Neurology Board review- Part 1

Valentina Joseph MD

Assistant Professor of Neurology

Neurology Clerkship director-USD School of medicine

Stroke

Stroke

- Sudden onset of a neurological deficit from the death of a brain tissue.
- Third most common cause of death in US
- Risk factors: **HTN, DM, HPL, Smoking**
- Etiology:

Ischemic- 85%→ Thrombus or embolus from

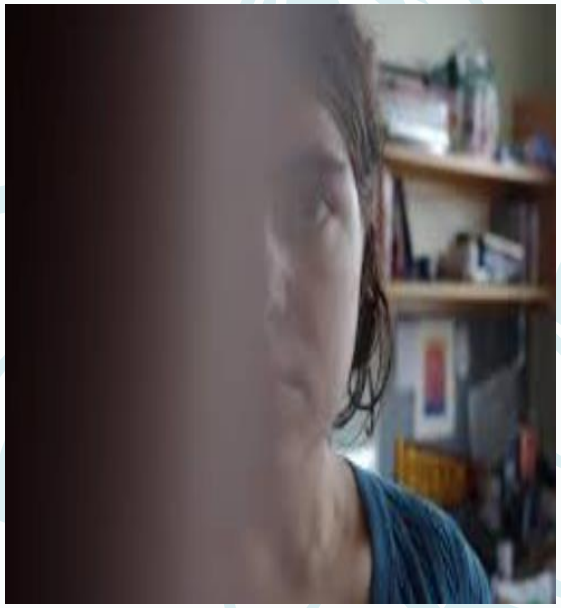
a) Heart→ Afib, Valvular heart disease, DVT causing paradoxical embolism
b) Carotid stenosis

Bleeding-15%

Thrombotic stroke	Embolic stroke
1. Atherosclerotic risk factors (uncontrolled HTN, diabetes)+/- h/o TIA 2. Local obstruction of artery (Carotid, cerebral or vertebral) 3. Stuttering progression (symptoms may alternate with periods of improvement)	1. H/o cardiac disease (Afib, endocarditis) or carotid atherosclerosis 2. Different vascular territories-multiple infarcts 3. Usually abrupt onset with and is maximal at the start.

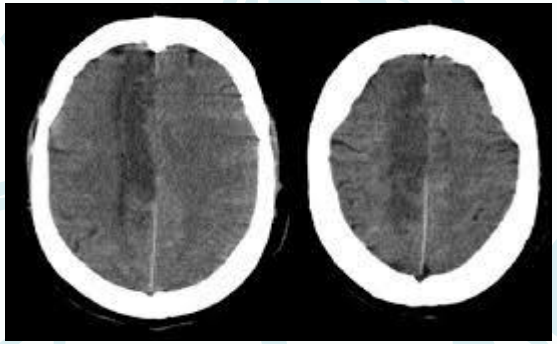
Infective endocarditis: IV antibiotics reduce the risk of septic cardio embolism within weeks of initiation of therapy
Valve surgery: If infection is persistent, difficult to treat medically, recurrent septic embolic or of heart failure

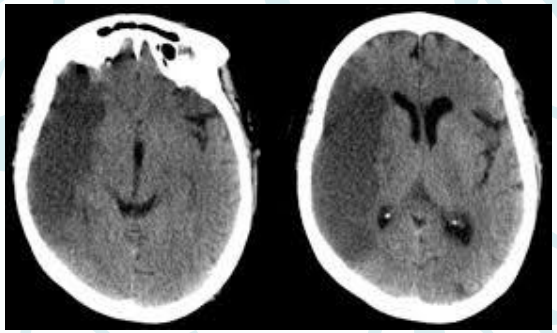
TIA(Transient ischemic attack)



- Symptoms last <24 hours.
- Initial treatment is modifying risk factors, starting aspirin/ statin and improving BP control.
- **Amaurosis fugax**: Painless, rapid, transient, monocular vision loss (curtain descending over the visual field) → suspect retinal ischemia due to atherosclerotic emboli from ipsilateral carotid artery → Carotid duplex is indicated

Presentation





ACA stroke	**1. Personality/cognitive defects** **2. Urinary incontinence** **3. C/L somatosensory and motor deficit(Legs> Arm weakness)** **4. Abulia (lack of initiative)**
MCA stroke	1. C/l weakness, sensory loss 2. C/L Homonymous hemianopsia-'eyes look towards the side of the lesion' 3. Aphasia if dominant side 4. Hemineglect if non-dominant side
Vertebrobasilar system stroke	1. Alternate symptoms (Ipsilateral cranial nerve and contralateral limb weakness/ numbness) 2. Limb ataxia

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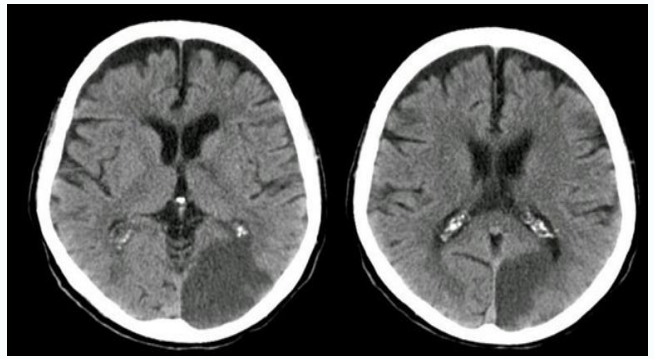
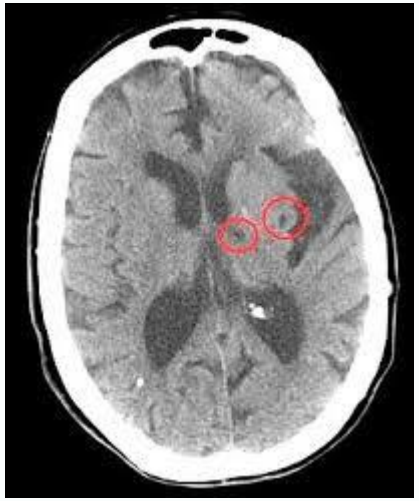
PCA stroke	**Homonymous hemianopia, alexia without agraphia (dominant hemisphere), visual hallucinations (calcarine cortex), sensory symptoms (thalamus), third nerve palsy, c/l motor deficits (cerebral peduncle, midbrain)**
ICA occlusion	Both ACA and MCA involvement (C/L dense hemiplegia with C/L visual, sensory, language or spatial impairments



Lacunar infarcts

- Small vessel infarcts affecting the deep cortical structures (<1.5 cm)
- Occlusion of single deep penetrating vessel of a large cerebral artery
- Some common presentations:
  1. Pure motor
  2. Pure sensory
  3. Sensorimotor
  4. Dysarthria clumsy hand
  5. Ataxic hemiparesis
- Face, arm and leg are equally involved without any cortical symptoms.
- Affected areas: Basal ganglia, subcortical white matter, Pons.
- Risk factors: Hypertension, DM, smoking, advanced age, increased LDL
- Pathology: **Small vessel lipohyalinosis**



Diagnosis and Treatment

- **Best initial test** → CT brain w/o contrast
- Most accurate test → MRI brain
- Why CT brain is done first?

Treatment:

General strategies:

1. < 3 hours → thrombolytics (IV TPA). Or even up to 4.5 hours. Thrombectomy up to 6 to 8 hours (or even 24 hours)

2. Stroke with no prior antiplatelet therapy→ Aspirin.

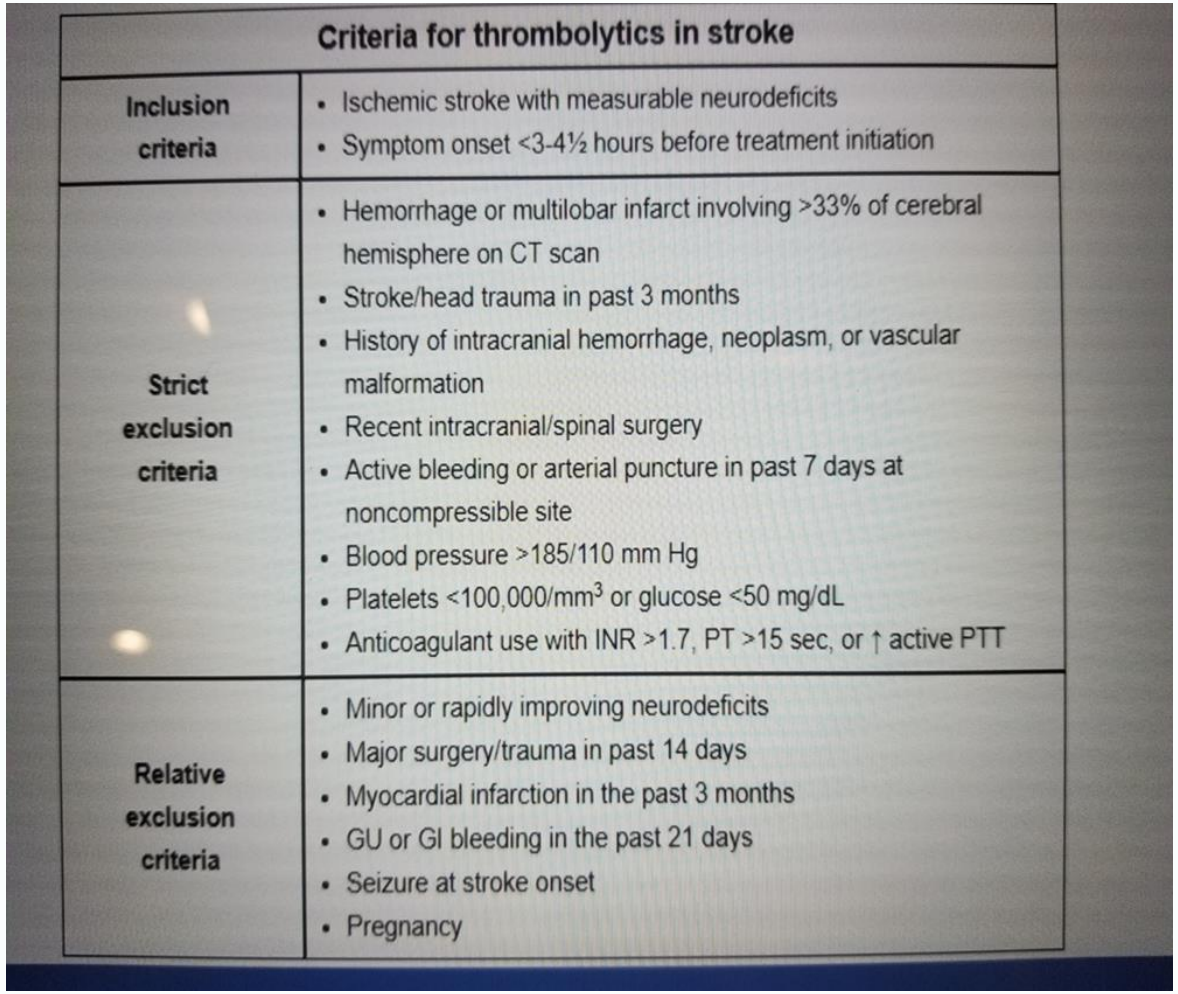
3. If the patient is already on ASA→ then add Dipyridamole or switch to Clopidogrel

4. If Afib- Long term anticoagulation (Warfarin, Dabigatran, Rivaroxaban)

5. Large intracranial atherosclerosis→ ASA + Plavix for 90 days, then only ASA

Statins:

- Every patient should be started regardless of LDL. Goal < 70.



Criteria for thrombolytics in stroke	
Inclusion criteria	• Ischemic stroke with measurable neurodeficits • Symptom onset <3-4½ hours before treatment initiation
Strict exclusion criteria	• Hemorrhage or multilobar infarct involving >33% of cerebral hemisphere on CT scan • Stroke/head trauma in past 3 months • History of intracranial hemorrhage, neoplasm, or vascular malformation • Recent intracranial/spinal surgery • Active bleeding or arterial puncture in past 7 days at noncompressible site • Blood pressure >185/110 mm Hg • Platelets <100,000/mm^3 or glucose <50 mg/dL • Anticoagulant use with INR >1.7, PT >15 sec, or ↑ active PTT
Relative exclusion criteria	• Minor or rapidly improving neurodeficits • Major surgery/trauma in past 14 days • Myocardial infarction in the past 3 months • GU or GI bleeding in the past 21 days • Seizure at stroke onset • Pregnancy

Remember!

No Aspirin 24 hours following IV TPA

BP should be kept **</=185/110** prior to TPA to avoid risk of hemorrhage(Use **Labetalol** if the BP is high)

Evaluation of causes of stroke and their treatment

Echocardiogram:

- Damaged valves-repair
- Thrombi: Heparin followed by Warfarin (INR 2-3) or Rivaraoxaban/Dabigatran
- PFO
- EKG: Atrial fibrillation/flutter
- Holter: If initial EKG is normal

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Carotid Duplex ultrasound:

If **symptomatic cerebrovascular disease +> 70% stenosis → surgical correction**

<50% stenosis (mild disease)- no surgery

Moderate stenosis (moderate disease)-unclear if surgery is beneficial

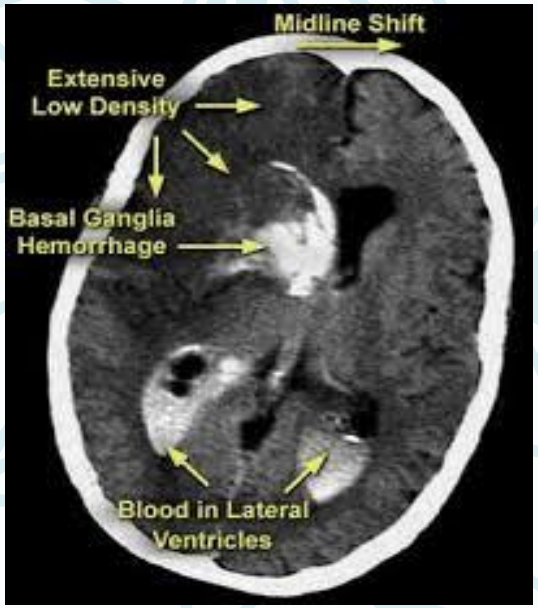
100% stenosis- no intervention

- Endarterectomy is superior to carotid angioplasty

Control of risk factors for stroke

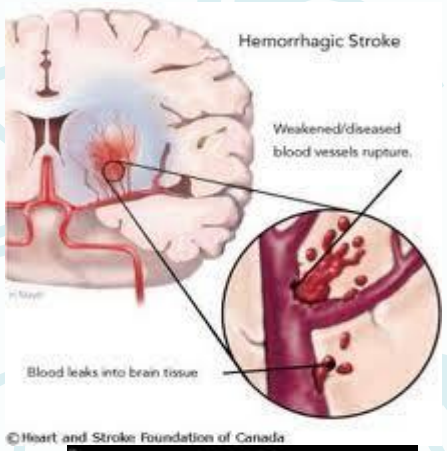
1. Diabetes to a HbA1C below 7%
2. Hypertension
3. Reduce LDL <70
4. Stop smoking

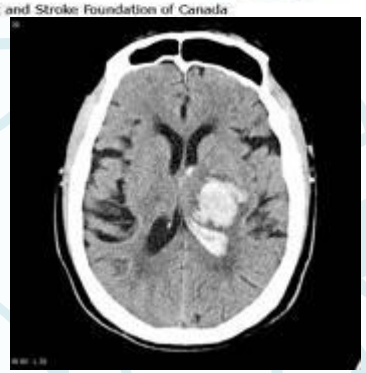
Complications post stroke



- **<u>Hemorrhagic transformation</u>**:
- Especially when stroke affects a large area or due to embolic stroke or treated with thrombolytics.
- Typically <48 hours after the initial stroke
- Manifests as deteriorating mental status.
- **Emergency CT brain w/o contrast is needed → may need urgent decompression**

Hemorrhagic stroke





- H/o **uncontrolled HTN, coagulopathy, illicit drug use (cocaine, amphetamines**)
- Hypertensive vasculopathy involving small penetrating branches of major cerebral arteries → causes spontaneous deep intracerebral hemorrhage
- Chronic hypertension leads to **Charcot-Bouchard aneurysms**→ that ruptures and causes the bleed
- **Quick progression (minutes to hours)**
- Focal neurological complaints appear early followed by symptoms of increased ICP
- Most common locations: Basal ganglia(Putamen), cerebellar nuclei, thalamus, Pons
- Brain CT confirms intracranial hemorrhage (highly sensitive)
- Although MRI brain can detect hemorrhage, CT is preferred (because of ready availability, cost and quick results)

Contd..

