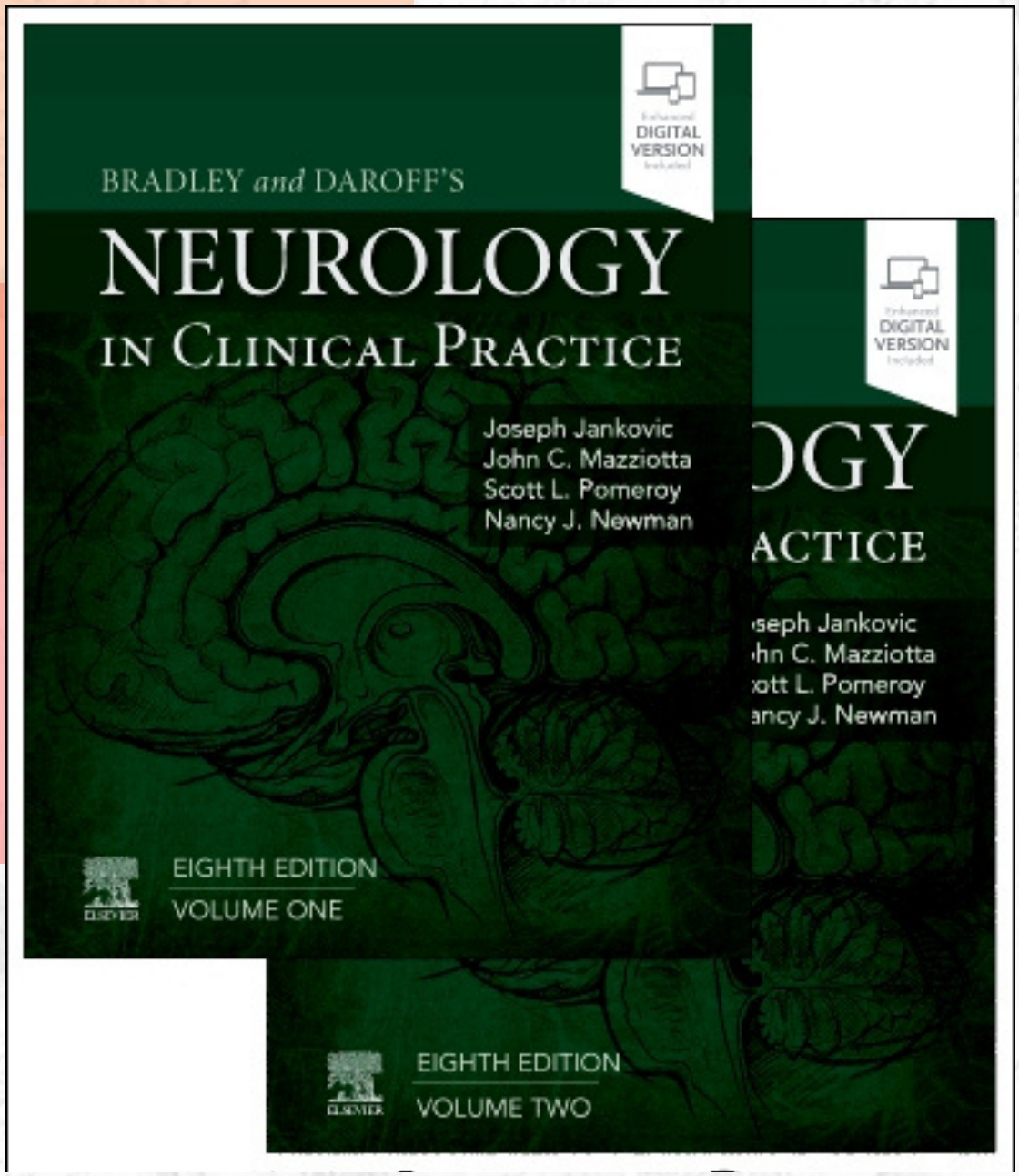
STEM-S

Last Minute Revision

LMR NOTES



INI-SS NEUROLOGY

PRESENTED BY
Stem-S

Asymmetric Pupils (Anisocoria)

1. The "Rule of Illumination" & Initial Localization
The critical first step in evaluating anisocoria is measuring pupil size in both bright
ambient light and darkness to determine which pupil is abnormal.

