Headaches

	Migraine	Cluster	Tension
	F > M	M > F	F > M
1. Severity	Moderate to Severe	Excruciating	Mild to Moderate
2. Frequency	Every few weeks	1-2/ Day, during cluster period	Variable
3. Peaks	Peaks in 1/2 hour	Peaks 2-15 minutes	Variable
4. Duration	Hours to 1-2/days	30-120 minute	30 min -7 days
5. Location	Unilateral (temporal or frontal)	Unilateral (orbital)	Bilateral diffuse
6. Type	Pulsating	Non- pulsating	Non-pulsating
7. Associated Features	Nausea, vomiting Photophobia, aura Phonophobia	Unilateral lacrimation Rhinorrhea or blockage Ipsilateral horner's	None
8. Treatment	Analgesics, Isomethepten Ergotamine, sumatriptan	100% O2, S/C sumatriptan Intra-nasal lidocaine	Analgesics
9. Prophylaxis	Aspirin, beta blockers Tricyclics (Amitriptyline) Calcium blockers Divalproex sodium	Prednisone 60 mg/day for one week Ergotamine	Tricyclics, Beta blockers

Oculogyric Crisis
- acute dystonic reaction
- common cause → compazine
- Sx:) Forced upward eye deviation
- Tx: Benztropine IV therapy

if allodynia (facial pain) Tx: Metoclopramide

Medication Overuse HA's
- chronic OTC use for HA's
- ↓ response to meds
- @ risk if using meds > 2x @ week
- Tx: - taper meds + migraine prophylaxis

Menstrual Migraine
- ↓ estrogen immediately preceding ovulation
- Tx: Estradiol 10 mcg

LP HA's
- usually remits w/in 1 week
- Tx: IV caffeine

Prophylaxis Indications:
① ≥ 2 days HA's Quick

(can be prolonged but this is more (Ex:) (e.Mrs.) may mimic a CVA/TIA

Transient focal neuro dysfx
- develops over mins (Ex:) Homonymous Hemianopsia
- last < 1 hr
- may precede HA's
- can be visual flashes of light
- assoc. w/ aphasia
- unilateral paresthesias or numbness
- unilateral weakness

+ Dopamine antag. (may be given SC or SL)

may see CSF pleocytosis w/ predominance of mono's

breaks Cluster period

Complications of Subarachnoid Hemorrhage

- can present as a "pop" in the head
- most important prognostic factor → condition of pt. @ arrival
- severe HA
- sudden onset
- w/out aura
- assoc. w/
  - brief LOC
  - diffuse HA's
- vomiting
- retinal hem's
- ↓ focal necrosis signs sometimes
- ptosis of one eye
- dilated pupils

1. Rebleeding
2. Hydrocephalus
3. Vasospasm → usually several days after initial event → can detect by transcranial Doppler → can lead to ↓ CVA

Cardiac Abnorms
- Deep T-wave inversions
- cMarker abnorm
- LV dysfx

cardiac stun

Dx: Non-contrast CT → then spinal tap if CT (-) → then angiogram (definitive dx of cause) → then NSurg → endovasc. coiling

Screening for SAH
- ⊕ FH
- AD PCKD
- ↑-risk pt's

Causes OF Pseudotumor Cerebri

- recent rapid wt gain

1. Obesity
2. Hypervitaminosis A
3. Tetracycline
4. Glucocorticoids
5. Oral contraceptives
6. Amiodarone

Complications
- visual field losses periphery → central
- ↓ serial visual field tests
- papilledema

Tx: - carbonic anhydrase inhibs
- shunts

AFTER LP confirms Dx

if pt. comatose: ventricular drain

Tx: oral Nimodipine CCB 60mg q4h x21 days
↑ transcranial Doppler velocities → HHH Tx
↑ BP → HTN
↑ CVP → Hypervolemia
↓ Hct → Hemodilution

Brain Tumors

optic chiasm tumors → (B) temporal hemianopsia

Grade I → 4 (worse) most important prognostic factor then cell type

solitary Metastases: - surgical resection then whole brain Rad Tx

- constant HA worse lying down, coughing or valsalva
- seizures can occur (esp. age >35)

Symptomatic Brain Tumor Tx
- IV Dexa + prophylactic anti-convulsants

50%	High grade or malignant astrocytoma
40%	low grade astrocytomas, ependymomas
	Oligodendroglioma (better prognosis than astrocytoma)
10%	Extraaxial tumors (meningioma, schwannoma)

Tx: Surgery then Rad Tx + chemo → resection then surveillance MRIs

↳ often asymptomatic → f/u Q3-6 months w/ reimaging

Frontal Lobe Meningioma
- personality Δ's
- irritability
- emotional lability
- often large size → surgical resection if <3cm → stereotactic Rad-Tx

C presentation

- Recurrent Tumors very poor prognosis

Neurological Evaluation

Upper Motor Neuron	Weakness, spasticity, hyperactive tendon reflexes, extensor planter reflex (Babinski's sign)
Lower Motor Neuron a) Ant. horn cells b) Motor roots c) Peripheral nerves	Weakness, hypotonia, atrophy, loss of tendon reflexes, fasciculations, sensory symptoms if mixed nerve is involved
Muscle Diseases	Weakness (proximal> distal), no sensory changes, reflexes preserved, atrophy, no fasciculations, eye and bulbar muscles are spared
Neuromuscular junction (Eti) Myasthenia	Weakness increases with activity, no sensory symptoms, eye and bulbar muscles are involved, reflexes normal, no atrophy or fasciculations