– Warfarin associated ICH:

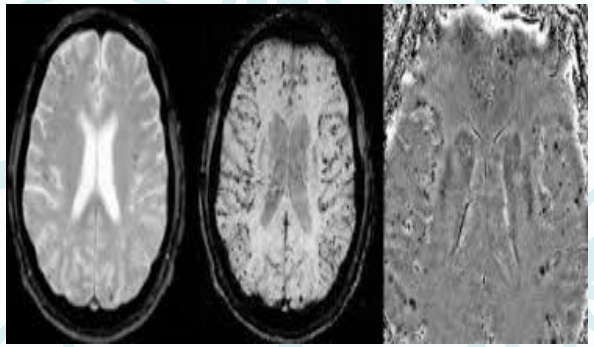
- Supratherapeutic INR
- Can be provoked by OTC cold medications (acetaminophen increases anticoagulation effect of warfarin and phenylephrine can increase BP)
- Symptoms of elevated intracranial pressure (headache, altered mental status, nausea/vomiting) as the hemorrhage expands
- Treatment: Immediately **reverse the anticoagulation effect**

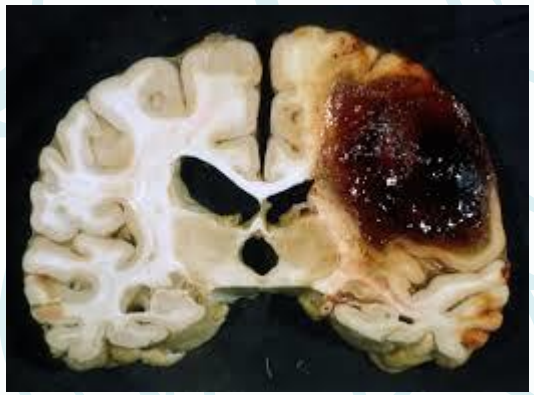
IV Vitamin K- takes 12-24 hours to be effective (promotes Vitamin K dependent clotting factors)

Prothrombin complex concentrate- contains Vitamin K dep clotting factors→ rapid and short term reversal of Warfarin

FFP can be given if PCC is not available but it takes longer time to prepare and is a larger volume infusion

Other facts



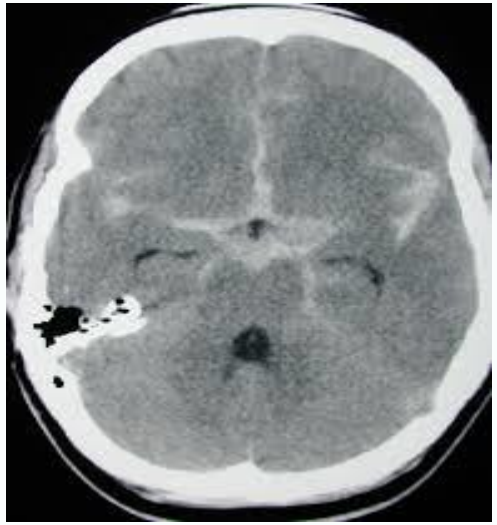


- Brain AVM is the most common cause of intraparenchymal hemorrhage in children
- **Cerebral amyloid angiopathy-** most common cause of spontaneous lobar/ cortical (Occipital/ parietal) hemorrhage in elderly >60 yrs (as a consequence of beta amyloid deposits in the walls of small to medium sized cerebral arteries→ vessel wall weakening → rupture.
- Similar kind of amyloid protein also seen in Alzheimer's dementia

Subarachnoid hemorrhage

- More frequent in patients with

a) Polycystic kidney disease
b) Tobacco smoking
c) Hypertension
d) Hyperlipidemia
e) High alcohol consumption

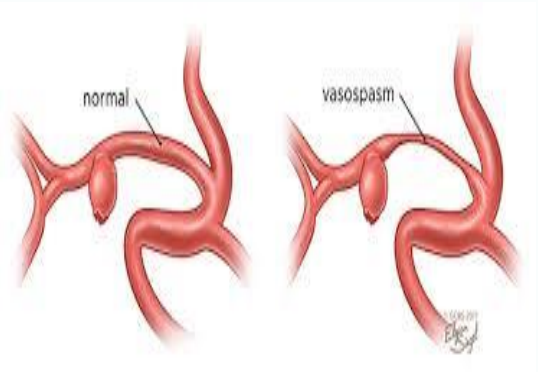


Bleeding from **berry aneurysm or AVM**

- Severe headache at the onset
- Neck stiffness due to meningeal irritation
- Focal deficits -30%
- Fever → secondary to blood irritating the meninges.

Complications of subarachnoid hemorrhage

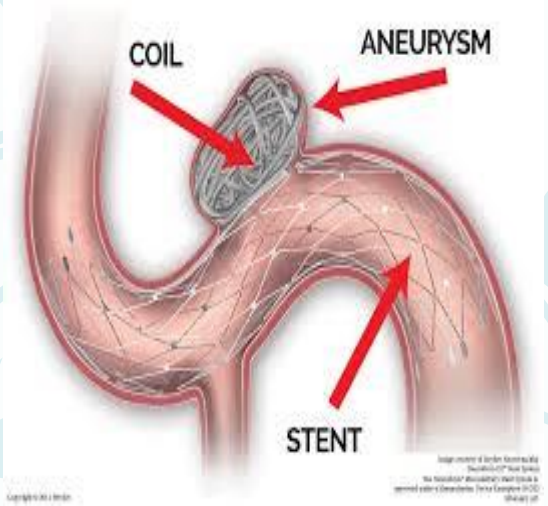
- Rebleeding (first 24 hours)
- **Vasospasm** (after 3 days up to 10 days)→ due to arterial narrowing at the base of the brain→ can cause infarction → can cause delayed morbidity and death
- Hydrocephalus/ Increased intracranial pressure
- Seizures
- **Hyponatremia (usually from SIADH)**



Diagnostic tests

- Best initial test → CT without contrast
- Most accurate test: Lumbar puncture showing blood **Xanthrochromia** (Yellow discoloration of CSF from the breakdown of RBCs)
- LP is necessary only for 5% that have a falsely negative CT scan.
- Ratio of WBCs to RBCs will be normal in CSF (1 WBC for every 500 to 1000 RBCs)
- Angiography- to determine the site of aneurysm
- CTA, MRA or standard angiography

Treatment



- **Nimodipine (Calcium channel blocker)** prevents subsequent ischemic stroke.
- **Hyperdynamic therapy** to reduce vasospasm(hypertension, hypervolemia, and hemodilution to optimize cerebral perfusion)
- **Embolization (Coiling/stenting)** → to clog up the site of bleeding to prevent repeated hemorrhage
- Embolization is superior to clipping in terms of survival and complications.
- VP shunt: Only if hydrocephalus develops.
- Seizure prophylaxis: Phenytoin for seizure prophylaxis (still controversial)
- 50-70% of those who rebleed will die.
- EKG: Large or inverted T waves suggestive of MI (cerebral T waves) from excessive sympathetic activity.

Cerebral venous thrombosis

- **Malignancy, Birth control pills, hypercoagulability**
- C/f: Headache, seizures, altered mental status and focal neurologic deficits.
- MRI/ MRV or CT venogram

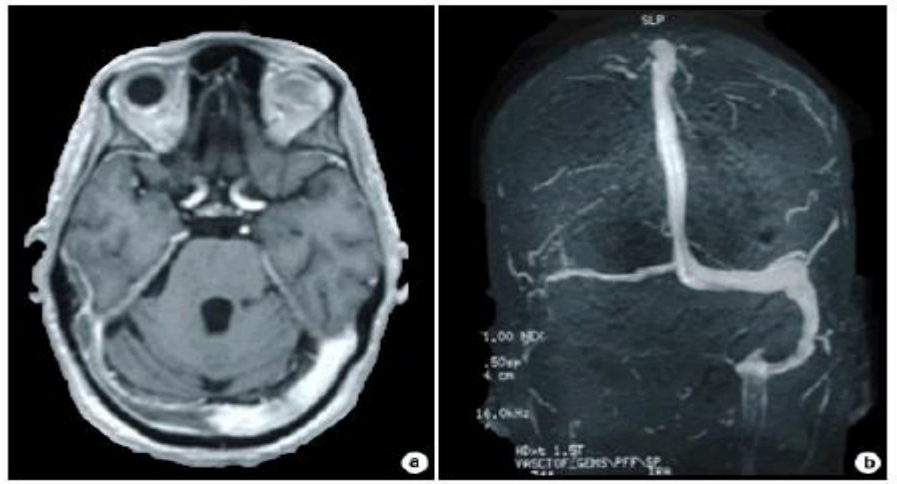


Figure 1: T1 weighted brain MRI after gadolinium injection (a) and MR angiography (b) showing right lateral sinus thrombosis and enhancement of the meninges.

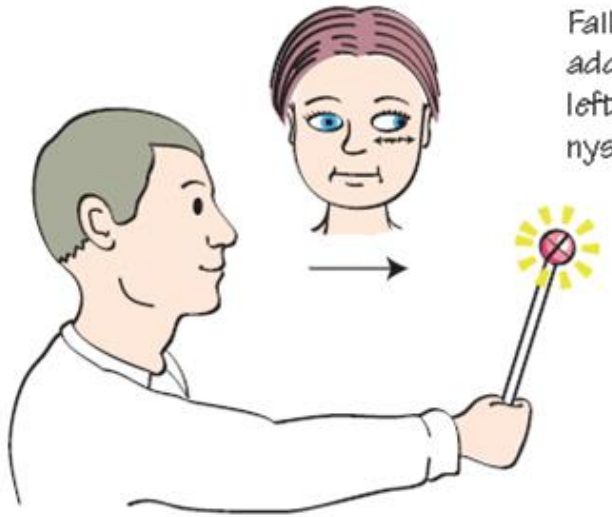

BREAK TIME

Multiple Sclerosis

Multiple Sclerosis

- Autoimmune inflammatory demyelinating disorder of CNS that presents with neurologic deficits disseminated in space and time.
- Onset: 15 to 50 years
- C/F: Optic neuritis, INO, RAPD, Lhermitte sign, Uhthoff phenomenon, sensory (numbness and paresthesia), motor (paraparesis, spasticity), bowel bladder symptoms.
- INO→**MLF lesion**→ inability to adduct on the side of lesion as a result the c/l eye abducts with nystagmus
- Transverse myelitis: Motor and sensory loss below the level of lesion with bowel bladder involvement. Initially have flaccid paralysis (spinal shock) followed by spastic paralysis with hyperreflexia
- Disease patterns: **Relapsing remitting, Primary progressive, secondary progressive, Progressive relapsing**

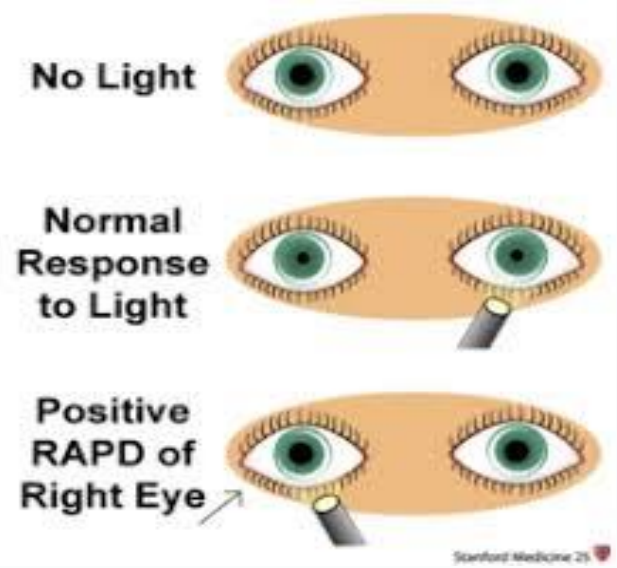
Right internuclear ophthalmoplegia (INO)



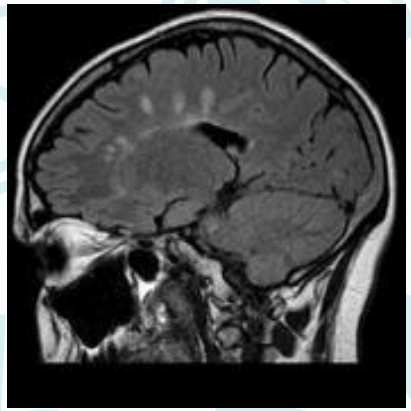
Failure of right adduction + left abducting nystagmus

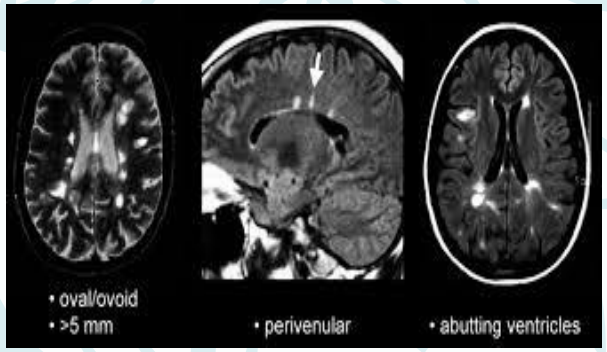
Aetiology

- Vascular (elderly)
- Demyelination of medial longitudinal fasciculus (MLF) in young people
- Needs MRI brainstem



Diagnosis and Treatment





- T2 MRI lesions **disseminated in space and time** (periventricular, juxtacortical, infratentorial, spinal cord)→ ovoid hyperintense white matter lesions.
- CSF: Oligoclonal Ig G bands (85-95% of patients with MS) with normal cell count (lymphocyte predominance) and normal protein count (high IgG compared to other proteins)
- Treatment:
- Acute exacerbation: **IV methylprednisone**. If no response→ **PLEX**
- Immunomodulators: Interferon beta, natalizumab, glatiramer

Optic neuritis

- Epidemiology:
  - ❖ Young women
  - ❖ Immune mediated demyelination.
  - ❖ Associated with MS
- C/F: Acute peak-2 weeks, monocular vision loss, eye pain with movement, 'Washed-out' color vision, RAPD, central scotoma
- Fundus: usually normal as the inflammation occurs behind the optic nerve head
- Diagnosis: MRI orbits and brain
- Treatment: IV steroids.
- 35% recurrence

Trigeminal neuralgia in MS:

- Bilateral
- Demyelination of the CN V nucleus or its nerve root → improper nerve signal → paroxysms of severe pain

Headaches

Headache	Presentation	Treatment	Prophylaxis
Migraine	+/- aura, photophobia, phonophobia, nausea, vomiting (Pulsatile and throbbing) Often unilateral Female> male	Avoid triggers NSAIDs **Triptans (5-HT1 agonists**)	If > 3 attacks/ month Propranolol Verapamil TCAs, SSRIs, Topiramate, Botox
Tension headache	-Bilateral band-like pressure -Lasts 4-6 hours (30 minutes to 7 days) (Dull, tight, persistent) Female> male	NSAIDs Acetaminophen	Amitriptyline, Nortriptyline and CBT to learn stress reduction techniques
Cluster headache	-Episodic pain -Unilateral periorbital intense pain -Lacrimation, eye redness, nasal stuffiness, ptosis, miosis (Excruciating, sharp and steady) Male > female	**100% Oxygen** Sumatriptan Octreotide Prednisone	Verapamil, Lithium Prednisone Sodium valproate

Other facts

- **Basilar migraine**:
- Cerebral vasospasm involving the brainstem
- C/F: Basilar aura (vertigo, dysarthria, tinnitus and diplopia) followed by migraine headache
- **Hemiplegic migraine**: Familial, can cause unilateral motor deficits
- **Cluster attacks**: Typically last for about 90 minutes, occurs up to 8 times daily for 6-8 weeks followed by remission lasting up to a year

Trigeminal neuralgia

- Recurrent, sudden onset severe pain involving V1, V2 and V3 branches of trigeminal nerve.
- Rare to involve V1 and can cause i/l tearing, rhinorrhea
- Lasts for few seconds to minutes
- Triggered by minor stimuli (touch, wind, chewing)
- **Usually unilateral**
- MS→ can be bilateral
- Herpes zoster: Viral reactivation → nerve inflammation → dermatomal vesicular rash.
- Herpes zoster ophthalmicus → involves V1 nerve and can cause blindness.