- Anisocoria Greater in the Light: Indicates that the larger pupil is abnormal because it fails to constrict appropriately. This localizes the defect to the parasympathetic efferent pathway, the iris sphincter muscle, or a pharmacological blockade.
- Anisocoria Greater in the Dark: Indicates that the smaller pupil is abnormal because it fails to dilate appropriately. This localizes the defect to the sympathetic efferent pathway (Horner syndrome), iris pathology, or pharmacological miosis.
- Physiological (Benign) Anisocoria: Affects up to 20% of the population, featuring a 0.4 to 1 mm difference that is usually the same in light and dark (though it can be slightly greater in darkness). Tip: It is never accompanied by ptosis, ophthalmoplegia, or abnormal light/near reflexes.

2. Anisocoria Greater in the Light: CN III & Life-Threatening Lesions
A large, poorly reactive pupil localizes to the parasympathetic fibers traveling with the
oculomotor nerve (CN III).

- Posterior Communicating (PCOM) Artery Aneurysm: Parasympathetic pupillary fibers are located superomedially on the surface of the third nerve, making them highly susceptible to external compression. A unilateral dilated pupil with an ipsilateral "down and out" eye and severe ptosis is a neurosurgical emergency.
- Uncal Herniation (Hutchinson Pupil): An expanding supratentorial mass pushes the temporal lobe uncus over the tentorial edge, compressing CN III. A sluggishly reactive pupil is an early warning sign, rapidly progressing to a completely dilated and fixed pupil.
- Pharmacological Testing (Distractor Check): To differentiate a CN III palsy from a pharmacologically dilated pupil (e.g., from atropine drops), instill 1% pilocarpine. A pupil with a CN III palsy will constrict fully, while a pharmacologically blocked pupil will not.

Carbamazepine and Oxcarbazepine

1. The Autoinduction & Metabolism

- Carbamazepine (CBZ) Autoinduction: Carbamazepine is a potent enzyme inducer that famously induces its own metabolism (autoinduction). This process increases its clearance over 2 to 4 weeks, resulting in a shortened half-life and lower serum concentration, meaning the dose must be titrated gradually to maintain therapeutic levels.
- The Oxcarbazepine (OXC): In contrast, oxcarbazepine does not induce its own metabolism.
- The CYP3A4 & Grapefruit Juice: Because CBZ is metabolized by CYP3A4, its levels will rapidly accumulate to toxic levels when coadministered with CYP3A4 inhibitors like erythromycin, fluoxetine, and grapefruit juice. Oxcarbazepine is not affectedby CYP3A4 inhibitors.
- The Epoxide: Carbamazepine is metabolized into an active metabolite, carbamazepine-10, 11-epoxide, which is responsible for many of its adverse effects. Coadministration with valproate, felbamate, or brivaracetam will specifically increase the levels of this epoxide metabolite.
- The Phenytoin Interaction: While OXC is a weak inducer of CYP3A4 (which can reduce oral contraceptive efficacy at doses >900 mg/d), it is uniquely a weak inhibitor of CYP2C19, which means high doses of OXC can paradoxically raise phenytoin levels.

2. The Seizure Exacerbation Rule (Avoid in IGE)

- Absolute Contraindication: While both drugs are excellent for focal-onset seizures, they can significantly exacerbate generalized absence and myoclonic seizures. Therefore, both CBZ and OXC must be strictly avoided in patients with idiopathic generalized epilepsy (IGE).

3. Hyponatremia

- Oxcarbazepine is more likely to cause symptomatic hyponatremia than carbamazepine.
- High-Risk Patients: You should be highly suspicious of this complication in older individuals and those who are concurrently taking a diuretic.