(+) Romberg's ↳ signal of post. column

Cerebellar signs
- (+) finger-nose test
- ↓ sensory abnorms or weakness

Sustained clonus

Adenocarcinoma of lungs w/ HA's / diplopia / dysphagia / foot drop

MRI → hydrocephalus

Think Leptomeningeal mets

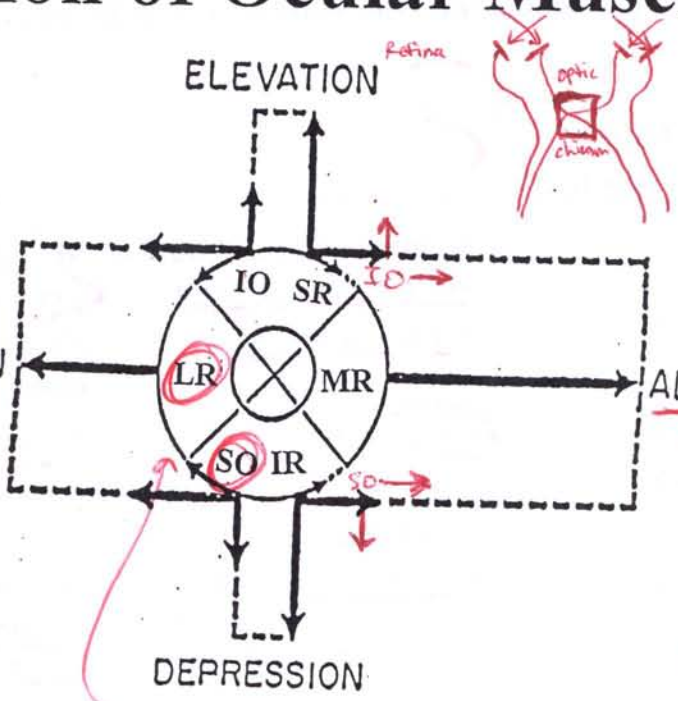
Glossopharyngeal Neuralgia (CN IX)
- paroxysmal pain
- tonsillar fossa
- precipitated by swallowing

Tx: Tegretol

Trigeminal Neuralgia
- knife-like pain in lips/gums/cheeks or chin
- lasts seconds @ a time
- triggered by touching certain areas of face

Tx: Carbamazepine

Action of Ocular Muscles



LR → abducts
MR → adducts
IR → pulls down
SR → pulls up

SO → adducts + pulls down
IO → adducts + pulls up

Retinal Ischemia
- transient blindness
- ↓ carotid u/s's

↳ lateral gaze

↳ towards nose

Amaurosis Fugax
- partial transient monocular blindness
↓ carotid u/s's

Binocular Diplopia
- disappears when covering 1 eye
↳ caused by misalignment of muscles or some paralysis of III, IV or VI.

Monocular Diplopia
- does not correct when covering 1 eye
↳ psychogenic

All muscles are supplied by 3rd nerve except LR (6th) & SO (4th)

pupil not involved
Age <50 → Postcommunicating aneurysm
>50 → vasc. infarction

IV Nerve Palsy
- inability to move eye outwards
- most common cause - DM

III Nerve Palsy
- ptosis
- eye classically move lateral + down
± dilated pupils
- may be painful if pain

Bell's Palsy
-abrupt onset
-pain behind ear
-mild CSF lymphocytosis common
-Tx: Prednisone (1mg/kg x7-10days) ± valacyclovir
-Cause: ?, HSV
-✓ Lyme Ab's

Facial Nerve Paralysis

Infranulear
-both upper + lower face is involved

Causes:
- Bell's palsy
- Ramsay Hunt Syndrome
- Acustic neuroma
- Pontine tumors or infarction
- Guillain-Barre, sarcoid, lyme

Ramsay Hunt Syndrome
-Hsv involving pharynx or auditory canal
-may involve 8th nerve
-vesicular eruption

Supranuclear
-upper face spared

Cause:
- CVA

more common w/ whole brain Rad Tx
↑
Radiation-Induced Leukoencephalopathy
-MRI → periventricular white matter disease
-triad: A) Apraxia (Inability to perform purposeful movements or use objects appropriately
B) Dementia
C) Urinary Incontinence
-usually presents within 1 yr. post-Rad Tx
-Tx: ϕ

Vertigo

	Peripheral (Vestibular nerve or inner ear)	Central (Brain stem or cerebellum)
1. Severity	+++ ++	+ +/-
2. Nausea/vomiting	some cases	-
3. Tinnitus/hearing loss		
4. Nystagmus	Unidirectional, horizontal vertical never present	Multidirectional Vertical +
5. Visual fixation	Inhibits nystagmus & vertigo	No inhibition of nystagmus & vertigo
6. Associated central abnormalities	None	Dysarthria, diplopia, ataxia, weakness
7. Causes	Vestibular neuritis labyrinthitis, Meniere's disease, acoustic neuroma benign positional	Brain stem dysfunction due to demyelinating, vascular or neoplastic diseases

Acoustic Neuroma
-vertigo w/ HL tinnitus + ear fullness
-✓ MRI

Tx: Abx

- Recurrent vertigo /N/V
- Episodes last mins/hrs
- Multiple episodes → hearing loss (high frequency)

Tx: Salt restriction, diuretics, prochlorperazine then surgical exploration of labyrinth

Vestibular Neuronitis
-usually post-URTI
-Tx: Methyl prednisolone x3wks.