Treatment

1. Carbamazepine, Oxcarbazepine
2. If unsuccessful **nerve decompression/ radiosurgery**

Side effects of carbamazepine: Nausea, vomiting, leukopenia, hyponatremia

Pseudotumor cerebri

- Associated with **obesity, venous sinus thrombosis, oral contraceptives, Tetracyclines, growth hormone and Vitamin A toxicity**.
- Features of increased ICP in an alert patient (Headache, transient vision loss , pulsatile tinnitus, diplopia)
- **Momentary vision loss that varies according to the change in head position**
- Exam: **Papilledema with diplopia from sixth nerve palsy**, peripheral visual field cut, **enlargement of blind spot**
- Diagnosis:

CT or MRI to exclude intracranial mass lesion +/- MRV

LP to show increased pressure**.(>250 mm water** with normal analysis)

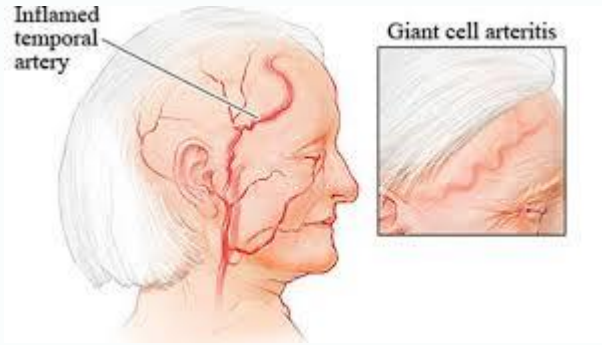
Treatment

1) Serial LPs or short term steroids (while awaiting surgery)
2) VP shunt, LP shunt
3) Optic nerve fenestration
4) Medications: **Acetazolamide** (carbonic anhydrase inhibitor that reduces CSF production), Lasix can be added
5) Weight loss

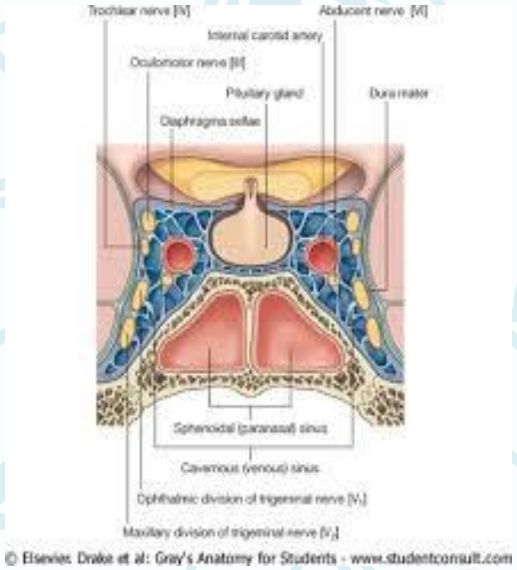
- Fundoscopy and routine eye exams required to prevent vision loss

Giant cell arteritis

- >50 years with new onset headaches **localized to temples**
- Frequently associated with fever, weight loss, vision changes and jaw claudication (fatigue and pain when chewing)
- Associated with **polymyalgia rheumatica**.



Cavernous sinus thrombosis



- Infection of medial face, sinuses, teeth → spreads through valveless facial venous system → cavernous sinus thrombosis
- C/F: headache, fever, periorbital edema, proptosis, vomiting, **CN 3,4,6, 5(V1,V2)** deficits. Fundoscopy may reveal papilledema.
- Diagnosis: MRI/ MRV brain
- Treatment: Broad spectrum IV antibiotics and prevention/ reversal of cerebral herniation

Warning signs in a patient with headache

- Seizures, change in consciousness, specific deficits
- Change in frequency, intensity or character of headache
- **New at age>40, sudden onset**, trauma, early morning headaches

If so, consider **early imaging**


Thank you

Neurology board review-2

Valentina Joseph MD,

Neurology Clerkship Director,

USD Sanford School of medicine.

Dementia

Delirium

- **Acute confusional** state with reduced or **fluctuating level of consciousness and inability to sustain attention. Reversible**
- Sleep wake disturbance (sundowning)
- MC in elderly with medical illness or conditions like PD, dementia, prior stroke
- Often associated with anxiety, agitation, hallucinations
- Precipitating factors:

  - Drugs: Narcotics, sedatives, antihistamines , relaxers , polypharmacy
  - Infections: Pneumonia, UTI, meningitis
  - Electrolyte disturbance (hyponatremia, hypercalcemia)
  - Metabolic derangements (volume depletion, Vitamin B12 deficiency, hyperglycemia)
  - Systemic illness (CHF, hepatic failure, malignancy)
  - CNS conditions (stroke, seizure, SDH, head injury)

Contd..

- Treatment of choice for elderly is **low dose Haloperidol**
- **Atypical antipsychotics** can also be used (Quetiapine, Risperidone)
- Prolonged use can increase mortality in elderly
- **Benzodiazepines are contraindicated in older patients** due to adverse events (withdrawal, dependence, motor impairment), may experience worsening agitation (paradoxical effect) because they metabolize slowly and hence can cause long lasting effects

Alzheimer's disease

- Progressive dementia
- Risk factors: Old age, female sex, family history, head trauma , Down syndrome
- Early stage:

➢ Memory loss for recent events (**anterograde amnesia-immediate recall affected and distant memory is preserved**), language difficulties

➢ Visuosaptial and executive problems (e.g getting lost in a familiar surroundings)

- Late stage:

➢ Neuropsychiatric (hallucinations, wandering, delusions, paranoia), apraxia**, lack of insight**, personality changes, impaired judgement

Contd..

Imaging: Early AD –MRI could be normal. Generalized cortical atrophy, reduced hippocampal volume, medial temporal lobe atrophy(**temporal and parietal atrophy**)

Other tests: VDRL/RPR, B12 and thyroid function test

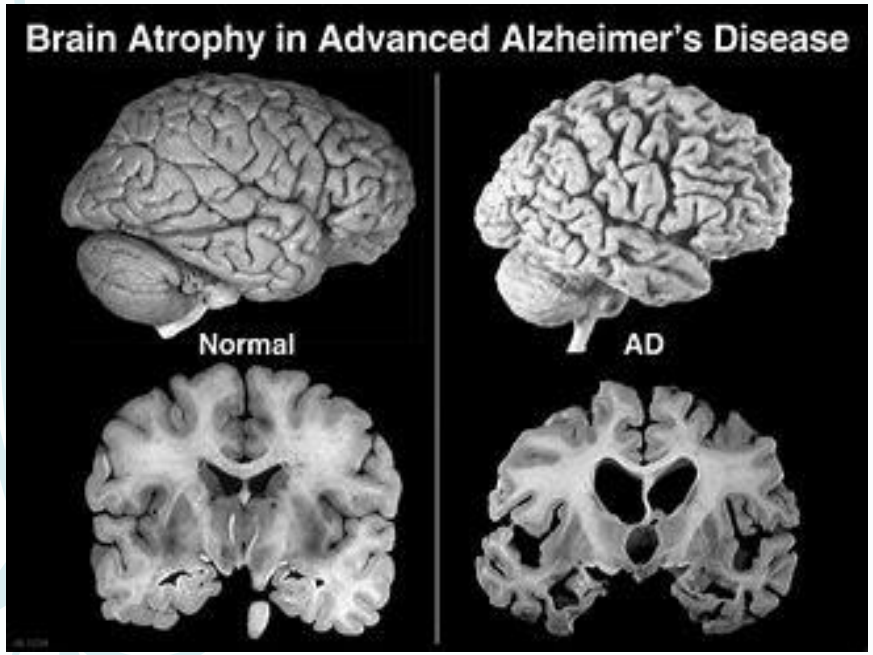
Treatment:

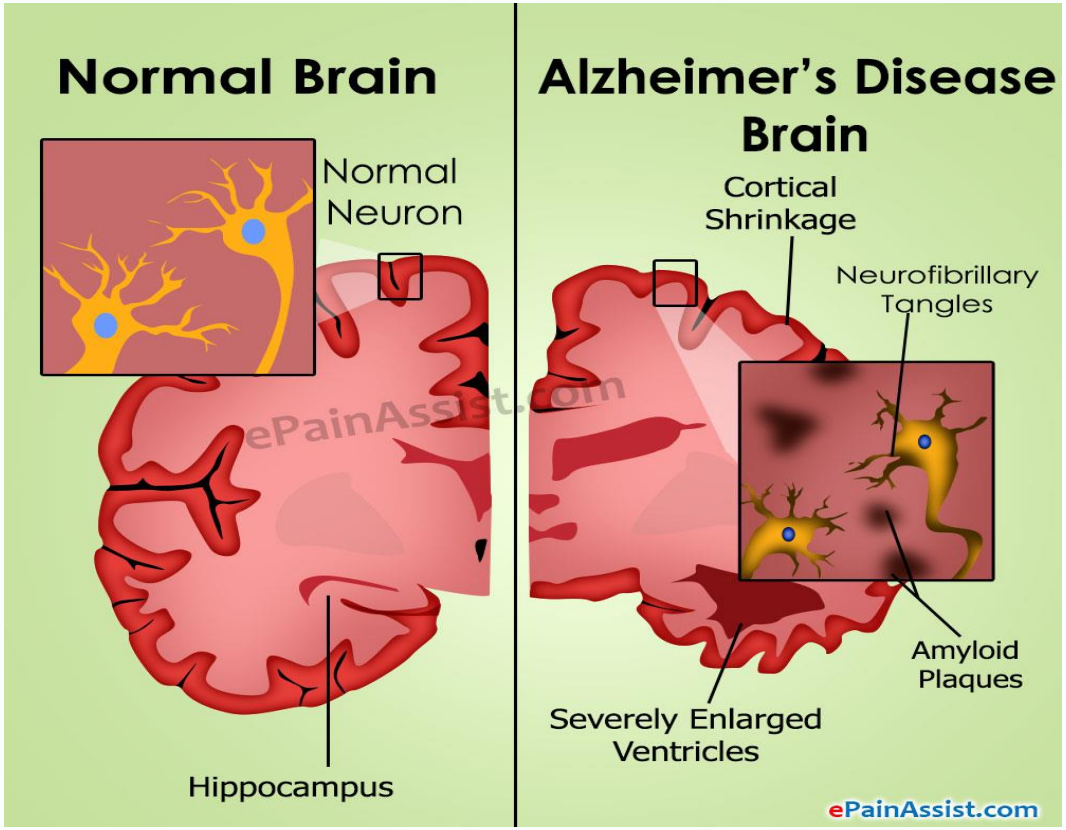
Psychosocial intervention and pharmacological therapy

- **Cholinesterase inhibitors may improve quality of life and cognitive functions (e.g memory, thought, language, reasoning) but they do not alter the course of dementia**

e.g: Donepezil, Rivastigmine, Galantamine for mild to moderate dementia

- Memantine (NMDA receptor antagonist) for moderate to severe dementia
- Selegiline- MAO inhibitor sometimes used in AD for antioxidant properties but evidence is weak.
- Vitamin E → limited evidence for treatment of mild to moderate dementia.


Brain Atrophy in Advanced Alzheimer's Disease
Normal
AD


Normal Brain
Normal Neuron
Hippocampus
Alzheimer's Disease Brain
Cortical Shrinkage
Neurofibrillary Tangles
Amyloid Plaques
Severely Enlarged Ventricles
ePainAssist.com

Lewy body dementia

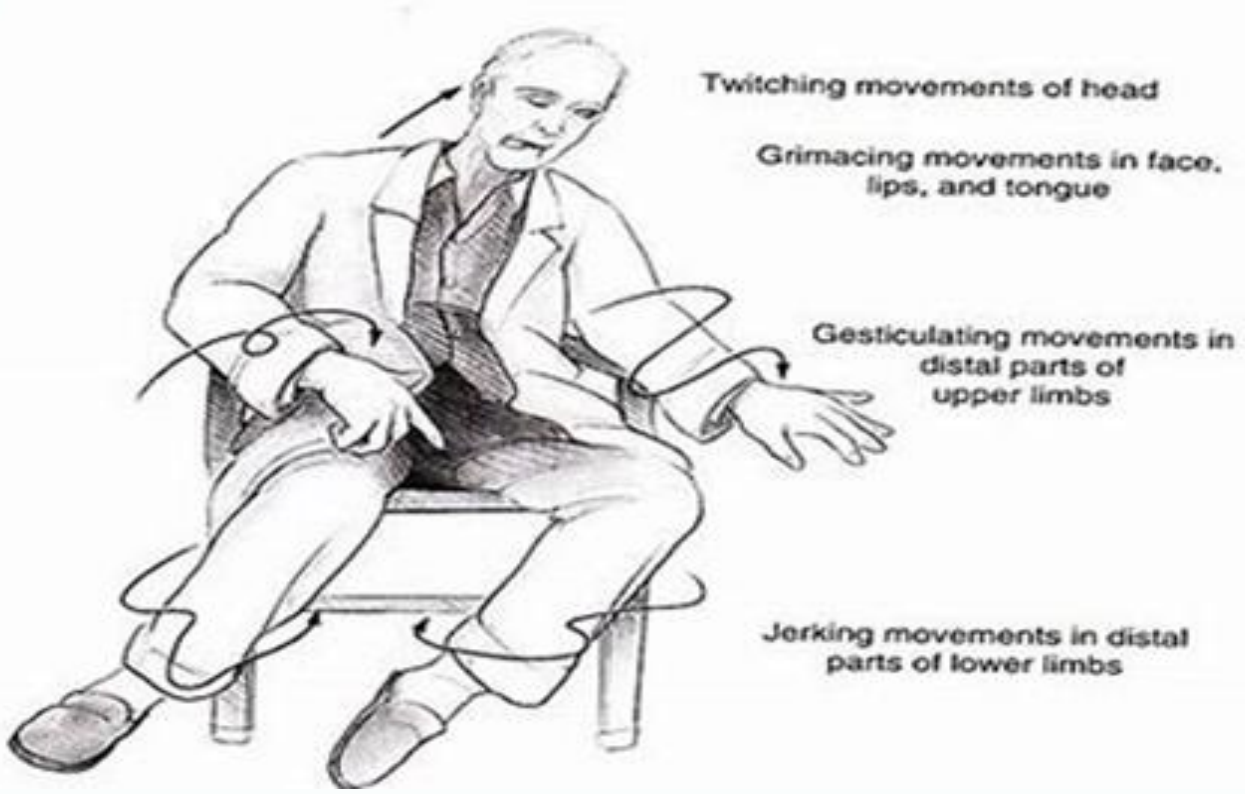
- **Fluctuating cognitive impairment, bizarre visual hallucinations and Parkinsonism** (if 2 positive-probable DLB, if 1 is positive- possible)
- Symptoms worsened with neuroleptic drug (severe sensitivity to dopamine antagonists (first generation antipsychotics, Risperidone)
- Deficits in attention , frontal-subcortical skills, visuospatial ability
- Other supportive features:

Repeated falls
Syncope/ transient LOC
Autonomic dysfunction
Neuroleptic sensitivity
Systematized delusions
Sleep disturbance (RBD)

Huntington's disease

- AD neurodegenerative disorder
- Typically present in 40s or 50s with chorea and/or behavioral disturbance
- **Chorea: Sudden jerky and irregular movements of the extremities.**
- Impaired judgement, executive function, awareness and attention (early stage)
- Memory impairment (late finding)
- Depression, irritability and social withdrawal
- Atrophy of the **caudate nucleus** (enlargement of lateral ventricles)

Huntington's Chorea



Contd..