4. The Dermatologic & Genetic profile:

● The HLA-B*1502 Allele: Both drugs can cause rare but life-threatening idiosyncratic skin reactions, such as Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN). The risk is significantly higher in individuals carrying the HLA-B*1502 allele, predominantly those of Asian descent. Genetic testing

for this polymorphism is explicitly indicated before initiating carbamazepine in these populations.
● The Cross-Reactivity: If a patient develops a rash on carbamazepine, switching to oxcarbazepine is risky because there is a 25% cross-reactivity for rash between the two drugs.
5. Withdrawal & Missed Doses
● Severe Rebound: Unlike drugs with very long half-lives, abrupt withdrawal or even just missing doses of carbamazepine or oxcarbazepine can precipitate severe rebound seizures.

6. The Overnight Switch Ratio

● An overnight conversion can be made using a ratio of 300 mg of oxcarbazepine for every 200 mg of carbamazepine, provided the total CBZ dose was 800 mg or less.

7. The "Third Generation" Relative: Eslicarbazepine

● The Active Metabolite: Eslicarbazepine acetate is a prodrug that is rapidly converted to S-licarbazepine, which is the exact same active enantiomer of the monohydroxy derivative of oxcarbazepine.
● The Pharmacokinetic Advantage: Unlike OXC, eslicarbazepine has a long half-life in the CSF, justifying once-daily dosing, and it explicitly lacks the "CSF spike" that is suspected to be responsible for the acute adverse effects seen with oxcarbazepine.

3. Lyme Neuroborreliosis

Caused by the spirochete Borrelia burgdorferi, neurological involvement typically manifests during Stage 2 of the disease (disseminated stage), weeks after the classic erythema chronica migrans rash.

- **Classic Presentations:** The most highly tested presentation is a cranial neuropathy, heavily favoring bilateral facial nerve palsies (seen in 25% of facial palsy cases), often accompanied by a lymphocytic (aseptic) meningitis.
- **Radiculoneuropathy:** Patients can also develop a painful multifocal radiculoneuropathy or mononeuritis multiplex, featuring burning radicular pain, weakness, sensory loss, and hyporeflexia corresponding to the involved nerve roots. Electrodiagnostic testing typically demonstrates a primarily axon-loss polyneuropathy.

4. Syphilis (Tabes Dorsalis)

Treponema pallidum causes late neurosyphilis, resulting in Tabes Dorsalis, which is characterized by extensive damage to the dorsal roots occurring 10 to 20 years after the primary infection.

- **The Classic Triad/Symptoms:** Patients experience severe, brief, stabbing pains in the legs known as lightning pains, combined with sensory ataxia and bladder disturbances.
- **Physical Exam:** The board vignette will frequently describe Argyll Robertson pupils (pupils that accommodate to near vision but do not react to light), profound loss of proprioception, areflexia, Charcot (neuropathic) joints, and trophic foot ulcers.
- **Visceral Crises:** Occurring in about 20% of patients, these are sudden, unprovoked episodes of severe epigastric pain that spread around the body.

5. Campylobacter jejuni (The GBS Trigger)

While C. jejuni causes bacterial enteritis, it is the most highly tested antecedent infection triggering Guillain-Barré Syndrome, specifically the Acute Motor Axonal Neuropathy (AMAN) subtype.

- **Molecular Mimicry:** The bacteria's lipo-oligosaccharide coat shares structural epitopes with host gangliosides. This triggers autoantibodies (predominantly anti-GM1 and anti-GD1a) that attack the axolemma at the nodes of Ranvier on motor nerve fibers, leading to acute motor conduction failure.