BPPV
-occurs w/ head movements
-confirm w/ Dix-Hallpike
-Tx: Epley Bedside maneuvers (otolith repositioning clears semicircular canal)

Evaluation of Hearing Loss

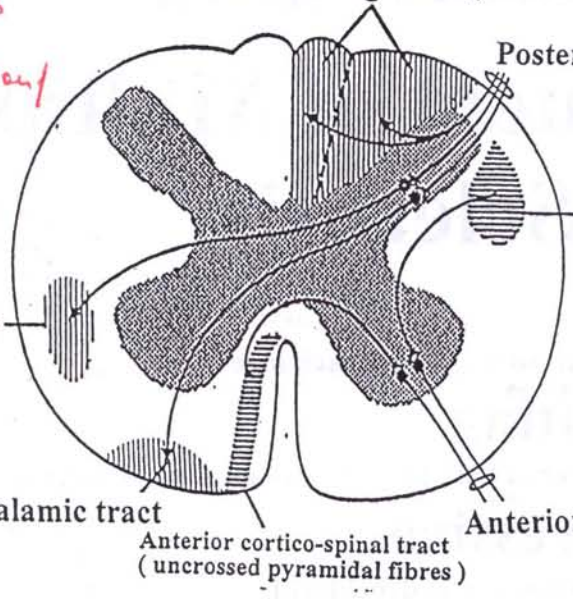
	Conductive (peripheral)	Sensory-Neural (central)
	Ext. auditory canal or middle ear	Inner ear, 8th nerve or central auditory pathway
	(Ex.) plug your ear	
1. Air Conduction	Abnormal	Abnormal
2. Bone Conduction	Normal	Abnormal
3. Rinne's Test		
a) Tuning fork in front of the ear (air conduction)	Abnormal	Abnormal
b) At mastoid process (bone)	Normal	Abnormal
4. Weber Test: Tuning fork over center of forehead	Hears better on affected side	Hears only on normal side

Normal: Air conduction longer than Bone conduction

otosclerosis
- tinnitus
- progressive HL
- otoscopy → ϕ cerumen
- conductive HL
- new formation of spongy bone @ the stapes

Normal
- Longer than Bone
- Shorter than Air
- ① Still present w/ air
- ② Sound Disappears
- Heard equally in both ears

Sensory and Pyramidal Tracts in the Spinal Cord



Syringomyelia
- Progressive myelopathy
- cavitation of central canal
- begins in early adulthood @ mid-cervical cord
- wasting of neck muscles, shoulders, arms
- Dissociated loss of sensation/ pain/ temperature
- touch/vibration preserved
- progressive spastic paraparesis can develop
- MRI C-Spine

Subacute Combined Degeneration (SCD)
- vit. B12 Defic.
- spastic paraplegia
- dementia
- peripheral neuropathy
- loss of position vibration
- affects:
  A) Post. columns
  B) Lateral columns
  C) peripheral nerves

Think cerebral cortex w/ LMN's

LMN → ↓DTR's
UMN → ↑DTR's (hyperreflexic)

B12 Defic.
- tingling
- unsteady gait
- poor memory
- ↓ vibratory sense in LE's
- (+) Romberg's

Cervical cord compression
- UMN below lesion + LMN @ lesion
- Hyperreflexic in LE's + triceps (C7)
- ↓DTR's in biceps (C5+6)
- neck/shoulder/arm pain (+)
- injury @ C5+6 level
- Lhermitte's Sign → shock sensation w/ neck flexion

- midthoracic back pain
- LE weakness/spasticity → Upper motor Neuron sign
- urinary retention → Upper motor Neuron sign
- hyperactive DTR's → Upper motor Neuron sign
- plantar extension (+ Babinski's) → Upper motor Neuron sign

Think → Epidural abscess (spine)

Dx: ✓MRI
Tx: Abx + surgery

Multiple Sclerosis

Most common demyelinating disease of the nervous system. The lesions (plaques) are widely distributed it the white matter of the brain and spinal cord.

Patterns of Multiple Sclerosis

1. Relapsing remitting
2. Secondary progressive
3. Primary progressive
4. Progressive relapsing

Treatment of Multiple Sclerosis

Acute Relapses

Methylprednisolone-prednisone, plasma exchange

Relapsing Remitting

Interferon beta 1b, Interferon beta 1a, Glatiramer acetate, Immune globulin

Secondary Progressive

Interferon beta 1b, Mitoxantrone hydrochloride

Primary Progressive

None

MS-Related Fatigue: Tx: Amantadine

Parkinson's Disease

Clinical Dx

Disorder of unknown etiology in which there is degeneration of the dopamine containing neurons in the basal ganglion

- Bradykinesia
- Drag Feet
- Shuffling Gait / Falls
- Expressionless Face
- Difficulty Turning
- Rigidity

Parkinson Tremors
- asymmetric
- more pronounced @ rest
- tongue / jaw / chin tremors can occur
- handwriting is small + irregular

Essential Tremors
- symmetrical
- more pronounced w/ activity
- handwriting is large + tremulous

- normal sensation
- normal DTR's
- normal strength

Secondary Causes of Parkinsonism

usually presents w/out tremor (in contrast to idiopathic Parkinsonism)

