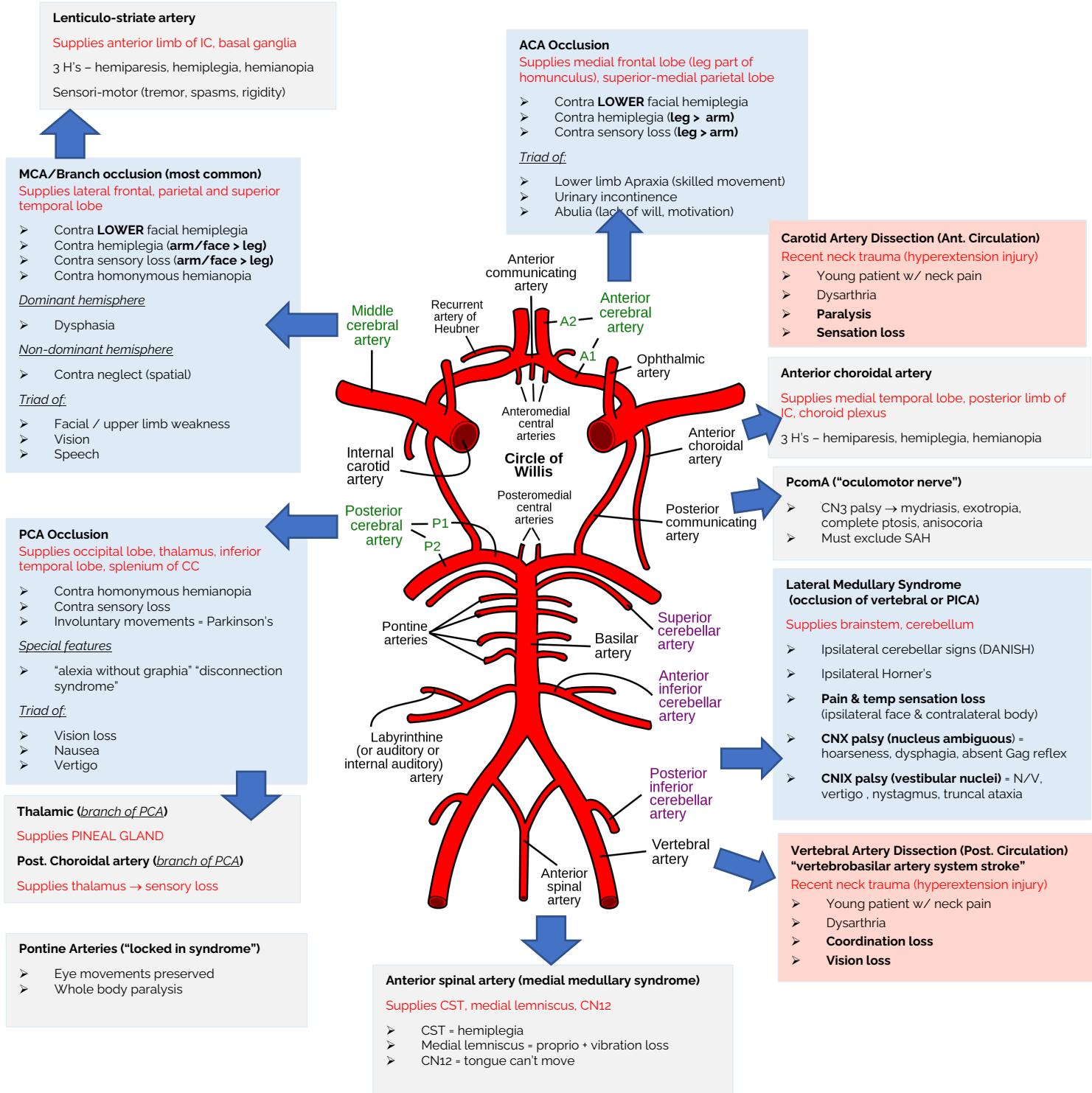
### Consider carotid endarterectomy if:

- 1) ipsilateral carotid stenosis > 50% OR
- 2) ipsilateral carotid stenosis > 70% (ECST)

**Long-term drugs = secondary prevention**  
(Anti-HTN, 75mg PO Clopidogrel, statins)

**F/U investigations include:**  
ECG, 24-hr ECG tape, ECHO, vasculitis and thrombophilia screen, carotid doppler USS

# STROKE: LOCATION



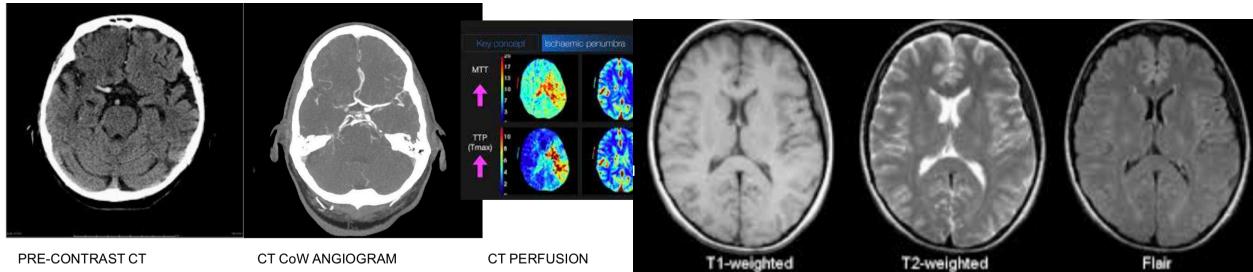
## LUMBAR PUNCTURE – MENINGITIS DDx

	LP fluid	Gram stain	Opening Pressure	Glucose (40-75)	Protein (15-45)	Cells on LP	Treatment
<b>Bacterial</b>	Cloudy	+	↑↑	low	High	<ul style="list-style-type: none"> <li>PMN (&gt;1000)</li> <li>Gram +ve = listeria (rods), pneumococci (diplococci)</li> <li>Gram -ve = HiB (coccobacilli), meningococcal (diplococci)</li> </ul>	<b>RF:</b> DM, immunocomp, EtOH, pregnant, > 50 <ul style="list-style-type: none"> <li>Senior help → ABCD → IVF</li> <li><b>Regardless</b> = Early IV 2g ceftriaxone + IV 10mg dexamethasone (+/- benzyl if listeria suspected)</li> <li><b>Viral</b> = IV acyclovir</li> <li><b>Fungal</b> = IV amphotericin B (check liver and renal function)</li> <li>Public NSW health notification</li> </ul> <u>Close contacts</u> <ul style="list-style-type: none"> <li>N. meningitis → prophylactic cef, cipro or rifampicin</li> <li>HiB → cef or rifampicin</li> </ul>
<b>Viral (PCR)</b>	Clear	-		normal	Low	<ul style="list-style-type: none"> <li>Lymphocytes (&gt;10-500)</li> <li>HSV = bloody tap + anosmia + confuse</li> </ul>	
<b>Fungal</b>	Cloudy	-	↑↑	low	High	<ul style="list-style-type: none"> <li>Lymphocytes (20-200)</li> </ul>	
<b>TB (acid-fast)</b>	Clear + spiderweb clot	+	↑↑	low	high	<ul style="list-style-type: none"> <li>Lymphocytes (mainly) +PMN</li> </ul>	
<b>SAH</b>	Yellow xanthochromia		↑↑				

# INTRACRANIAL BLEEDS

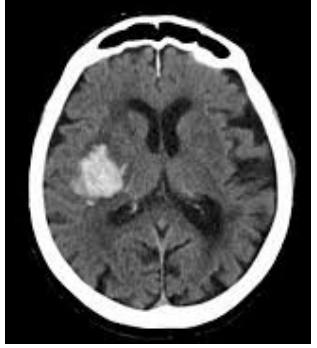
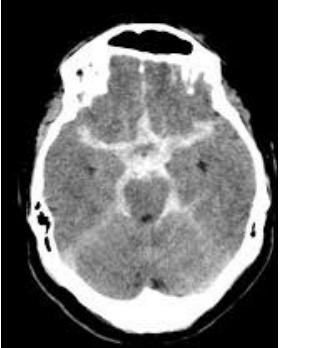
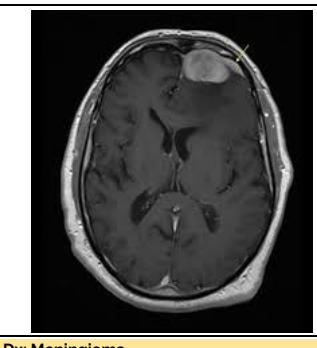
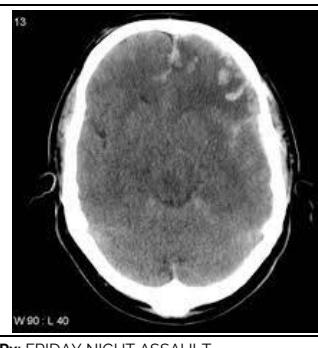
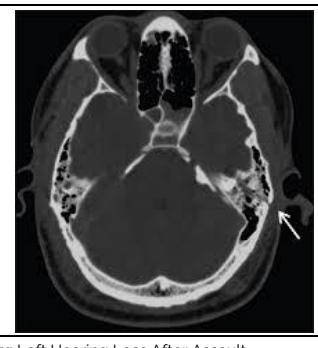
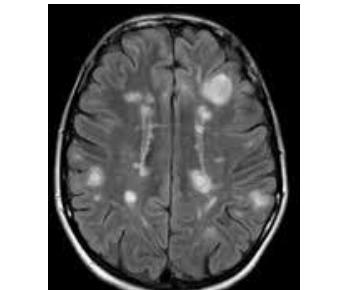
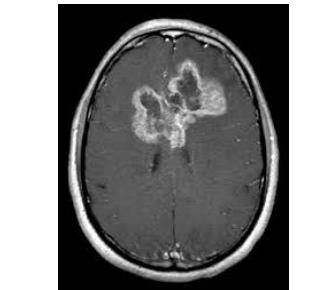
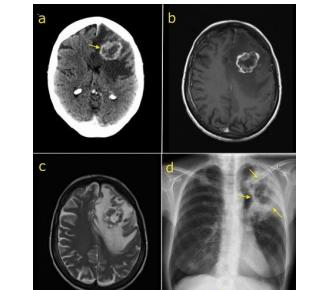
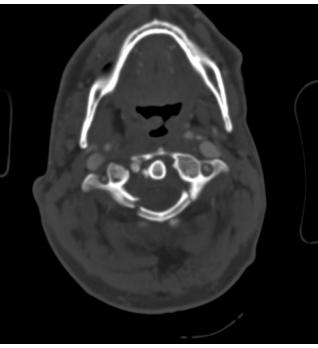
	EDH	SDH	SAH	ICH	General Mx
Where	Ruptured middle meningeal artery Assoc.	Rupture of bridging emissary veins	Subarachnoid space (in CSF) ➤ Meningeal signs without fever ➤ LP – xanthochromia ➤ CTA or MRA	• Lobar ICH • Deep ICH • IVH • Basal ganglia • Cerebellar	<b>Assess risk factors:</b> 4. Head injury 5. Hypertension 6. Aneurysms 7. Ischaemic stroke can progress to haemorrhage 8. Brain tumours 9. Anticoagulants e.g. warfarin
Key features (RF)	• temporal bone # • young pt • brief Lucid period then sudden deterioration	• elderly • alcoholic (more brain atrophy)	• Ruptured aneurysm • Cocaine • Sickle cell anaemia • ADPKD, NF, Marfan, Ehler • XS EtOH • <b>Black females aged 45-70</b>	Spontaneous secondary to: ➤ Infarct, tumour, aneurysm rupture	<b>Senior help</b> 10. GCS score 11. Urgent non-contrast CT head 12. FBC + Coags 13. Admit under ASU OR neurosurgery 14. Consider I+V, and ICU care (if reducing LOC)
Shape	Bi-convex	Crescent	Continuous w/ ventricles	Central dot sign	
Suture cross	No	Yes	NA	NA	
Midline cross	Yes	No	NA	NA	
Rx	Clot removal	Burr-hole washout	1. <b>Nimodipine</b> = prevent vasospasm post-SAH 2. <b>Endovascular coiling OR surgical clipping</b> 3. <b>Shunt insertion</b> (for hydrocephalus) 4. <b>Anti-epileptics</b> (if seizures)		<b>E/U management once stabilised:</b> 15. Correct clotting issue, severe HTN (but avoid hypoTN) 16. Consider LP 17. Consider CTA or MRI-angio