Neurology

1. **HLA-B*1502**
   - **Skin reaction to phenytoin & CBZ (carbamazepine)**
2. **HLA-DQB1*0602**
   - **Present in 90% of Narcolepsy**

~25% of general population

3. **HLA-DRB1*1501**
   - **Class II**

~10% increased risk of MS

ERP:

- P300 / P3: Event-related or endogenous
- Related to attention & decision-making
- Reduced in depression
- SSEP (Somatosensory Evoked Potentials)
- N13 = Cervical spine (dorsal horn)
- N9 = Erb's point
- N9–N13 = Upper limb
- P14 = Brainstem (medial caudal medulla) / meniscus
- N20 = Cortical response
- N22–P34 = Inter-peak latency
- Useful in lumbar roots & brainstem

VEP

- N70
- P100

BAER (Brainstem Auditory Evoked Response)

Wave I – Distal auditory nerve
Wave II – Proximal auditory nerve
Wave III – Cochlear nucleus / trapezoid body
Wave IV – Superior olivary complex / lateral lemniscus (pons)
Wave V – Inferior colliculus (midbrain)
V–VII – Thalamus (MGB)

• Side Effects

Drug	Important Side Effects / Key Points
Ezogabine	QT prolongation
Lacosamide	PR prolongation; Linear pharmacokinetics; 100% bioavailability; Protein binding <10%
Rufinamide	QT shortening
Phenytoin	Hyperglycemia
Felbamate	Aplastic anemia
Topiramate	Hypohidrosis; Renal stones
Zonisamide	Hyperthermia
Valproate	Tremors; Pancreatitis; Hair loss; Weight gain
Gabapentin	Weight gain
Carbamazepine (CBZ)	Induces warfarin metabolism
Valproate + Topiramate	Teratogenicity; Metabolic acidosis
Lithium	AV block; Plaque psoriasis; Tremors; Diarrhea; Hypothyroidism; Nephrogenic diabetes insipidus; Lupus-like lesions; Onycholysis; Milia; Focal myoclonus; ↑ CK in SSS; Chorea; Weight gain; Acne
OKT3	Akinetic mutism
Methotrexate (MTX)	Leukoencephalopathy
Mitoxantrone	Irreversible cardiomyopathy
β-Interferon	Headache
DMF / Fingolimod / Natalizumab	Progressive multifocal leukoencephalopathy (PML)