1. Drugs : Phenothiazines, reserpine, metoclopramide
   - ↳ blocks both peripheral + central Dopamine receptors
2. CO Poisoning
3. Wilson's disease
4. Illicit drug MPTP (methyl phenyl tetrahydro pyridine)
5. Post-encephalitis

Huntington's Chorea
- brief / brisk unpredictable movements
- assoc. cognitive / psychiatric changes
- usually develops in 30's-40's

Restless leg Syndrome
- ↓ ferritin level if < 50
- Tx: Fe

Progressive Supranuclear Palsy
- Parkinsonism
- vertical gaze palsy
- dysarthric
- dysphagia

Multiple System Atrophy
↓
Shy-Drager Syndrome (dysautonomia)

Treatment of Parkinson's Disease

- Start Tx when symptoms bother pt. or disability

First-Line Dopaminergic Agents

1. Levodopa (Dopamine precursor) + Carbidopa (Peripheral dopa decarboxylase inhibitor)
   - Dx's for pt's > 70 y.o.
   - prevents peripheral Dopamine breakdown
   - common side effect: nausea
     - ↓ due to peripheral Dopamine breakdown
     - ↑ carbidopadase
     - ↓ peripheral breakdown
2. Levodopa + Carbidopa + Entacapone (COMT inhibitor)
   - blocks degradation of L-Dopa + Dopamine
3. Dopamine agonist → 1st choice for younger pt's < 70 y.o.
   - Ergot: Bromocriptine, Pergolide → not available
   - Preferred → Nonergot: Pramipexole, Ropinirole, Rotigotine transdermal patch

Second-Line Alternatives

1. MAO-B inhibitor: Selegiline (deprenyl)
   - prevents Dopamine breakdown
2. Anticholinergics: Trihexyphenidyl, Benztropine
3. NMDA antagonist: Amantadine (Symetrill)

Surgical Treatment

↳ Deep brain stimulation

Drug-Induced Psychosis (Hallucinations)
- avoid Haldol → worsens Parkinsonism
- Tx: Seroquel or Clozapine

Complications of Levodopa Therapy

1. On-off phenomenon
2. Nausea-vomiting
3. Dyskinesias
4. Psychosis

imbalanced Levodopa/Carbidopa dosing → ↑Carbidopa dose → ↓'d peripheral Dopamine breakdown

Sudden weakness, loss of mobility, ϕ relation to dose schedules
Tx: COMT Inhibs or Dopamine Agonists or Δ dosing schedule

Dystonia, Choreathetosis

Muscle Diseases

Myasthenia gravis
- assoc. w/ autoimmune d/o's
- females > males
- diplopia @ end of day (worsens w/ fatigue)
- occasional dysphagia
- asymmetric ptosis
- Dx: Edrophonium Test → given IV will reverse symptoms
- Ach receptor Ab's confirms Dx
- Repetitive nerve stimulation → ↓'ing action potentials
- chest/Abd. → look for thymoma

Myasthenia crisis → weakness, dysphagia, resp. insufficiency
- ↓vital capacity → if <20 ml/kg → intubation
- Tx: ICU, plasmapheresis, IV Ig, Immunosuppressives

Tx: - ocular disease → pyridostigmine (method. increase)
- generalized disease → pyridostigmine plus thymectomy, then steroids, then Azathioprine

Lambert-Eaton syndrome
- symmetric proximal weakness
- strength improves w/ activity
- ↓CK → look for oat-cell cancer
- autonomic dysfunction (small)

EMG → action potentials ↑
NCS → action potentials ↓↑↑ confirms Dx

Muscular dystrophies

Duchenne
- chr-by pseudohypertrophy
- abnormal dystrophingene
- mental retardation
- x-linked
- ↑↑CK
- onset @ 3-5 yrs.
- unable to walk by 12 yrs.
- survival <25 yrs.
- Death Causes: A) CMyopathy B) Resp. Failure

Becker
- milder than Duchenne
- x-linked too

Myotonic
- AD inheritance
- Sustained contraction/inability to relax
- normal CK
- complications: A) cataracts B) cardiac conduction defects C) Insulin Resistance D) Frontal Balding

inability to relax after handshake

Facioscapulohumeral
- slightly ↑CK
- AD-inheritance
- assoc./complicated by HTN

Limb-girdle
- AR-inheritance
- complicated by cmyopathy

Thyroid Myopathy (Hypothyroidism)
- paresthesias/pain
- CK/MB can both be ↑'d
- ✓TFT's

Mitochondrial myopathy → progressive eye movement paralysis

Toxic myopathies

Lipid lowering drugs: Fibric acid derivatives, Statins, Niacin
- stop drug w/ myalgias
- leg pains
- normal CK seen @ times

Proximal myopathy

Steroids, Zidovudine, Colchicine

Drugs of abuse: Alcohol, Amphetemine, Heroin, Phencyclidine, Meperidine

Botulism
- ingested preformed toxin
- blurred vision/diplopia
- vomiting/diarrhea
- progressive descending weakness
- fixed/dilated pupils