	CT: Computed Tomography	MRI: Magnetic Resonance Imaging
Modality	• <b>Electron imaging</b> • Utilizes X-rays – ionizing radiation → cancer risk • ED workhorse	• <b>Proton imaging</b> (any structure with water lights up = oedema, tumour etc.) • <b>Radio waves &amp; high energy magnet</b> → non-ionizing radiation (but heat up skin) • <b>Patient safety questionnaire</b> (E.g. metal implant, pacemaker, cochlear implant, neurostimulator, braces)
Indication	• <b>Bone/fractures</b> [use bone window] • <b>Acute blood</b> (bright = hyperattenuating Hb/Fe) • <b>Established stroke</b> • <b>Vessel blockage or aneurysm</b> (CTA esp. CoW) • <b>Ventricle size</b> (hydrocephalus)	• <b>Acute stroke detection</b> (DWI) • <b>Brain &amp; pituitary masses/tumour</b> • <b>Epilepsy Foci</b> • <b>Spinal cord injury – T2 Flair</b> • <b>Ligamentous injury</b> • <b>White matter lesions</b> (e.g. MS) – – T2 Flair
Avoid	• <b>Acute infarct/stroke</b> (need CT angiogram) • <b>Masses/tumour</b> (need iodinated contrast CT) • <b>Demyelinating disease</b> • Pregnant women • Young children	• Fractures • <b>Minimise contrast</b> (i.e. Gadolinium may cause rare and incurable nephrogenic systemic fibrosis) esp. if eGFR < 30 (CKD stage 4) • <b>Unsecured metal</b> (e.g. shrapnel, metal fragments in eyes) NB: pacemakers OK (if allowed to be switched off)
Differences	• Bright skull bone • Fat is dark on CT = dark white matter  Metal > contrast > bone > blood > soft tissues > fat > thin fluid > air Hyperattenuating -----> Hypoattenuating	• Scalp is white Hyperintense ----> Isointense (same as cortex/muscle)----> Hypointense • <b>T1</b> = grey matter (grey), white matter (white) → <b>fat melanin, heavy metals</b> • <b>T2</b> = grey matter (white), white matter (grey), → <b>CSF, SC compression</b> • <b>Flair</b> = T2 without bright CSF → <b>demyelinating diseases (MS)</b> • <b>DWI</b> = best to detect <b>acute strokes or cytotoxic oedema</b> o Water molecules restricted within the cells o <b>Susceptible to artefacts from blood/metal/braces</b>
Advantages	1. Widely available 2. Less contraindications (metals, implants, claustrophobia) 3. Much faster acquisition or less motion artefacts 4. More sensitive in detecting acute bleed/skull and spine fractures 5. Routine multiplanar reconstruction 6. CT radiographers more often available	1. <b>No Radiation</b> → children, pregnant women, young adults 2. Superior contrast resolution (with gadolinium) 3. Many sequences to assess different pathology 4. No contrast needed to assess vascular structure 5. Image in any plane
Disadvan	1. Radiation 2. Poor to assess infection, tumours, demyelination and SCI 3. Damage to lens, skin, cancer and foetus	1. Safety questionnaire needed → Be careful of pacemakers, metal implants 2. Claustrophobia 3. Not good for anxious/claustrophobic patients → need oral sedation 4. MRI radiographers longer training (less available) 5. Long preparation and acquisition time

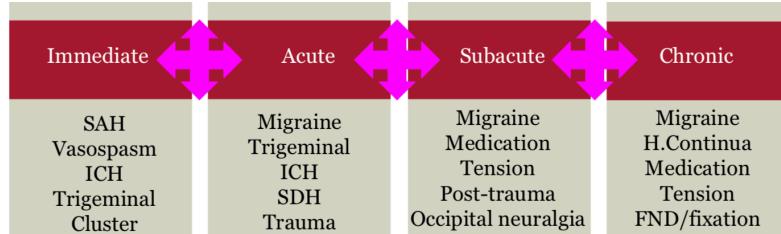


Scenario	Imaging of choice	Reason
Acute head injury brought in by ambulance to ED	<b>CT brain and cervical spine non-contrast</b>	Exc. intracranial bleed and skull/C-spine fracture)
50 yo patient with sudden onset left hemiplegia	<b>CT brain pre-contrast</b> (exc. haemorrhage) + CT CoW angiogram (aortic arch to vertex), CT perfusion	acute stroke +/- occluded MCA or acute haemorrhage in right cerebrum
30 year old woman with 3 weeks history of numbness in left arm and previous episode of visual deterioration	<b>MRI</b>	MS or less likely a tumour
6 year old boy with recurrent seizures	<b>MRI</b>	epileptogenic foci, congenital anomalies + exc. tumour

\*Not uncommon for patient to get both – initial CT in ED to triage and decide which team coming in under (e.g. neurology vs neurosurgery) overnight, and then MRI next day for detailed assessment

			
<p><b>Px:</b> Progressive Confusion And Right Weakness: Elderly Patient</p> <p><b>Dx: Subacute subdural haematoma</b></p> <p><b>Desc:</b> Non-contrast axial CT brain scan showing significant subdural haemorrhage causing significant midline shift and effacement of left lateral ventricles</p>	<p><b>Px:</b> Headache And Left Weakness: History Of Hypertension</p> <p><b>Dx: intracerebral hemorrhage</b></p> <p><b>Desc:</b> Non-contrast axial CT brain scan showing right intracerebral hemorrhage of basal ganglia with no midline shift</p>	<p><b>Px:</b> Sudden Onset Worst Headache Of Life</p> <p><b>Dx: Subarachnoid hemorrhage</b></p> <p><b>Desc:</b> Post-contrast axial CT brain scan showing subarachnoid hemorrhage with blood in the basal cisterns</p>	<p><b>Px:</b> MVA – No Seatbelt – Head Through Windscreen</p> <p><b>Dx: acute subdural haematoma</b></p> <p><b>Desc:</b> Post-contrast axial CT brain scan showing right SDH causing sig. midline shift with IVH causing obstructed hydrocephalus in the left occipital horn as well as calcification in the right occipital horn + effaced R lateral ventricles</p>
			
<p><b>Px:</b> Fall From Ladder</p> <p><b>Dx: Extradural hematoma</b></p> <p><b>Desc:</b> Non-contrast axial CT brain scan showing right parietal extradural haemotoma with overlying scalp swelling and significant midline shift req. neurosurgical referral</p>	<p><b>Dx: Meningioma</b></p> <p><b>Desc:</b> Axial MRI brain scan showing a well-demarcated hyperattenuating lesion in the left frontal lobe that is adhered to the dural wall with a poorly demarcated homogenous hypoattenuating oedematous region</p>	<p><b>Px:</b> FRIDAY NIGHT ASSAULT</p> <p><b>Dx: Cerebral contusion</b></p> <p><b>Desc:</b> Non-contrast axial CT brain scan of poor resolution due to patient movement. Evidence of <b>contre-coup injury</b>, slight midline shift and SAH in the left frontal lobe and basal cisterns. <b>[Salt + pepper appearance – hypo/hyperattenuating]</b> Also effacement of CSF on left midbrain suggestive of L-sided uncal herniation</p>	<p><b>Px:</b> Left Hearing Loss After Assault → localised bruising at back of mastoid</p> <p><b>Dx: Base of skull fracture</b></p> <p><b>Desc:</b> Non-contrast axial CT scan in bone window showing an oblique fracture of the left petrous portion of the temporal bone with Ethmoid air sinus damage</p>
			