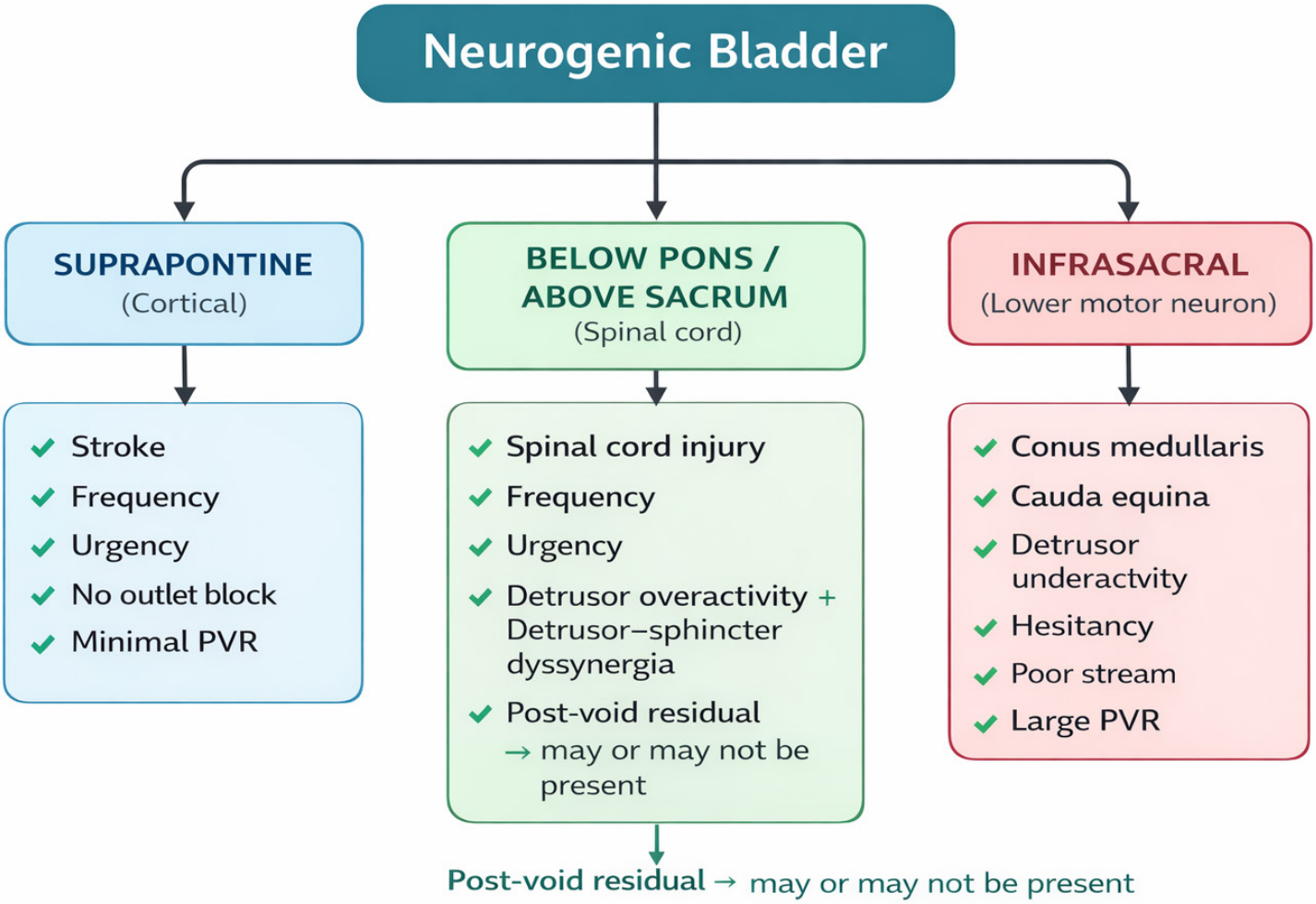
Drug	Key Effects / Important Points
Citalopram (SSRI)	Hyponatremia; Due to drug–drug interactions
Nortriptyline / Trazodone	Somnolence
TCA	**Fatal cardiac arrhythmia**
Aripiprazole	Partial dopamine (D2) agonist; ↓ EPS
Phenelzine	Non-selective MAO inhibitor; Anorgasmia
Olanzapine	Weight gain; Metabolic syndrome; Diabetes mellitus
Bupropion	↑ Seizure risk
Trazodone	α1-receptor blockade → Vasodilation; Priapism; Orthostatic hypotension; Sedation
Mirtazapine (Uses)	Depression; ↑ Appetite; Improves sleep
Loperamide	Safe in hepatic dysfunction
Risperidone	↑ Prolactin; Sexual dysfunction
Tacrolimus	TNF-α inhibition; Used in **CIDP**

- **Sensory Receptors**

Receptor	Encapsulation	Stimulus	Adaptation
Free nerve endings	Unencapsulated	Pain, temperature	—
Pacinian corpuscle	Encapsulated	Pressure, vibration	Rapid
Merkel discs	Unencapsulated	Light touch, pressure	Slowly adapting
Meissner corpuscle	Encapsulated	Vibration	Rapidly adapting
Ruffini endings	Encapsulated	Stretch	Slow

- **Hypothalamic Nuclei**

Nucleus	Primary Function / Hormone	Lesion Effect / Key Point
Lateral nucleus	Feeding center	Lesion → anorexia
Ventromedial nucleus	Satiety center	Lesion → hyperphagia
Arcuate nucleus	↑ GnRH, ↑ GHRH, ↑ Dopamine	Dopamine ↓ prolactin
Anterior nucleus	Heat dissipation	Lesion → **hyperthermia**
Posterior nucleus	Heat conservation	Lesion → **hypothermia**
Paraventricular nucleus	CRH, TRH → ↑ TSH, Somatostatin → ↓ GH	Endocrine regulation
Supraoptic nucleus	Vasopressin (ADH)	Water balance