- Autopsy: **Eosinophilic intracytoplasmic inclusions (alpha synuclein)** in neurons of substantia nigra , locus coeruleus, dorsal raphe, substantia innominata
- Treatment:

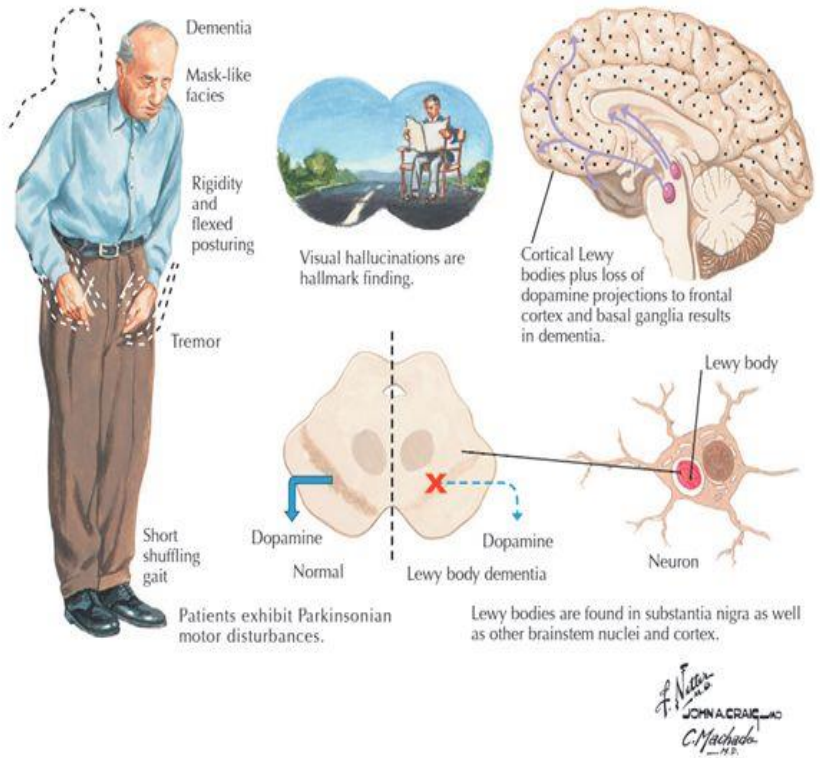
➢ Parkinsonism: Carbidopa-levodopa

➢ Dementia: acetylcholinesterase inhibitors

➢ Psychotic features: low dose second generation antipsychotic (avoid first generation due to severe neuroleptic sensitivity)

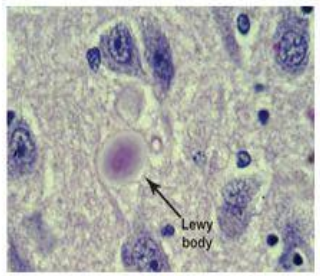
Note: **In patients with Parkinson's disease with dementia, dementia appears much later in the course and also visual hallucinations are not that common unless they are on dopaminergic medications**

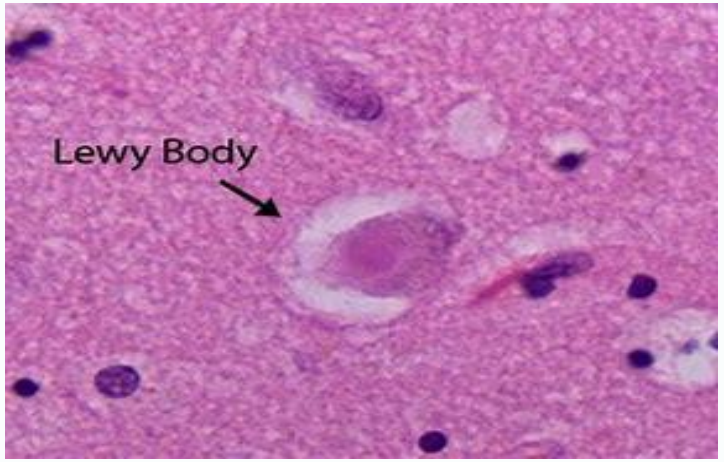
Dementia with Lewy bodies



Demencia +

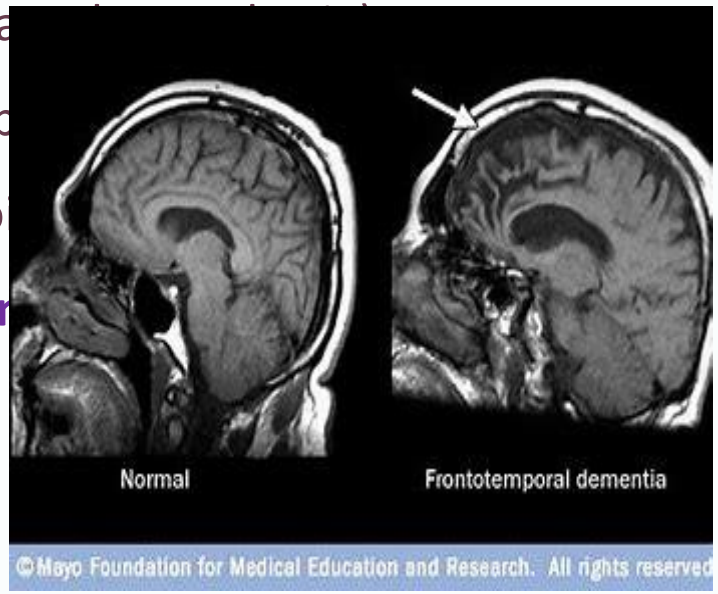
- Fluctuating cognition
- Visual hallucinations
- Parkinsonism





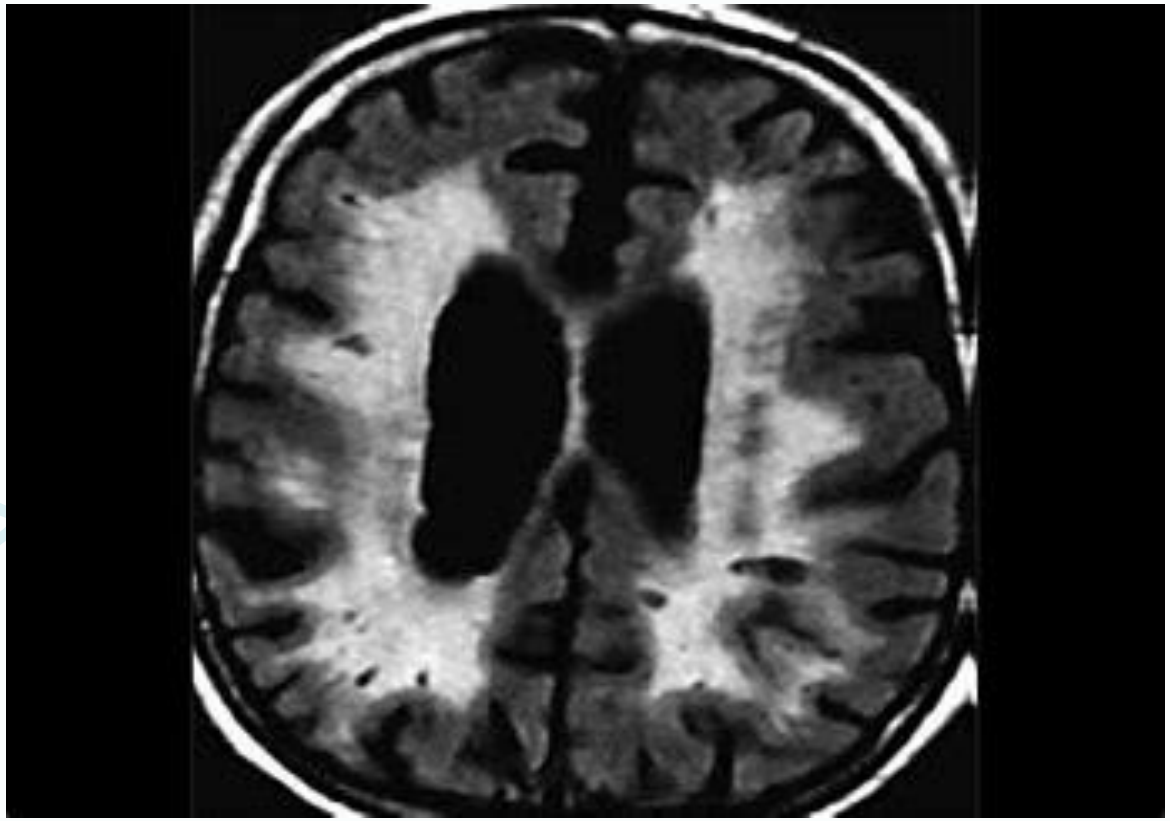
Frontotemporal dementia (Pick disease)

- **Earlier onset** than AD (usually 50-60 years)
- Early **personality changes** (e.g. disinhibition, a
- Compulsive behaviors (e.g peculiar eating hab
- **Hyperorality, frontotemporal atrophy** (neuro
- **Behavioral changes precede memory impairr**
- 25% have family history
- MRI: Atrophy of frontal and temporal lobes



Vascular dementia

- 15-20% of dementia cases
- Cognitive decline with focal neurological symptoms (**stepwise decline**)
- Deficits in **executive function > memory impairment**
- Brain imaging: **Multiple small cortical (large artery) and subcortical infarcts (small artery)**
- Risk factors: Older age, Male sex, Black race, smoking, HTN, diabetes, vasculitis.
- Treatment is directed towards modifying the risk factors



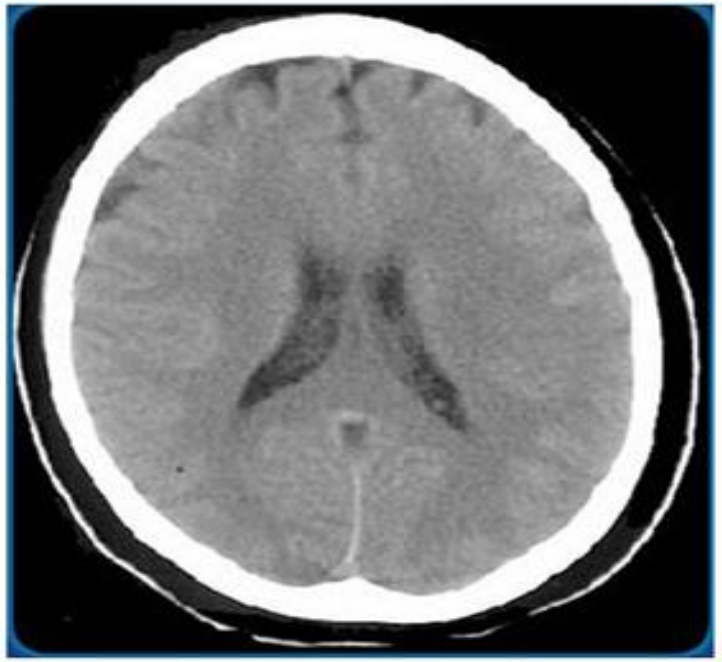
General paresis

- Form of **tertiary neurosyphilis**
- 15-20% of late syphilis
- C/F: Decreased concentration, memory loss, personality changes, dysarthria, tremors of fingers and lips, irritability, mild headaches
- **RPR may be non reactive but FTA-ABS will confirm the test**.

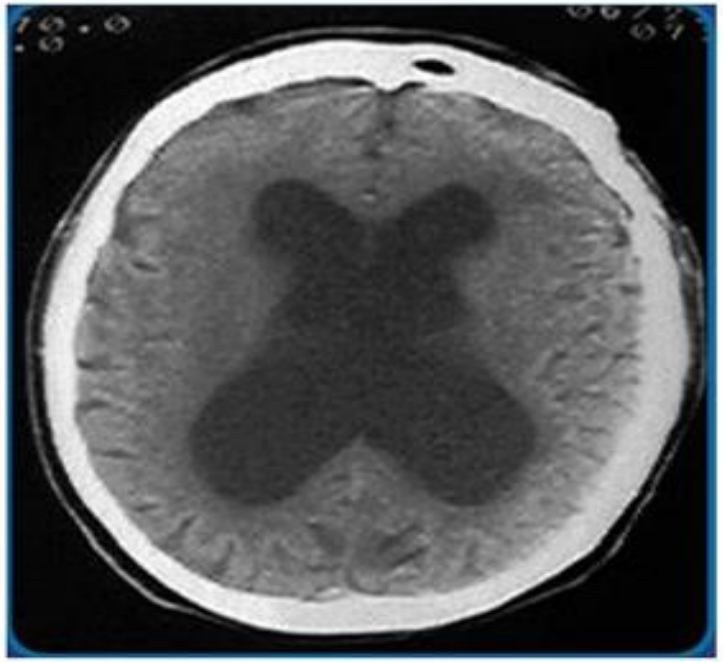
Normal Pressure hydrocephalus

- **Triad of gait disturbance (magnetic gait/ gait apraxia, slow shuffling), dementia and urinary incontinence (but fecal incontinence can happen in advanced cases**)
- Symptoms arise due to distortion of brain matter adjacent to the ventricles.
- CT or MRI shows dilated ventricles
- CSF opening pressure is normal
- If repeated spinal taps leads to improvement, then **VP shunt** can be considered

Brain Imaging



Normal Brain



NPH Brain

Prion disease

- Behavioral changes
- **Rapid** progression
- Myoclonus and seizures

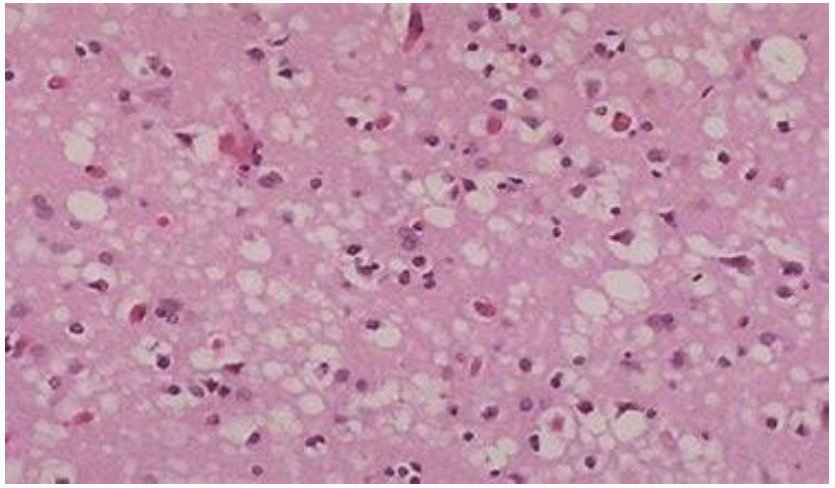
Probable diagnosis:

- Rapidly progressive dementia
- 2 out of 4 clinical features (myoclonus, akinetic mutism, cerebellar or visual disturbance, pyramidal/ extrapyramidal dysfunction-hypokinesia)
- **Periodic sharp wave** complexes on EEG and/or **Positive 14-3-3 CSF assay**

Definitive diagnosis : Above features + **brain biopsy (gold standard) or demonstrated PRNP gene mutation**

Contd..