<p><b>Px:</b> Intermittent Limb Paraesthesia, Fatigue, Visual Loss</p> <p><b>Dx: Multiple sclerosis</b></p> <p><b>Desc:</b> Non-contrast axial MRI brain depicting multiple areas of hyperintense regions of white matter bilaterally especially at <b>Juxtacortical, periventricular and corpus callosum regions</b></p>	<p><b>Px:</b> 4 Months Headache And New Seizures</p> <p><b>Dx: Diffuse astrocytoma: WHO II</b></p> <p><b>Desc:</b> Non-contrast axial MRI brain scan showing a large left sided hyperintense temporal lobe associated with oedema centred in white matter. To confirm, use post-gadolinium image and lesion should NOT enhance</p>	<p><b>Px:</b> Headache For 3 Months, Confusion, Incontinence</p> <p><b>Dx: Glioblastoma: WHO IV</b></p> <p><b>Desc:</b> Axial MRI depicting <b>intra-axial</b> poorly demarcated ring enhancing lesion/mass across the midline in both frontal lobes. There is a heterogenous hyperintense oedematous region around these lesions.</p>	<p><b>Dx: Metastatic lung cancer into brain</b></p> <p><b>Desc:</b> Contrast axial CT and MRI brain scans depicting ring-enhancing lesion in the left frontal lobe with surrounding homogenous poorly demarcated area of <b>disproportionate oedema</b> *CXR confirmation of apical lung tumour metastasis.</p>
			
<p><b>Px:</b> Intractable Seizures And Developmental Delay</p> <p><b>Dx: Migrational anomaly ( grey matter heterotopia)</b></p> <p><b>Desc:</b> Coronal brain MRI showing possible migrational anomaly with heterogenous basal ganglia gray matter near the lateral ventricles in abnormal anatomical location.</p>	<p><b>Px:</b> Sudden Onset Left Weakness</p> <p><b>Dx: MCA occlusion</b></p> <p><b>Desc:</b> Non-contrast axial CT scan showing a hyperattenuating lesion suggestive a right MCA occlusion</p>	<p><b>Px:</b> Neck Pain, Ataxia, Upper Limb Weakness</p> <p><b>Dx: Spinal cord compression</b></p> <p><b>Desc:</b> Sagittal MRI of the cervical spine showing effacement of CSF anteriorly and posteriorly from C3-C6 and <b>spinal cord oedema (needs surgical intervention)</b></p>	<p><b>Px:</b> Hit By Falling Object From Building</p> <p><b>Dx: Multiple Fracture of C1 (Jefferson Fracture)</b></p> <p><b>Desc:</b> Axial CT of cervical spine showing multiple fractures of the atlas/C1 along the posterior and anterior arch.</p> <p>Nb: Single fracture of posterior arch may be congenital and physiological</p>

# HEADACHES



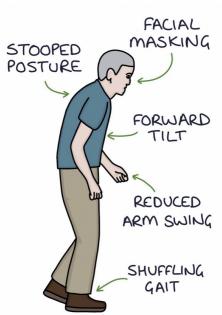
	Site	Onset/Timing	Assoc.	Treatment
<b>Common Migraine</b> [no aura]	<b>Unilateral</b>	Prodrome (3/7 before) Aura (lasts 60 mins) headache(4-72 hrs) resolution Recovery / post-drome	<ul style="list-style-type: none"> <li>Pulsatile headache</li> <li>4-72 hours duration</li> <li>Unilateral, not bilateral</li> <li>Nausea and / or vomiting &amp; mental clouding</li> <li>Disabling headache</li> </ul>	<b>Acute Mx:</b> <ul style="list-style-type: none"> <li>paracetamol</li> <li>Triptans → 50mg sumi</li> <li>NSAIDs</li> <li>Anti-emetics (maxolon)</li> </ul> <b>Long-term Mx:</b> <ul style="list-style-type: none"> <li>Avoid trigger (stress, lights, smells, dehydration, choc, citric acid, poor sleep)</li> <li>CBT</li> <li>Headache diary</li> <li>Relaxation (massage)</li> <li><b>Vitamin B<sub>2</sub> (riboflavin)</b> – reduce freq. + severity</li> <li><b>Amitriptyline (TCA)</b> but AE – fatigue, dizzy, depression, insomnia</li> <li><b>Prophylaxis (with Panadol + propranolol)</b></li> </ul>
<b>Classical Migraine Headache (+aura)</b>	<b>Unilateral</b>	Gradual onset <1 hr	<ul style="list-style-type: none"> <li>Females (20%)</li> <li>Preceded by aura -sparks, blurry, lines across vision or loss of visual fields</li> <li><b>Photophobia, phonophobia</b></li> <li>Visual scotoma, scintillations,</li> <li>Disequilibrium</li> </ul>	
<b>Hemiplegic migraine</b>	<b>Unilateral</b>	Sudden or gradual	<ul style="list-style-type: none"> <li>Hemiplegia (weakness + migraine)</li> <li>Ataxia</li> <li>Altered level of consciousness</li> </ul>	
<b>Tension-Type Headache</b>	<b>Bilateral</b> (bitemporal, bifrontal)	Recurrent/daily with variable duration (30 mins – 7days )	<ul style="list-style-type: none"> <li>worse by walking</li> <li>Tight dull aching pressure headache</li> <li>Trigger = A+D, alcohol, dehydrated, skip meals, dehydration</li> </ul>	<ul style="list-style-type: none"> <li>Reassure</li> <li>Basic analgesia</li> <li>Relaxation techniques</li> <li>Hot towels to local area</li> </ul>
<b>Cluster Headache</b> ('Alarm Clock')	<b>Unilateral 10/10</b> (e.g. one eye) (15mins-3hrs)  "trigger = EtOH, strong smell, exercise"	Wake from sleep <b>same time each day</b>  	<ul style="list-style-type: none"> <li>30-50yo Men (+ smoking!)</li> <li>Attacks of severe orbital/ supraorbital pain (3-4x attacks per day for weeks followed by pain free period lasting 1-2 years)</li> <li><b>ANS</b> issues (Lacrimation, rhinorrhoea)</li> <li>Agitation</li> <li>At least 8x/day</li> <li>Alternate sides</li> <li>Akathisia</li> <li>Anxiety</li> <li>At-risk suicide</li> </ul>	<ul style="list-style-type: none"> <li><b>100% High flow FiO<sub>2</sub> [BEST]</b></li> <li><b>Triptans</b> (8mg sumi SC)</li> </ul> <p>Prophylaxis:</p> <ul style="list-style-type: none"> <li>Verapamil</li> <li>Lithium</li> <li>Prednisone for 2-3wks to break cycle</li> </ul>
<b>Temporal Arteritis</b> [Giant cell arteritis - GCA]	<b>"Unilateral</b> over temporal area	Persistent [mild and severe]	<ul style="list-style-type: none"> <li><b>Polymyalgia rheumatica</b> (common cause) → features shoulder and hip girdle pain + (RAISED ESR)</li> <li>&gt; 50 → tender <u>temporal artery</u></li> <li><b>Vision loss [worst outcome]</b></li> <li><b>Locked Jaw stiffness/pain</b> esp. when eating → weight loss</li> </ul>	<ul style="list-style-type: none"> <li>Prednisone</li> <li>Rheumatological review</li> </ul>
<b>Subarachnoid Haemorrhage</b>	initially localised then generalised	<b>Sudden onset</b> "thunderclap"	<ul style="list-style-type: none"> <li>Worst ever headache 10/10</li> <li>neck stiffness</li> </ul>	Surgery clipping of ruptured aneurysm
<b>Reversible cerebral vasoconstriction syndrome (RCVS)</b>	sudden constriction (tightening) of the vessels that supply blood to the brain		<ul style="list-style-type: none"> <li>hemiparesis</li> <li>receptive and expressive dysphasia</li> <li>altered vision</li> </ul>	<b>NONE</b> <ul style="list-style-type: none"> <li>CTA = string and bead appearance</li> </ul>
<b>IIH</b>	Bilateral	Insidious onset	<ul style="list-style-type: none"> <li>Obese young women of child-bearing age</li> <li>Papilloedema (fundoscopy)</li> </ul>	<ul style="list-style-type: none"> <li>LP to relieve pressure if no tumour present</li> </ul>
<b>Posterior reversible encephalopathy syndrome</b>	Due to HT, CKD, pre-eclampsia	Insidious rapid onset	<ul style="list-style-type: none"> <li>Headache</li> <li>Seizures</li> <li>altered consciousness,</li> <li>visual disturbance</li> </ul>	Short-term management of seizures using anti-epileptic drugs
<b>Subdural / epidural haemorrhage</b>	Site of head trauma	Freq. + severity increases over weeks	After head trauma	Surgical evacuation of haematoma
<b>Acute Sinusitis</b>	<b>Unilateral/ Bilateral</b>	Preceding infection	Facial pain + Forehead flush when bending down (better in evening)	<ul style="list-style-type: none"> <li>NSAIDs, decongestants, nasal sprays, hydration</li> <li>resolves within 2-3 wks</li> </ul>
<b>Meningitis</b> [like migraines]	General headache	Gradual + infection	<ul style="list-style-type: none"> <li>Photophobia/phonophobia + <b>fever</b> + neck stiffness</li> <li>+/- HIV or cancer</li> </ul>	<ul style="list-style-type: none"> <li>Bacterial = vancomycin</li> <li>Viral = antivirals</li> </ul>
<b>Raised ICP</b>	General Pressure headache	AM headache (after exertion)	<ul style="list-style-type: none"> <li><b>CUSHING'S TRIAD:</b> widened PP, bradycardia and irregular RR</li> </ul>	<ul style="list-style-type: none"> <li>Non-contrast CT</li> <li>MRI</li> </ul>