- **Tumor Pathology**

Tumor	Key Histopathological Features
Meningioma	• Psammoma bodies • Meningothelial whorls
Glioblastoma	• Pseudopalisading pattern around necrosis
Pilocytic astrocytoma	• Glioma • Rosenthal fibres
Ependymoma	• Perivascular pseudorosettes
Medulloblastoma	• Homer–Wright rosettes
Oligodendroglioma	• Fried-egg appearance • Chicken-wire capillary pattern

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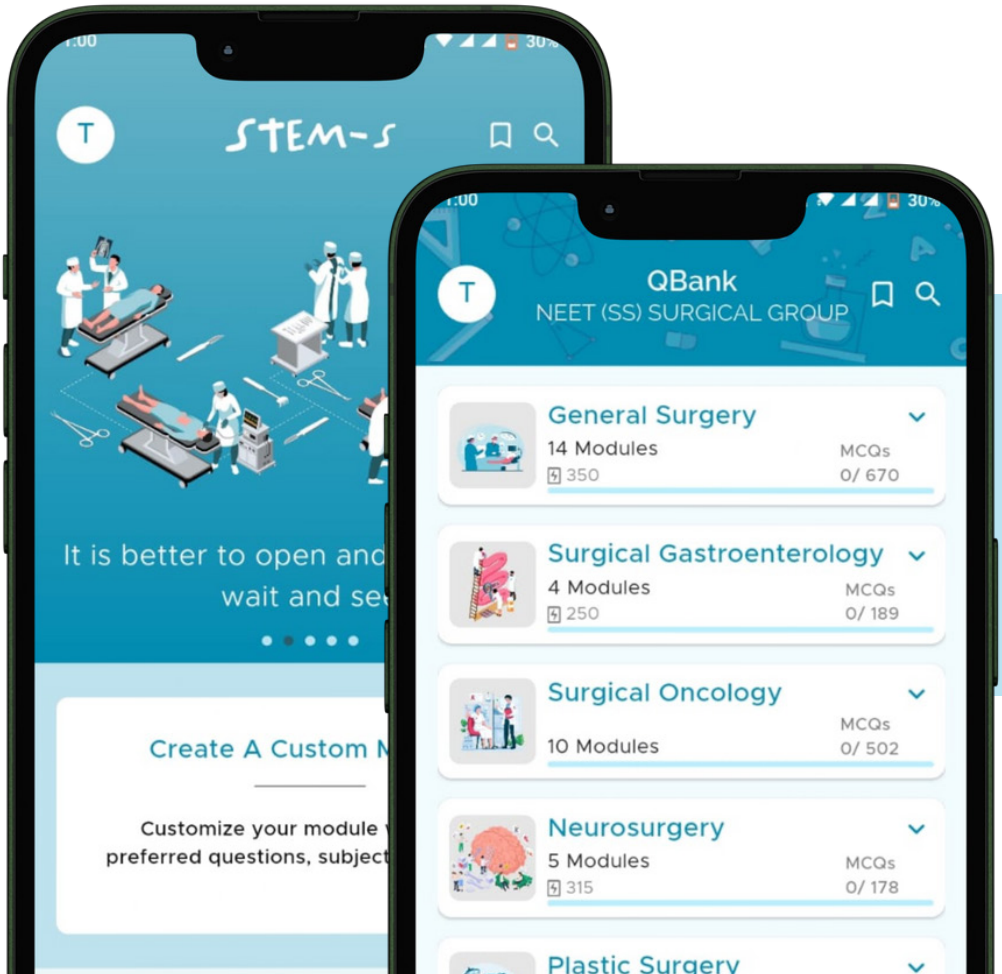

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