- Key features: Long incubation period, characteristic **spongiform changes** and lack of inflammatory response
- Most cases are **sporadic (85%) and remainder (hereditary or iatrogenic due to contaminated transplants or surgical instruments**)
- Most patients die **within 1 year** of onset of symptoms.

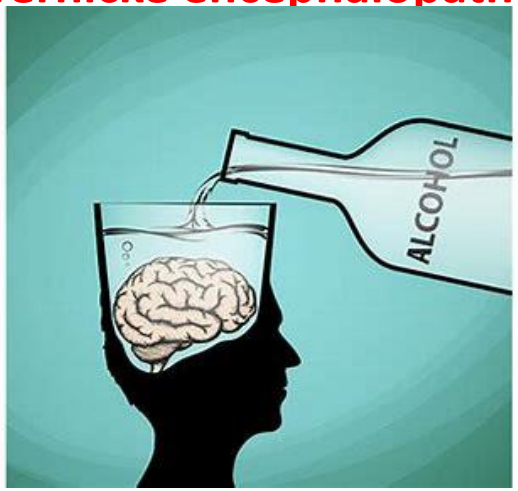


Depression related cognitive impairment

- Features of major depression (anhedonia, poor appetite, fatigue, difficulty to focus, feeling of worthlessness) + memory impairment
- **Pseudodementia**
- On cognitive testing: **Deficits in attention, concentration, memory and executive function**
- **Poor effort/ difficulty completing the test**
- **Patients are more concerned** about their memory problems unlike other dementias where patients are brought by family members for clinical attention.
- Treatment: Underlying depression management (e.g: SSRIs and psychotherapy)

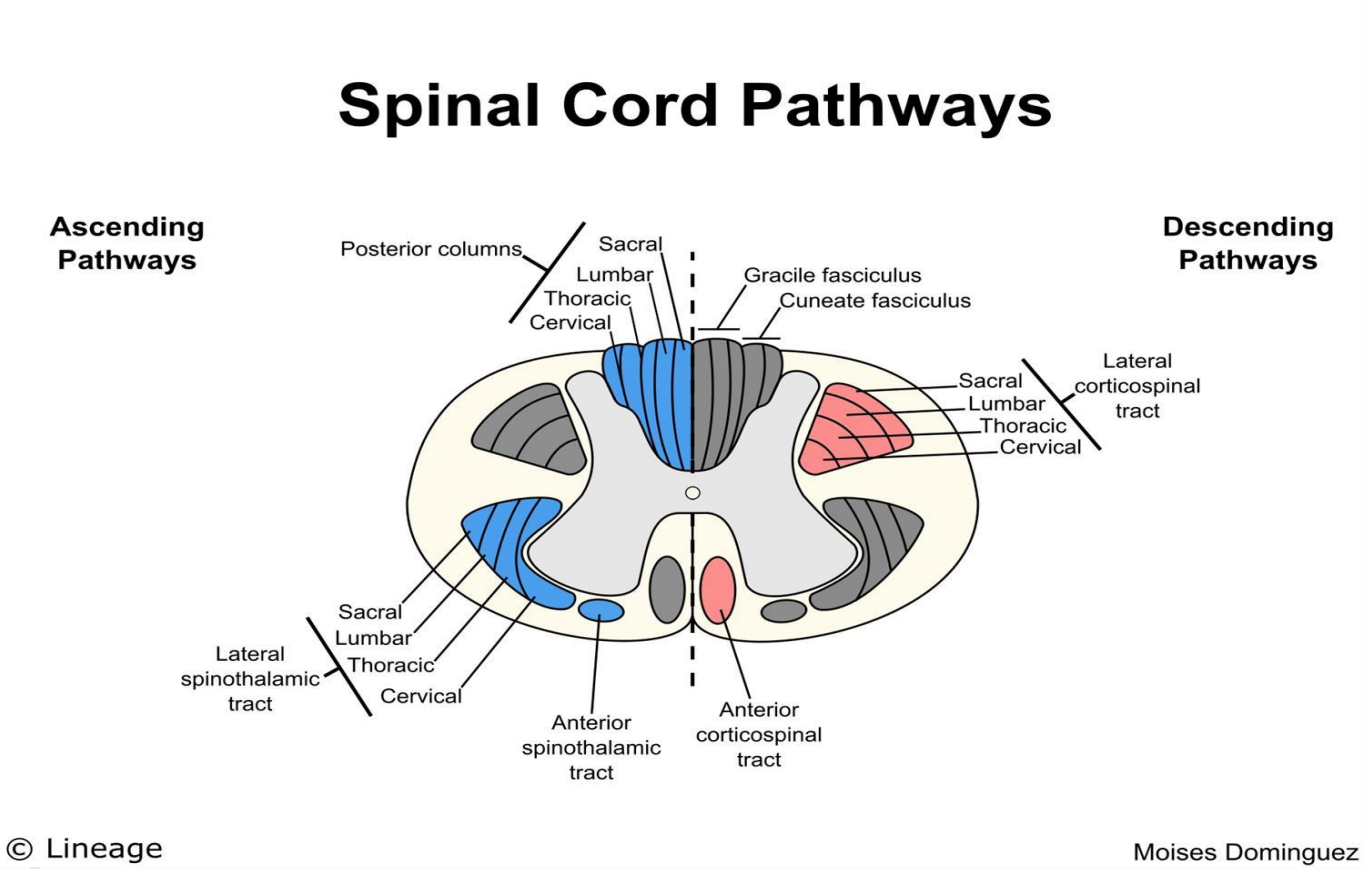
Korsakoff syndrome

- Acute onset severe memory impairment seen in **Thiamine deficiency**
- Common is **alcoholics**
- Can occur concurrently with or following the **Wernicke encephalopathy (ocular disturbances, alteration of consciousnes**



Spinal cord disorders and trauma

Spinal cord disorders



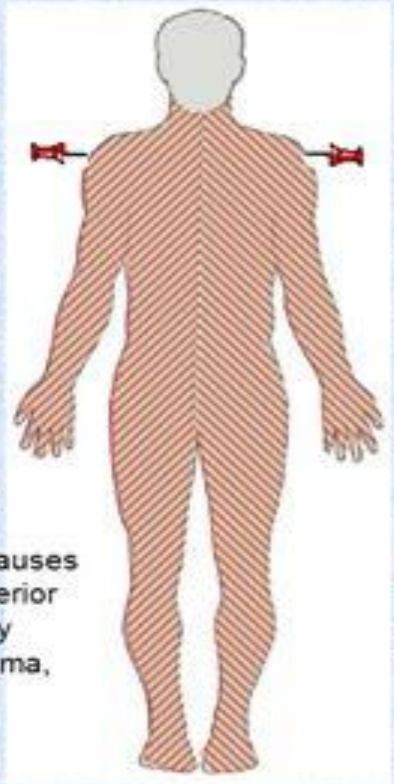
Anterior cord syndrome

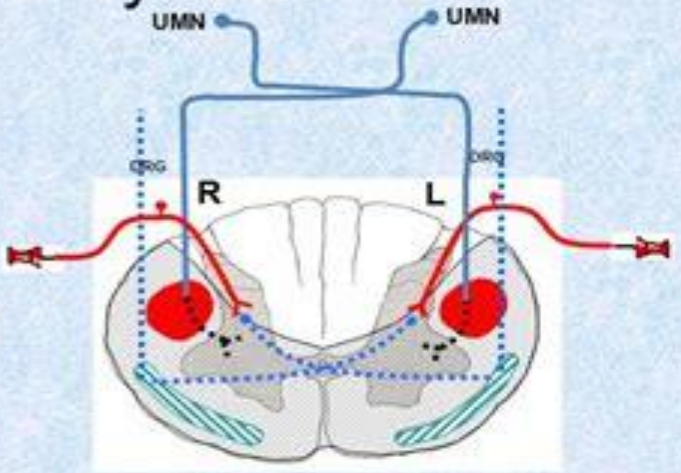
- Usually after injury to anterior spinal artery (e.g: disc retropulsion, fragments of bone from vertebral burst fracture) affecting **anterior 2/3 rds of spinal cord**.
- C/F: 1) Bilateral hemiparesis (Lateral corticospinal tract, **at the level** of cord injury and below)

2) Diminished b/l pain and temperature sensation (lateral spinothalamic tract, **1-2 levels below the cord injury because LST decussates 1-2 levels before the corresponding level**

3) Intact b/l proprioception and vibration, light touch (dorsal columns are supplied by posterior spinal arteries which are bilateral and reinforced by radicular segmental branches)

Anterior Cord Syndrome





Common causes include anterior spinal artery infarct, trauma, and MS.

Anterior cord lesion

Lateral corticospinal tract lesion
Ipsilateral upper motor neurons signs

Lateral spinothalamic tract lesion
Contralateral loss of pain and temperature sense

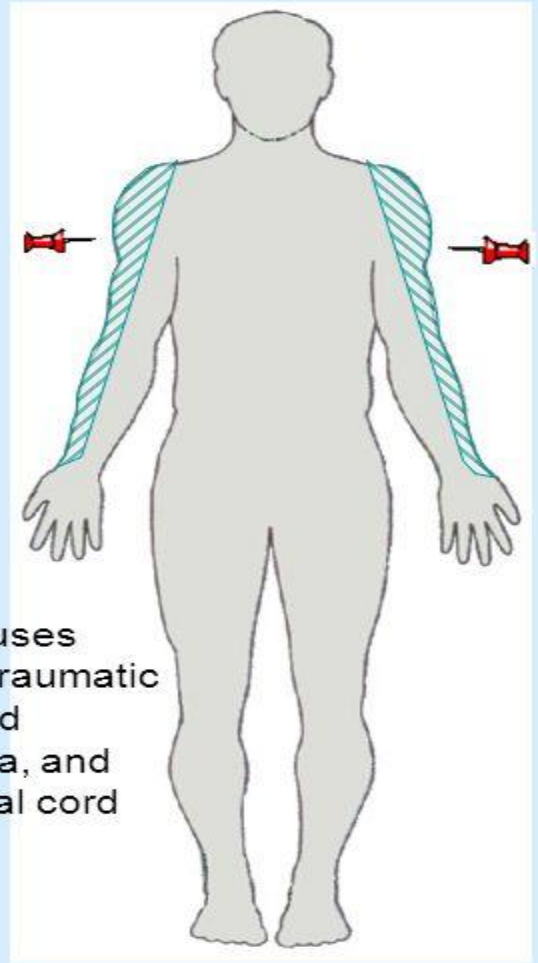
Central cord syndrome

- Decreased sensation and motor functions in the arms with relative sparing of the legs after forced hyperextension (fall, whiplash)
- Can be associated with bladder dysfunction
- Seen in elderly with underlying cervical spondylotic myelopathy
- Damages the central portion of corticospinal tract and decussating fibers of lateral spinothalamic tract
- UE has more weakness than lower (arm fibers are closer to the center)
- Dissociative anesthesia (selective loss of pain and temperature sensation in arms- cape like distribution)

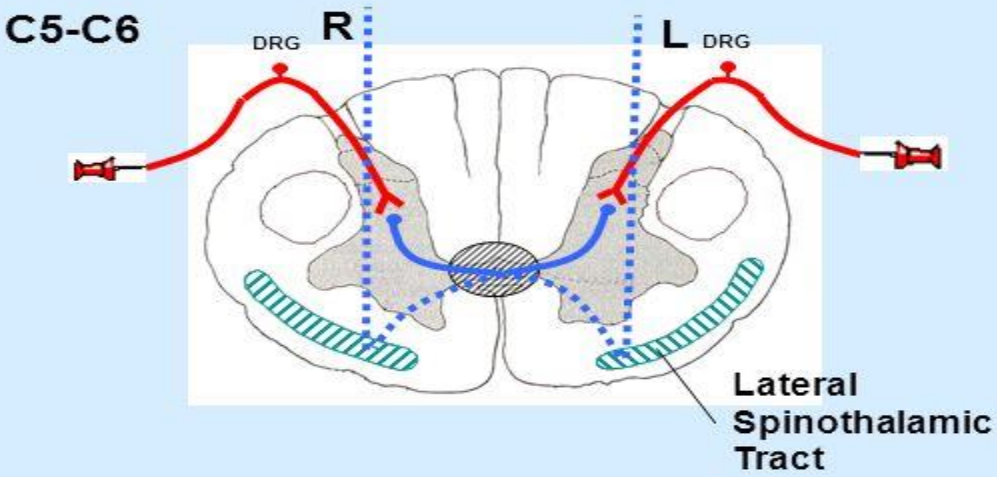
Contd..

- Syringomyelia: Fluid filled cavity in the cord that represents dilatation of central canal or a separa**Cervical or thoracic** te cavity within the spinal parenchyma
- but may involve brainstem (Syringobulbia)
- Commonly associated with **Arnold Chiari malformation type 1** but can also be due to inflammation, tumor, infection or trauma
- As the cavity enlarges→ interruption of anterior gray matter →LMN signs of UE (areflexic weakness)

Central Cord Syndrome



Common causes include posttraumatic contusion and syringomyelia, and intrinsic spinal cord tumors.