			<ul style="list-style-type: none"> <li>• drowsiness or vomiting</li> <li>• Papilledema (if severe)</li> <li>• <b>Basal skull # (Raccoon's eyes and Battle' sign)</b></li> </ul>	<ul style="list-style-type: none"> <li>• LP (if no tumour)</li> </ul>
<b>Low CSF pressure</b>	Trauma site → CSF leak through tear or cribriform fracture	Worse in evening	<ul style="list-style-type: none"> <li>• Positional exacerbation (most severe when upright)</li> <li>• Similar to post-dural puncture headache</li> </ul>	<ul style="list-style-type: none"> <li>• Bed rest</li> <li>• Caffeine</li> <li>• Increased fluid intake</li> </ul>
<b>Tolosa Hunt syndrome</b>	Unilateral orbital pain [CNIII, CNIV and CNVI]		<ul style="list-style-type: none"> <li>• Paresis/damage to CNIII, CNIV and CNVI → due to granulomatous inflammation of cavernous sinus, SOF or orbit</li> <li>• Reduced and painful eye movement</li> </ul>	High-dose glucocorticoid (response in 24-72 hrs)
<b>Trigeminal neuralgia /injury</b> 	<b>V1, V2, V3 dermatomes</b> [vascular compression of CNV nerve root → usually (supra/infraorbital branch)]	<b>Recurrent</b> (secs and mins) <b>Sudden onset</b> (short lasting)	<ul style="list-style-type: none"> <li>• Pain on brushing teeth, eating</li> <li>• <b>Excruciating Electric +++ shocks on face</b></li> <li>• <b>Allodynia</b>,</li> <li>• May be Herpes Zoster</li> </ul> <p>Triggers:</p> <ul style="list-style-type: none"> <li>➢ Cold weather</li> <li>➢ Spicy food</li> <li>➢ Citrus fruits</li> </ul>	<ul style="list-style-type: none"> <li>• <b>1<sup>st</sup> line = carbamazepine</b></li> <li>• Surgical nerve decompression</li> <li>• Post-herpes = gabapentin, lyrica, carbamazepine</li> <li>• CaB (verapamil) = prophylaxis</li> </ul>
<b>Occipital neuralgia</b>	<b>Site of nerve injury [greater occipital nerve]</b>	Subacute	<ul style="list-style-type: none"> <li>• Aching, pressure, stabbing or throbbing pain</li> <li>• Pain reproduced with digital pressure over greater occipital nerve (rad. Ipsilateral fronto-orbital region)</li> </ul>	<ul style="list-style-type: none"> <li>• <b>Massage</b></li> <li>• <b>Heat</b></li> <li>• <b>Nerve block</b></li> </ul>
<b>Concussion</b>	TBI	Sudden (within 1 <sup>st</sup> 24 hrs)	<ul style="list-style-type: none"> <li>• headache,</li> <li>• FND – speech, weakness, sensation</li> <li>• Head and neck injury</li> </ul> <p><b>How to investigate?</b></p> <ul style="list-style-type: none"> <li>• Exclude ICH, SDH, SAH =</li> <li>• MRI &gt; CT</li> <li>• F/U within 1 week (as neurophysiological changes may take 30 days to occur)</li> </ul>	<p><b>Educate</b></p> <ul style="list-style-type: none"> <li>➢ <b>Simple analgesia</b></li> <li>➢ <b>No ETOH, driving</b></li> </ul> <p><b>When to refer?</b></p> <ul style="list-style-type: none"> <li>➢ Insight loss</li> <li>➢ Mood + memory change</li> <li>➢ CTE – chronic traumatic encephalopathy (footballer)</li> </ul>
<b>Post-concussion syndrome (PCS) after traumatic brain injury (TBI)</b>	Worse in those with mild head injury compared to severe trauma	<b>&gt; 2 weeks after concussion</b> [greatest within 7-10 days]	<ul style="list-style-type: none"> <li>• headache,</li> <li>• dizziness,</li> <li>• neuropsychiatric symptoms,</li> <li>• cognitive impairment</li> </ul>	<ul style="list-style-type: none"> <li>• CBT</li> <li>• Antidepressants?</li> </ul>
<b>TMJ injury</b>	Site of trauma		<ul style="list-style-type: none"> <li>• Jaw claudication</li> <li>• Ipsilateral hemicranial aching</li> </ul>	<ul style="list-style-type: none"> <li>• NSAIDs</li> <li>• TCAs</li> </ul>
<b>Cervical spondylosis</b>	Degenerative change in cervical spine		<ul style="list-style-type: none"> <li>• Neck pain worse on movement</li> </ul>	<ul style="list-style-type: none"> <li>• CT and MRI spine to exclude nerve root lesions</li> </ul>
<b>SUNCT</b>	unilateral	short-lasting [+/- triggered by skin contact]	<ul style="list-style-type: none"> <li>• <b>short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing</b></li> <li>• <b>SUNA =</b>if autonomic symptoms (e.g. lacrimation,</li> </ul>	<ul style="list-style-type: none"> <li>• <b>Gabapentin</b></li> </ul>
<b>Psychologically induced headache</b>	Generalised		<ul style="list-style-type: none"> <li>• Insomnia (daytime somnolence)</li> <li>• OSA</li> <li>• Cannot tolerate noise or large crowds</li> <li>• Assoc. with PTSD, acute stress, panic disorder, depression</li> </ul>	<ul style="list-style-type: none"> <li>• Address stresses in life</li> </ul>
<b>Dysesthesias</b> (abnormal sensations)	site of scalp laceration /soft tissue injury	Persist over months	<ul style="list-style-type: none"> <li>• Aching, sore, tingling or shooting pain</li> </ul>	<ul style="list-style-type: none"> <li>• <b>Gabapentin</b></li> <li>• <b>Pregabalin</b></li> <li>• <b>carbamazepine</b></li> </ul>
<b>Coital Headache</b>	Generalised	Sudden onset during sex [esp. Middle-aged men]	<b>NOT</b> SAH [no nausea or neck stiffness]	<ul style="list-style-type: none"> <li>• indomethacin <b>BEFORE</b> sex</li> </ul>
<b>Hormonal headache</b>		2 days before and first 3 days of menstrual period OR Around menopause	<p>Non-specific TTH</p> <ul style="list-style-type: none"> <li>➢ assoc. with <b>low estrogen</b></li> </ul>	<ul style="list-style-type: none"> <li>• OCP</li> <li>• Mirena</li> </ul>
<b>Analgesia Headache</b>	Generalised		Long-term or XS analgesia usage (Analgesic overuse (i.e. Panadol & codeine))	<ul style="list-style-type: none"> <li>• Psych counsel</li> <li>• Slow wean</li> </ul>