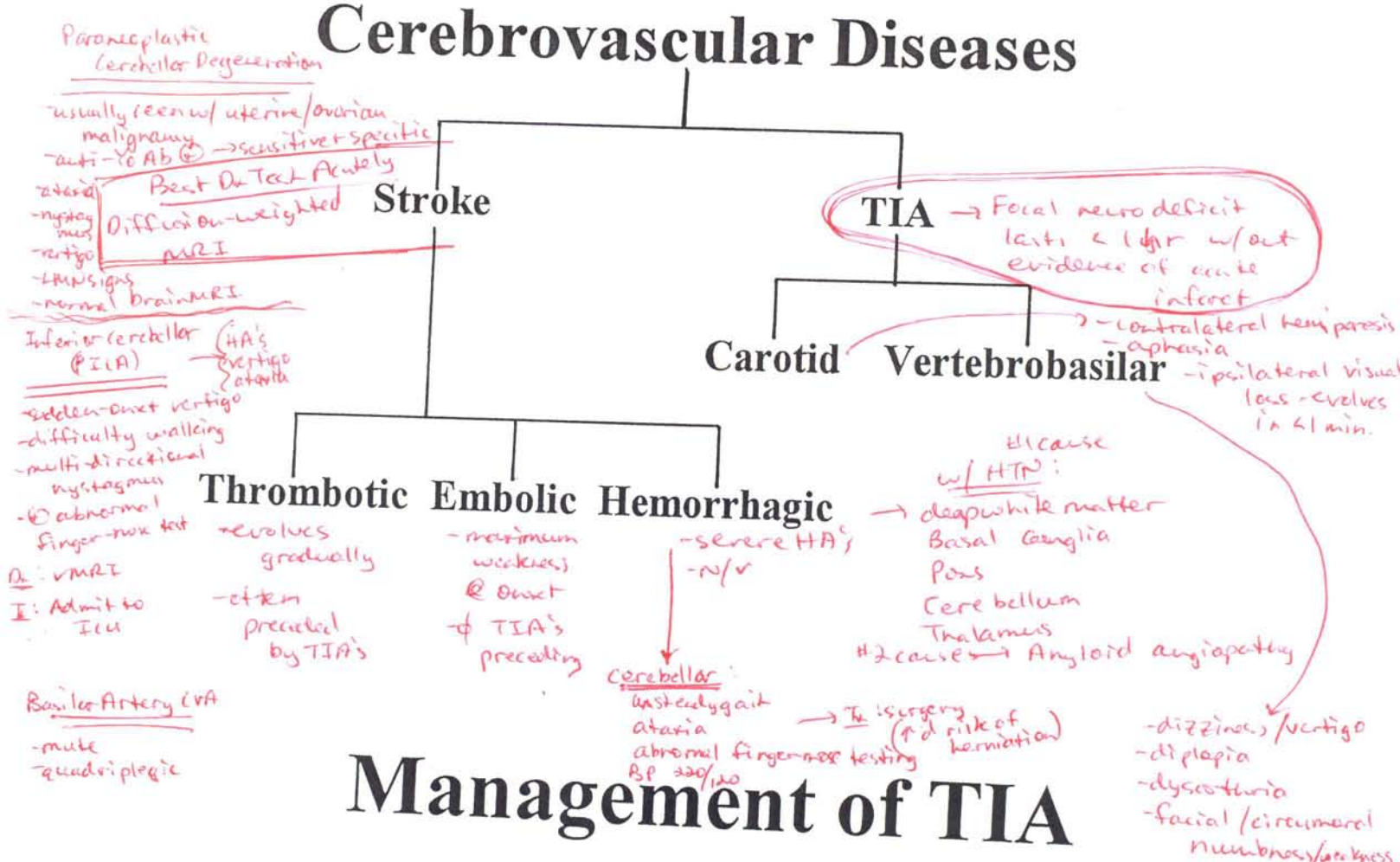
Dx → serum/vomitus toxin ⊕

Tx: Anti-toxin

muscle tension/stiffness

- can be used to tx dystonia
- inj. given @ 3-6 months
- corrects posture + ↓'s pain

Cerebrovascular Diseases



Management of TIA

Risk of Stroke: Highest in the first few days, evaluate patient immediately

Evaluation: Glucose, Na, platelet count, sed rate, EKG, MRI or CT scan
Imaging of carotid artery by Doppler ultrasonography or magnetic resonance angiography