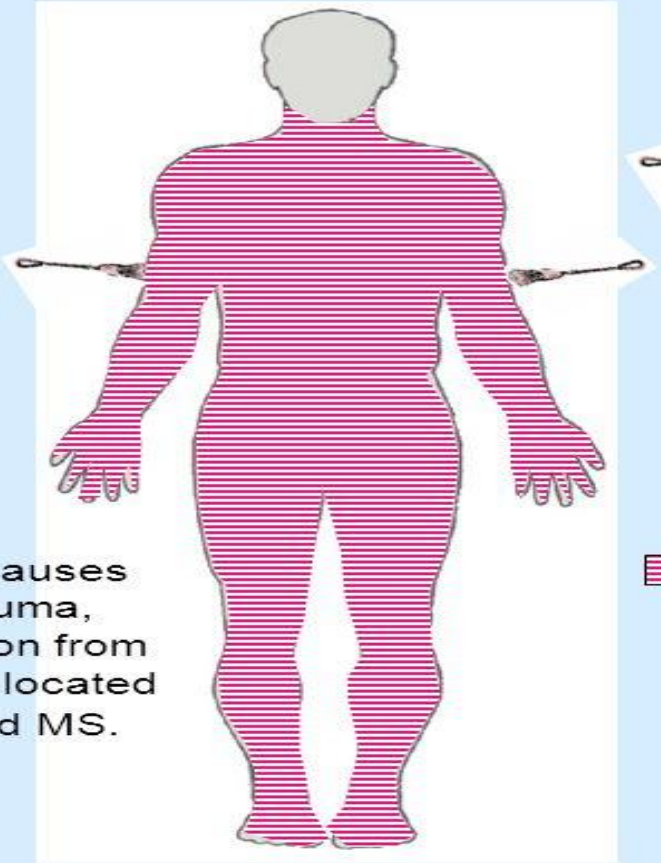


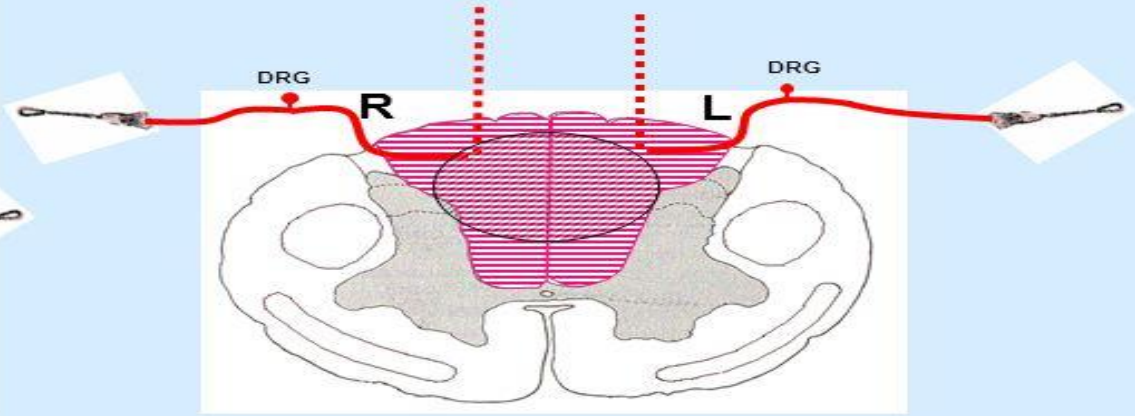
Impaired pain and temperature sensation, C5-C6 dermatomes, ***bilaterally***

Posterior cord syndrome

- B/L loss of position and vibration often with weakness, parasthesias , urinary incontinence or retention
- MC causes are MS and vascular disruption (e.g Vertebral artery dissection)

Posterior Cord Syndrome



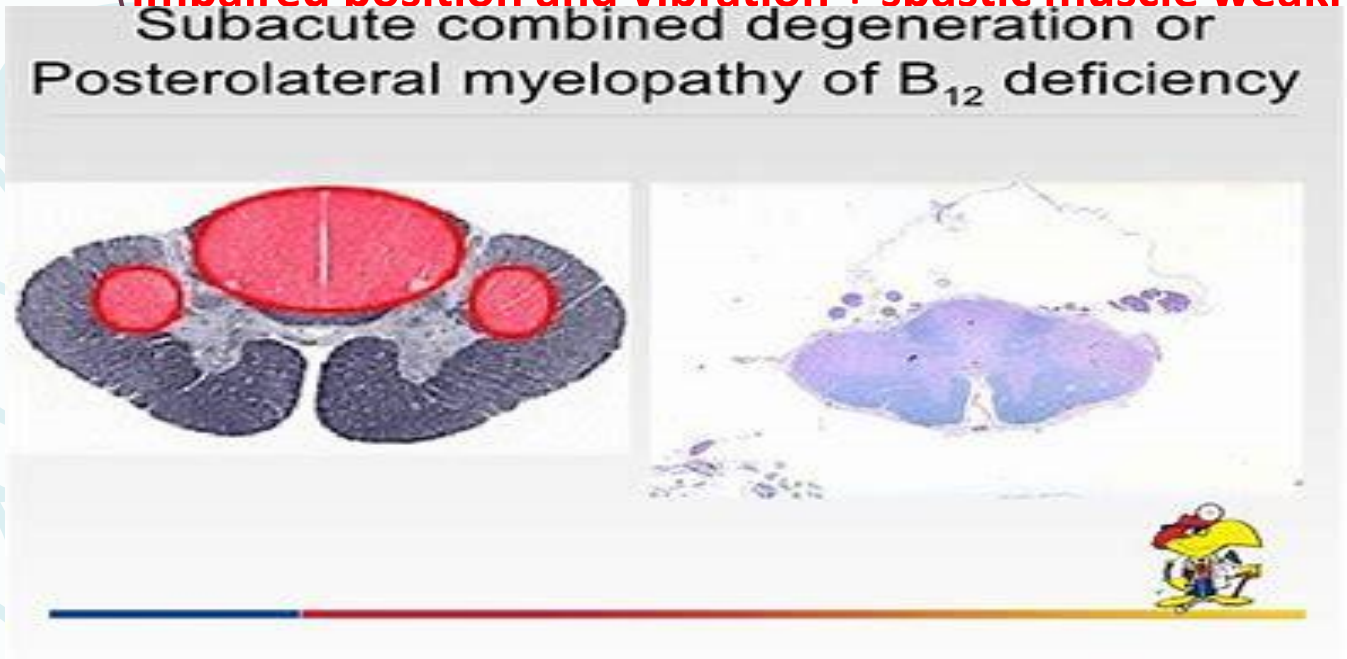


Common causes include trauma, compression from posteriorly located tumors, and MS.

Dorsal column lesion (bilateral)
Bilateral loss of light touch, vibration, and position sense, generalized below lesion level

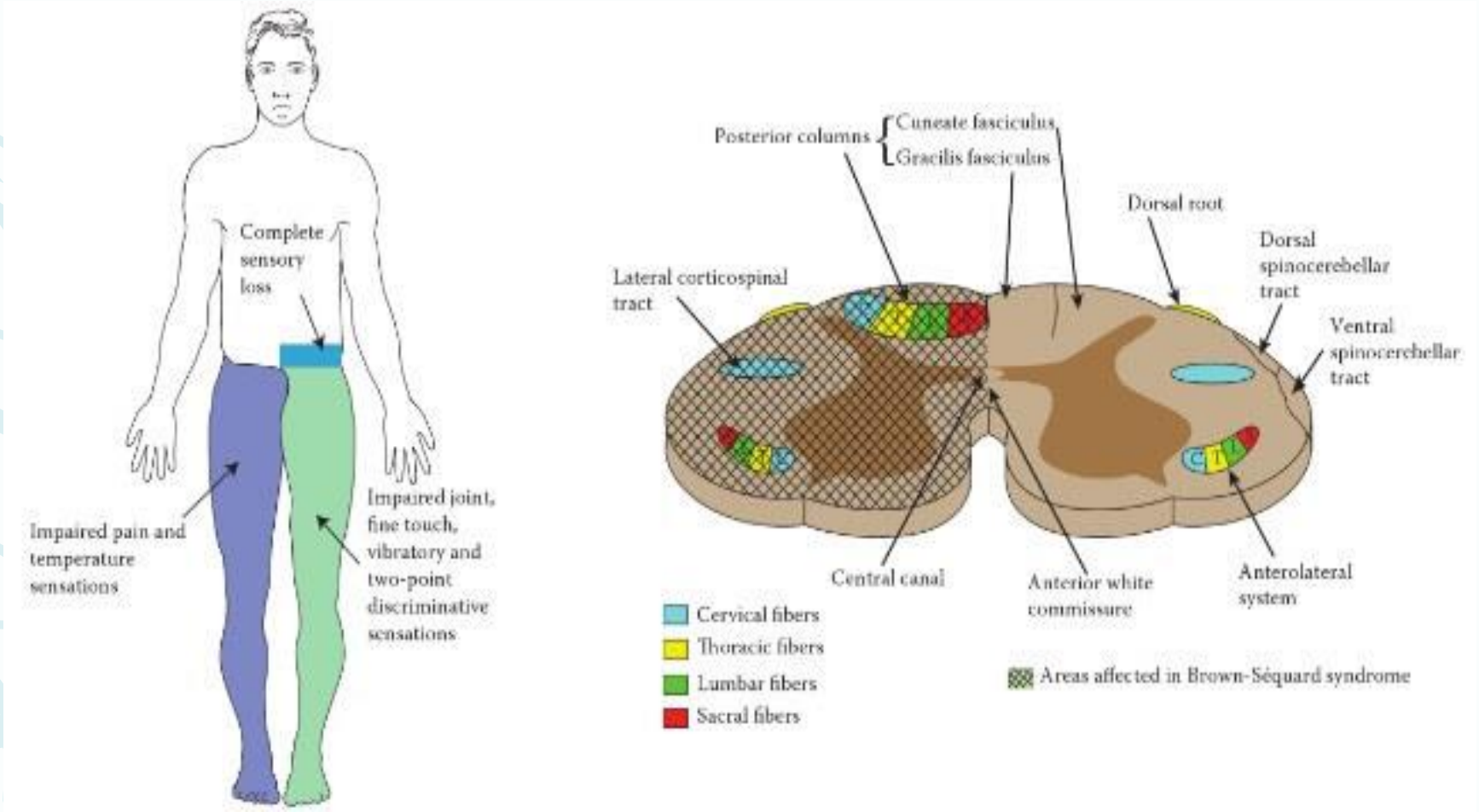
Subacute combined degeneration

- Subacute combined degeneration can occur in Vitamin B12 deficiency (**Impaired position and vibration + spastic muscle weakness**)→ sensory ataxia



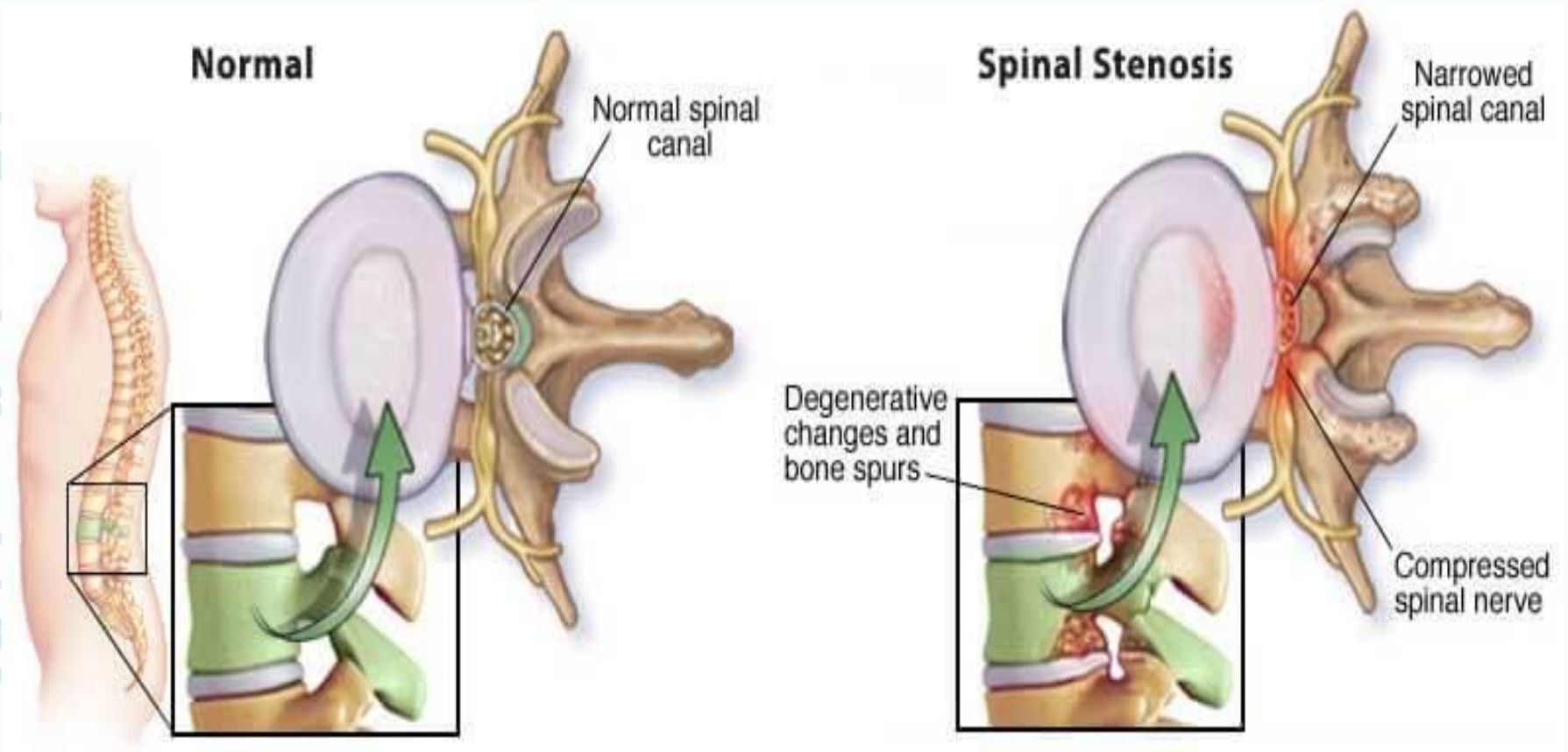
Cord hemisection

- Mostly due to penetrating injury
- Leads to **Brown-Sequard syndrome**
  - Ipsilateral: Weakness, spasticity, loss of position and vibration
  - **Contralateral: Loss of pain and temperature**


Complete sensory loss
Impaired pain and temperature sensations
Impaired joint, fine touch, vibratory and two-point discriminative sensations
Posterior columns
Cuneate fasciculus
Gracilis fasciculus
Dorsal root
Dorsal spinocerebellar tract
Ventral spinocerebellar tract
Lateral corticospinal tract
Central canal
Anterior white commissure
Anterolateral system
Cervical fibers
Thoracic fibers
Lumbar fibers
Sacral fibers
Areas affected in Brown-Séquard syndrome

Lumbar spinal stenosis

- Can cause low back and leg pain.
- Narrowing spinal canal → compression of spinal nerve roots (**neuropathic claudication**)
- MC cause is degenerative joint disease (disc herniation and facet osteophytes impinge upon the spinal cord)
- **Flexion of spine causes widening of spinal canal (improvement of symptoms like sitting and uphill walking)**
- **Extension causes narrowing of spinal canal (therefore exacerbation of symptoms, standing and downhill walking)**
- Weakness, sensory loss, numbness/tingling and leg discomfort
- Neuro exam can be normal and **only 10% will have positive straight leg test**
- MRI is the study of choice.