# EPILEPSY

	EPILEPSY				
Def	<ul style="list-style-type: none"> <li>Umbrella term for condition where there is a tendency to have seizures</li> <li>Seizure = transient episodes of abnormal electrical activity in brain</li> </ul>				
ix	<b>General Bloods</b> <ul style="list-style-type: none"> <li>Postural BP</li> <li>Capillary glucose &amp; Hb<sub>a1C</sub> (exc. hypoglycaemia)</li> <li>EUC + VBG (measure lactate + acidosis + BSL)           <ul style="list-style-type: none"> <li>?hypoBSL, hypoCa, hyperUrea</li> </ul> </li> <li><b>CK &amp; Prolactin</b> (both elevated in generalised tonic-clonic seizures)</li> <li>ACE (sarcoidosis)</li> <li>Urine MCS &amp; drug screen</li> <li>B<sub>12</sub>/Folate (reduced = encephalopathy)</li> <li>Fe (haemochromatosis – Fe deposition in brain)</li> <li>ESR/CRP (septic)</li> <li>Cholesterol (risk of stroke)</li> <li>Autoimmune panel, HIV/Viral panel, acanthocytes, porphyrins</li> </ul>	<b>Imaging</b> <ul style="list-style-type: none"> <li>MRI → visualise brain structure = Sol, infarcts</li> <li>MRA <i>Magnetic resonance angiography</i> → vasospasms, RCVS</li> <li>MRV <i>Magnetic resonance venography</i> → venous thromboembolisms (esp. SSS, transverse sinus)</li> <li>CT/CXR → check for possible fractures</li> <li>EEG → DDX: idiopathic generalized epilepsy from PNES,           <ul style="list-style-type: none"> <li><b>NB: low sensitivity/specificity</b> → abnormal EEG does NOT indicate brain pathology NOR does a normal EEG rule out epilepsy/seizure disorder</li> </ul> </li> </ul>	<b>Other</b> <ul style="list-style-type: none"> <li>ECG (prolonged QT interval)</li> <li>LP if meningitis/encephalitis suspected</li> <li><b>Neurology referral if:</b> <ul style="list-style-type: none"> <li>1<sup>st</sup> seizure → most epilepsies treatable with 1<sup>st</sup> agent</li> <li>Refractory epilepsy</li> <li>Surgery</li> </ul> </li> </ul>		
Types	<b>Description</b>		Duration	1 <sup>st</sup> line med	2 <sup>nd</sup> line med
	<b>Focal seizures</b> <ul style="list-style-type: none"> <li><b>Begin in temporal lobe:</b> <ul style="list-style-type: none"> <li>Hallucinations, memory flash backs</li> <li>Déjà vu</li> <li>Automatism = strange actions on autopilot</li> </ul> </li> </ul>		30-180s	Lamotrigine Carbamazepine	<b>Sodium valproate</b> Levetiracetam
	<b>Generalised tonic-clonic "Grand mal"</b> <ul style="list-style-type: none"> <li>LOC + tonic-clonic jerks (tonic before clonic)</li> <li>WILD – post-ictal period</li> </ul>		3-5mins	<b>Sodium valproate</b>	Lamotrigine Carbamazepine
	<b>Absence "petit mal"</b> <ul style="list-style-type: none"> <li><b>Typically children</b></li> <li>Typical = Blank stare into space ONLY</li> <li>Atypical = plus automatisms</li> </ul>		< 10s	<b>Sodium valproate</b>	Ethosuximide
	<b>Atonic "drop attacks"</b> <ul style="list-style-type: none"> <li>"drop attacks" = brief lapses in muscle tone</li> <li>Seen in Lennox-Gestaut syndrome</li> </ul>		< 3mins	<b>Sodium valproate</b>	Lamotrigine
	<b>Myoclonic seizures</b> <ul style="list-style-type: none"> <li>Sudden brief muscle contraction like a "sudden jump"</li> <li>Remains conscious</li> <li>Seen in juvenile myoclonic epilepsy</li> <li>DDx: CJD (mad cow disease)</li> </ul>			<b>Sodium valproate</b>	Lamotrigine Levetiracetam Topiramate
	<b>Infantile spasms (west syndrome)</b> <ul style="list-style-type: none"> <li>Rare (1 in 4000)</li> <li>Infancy → 6/12 old</li> <li>Full body spasms</li> <li>Bad prognosis – 1/3<sup>rd</sup> die by age 25</li> </ul>			Prednisone Vigabatrin	
	<b>Status epilepticus (medical emergency)</b> <ul style="list-style-type: none"> <li><b>Defined as any seizure that:</b> <ul style="list-style-type: none"> <li>Lasts longer than 5 mins OR</li> <li>More than 3 seizures in 1 hour</li> </ul> </li> <li><b>Stages:</b> <ul style="list-style-type: none"> <li>stage 1 = acidosis</li> <li>stage 2 = hypoglycaemia</li> </ul> </li> </ul>		>5mins	<b>ABCDE</b> <ol style="list-style-type: none"> <li>Secure airway</li> <li>High FIO<sub>2</sub></li> <li>Assess CV and Resp</li> <li>Check BSL</li> <li>IV lorazepam 4mg or 0.3mg/kg buccal or intranasal midazolam (repeat after 5 mins if seizure persists)</li> <li>Switch to IV 20mg/kg phenytoin (if seizure persists at 15 mins)</li> </ol>	*Can use buccal midazolam or PR diazepam (in community) → flumazenil (antidote to midazolam)
	<b>Non-epileptic seizures (PNES)</b> <ul style="list-style-type: none"> <li>Type of conversion disorder</li> <li>May be caused by Factitious disorder and malingering where patient is purposely deceiving the physician</li> <li><b>NO AUTOMATISM, NO TONGUE BITING AND LASTS OVERLY LONG!</b></li> </ul>		5-10mins	About 9% to 15% of patients with psychogenic events have coexistent seizure disorders ➤ Psych counselling	
A/E of meds	Sodium valproate	Carbamazepine	Ethosuximide	Lamotrigine	Phenytoin
	Increases GABA activity inhibiting GABA transaminase	Na channel blocker	Ca channel blocker	Na channel blocker	Na channel blocker
Lifestyle impacts & legal considerations	<ul style="list-style-type: none"> <li>Teratogenic so patients need careful advice about contraception</li> <li>Liver damage and hepatitis</li> <li>Hair loss</li> <li>Tremor</li> </ul>	<ul style="list-style-type: none"> <li>Agranulocytosis</li> <li>Aplastic anaemia</li> <li>Induces the P450 system so there are many drug interactions</li> <li>SJS (if mutant HLA B1502)</li> </ul>	<ul style="list-style-type: none"> <li>Night terrors</li> <li>Rashes</li> </ul>	<ul style="list-style-type: none"> <li>Stevens-Johnson syndrome or DRESS syndrome. These are life threatening skin rashes.</li> <li>Leukopenia Nb: hormonal agents reduce effectiveness</li> </ul>	<ul style="list-style-type: none"> <li>Folate and vitamin D deficiency</li> <li>Megaloblastic anaemia (folate deficiency)</li> <li>Osteomalacia (vitamin D deficiency)</li> <li>Affect cerebellar function</li> </ul>
	<ul style="list-style-type: none"> <li><b>Driving (no driving for 6/12)</b></li> <li><b>No swimming &amp; driving (loss of independence)</b>, limited options for occupation/work           <ul style="list-style-type: none"> <li>Doctors responsible to encourage patient to self-notify RMS [but can legally notify RMS without consent]</li> </ul> </li> <li><b>Need for counselling</b> (explaining that it is a medical condition that was out of anyone's control)           <ul style="list-style-type: none"> <li>Seizure action plan → call OOO → keep attended and away from danger (administer benzo ASAP)</li> </ul> </li> <li><b>Medical costs</b> (i.e. consultations and medications) → (aim to wean off AEDs after ≥2 year seizure free period)           <ul style="list-style-type: none"> <li>If frequent → carer requirements</li> </ul> </li> <li><b>Disruptions to social life</b> → encouraging isolated, anxiety and depressive behaviour (also personality changes)</li> <li><b>Fear of losing control and hurting one's self during an episode</b> (esp. falls) with subsequent comp. (e.g. fractures, bruises, hemorrhages if brain trauma)</li> </ul>				

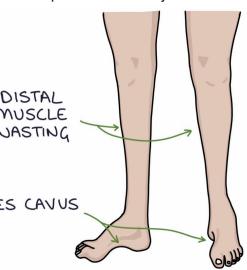
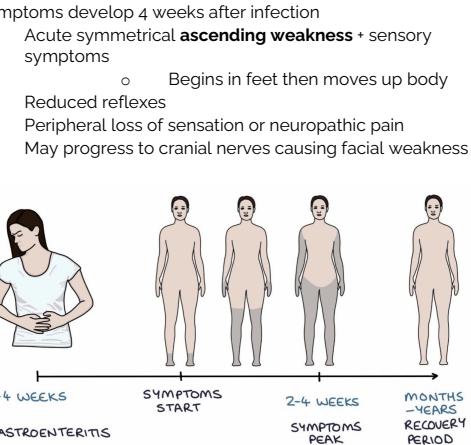
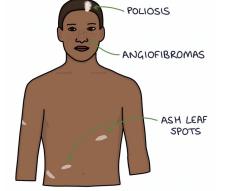
# MS, MND, Parkinson's