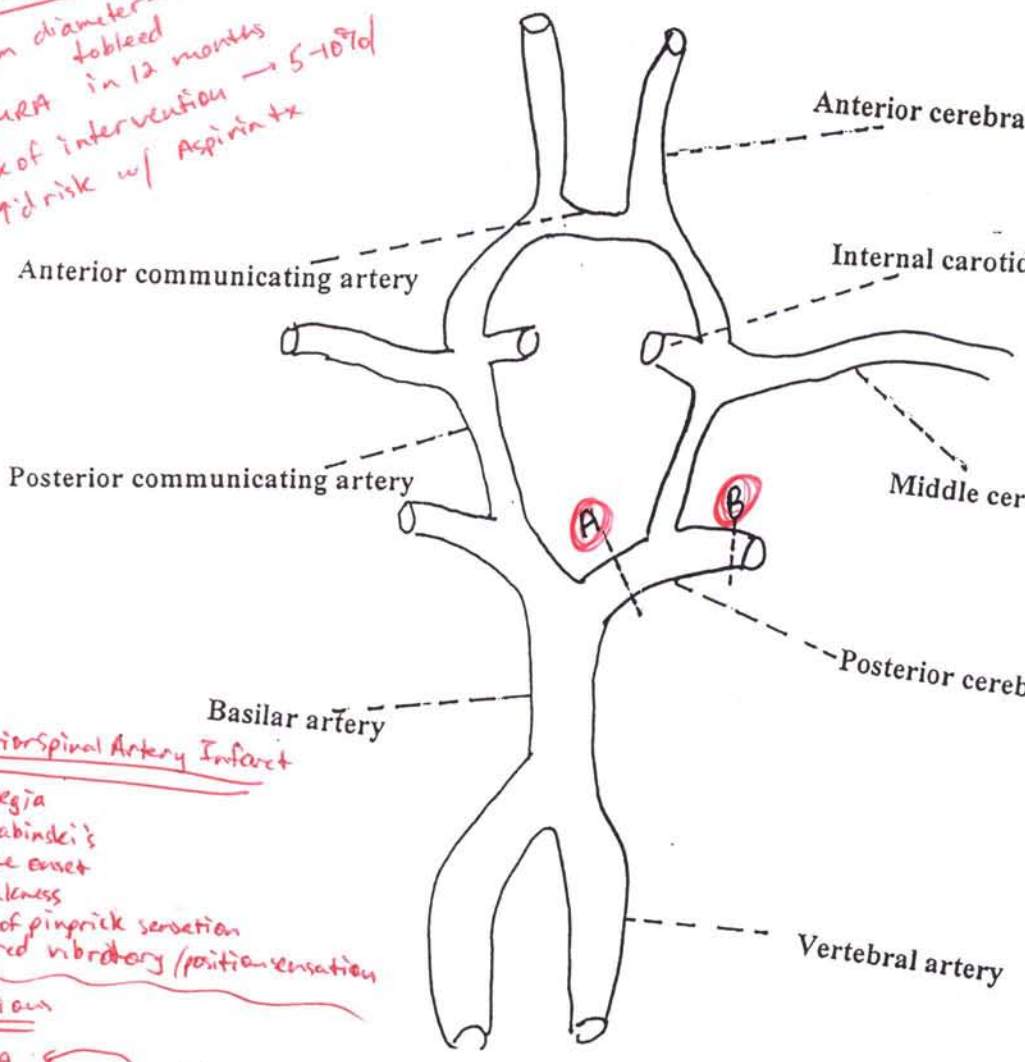
Treatment: Aspirin (80-325mg/day)
Clopidogrel, or asprin + dipridamole → preferred 25/200 BID
Warfarin if due to AF (add heparin until adequate INR reached)
Urgent endarterctomy for patients with ICA stenosis of 70-99%
Carotid artery stenting

Risk Factor Management:

BP < 140/90 (<130/80 with diabetes), glucose < 126 mg/dL, LDL < 100 mg
Smoking cessation, exercise 30-60 minutes 3-4 times /week
Avoid excessive use of alcohol (mild to moderate use beneficial)
Diet and exercise to reduce weight to less than 120% of ideal body weight

Blood Supply OF The Brain

Aneurysms
<7mm diameter → ↑risk → <10%/yr
↑bleed
✓MRA in 12 months
risk of intervention → 5-10%/yr
↓'d risk w/ Aspirin tx



supplies medial cerebral hemisphere
- contralateral leg weakness > arm

- ipsilateral visual loss

Anterior Spinal Artery Infarct
- paraplegia
- (B) Babinski's
- acute onset
- weakness
- loss of pinprick sensation
- spared vibratory/position sensation

Dissection
(C) ICA
- hemiplegia
- aphasia
- ptosis
- ipsilateral Horner's

(D) Vertebral Artery:
- severe occipital HA
- vertigo/ataxia
- N/V
- horizontal nystagmus
- dysmetria (contralateral)
- inability to control range of movement
- ✓MRA

Lacunar Infarct involving Internal capsule
↓
Branch of MCA

Localization of Cerebral Lesions in Stroke

Anterior Cerebral Artery (Medial cerebral hemisphere)
Contralateral weakness and sensory loss in leg > arm, behavioral changes

Internal Carotid Artery
Ipsilateral visual loss, contralateral hemiparesis and sensory loss, homonymous hemianopia, aphasia (if involvement of dominant hemisphere)

Middle Cerebral Artery (Lateral cerebral hemisphere)
Contralateral hemiplegia and sensory loss, homonymous hemianopia, aphasia (DH)

Branch of Middle Cerebral Artery ← dominant hemisphere
Contralateral hemiplegia without sensory loss or sensory loss without hemiplegia (pure motor)

Posterior Cerebral Artery (Inferior cerebral hemisphere)
(A) Ipsilateral 3rd nerve and contralateral hemiplegia (Weber's Syndrome)
Ipsilateral 3rd nerve and contralateral ataxia (Claude's syndrome) → visual cortex OK
(B) Homonymous hemianopia, cortical blindness if bilateral involvement → visual cortex affected

Basilar Artery (Brainstem, pontomedullary junction)
Bilateral sensory and motor signs, cranial nerves involvement (5, 6, 7), cerebellar signs
Diminished pain and thermal sense over half body, Horner's syndrome → "locked in" → basilar pontine infarct

Vertebral Artery (Brainstem, lateral medulla)
Vertigo, ataxia, nystagmus, Horner's syndrome
Diminished pain & thermal sense over half of body