Normal
Normal spinal canal
Spinal Stenosis
Narrowed spinal canal
Degenerative changes and bone spurs
Compressed spinal nerve

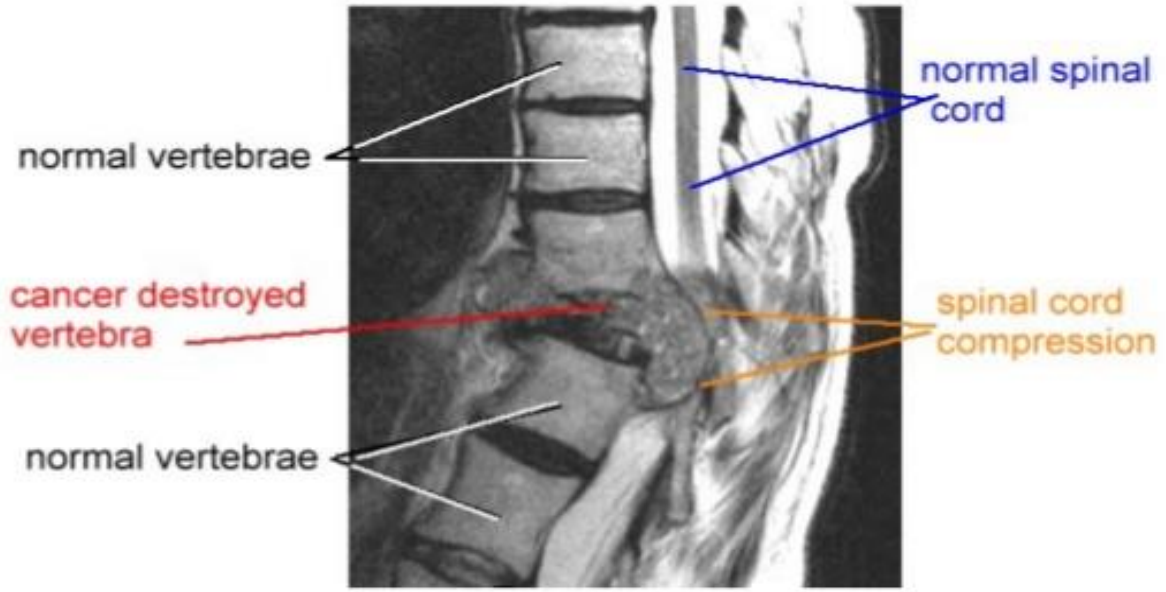
Spinal cord compression

- Causes: Spinal injury (MVA), malignancy (lung, breast, prostate cancers, myeloma), infection (epidural abscess-IV drug abuser)

- Gradual worsening, severe local back pain
- Pain worse in the **recumbent position/ at night**
- Early signs: Symmetric LE weakness (Corticospinal tract), **hypoactive/ absent DTRs**
- Late signs: B/L Babinski reflex, decreased rectal sphincter tone, urinary retention, flaccid bladder, bladder shock (autonomics in reticulospinal tract) paraparesis/paraplegia with increased DTRs, sensory loss (spinothalamic tract-sensory level is 2 spinal segments below the level of lesion)

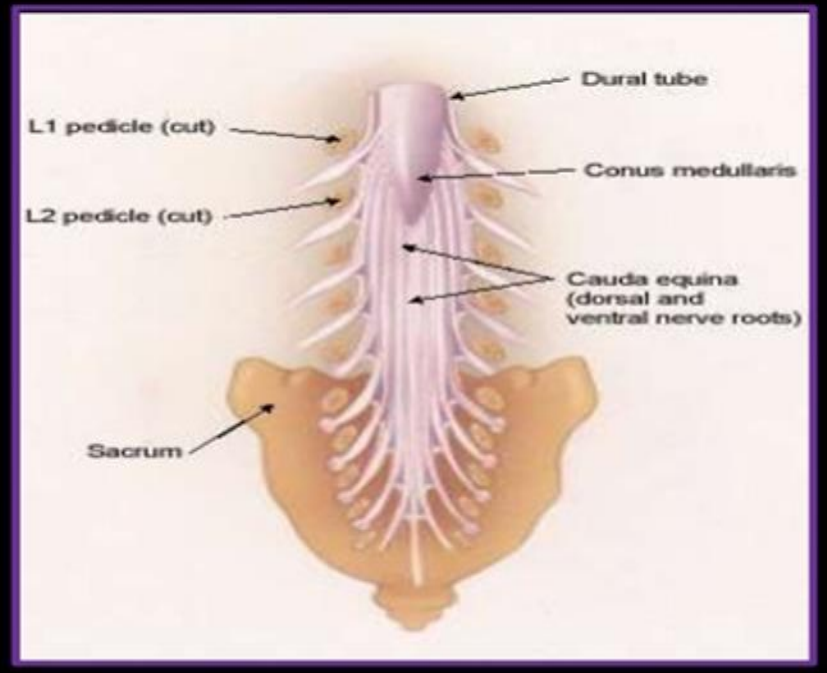
- Management: **Emergency MRI, IV glucocorticoids, radiation oncology/neurosurgery consultation**

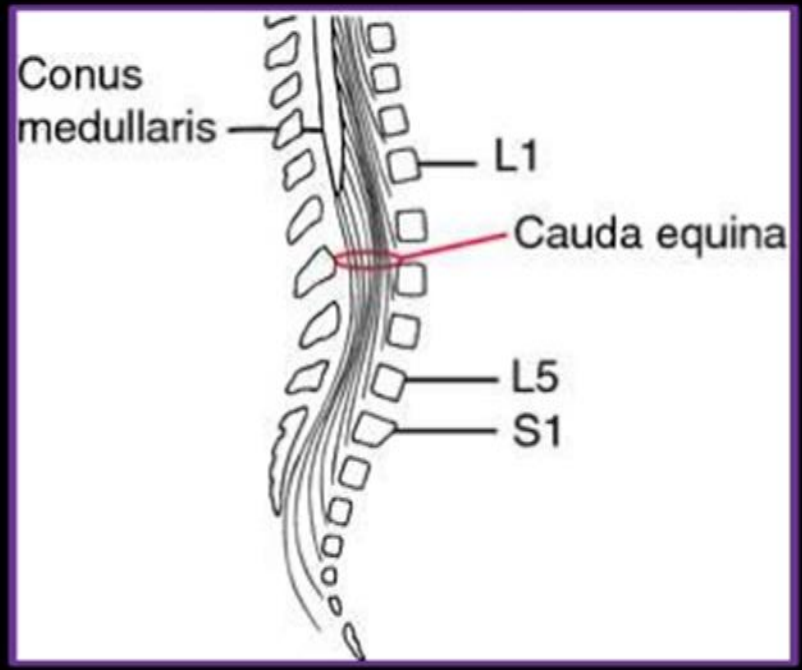
Spinal Cord Syndrome



Cauda equina syndrome	Conus medullaris
Usually bilateral, severe radicular pain	Sudden onset severe back pain
Saddle hypo/anesthesia	Perianal hypo/anesthesia
Asymmetric motor weakness	**Symmetric** motor weakness
Hypo/areflexia	**Hyperreflexia**
Late onset bowel bladder dysfunction	**Early onset** bowel bladder dysfunction

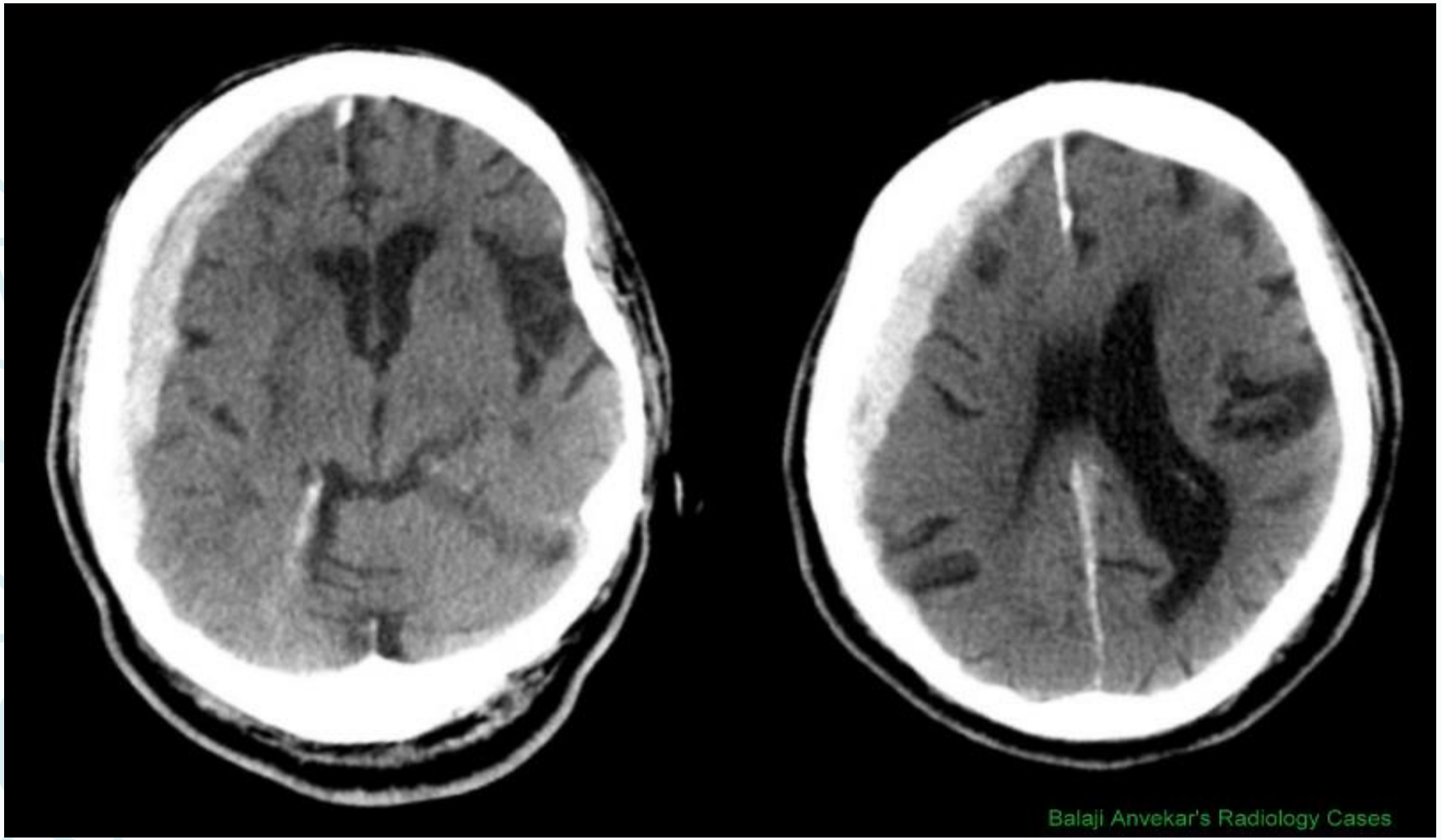
CONUS MEDULLARIS & CAUDA EQUINA SYNDROMES





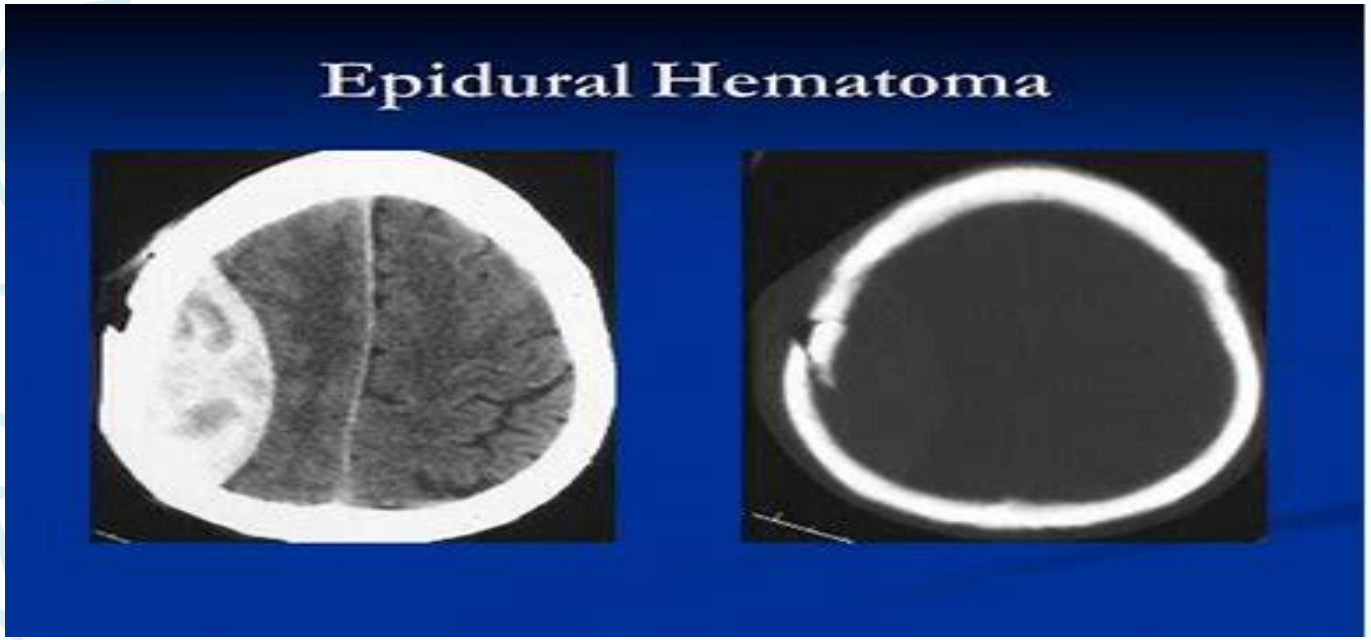
Subdural hematoma

- Tearing of bridging veins → slow bleed into subdural space
- Patients with cerebral atrophy (elderly and alcoholics) are at high risk because the bridging veins must traverse long distance → more susceptible to tears
- Anticoagulants increase the risk of bleeding
- C/F: gradual onset of symptoms (1-2 days after the initial injury), impaired consciousness, confusion and symptoms of intracranial hypertension (headaches, nausea , vomiting)
- CT brain: **Crescent shaped** hyperdensity that crosses suture lines
- Treatment: Conservative if small and surgical evacuation if large


Balaji Anvekar's Radiology Cases

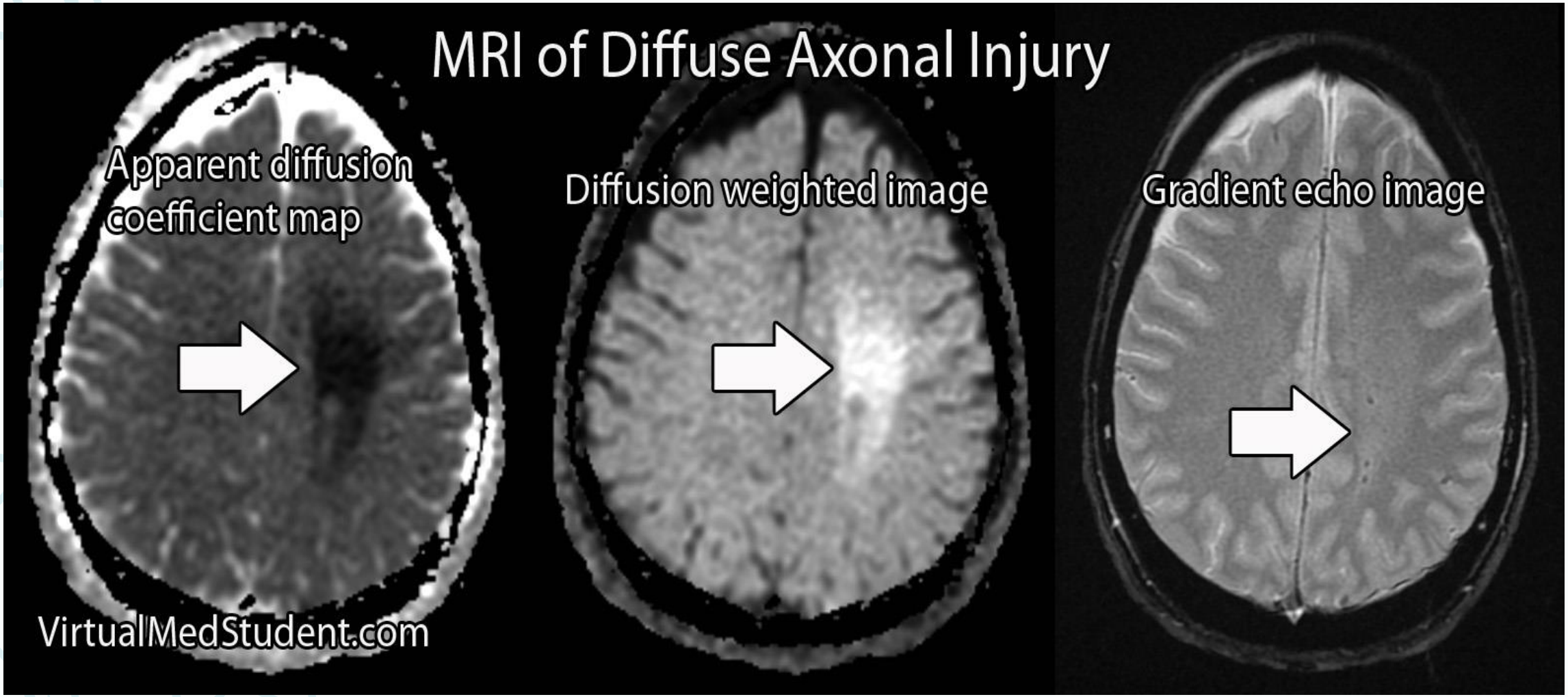
Epidural hematoma

- **Middle meningeal artery injury** (from trauma to sphenoid bone)
- CT brain: **Biconvex** hematoma