	MULTIPLE SCLEROSIS	MOTOR NEURON DISEASE	PARKINSON'S DISEASE																														
Def	<ul style="list-style-type: none"> <li>Chronic progressive autoimmune demyelination of nerves in CNS by myelin-reactive T cells</li> </ul>	<p><b>MND</b> = umbrella term that encompasses progressive deterioration of UMN and LMN with NO effect on sensation</p>	Degeneration of the dopaminergic neurons in the pars compacta in the substantia nigra and basal ganglia																														
Types	<p><u>Types:</u></p> <ol style="list-style-type: none"> <li><b>Clinically isolated</b> (not disseminated in time and space) = 1<sup>st</sup> MS attack</li> <li><b>Relapse and remitting (most common)</b></li> <li><b>Secondary progressive</b> – initially relapse and remitting but now symptoms worsen and more permanent</li> <li><b>Primary progressive</b> – worsening Sx from point of diagnosis (no relapse or remitting period)</li> </ol>	<p><u>Types</u></p> <ul style="list-style-type: none"> <li><b>ALS</b> (UMN + LMN signs) = most common (Stephen Hawking)</li> <li><b>Progressive bulbar palsy</b> (CNIX, X, XI, XII) = 2<sup>nd</sup> most common</li> <li><b>Progressive muscular atrophy</b> (LMN, anterior horn of medulla)</li> <li><b>Primary lateral sclerosis -rare</b> (UMN, <b>BETZ motor neurons</b> → CST degeneration)</li> </ul>	<p><b>Types: Parkinson-PLUS syndromes</b></p> <ul style="list-style-type: none"> <li><b>Multiple system atrophy (rare)</b> – neurons in multiple areas of brain degenerate <ul style="list-style-type: none"> <li><b>ANS dyfn</b> = postural HypoTN (falls risk), constipation, unregulated sweating, sexual dyfn</li> <li><b>Cerebellar dyfn (ataxia)</b></li> </ul> </li> <li><b>Lewy Body dementia</b> <ul style="list-style-type: none"> <li><b>Progressive cognitive decline</b></li> <li><b>Visual hallucinations</b></li> <li><b>REM sleep disorder (e.g. kicking in bed)</b></li> <li><b>Fluctuating consciousness</b></li> <li><b>Delusions</b></li> </ul> </li> <li><b>Progressive supranuclear palsy</b> =</li> </ul>																														
Cause	<ul style="list-style-type: none"> <li>Multiple GENES</li> <li>EBV</li> <li>Low vitamin D</li> <li>Smoking</li> <li>Obesity</li> <li><b>Risks:</b> Female, &lt;50, obese, northern hemisphere</li> <li><b>Protective:</b> pregnancy, early age, breastfeeding</li> </ul>	<ul style="list-style-type: none"> <li>FHx <ul style="list-style-type: none"> <li>Genes = SOD1, ALS2, SETX</li> </ul> </li> <li>Smoking</li> <li>Heavy metals</li> <li>Pesticides</li> </ul>	<ul style="list-style-type: none"> <li>Idiopathic</li> </ul>																														
S+S	<p>Symptoms &gt; 24 hrs (lasting days to weeks before improving)</p> <ol style="list-style-type: none"> <li><b>Optic neuritis</b> – new onset unilateral vision loss and colour vision + painful eye movements + RAFD</li> <li><b>6<sup>th</sup> CN palsy</b> = Internuclear ophthalmoplegia (MLF lesion) and conjugate lateral gaze palsy</li> <li><b>Focal weakness</b> = Bell's, Horner's, limb paralysis, incontinence</li> <li><b>Focal sensory</b> = trigeminal neuralgia, numb, parasthesia, <b>Lhermitte's sign</b> = shooting pain down spine on neck flexion</li> <li><b>Sensory ataxia</b> = loss of proprioception (Romberg's +)</li> <li><b>Cerebellar ataxia</b></li> </ol>	<p><u>UMN signs:</u></p> <ul style="list-style-type: none"> <li>Spasticity, rigidity, hyperreflexia</li> </ul> <p><u>LMN signs</u></p> <ul style="list-style-type: none"> <li>Muscle wasting, reduced tone, hyporeflexia, fasciculations</li> </ul> <p><u>Key signs:</u></p> <ol style="list-style-type: none"> <li><b>Eyes spared (ALL)</b></li> <li><b>sensation spared (ALL)</b></li> <li><b>ALS</b> – progressive paralysis</li> <li><b>progressive bulbar palsy</b> = dysphagia and dysphonia</li> <li><b>Progressive muscular atrophy</b> = clumsy hand movements, fasciculations</li> <li><b>Primary lateral sclerosis</b> = akinesia</li> </ol>	<p><b>ASSYMETRICAL MOTOR SYMPTOMS</b></p> <ul style="list-style-type: none"> <li>Unilateral resting Pill-rolling tremor (4-6Hz) – improves on movement</li> <li>Rigidity "cogwheel"</li> <li>Akinesia – "shuffling gait", hard to start walking, smaller and smaller handwriting, reduced facial expression (<b>hypomimia</b>)</li> <li>Postural instability</li> <li>"glabellar tap" = Meyerson's sign "eyes shut when tapped lightly between eyebrows"</li> </ul> <p><b>Non-motor symptoms:</b></p> <ul style="list-style-type: none"> <li>Depression</li> <li>Insomnia</li> <li>Anosmia</li> <li>Cognitive issues and memory problems</li> </ul> 																														
IX	<p><b>McDonald's criteria</b></p> <ul style="list-style-type: none"> <li>≥ 2 attacks + ≥ 2 lesions w/ objective evidence <b>OR</b></li> <li>1 lesion + clinical Hx suggesting previous lesion <b>OR</b></li> <li>dissemination in space on MRI</li> </ul> <p><b>MRI</b></p> <ul style="list-style-type: none"> <li>T1 = atrophy &amp; axonal death</li> <li>T2 = Hyperintense fingerprints</li> </ul> <p><b>CSF</b> = +ve oligoclonal IgG bands</p> <p><b>Autoantibody test</b> (Serum aquaporin 4) = marker for neuromyelitis optica</p>	<p>Diagnosis based on clinical presentation and exclusion of other causes</p> <ul style="list-style-type: none"> <li>Formal diagnosis by neurologist</li> </ul>	<p>Diagnosis based on clinical presentation and exclusion of other causes</p> <ul style="list-style-type: none"> <li>Formal diagnosis by neurologist</li> </ul>																														
Comp.	<ul style="list-style-type: none"> <li><b>Death</b></li> </ul>	<p>Death – respiratory failure or pneumonia</p>																															
Rx	<p><b>MDT approach</b> neurologist, specialist nurse, PT, OT</p> <p><b>Lifestyle:</b></p> <ul style="list-style-type: none"> <li>Stop smoking + vit D supp.</li> <li>Regular exercise</li> </ul> <p><b>DMARDs and biologics</b></p> <ul style="list-style-type: none"> <li>Rituximab</li> <li>Fingolimod</li> </ul> <p><b>Symptom Mx:</b></p> <ul style="list-style-type: none"> <li><b>Gabapentin</b> (neuropathic pain)</li> <li><b>SSRI</b> (depression)</li> <li><b>Ant-chol</b> (urge incontinence)</li> <li><b>Baclofen, Gabapentin</b> (spasticity, painful cramps)</li> </ul> <p><b>Avoid giving vaccines during attacks</b></p>	<p>No cure</p> <ul style="list-style-type: none"> <li>Palliative care <ul style="list-style-type: none"> <li>Organise ACD</li> <li>End-of life care planning</li> </ul> </li> <li>Symptom management</li> <li><b>Experimental drugs to slow progression:</b> <ul style="list-style-type: none"> <li>Riluzole (UK)</li> <li>EdaravOne (US)</li> </ul> </li> </ul>	<p><b>MDT approach (NO cure)</b> neurologist, specialist nurse</p> <table border="1"> <thead> <tr> <th>Drugs</th> <th>Class</th> <th>MoA</th> <th>Indication</th> <th>Adverse Effects</th> </tr> </thead> <tbody> <tr> <td>L-DOPA (Levodopa)</td> <td>Natural dop precursor</td> <td>Crosses BBB → <b>BUT</b> broken down outside BBB by AACD or COMT</td> <td>1<sup>st</sup> line (useless after 5 years)</td> <td> <p><b>Dyskinesias (XS motor activity):</b></p> <ul style="list-style-type: none"> <li>Dystonia – abnormal postures</li> <li>Chorea / invol. jerks</li> <li>Athetosis – twisting/ writhing fingers, feet</li> </ul> </td></tr> <tr> <td><b>Carbidopa</b></td> <td>AACD inhibitor</td> <td>Block L-DOPA breakdown in periphery extend duration [<b>cannot cross BBB</b>]</td> <td>Combined with L-Dopa + sinemet</td> <td></td></tr> <tr> <td><b>Entacopone</b></td> <td>COMT inhibitor</td> <td></td> <td>Combined with L-Dopa</td> <td></td></tr> <tr> <td><b>Selegiline, Rasagiline</b></td> <td>Irreversible MAO inhibitor</td> <td>Prevent dopamine breakdown</td> <td>Used early to delay L-DOPA use OR combined with L-DOPA</td> <td>Selegiline is metabolized to methamphetamine = XS SNS activity</td></tr> <tr> <td><b>Bromocryptine, pramipexole</b></td> <td>D2/D3 agonists</td> <td>Directly stimulate dopamine receptors</td> <td></td> <td><b>Pulm. Fibrosis (long-term use)</b></td></tr> </tbody> </table>	Drugs	Class	MoA	Indication	Adverse Effects	L-DOPA (Levodopa)	Natural dop precursor	Crosses BBB → <b>BUT</b> broken down outside BBB by AACD or COMT	1 <sup>st</sup> line (useless after 5 years)	<p><b>Dyskinesias (XS motor activity):</b></p> <ul style="list-style-type: none"> <li>Dystonia – abnormal postures</li> <li>Chorea / invol. jerks</li> <li>Athetosis – twisting/ writhing fingers, feet</li> </ul>	<b>Carbidopa</b>	AACD inhibitor	Block L-DOPA breakdown in periphery extend duration [ <b>cannot cross BBB</b> ]	Combined with L-Dopa + sinemet		<b>Entacopone</b>	COMT inhibitor		Combined with L-Dopa		<b>Selegiline, Rasagiline</b>	Irreversible MAO inhibitor	Prevent dopamine breakdown	Used early to delay L-DOPA use OR combined with L-DOPA	Selegiline is metabolized to methamphetamine = XS SNS activity	<b>Bromocryptine, pramipexole</b>	D2/D3 agonists	Directly stimulate dopamine receptors		<b>Pulm. Fibrosis (long-term use)</b>
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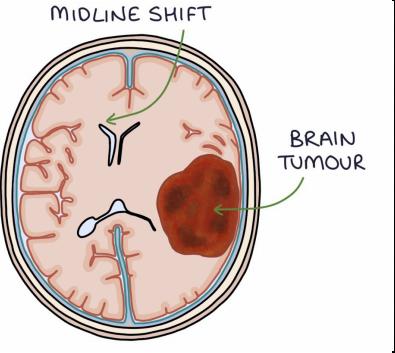
## RARE NEUROLOGICAL DISEASES