Posterior Inferior Cerebellar Artery (Lateral medulla)
Ipsilateral facial sensory loss, contralateral body sensory loss, vertigo, ataxia, dysarthria

Prevention of Stroke

Each ↓'s stroke recurrence by 20-30%
Statins - even w/ ↓ LDL
ACEI's - even w/ ok BP's
Anti-Platelet Regimens

1. Treatment of hypertension
2. Smoking cessation
3. Regular Exercise
4. Diet high in fruits, vegetables, fiber and low in saturated fat
5. Anticoagulation for AF
6. Carotid endarterectomy for symptomatic ICA stenosis >70%
7. Statin therapy (LDL goal < 100 mg/dL)
8. Antiplatelet therapy for secondary prevention (aspirin or low-dose aspirin +dipyridamole, or clopidogrel)
9. Glucose control with diabetes

If aspirin allergy → go to Plavix (± dipyridamole alone)

→ Do not combine Aspirin/Plavix here

Treatment of Ischemic Stroke

Nonischemic CVA
If - ↓ coumadin
- Aspirin 325 QDay

Embolic CVA
- do not use early UFH tip ↑'d risk of hemorrhagic
- warfarin for small infarcts
- large infarcts → delay coumadin x 2 wks.
↓ Heparin

1. Treat high BP only if > 220/120
2. Aspirin (160-325mg)---→ after 2 weeks change to low-dose aspirin + dipyridamole) → common side effect → HA's - change to Plavix
3. S/C heparin 5000 units BID for DVT prophylaxis in immobile patients ↓ IV Heparin
4. Treatment of cerebral edema
5. IV thrombolytic therapy → if appropriate
6. Intraarterial thrombolytic therapy
7. Neurosurgical evaluation for cerebellar infarct or bleeding → ↑'d herniation risk

- Young pt (female)
- sudden-onset CVA
- ↓ on OCP's
↓
Think PFO w/ paradoxical emboli
↓
TEE

Thrombolytic Therapy in CVA

→ Doubles odds of independent function

Indicated for patients presenting within 3 hours of the onset of symptoms and a CT scan shows no evidence of intracranial hemorrhage

Dose: t-PA .9mg/kg (maximum 90 mg)

After 3 hrs. → intra-arterial tPA (w/in 3-6 hrs. of onset)

Contraindications

1. Previous stroke or serious head trauma in 3 months
2. BP> 185/110 (despite treatment)
3. Recent invasive surgical procedure in prior 2 weeks
4. PT > 15 or INR > 1.7
5. Increased PTT if heparin was given in the preceding 48 hr
6. GI or GU bleeding in prior 21 days
7. Platelet count < 10000, HCT < 25, glucose <50 or >400
8. Recent MI
9. Coma or stupor

Aphasia → disturbance of comprehension or production of spoken/written language

- difficulty w/ speech
- dominant lobe
- branch of MCA

1. Global (both sensory & motor) – large infarct of ICA or MCA dominant hemisphere
2. Broca's (motor or nonfluent)
3. Wernicke's (sensory or fluent) → does not understand but speech is fluent – branch of MCA
4. Conduction
5. Pure word deafness → ϕ auditory comprehension – branch of MCA
6. Pure word blindness → inability to read or name colors – branch of PCA
7. Isolation of speech area → due to hypotension/shock – extensive brain damage – parrot-like repetition

- repetition is affected
- branch of MCA

Asterixis → Toxic Encephalopathy as cause of

Dementia

- progressive impairment of cognitive functions
- Mild Cognitive Impairment → Neuropsych testing

1. Alzheimer's disease
2. Multi- infarct dementia → usually h/o HTN → multiple hypo-dense areas of white/gray matter
3. Deficiency of vitamin B1 or B12
4. Hypothyroidism
5. Normal- pressure hydrocephalus → Dementia, Gait Disturbance, Urinary Incont.
6. CNS infections : Cryptococcus, syphilis, HIV
7. Creutzfeld-Jakob disease – Rapidly-developing dementia – myoclonus – cause → prions – Normal MRI – ↑ 14-3-3 protein in CSF

CT → dilated ventricles, ϕ sulcal enlargement

Tx: VP shunt

Lewy Body Dementia
- fluctuating cognition
- Parkinsonism
- Hallucinations
- sometimes co-exists w/ Alzheimer's
- ↑'d β-amyloid peptide
- results in cell dysfx
- deficiency of Acetylcholine
- CT → atrophy / ↑'d ventricles / sulci
- PET → ↓ parietal glucose metabolism

Tx: Donepezil

Tx: centrally-acting cholinesterase inhibs. Donepezil (Aricept) → Bradycardia + HBlock
Vit. B + C
Ginkgo biloba
- familial form → ✓ presenilin-1 & presenile dementia
- normal neuroexam

Frontotemporal Dementia
- personality Δ's
- lost initiative
- slow thoughts
- <12 verbal fluency score
- MRI → disproportionate atrophy of frontal/temporal lobes
- memory ϕ affected

Neurological Complications of Diabetes

1. Polyneuropathy → Tx: Pregabalin / Gabapentin
2. Mononeuropathy
3. Radiculopathy
4. Autonomic neuropathy
5. Diabetic amyotrophy – proximal muscle weakness (Ex: thighs)

- wristdrop
- Footdrop
- VI Nerve
- IV Nerve
- III Nerve

Critical Illness Polyneuropathy
- generalized or distal flaccid paralysis
- ↓'d reflexes
- distal sensory loss
- spared CN functions
- normal CSF