Diffuse axonal injury

- Traumatic acceleration/ deceleration shearing forces that diffusely damages axons in the brain
- C/F: Instantly lose consciousness (Coma) and then persistent vegetative state
- CT brain: May show diffuse small bleeds at the grey-white matter junction but MRI is more sensitive


MRI of Diffuse Axonal Injury
Apparent diffusion coefficient map
Diffusion weighted image
Gradient echo image
VirtualMedStudent.com

Post concussive syndrome

- Headache, confusion, amnesia, difficulty concentrating or multitasking, vertigo, mood changes, sleep disturbance and anxiety
- Usually follows a mild TBI
- Symptoms resolve within few weeks to months following TBI
- Some patients have persistent symptoms lasting > 6 months

Movement disorders

Parkinson's disease

- **Resting tremor, bradykinesia, rigidity (lead pipe or cogwheel), postural instability (at least 2 signs –clinical diagnosis)**
- **4-5 Hz rest tremor**, asymmetric
- Mask-like facies, decreased arm swing, stooped posture, micrographia, hypophonia
- Degeneration of neurons of substantia nigra → decreased dopaminergic activity, increased cholinergic activity
- Trihexyphenidyl : young patients with tremor predominance

Medications

Drug	Action	Side effects
Levodopa plus carbidopa	Dopamine precursor	Somnolence, confusion ,hallucinations , dyskinesia
Trihexyphenidyl or Benztropine	Anticholinergic	Dry mouth, blurry vision , constipation, urinary retention , nausea
Amantadine	Unclear mechanism	Ankle edema, livedo reticularis
Apomorphine, Bromocriptine, Pramipexole, Ropinirole	Dopamine agonist	Somnolence, hypotension, confusion Hallucinations (older pts)
Entacapone, Tolcapone	COMT inhibitor	Dyskinesia, hallucinations , confusion, nausea, orthostatic hypotension
Selegiline	MAO B inhibitor	Insomnia, confusion (elderly patients)

PARKINSON'S DISEASE

- Onset usually gradual, after age 50. (Slowly progressive)

- Mask-Like, Blank Expression
- Stooped Posture
- Pill Rolling Tremors

Bradykinesia
- Loss of normal arm swing while walking
- ↓ Blinking of the eyelids
- Loss of ability to swallow
- Blank expression
- Difficulty initiating movement

- Possible Mental Deterioration
- Depression

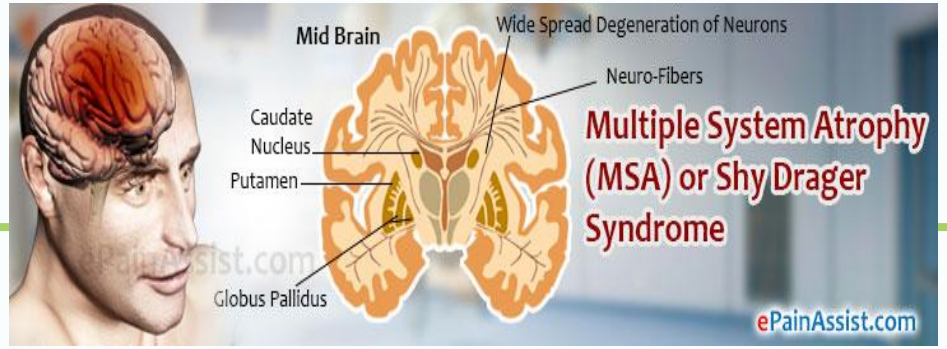
Tremor
- Commonly in hands and arm
- Pill rolling motion with the fingers
- Occurs most often at rest
- May involve diaphragm, tongue, lips and jaw
- Increases with stress

Muscle Rigidity
- ↑ Resistance to passive movement
- Cog wheel, jerky slow movement

- Rarely Occurs In Black Population

- Shuffling, Propulsive Gait

Multiple system atrophy



- **Shy Drager syndrome** is a degenerative disease
- C/F:
  - Parkinsonism
  - **Autonomic dysfunction** (postural hypotension, bladder bowel disturbance, impotence, abnormal salivation/lacrimation, gastroparesis)
  - Widespread neurological signs (cerebellar, pyramidal or lower motor neuron)
  - Bulbar symptoms and **laryngeal stridor are fatal**
- Antiparkinsonian treatment are usually ineffective
- Orthostatic hypotension: Intravascular volume expansion, Fludrocortisone, salt supplementation, alpha adrenergic agonists and constrictive garments to lower body

Tremor

Tremor	C/F
Essential tremor	Bilateral action tremor of hands without leg involvement Possible isolated head tremor without dystonia No other neurological signs Improves with alcohol
Parkinson's disease	Rest tremor (4-6 Hz) that decreases with voluntary movement. Pill rolling quality. Usually involves legs and hands Facial involvement less common (jaw, face,tongue, lips) and not the whole head More pronounced with distractibility and re-emergence when movement is stopped
Cerebellar	Associated with ataxia, dysmetria or gait disorder Tremor increases steadily as the hand reaches its target
Physiologic	Low amplitude (10-12 Hz) not visible under normal conditions Acute onset with increased sympathetic activity (drugs , hyperthyroidism, anxiety, caffeine) Usually worse with movement and can involve face and extremities

Essential tremor

- Intention tremor affecting the upper extremities, head, voice and other body parts
- **Resolves during sleep and improves with alcohol**
- Positive family history
- 5% of population

C/F:

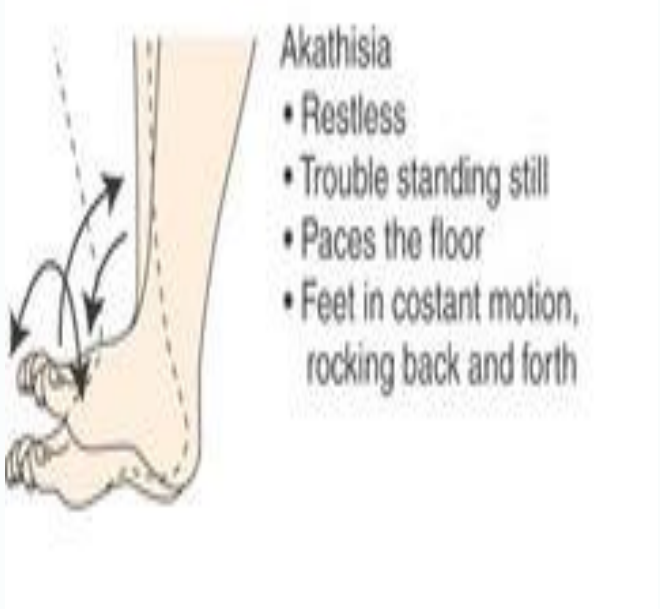
- Action and postural tremors
- Bilateral
- **Hands> arms>head >>legs**
- Treatment: **Propranolol, Primidone, Clonazepam**

Chorea

- Brief, irregular, unintentional muscular contractions.
- Movements flow from one location to another but are not repetitive or rhythmic.
- Huntington's disease: Autosomal dominant, affects both sexes equally(30-50 yrs)
- C/f: Mood disturbance(depression, apathy),dementia, choreiform movements (facial grimacing, ataxia, dystonia, tongue protrusion, writhing movements)
- Progressive eventually disabling

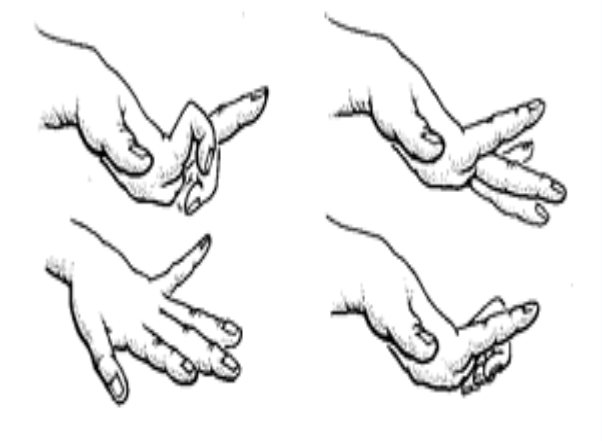
Akathisia

- Sensation of restlessness that causes patients to move frequently



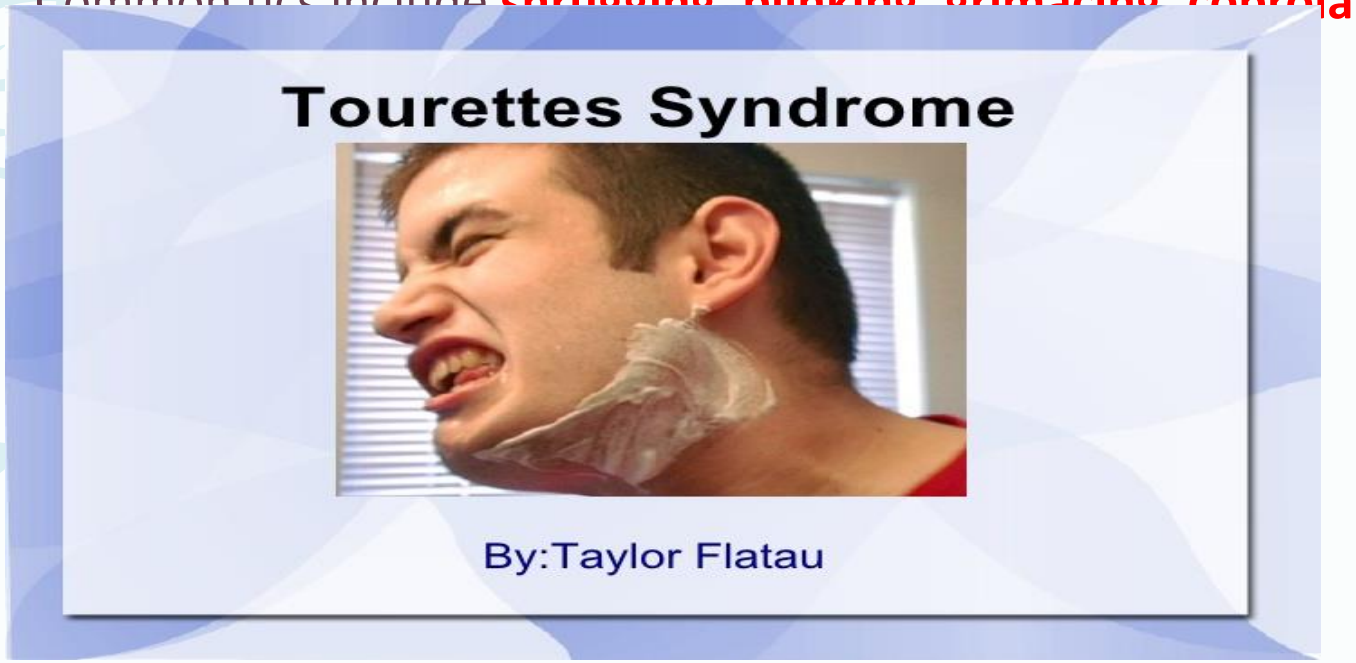
Athetosis

- Slow, writhing movements that affect the hands and feet
- Athetoid movements are a characteristic of Huntington disease
- Chorea and athetosis can happen



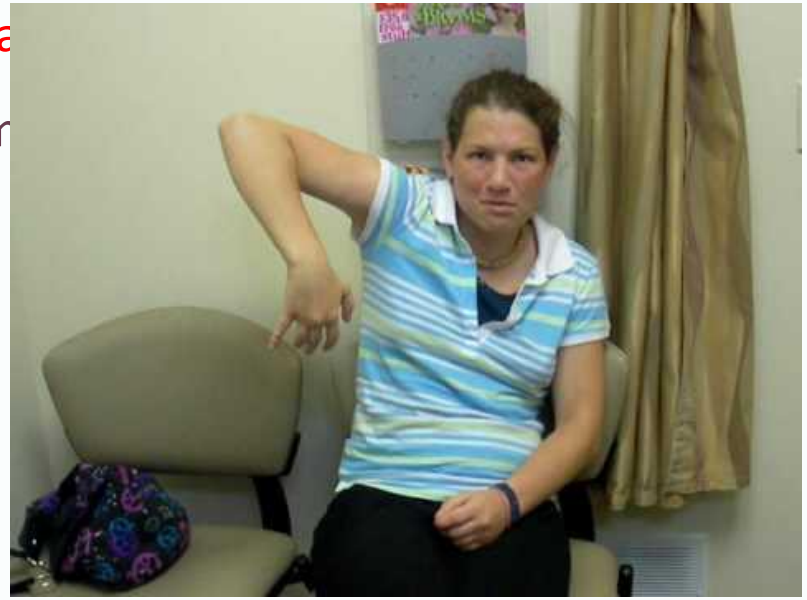
Tourette syndrome

- Involuntary, stereotyped, repetitive movements and vocalizations called tics.
- Common tics include **shrugging, blinking, grimacing, coprolalia (swearing)**



Hemiballismus

- Unilateral, violent , arm flinging movements
- Due to damage to contralateral subtha
- Very disruptive but tends to be self lim



Myoclonus

- Involuntary **jerking of muscle or muscle group**
- Usually rhythmic or patterned (unlike chorea)
- Usually initiated by contraction or relaxation

Wilson's disease

- Autosomal recessive
- Abnormal **copper deposition** in tissues like liver, cornea, basal ganglia.
- Children and young adults usually present with liver disease (asymptomatic aminotransferase elevation to fulminant hepatic failure)
- Young adults →Neuropsychiatric disease (**tremor, rigidity to depression, paranoia and catatonia**)
- Diagnosis: Low serum ceruloplasmin (<20 mg/dL) +increased copper excretion/ KF rings (Kayser-Fleischer → slit lamp exam)

Torticollis

- Focal dystonia of sternocleidomastoid muscle.
- Dystonia : Sustained muscle contraction resulting in twisting, repetitive movements or abnormal postures
- Focal (affecting only one muscle) or diffuse.
- Can be congenital, idiopathic, secondary to trauma, local[illegible]ug induced, medications (typical antipsychotics, metoclopra[illegible] prochlorperazine)


Thank
You