	Huntington's chorea	Myasthenia Gravis	Lambert Eaton Myasthenic syndrome	Facial Nerve Palsy	Neuropathic Pain
Cause	<p>Genetic – autosomal dominant</p> <ul style="list-style-type: none"> <li>➤ <b>Trinucleotide repeat disorder (HTT gene)</b> – 'CAG'</li> <li>➤ <b>Anticipation</b> = successive generations have more repeats in gene → earlier age of onset and increased disease severity</li> </ul>	<ul style="list-style-type: none"> <li>Type 2 Autoimmune attack on ACh receptors in NMJ</li> <li>Progressive muscle weakness across day that improves with rest</li> <li>Affects women at an earlier age than men</li> </ul>	<ul style="list-style-type: none"> <li>Progressive muscle weakness due to antibodies targeting voltage Ca channels on pre-synaptic terminals of LMN</li> </ul>	<p>Dysfunctional facial nerve</p> <ul style="list-style-type: none"> <li>➤ <b>Motor</b> = muscles of facial expression, stapedius, post. digastric stylohyoid, platysma</li> <li>➤ <b>Sensory</b> = taste anterior 2/3<sup>rd</sup></li> <li>➤ <b>PSNS</b> = submandibular, sublingual and lacrimal gland</li> </ul> <p><u>Pathway of facial nerve:</u></p> <ul style="list-style-type: none"> <li>➤ Exit cerebellopontine angle</li> <li>➤ Passes temporal bone</li> <li>➤ Passes parotid gland</li> </ul>	<p><b>Abnormal functioning of sensory nerves causes delivery of painful and abnormal signals to the brain</b></p> <p><b>Causes:</b></p> <ul style="list-style-type: none"> <li>➤ Post-herpetic neuralgia (HZV – shingles)</li> <li>➤ Post-op nerve damage</li> <li>➤ MS</li> <li>➤ Trigeminal neuralgia</li> <li>➤ Complex regional pain syndrome (CRPS)</li> </ul>
S+S	<p>Asymptomatic until symptoms begin around aged 30-50</p> <ul style="list-style-type: none"> <li>➤ Chorea (involuntary, abnormal movements)</li> <li>➤ Eye movement disorders</li> <li>➤ Dysarthria</li> <li>➤ Dysphagia</li> </ul>	<p>Symptoms <b>affect proximal muscles</b> and small muscles of head and neck:</p> <ul style="list-style-type: none"> <li>➤ Extraocular weakness = diplopia</li> <li>➤ Eyelid weakness = ptosis</li> <li>➤ Facial weakness</li> <li>➤ Dysphagia</li> <li>➤ Slurred speech</li> <li>➤ Jaw fatigue</li> </ul> <p><b>TEST FATIGABILITY BY ASKING PATIENT TO:</b></p> <ul style="list-style-type: none"> <li>➤ GAZE UPWARDS OR abduct shoulder and hold it</li> </ul>	<p>Paraneoplastic syndrome</p> <ul style="list-style-type: none"> <li>➤ SCLC</li> <li>➤ RCC</li> </ul> <p>Symptoms <b>affect proximal muscles</b> and small muscles of head and neck:</p> <ul style="list-style-type: none"> <li>➤ Extraocular weakness - diplopia</li> <li>➤ Eyelid weakness (levator muscle) = ptosis</li> <li>➤ ANS dysfn = dry mouth, blurred vision, ED, dizzy</li> <li>➤ <b>Reduced tendon reflex</b></li> <li>➤ <b>Post-tetanic potentiation</b> → after contracting muscles, reflexes tested after are actually normal</li> </ul>	<ul style="list-style-type: none"> <li>➤ <b>Unilateral</b> facial weakness</li> <li>➤ <b>Ptosis</b></li> <li>➤ <b>Loss of nasolabial fold</b></li> </ul> <p><b>UMN lesion (forehead sparing)</b></p> <ul style="list-style-type: none"> <li>➤ Unilateral = CVA, tumour</li> <li>➤ Bilateral = MND, pseudobulbar palsy</li> </ul> <p><b>LMN lesion</b></p> <ul style="list-style-type: none"> <li>➤ <b>Bell's palsy</b> (HZV) = idiopathic</li> <li>➤ <b>Ramsey Hunt</b> (VZV) = painful vesicular rash</li> <li>➤ <b>Infection</b> (AOM, HIV, lyme, malignant otitis externa)</li> <li>➤ <b>Systemic</b> (Sarcoid, MS, GBS, DM)</li> <li>➤ <b>Tumour</b> (acoustic neuroma, cholesteatoma, parotid tumour)</li> <li>➤ <b>Trauma</b> (post-op, base of skull or temporal bone #)</li> </ul>	<p><b>General Symptoms:</b></p> <ul style="list-style-type: none"> <li>• Burning</li> <li>• Tingling</li> <li>• Parasthesia (pins and needles)</li> <li>• Electric shocks</li> <li>• Loss of touch sensation to affected area</li> </ul> <p><b>Complex regional pain syndrome</b></p> <ul style="list-style-type: none"> <li>➤ Allodynia</li> <li>➤ Skin flushed</li> <li>➤ Temperature change</li> <li>➤ Swelling</li> <li>➤ Abnormal sweating</li> <li>➤ Abnormal hair growth</li> </ul>
Ix	<ul style="list-style-type: none"> <li>➤ <b>Genetic testing</b></li> </ul>	<ul style="list-style-type: none"> <li>➤ Lung function test = forced vital capacity (FVC)</li> <li>➤ ACh-R antibodies (85% patients)</li> <li>➤ Muscle-specific kinase (MuSK) antibodies (10% patients)</li> <li>➤ CT/ MRI thymus</li> </ul> <p><b>EDROPHONIUM</b> test helpful if diagnosis unsure</p> <ul style="list-style-type: none"> <li>- Patient given IV dose of neostigmine (edrophonium Cl) to briefly and temporarily relieve the weakness</li> </ul>	<p>Investigate cause</p> <ul style="list-style-type: none"> <li>➤ CXR</li> <li>➤ HRCT</li> </ul>	<p><b>CLINICAL diagnosis</b></p> <ul style="list-style-type: none"> <li>➤ <b>Brain MRI</b></li> </ul>	<p><b>Clinical diagnosis</b></p>
Comp.	<ul style="list-style-type: none"> <li>➤ Suicide (leading cause of death)</li> <li>➤ Death due to respiratory disease (pneumonia)</li> </ul>	<ol style="list-style-type: none"> <li>1. Strong link to <b>thymomas</b> (20-40%) and <b>converse is true</b> (i.e. those with thymomas are more likely to develop MG)</li> <li>2. <b>Myasthenic crisis (life-threatening)</b></li> </ol> <ul style="list-style-type: none"> <li>➤ Triggered by acute illness (e.g. URTI)</li> <li>➤ Leads to respiratory failure due to respiratory fatigue</li> <li>➤ May need PEEP or I+V</li> <li>➤ <b>Rx: IVIg and plasma exchange</b></li> </ul>		<ul style="list-style-type: none"> <li>➤ HSV → meningitis, encephalitis</li> <li>➤ <b>Ramsey Hunt</b> (VZV) = painful vesicular rash may extend to ear canal, pinna and around ipsilateral ear</li> </ul>	<ul style="list-style-type: none"> <li>➤ Reduces QoL</li> <li>➤ Suicide</li> </ul>
Rx	<ul style="list-style-type: none"> <li>➤ <b>No treatment</b> - life expectancy 15-20 years after onset of symptoms</li> </ul> <p><b>MDT approach:</b></p> <ul style="list-style-type: none"> <li>➤ OT, PT and psychologist</li> <li>➤ Speech therapies</li> <li>➤ Genetic counselling (relatives, pregnancy, children)</li> <li>➤ Advanced care planning</li> <li>➤ End of life care planning</li> </ul> <p><b>Symptomatic treatment:</b></p> <ul style="list-style-type: none"> <li>➤ Anti-psychotics (olanzapine)</li> <li>➤ Benzos (diazepam)</li> <li>➤ Dopamine -depleting agents (e.g. tetrabenazine)</li> <li>➤ Antidepressants</li> </ul>	<p><b>Medical treatment:</b></p> <ol style="list-style-type: none"> <li>1. <b>Neostigmine</b> (reversible AChE inhibitors)</li> <li>2. <b>Immunosuppression</b> (e.g. prednisone) – suppress antibody production</li> <li>3. <b>Thymectomy</b> – may improve symptoms even in patients without thymoma</li> </ol> <p><b>Alternatives (MABs):</b></p> <ul style="list-style-type: none"> <li>➤ <b>Rituximab</b> – (anti-CD20) – inhibit B cell antibody production</li> <li>➤ <b>Exulizumab</b> (anti-C5) = prevent complement activation and stop AChR destruction</li> </ul>	<p><b>Medical treatment:</b></p> <ol style="list-style-type: none"> <li>1. <b>Amifampridine</b> (block voltage gated K channels in pre-synaptic terminals) → prolongs depolarization of cell membrane</li> <li>2. <b>IVIg</b></li> <li>3. <b>Plasma exchange</b></li> <li>4. <b>Immunosuppression</b> (e.g. prednisone) – suppress antibody production</li> </ol>	<p><b>For UMN:</b></p> <ul style="list-style-type: none"> <li>➤ Surgical resection if tumour</li> </ul> <p><b>For LMN:</b></p> <ul style="list-style-type: none"> <li>➤ <b>Bell's palsy</b> = start <b>50mg prednisolone PO for 10 day</b> within 72 hours then <b>5 days high does 60 mg</b> before weaning for next 5 days <b>NO acyclovir</b> <ul style="list-style-type: none"> <li>○ Lubricating eye drops to prevent exposure keratopathy</li> <li>○ May take 12 months recovery with some residual weakness</li> </ul> </li> <li>➤ <b>Ramsey Hunt</b> = start <b>prednisolone and acyclovir</b> within 72 hours</li> </ul>	<p><b>1) Referral to pain specialist:</b></p> <p><b>2) medical therapy</b></p> <ul style="list-style-type: none"> <li>➤ Antidepressants (<b>amitriptyline, SNRI</b>)</li> <li>➤ <b>Anti-convulsant</b> (gabapentin, pregabalin)</li> <li>➤ <b>Carbamazepine</b> (if trigeminal neuralgia)</li> </ul>