Risk Factors:
1) Multi-organ Failure
2) Sepsis

→ if atrophy present + ↑CK → critical illness myopathy

Seizure Disorder

RR=Relative Risk

Risk Factors for
CNS Infex → ↑RR ×10
Head Trauma w/ LOC ×30 mins → ↑RR ×10
cerebral palsy
MRetardation
Alzheimer's
CVA (infarct)
↑RR ×10

ϕ↑RR w/ concussions

Incidence ~1%

Cocaine can induce

Primary vs. 2nd-ary
which happens 1st

Partial

Simple
-symptoms localized to 1 area or ipsilateral body
-consciousness ϕ impaired

Complex
-impaired consciousness
(nonconvulsive)

Generalized

Tonic-clonic

Absence
(petit mal)
(nonconvulsive)

Treatment of Seizures

Psychogenic → last longer → anxiety is prominent
maintain consciousness → normal EEG (Ex.) 10 mins.

-SSRI's OK in seizure D/O's
Tricyclics ↓seizure threshold

Frontal Lobe Seizure
-originate in Broca's area (if dominant)
-may spread to motor area
-usually brief
-ϕ postictal state

Pregnancy Regimen
carbamazepine
ϕ valproate
↓
bi-th defects

First-Line Drugs

Tonic-clonic seizures	Valproic acid, lamotrigine
Partial Seizures	Gabapentin (OOC) → better tolerated or Carbamazepine, phenytoin, lamotrigene, valproic acid
Absence seizures	Ethosuximide, valproic acid
Juvenile myoclonic	Valproic acid (lifelong)

-myoclonic jerks then generalized tonic/clonic seizure

set-off factors: ① Sleep deprivation
② Stress
③ EtOH

Treatment of Status Epilepticus

ABCs

① IV Lorazepam (.1 mg/kg IV at 2mg/min)

↓ → may give both initially but phenytoin has longer onset of action; benzo's are key initially

Phenytoin (20 mg/kg IV at 50mg/min) or Fosphenytoin (20mg/kg at 150 mg/min)

↓

Additional Phenytoin or Fosphenytoin (5-10 mg/kg IV)

↓

Phenobarbital (20 mg/kg IV at 50-75 mg/min)

↓

Additional Phenobarbital (5-10 mg/kg IV)

↓

Anesthesia with Midazolam or Propofol

Failure to respond to 3 med trials
↓
Refer for surgery

Paraneoplastic Limbic Encephalitis
-occasionally associated w/ small-cell lung
-rapidly progressive cognitive decline ea.
-seizures are common
anti-Hu Ab ⊕
-MRI: Hippocampal abnorms.

Vegetative states

① Locked-In : caused by lesions @ base of pons
- quadriplegia
- communicate via moving eyes / blinking
  ↳ can only move eyes vertically

② Minimally Conscious State:
- evidence of awareness (minimal) of self or environment

③ Persistent Vegetative State:
- present 1 month after nontraumatic or traumatic injury

④ Permanent Vegetative State:
- present 3 months after non traumatic injury & (2 months after traumatic injury

} complete unawareness of self or environment

CT Scan Findings in Various Diseases

Infarct	: Hypodense area, no enhancement
Multi-infarct dementia	: Multiple hypodense areas, no enhancement
Tumor or lymphoma	: Diffuse enhancing lesion or ring enhancement with central necrosis
Brain abscess	: Ring enhancing lesion
Toxoplasmosis	: Multiple ring enhancing lesions
Cerebral atrophy	: Dilated ventricles, dilated sulci
Alzheimer's disease	: Atrophy ± non-enhancing periventricular white matter lesions
AIDS dementia	: Atrophy ± non enhancing white matter lesions
Multiple sclerosis	: Periventricular & sub-cortical white matter lesions, lesions enhance with active disease
NPH	: Dilated ventricle, normal sulci
Multi-focal leucoencephalopathy	: Large non-enhancing white matter lesions

tough to distinguish

ALS
- progressive cerebral cortex / ant. horn s cord motor neuron loss
- begins ~50 yrs. of age
- asymmetric UMN & LMN weakness / signs → purely motor dis.
- bulbar signs as well → difficulty chewing / swallowing
- fasciculations / hyperreflexia / spasticity / ⊕ Babinski
- weakness / atrophic limbs

BOTH UMN + LMN signs

Tx: Riluzole (if indicated acutely esp. w/ respi insufficiency)
↓ benefit of IVIg

Indications For NIPPV → may prolong survival by ~20 months
- FVC <50% (predicted)
- symptoms of nocturnal hypoventilation

Dx: ✓ MRI C-spine
differentiating ALS vs. C-spine cord compression

ALS:
- ↓ pain
- ↓ sensory abnorms
- normal bowel / bladder functions
- normal spinal films / images

+ sensory symptoms
+ dersnot effect ocular muscles
+ fasciculations

Charcot Marie-Tooth Disease
- hereditary sensorimotor neuropathy
- distal muscle atrophy
- weakness
- sensory loss
- assoc. w/ : hammertoes / ↑ arches

Primary CNS Lymphoma (PCNSL)
- get an ophtho eval → may be able to dx w/out brain Bx
- avoid steroids → cytotoxic → may distort tissue dx
- Tx: Methotrexate then Rad Tx if fails