# RARE NEUROLOGICAL DISEASES

	Charcot Marie-Tooth Disease	Gullian-Barre Syndrome	Neurofibromatosis	Tuberous Sclerosis	ESSENTIAL TREMORS
Cause	<ul style="list-style-type: none"> <li>Inherited – autosomal dominant</li> <li>1 in 2500 people</li> <li>Affects <b>peripheral motor and sensory nerves</b> (dysfunctional myeline)</li> <li></li> </ul>	<ul style="list-style-type: none"> <li>Acute paralytic polyneuropathy that affects PN</li> <li>Triggered by infection (esp. C. jejuni, CMV, EBV)</li> <li>Cause: <b>MOLECULAR MIMICRY</b> ➔ B cells create antibodies against antigens on pathogen from preceding infection. Same antibodies target myeline sheath of LMN</li> </ul>	<ul style="list-style-type: none"> <li>Inherited – autosomal dominant</li> <li>NF1 – chr 17 (neurofibromin -TSP)</li> <li>NF2 – chr 22 (merlin - TSP)</li> </ul>	<ul style="list-style-type: none"> <li>Inherited – autosomal dominant</li> <li>TSC1 = Chr 9 (hamartin)</li> <li>TSC2 = Chr 16 (tuberin)</li> </ul> <p>Abnormal development of hamartomas (benign neoplastic growth of tissue they originate from → skin, brain, lung, heart, kidneys and eyes)</p>	<ul style="list-style-type: none"> <li><b>VERY COMMON</b> condition assoc. with older age</li> <li><b>Fine tremor</b> affecting <b>ALL voluntary muscles</b> (e.g. hand, jaw and vocal tremor)</li> </ul>
S+S	<p>Symptoms develop before 10yo but can be <u>delayed up to 40yo</u></p> <ul style="list-style-type: none"> <li>Pes cavus (high foot arch)</li> <li>Inverted champagne bottle leg (distal muscle wasting)</li> <li>Weak lower leg (loss of ankle DF)</li> <li>Weakness in hands</li> <li>Reduced tendon reflexes</li> <li>Reduced muscle tone</li> <li>Peripheral sensory loss</li> </ul> 	<p>Symptoms develop 4 weeks after infection</p> <ul style="list-style-type: none"> <li>Acute symmetrical <b>ascending weakness</b> + sensory symptoms <ul style="list-style-type: none"> <li>Begins in feet then moves up body</li> </ul> </li> <li>Reduced reflexes</li> <li>Peripheral loss of sensation or neuropathic pain</li> <li>May progress to cranial nerves causing facial weakness</li> </ul> 	<p><b>NF1 criteria (at least 2 out of 7)</b></p> <ul style="list-style-type: none"> <li><b>C – Café-au-lait spots</b> (≥ 6) measuring ≥ 5mm in children or ≥ 15mm in adults</li> <li><b>R – Relative with NF1</b></li> <li><b>A – Axillary or inguinal freckles</b></li> <li><b>BB – Bony dysplasia</b> such as Bowing of a long bone or sphenoid wing dysplasia</li> <li><b>I – Iris hamartomas</b> (≥2x Lisch nodules) = yellow brown spots on the iris</li> <li><b>N – Neurofibromas</b> (2 or more) <b>or</b> 1 plexiform neurofibroma</li> <li><b>G – Glioma of optic nerve</b></li> </ul> <p><b>NF2 classic signs (acoustic neuroma)</b></p> <ul style="list-style-type: none"> <li>Hearing loss</li> <li>Tinnitus</li> <li>Balance problems</li> </ul>	<ul style="list-style-type: none"> <li><b>Ash leaf spots</b> = depigmented areas of skin shaped like an ash leaf</li> <li><b>Shagreen patches</b> = thickened, dimpled, pigmented patches of skin</li> <li><b>Angiofibromas</b> = small skin coloured or pigmented papules on nose and cheeks</li> <li><b>Subungual fibromata</b> = circular painless fibromas growing from the nail bed.</li> <li><b>Cafe-au-lait spots</b> = light brown "coffee &amp; milk" coloured flat pigmented lesions on skin</li> <li><b>Poliosis</b> = isolated patch of white hair on the head, eyebrows, eyelashes or beard</li> </ul> 	<p>Autosomal dominant inheritance</p> <ul style="list-style-type: none"> <li>Fine symmetrical tremor</li> <li>Worse on movement</li> <li>Worse when tired, stressed</li> <li>Improved with alcohol</li> <li>Absent during sleep</li> </ul>
Ix	<p><b>Exclude other causes of peripheral neuropathy:</b></p> <ul style="list-style-type: none"> <li>A) Alcohol</li> <li>B) B12 deficiency</li> <li>C) Cancer or CKD</li> <li>D) Diabetes and drugs (e.g. isoniazid, amiodarone, cisplatin)</li> <li>E) Every vasculitis</li> </ul> <p><b>Supporting Ix:</b></p> <ul style="list-style-type: none"> <li>Nerve conduction studies (reduced signal through nerves)</li> <li>LP in CSF (<b>elevated proteins</b> with normal FBC and BSL)</li> </ul>	<p><b>Brighton criteria for GBS</b></p> <ul style="list-style-type: none"> <li>Bilateral and flaccid limb weakness</li> <li>Loss of deep tendon reflexes</li> <li>CSF cell count (normal) and CSF protein (high)</li> <li>Nerve conduction study consistent with GBS</li> </ul> <p><b>Supporting Ix:</b></p> <ul style="list-style-type: none"> <li>Nerve conduction studies (reduced signal through nerves)</li> <li>LP in CSF (<b>elevated proteins</b> with normal FBC and BSL)</li> </ul>	<p><b>Clinical diagnosis:</b></p> <ul style="list-style-type: none"> <li>Genetic testing</li> <li>XR – to investigate bone pain</li> <li>CT and MRI → check for lesions / masses in brain, SC</li> </ul>	<p><b>Clinical diagnosis:</b></p> <ul style="list-style-type: none"> <li>Genetic testing</li> </ul>	<p><b>DDx:</b></p> <ul style="list-style-type: none"> <li>Parkinson's</li> <li>MS</li> <li>Huntington's</li> <li>Hyperthyroidism</li> <li>Wilson (&lt; 30 yo)</li> <li>Fever</li> <li>Meds (e.g. anti-psychotics)</li> </ul>
Comp.	<ul style="list-style-type: none"> <li>Reduced QoL</li> </ul>	<ul style="list-style-type: none"> <li>80% fully recover</li> <li>15% with some neurological deficit</li> <li>5% die (usually secondary to PE)</li> </ul>	Mainly for NF1 <ul style="list-style-type: none"> <li>Migraines</li> <li>Epilepsy</li> <li>Renal artery stenosis (HTN)</li> <li>ADHD</li> <li>Malignant PNS sheath tumours</li> <li>GIST (sarcoma)</li> <li>++cancers (brain, breast, leukemia, spinal cord)</li> </ul>	<ul style="list-style-type: none"> <li>Epilepsy</li> <li>Learning disability</li> <li>Developmental delay</li> </ul> <p>Extra-organ manifestations:</p> <ul style="list-style-type: none"> <li>Rhabdomyomas in heart</li> <li>Gliomas (tumours in brain and spinal cord)</li> <li>Polycystic kidneys</li> <li>Retinal hamartomas</li> <li>Lymphangioleiomyomatosis (SMC growth in lungs)</li> </ul>	<ul style="list-style-type: none"> <li>Disrupted QoL</li> </ul>
Rx	<p><b>MDT approach (NO cure)</b></p> <ul style="list-style-type: none"> <li><b>Neurologist and geneticist</b> (make dx)</li> <li><b>PT</b> – maintain muscle strength and ROM</li> <li><b>OT</b> – assist ADLs</li> <li><b>Podiatrist</b> – orthoses to improve Sx</li> <li><b>Orthopaedic surgeons</b> – correct disabling joint deformities</li> </ul>	<ul style="list-style-type: none"> <li>IVIg</li> <li>Plasma exchange</li> <li>Supportive care</li> <li>VTE prophylaxis (PE is a leading cause of death)</li> </ul>	<p>No CURE</p> <p><b>NF1:</b></p> <ul style="list-style-type: none"> <li>Control Sx / complications</li> </ul> <p><b>NF2- Schwannomas:</b></p> <ul style="list-style-type: none"> <li>Surgical resection (risk of permanent damage)</li> </ul>	<p>No CURE</p> <ul style="list-style-type: none"> <li><b>SUPPORTIVE MANAGEMENT</b></li> <li>Prevent and treat complications (esp. epilepsy)</li> </ul>	<ul style="list-style-type: none"> <li>No definitive treatment</li> </ul> <p><b>Symptomatic improvement:</b></p> <ul style="list-style-type: none"> <li>Propranolol (non-selective BB)</li> <li>Primidone (barbiturate anti-epileptic)</li> </ul>

# BRAIN TUMOURS

Cause	<p>Abnormal growths in brain that can be benign or malignant</p> <ul style="list-style-type: none"> <li>➤ Benign = meningioma, pilocystic astrocytoma</li> <li>➤ Malignant = glioblastoma, medulloblastoma</li> <li>➤ Secondary METs = lung, breast, RCC, melanoma</li> </ul>	
S+S	<p><u>Asymptomatic (especially when they are small)</u></p> <ul style="list-style-type: none"> <li>➤ <b>FND</b></li> <li>➤ <b>Raised ICP</b> – AM headache, altered mental state, CN palsies (III and VI)</li> <li>➤ <b>Visual disturbances</b> – diplopia</li> <li>➤ <b>Personality change</b> → frontal lobe tumour</li> <li>➤ <b>Headache:</b> <ul style="list-style-type: none"> <li>○ constant,</li> <li>○ nocturnal,</li> <li>○ new</li> <li>○ worse on coughing or straining</li> <li>○ AM headache</li> </ul> </li> <li>➤ <b>Seizures</b> (usu. low grade gliomas)</li> </ul>	<p><b>DDx:</b></p> <ul style="list-style-type: none"> <li>➤ Psychological stress</li> <li>➤ Migraines, cluster</li> <li>➤ Raised ICP - DDx: tumour, ICH, IIH, abscess, infection, hydrocephalus</li> <li>➤ Infection</li> <li>➤ Stroke</li> <li>➤ Temporal arteritis</li> <li>➤ Autoimmune (MS)</li> <li>➤ Drugs (nitrates)</li> </ul>
Ix*	<p><b>Bloods:</b></p> <ul style="list-style-type: none"> <li>➤ FBC (WCC = infection, low plts = haemorrhage)</li> <li>➤ EUC (SIADH)</li> <li>➤ CRP (inflammation)</li> <li>➤ INR - coagulation</li> </ul> <p><b>Imaging:</b></p> <ul style="list-style-type: none"> <li>➤ <b>Non-contrast head CT</b></li> <li>➤ <b>MRI Brain (better delineation)</b></li> <li>➤ <b>PET-CT (mets? - → conduct breast, resp and abdo exam)</b></li> <li>➤ <b>Fundoscopy – papilloedema (optic disc swelling)</b> - <i>When looking for elevation of the optic disc, look at the way the retinal vessels flow across the disc. Vessels are able to flow straight across a flat surface, whereas they will curve over a raised disc.</i></li> </ul> <p><b>Invasive:</b></p> <ul style="list-style-type: none"> <li>➤ <b>Brain biopsy</b></li> <li>➤ <b>Frozen section + biopsy (confirm on the spot)</b> <ul style="list-style-type: none"> <li>○ DDx: is it glioblastoma (resection) or CNS lymphoma (chemo)? (as management is different)</li> </ul> </li> <li>➤ <b>Genetic profiling = p53, MDMT methylation and IDH status</b> <ul style="list-style-type: none"> <li>○ IDH testing → <b>IDH WT = highly aggressive and malignant glioblastoma</b></li> </ul> </li> </ul>	
Comp.	<ul style="list-style-type: none"> <li>➤ Death</li> </ul>	
Rx	<p><b>MDT approach for any brain tumour</b></p> <ul style="list-style-type: none"> <li>➤ Chemo</li> <li>➤ Radiotherapy w/ temozolamide (alkylating agent) → for Glioblastoma</li> <li>➤ Surgical resection (neurosurgery) → risk of irreversible brain damage depending on location <ul style="list-style-type: none"> <li>○ Corticosteroids → reduce inflammation</li> <li>○ Stop taking aspirin prior to surgery</li> </ul> </li> <li>➤ Palliative care</li> </ul> <p><b>Specific approach for PITUITARY tumour</b></p> <ul style="list-style-type: none"> <li>➤ Trans-sphenoidal Surgical resection</li> <li>➤ Radiotherapy</li> <li>➤ Medications: <ul style="list-style-type: none"> <li>○ <b>Bromocriptine</b> to block Prolactin-secreting adenomas</li> <li>○ <b>Somatostatin analogues (octreotide)</b> to block GH-secreting adenomas</li> </ul> </li> </ul>	

