

QUESTION

A 28-year-old man reports arm and hand weakness and numbness after sleeping with his arm over a chair post heavy drinking. What is the correct order of susceptibility of different nerve Fibers to pressure?

A A Fiber is less susceptible than B Fibers

B B Fiber is more susceptible than A Fibers

C C Fiber is less susceptible than B Fibers

CORRECT

D C Fiber is more susceptible than A Fibers

RIGHT ANSWER

OK C. C Fiber is less susceptible than B Fibers

WHY THIS IS RIGHT

Pressure neuropathy preferentially affects larger, myelinated Fibers.

B Fibers (preganglionic autonomic, myelinated) are more susceptible to pressure than C Fibers (unmyelinated).

C Fibers are relatively resistant to compression and hypoxia.

| Fiber | Myelination | Size | Function |

| ---- | ----- | --- | ----- |

| A Fibers | Heavy | Large | Motor, proprioception |

| B Fibers | Light | Small | Autonomic |

| C Fibers | None | Very small | Pain, temperature |

QUESTION

Which autoimmune disorder involves antibodies against post-synaptic ACh receptors?

- A Multiple sclerosis
- B Myasthenia gravis
- C Lambert-Eaton syndrome
- D Guillain-Barré syndrome

CORRECT

RIGHT ANSWER

- OK
- B. Myasthenia gravis

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$19” here and does not include a written explanation for this item.

QUESTION

A person after sleeping overnight with the arm under his head now experiences paresis but no numbness in the morning. Which of the following is the best explanation for it?

- A** C Fibers are more sensitive to pressure than A Fibers
- B** A Fibers are more sensitive to hypoxia than B Fibers
- C** A Fibers are more susceptible to pressure changes than C Fibers CORRECT
- D** A Fibers are more susceptible to hypoxia than C Fibers

RIGHT ANSWER

- OK** C. A Fibers are more susceptible to pressure changes than C Fibers

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1a” here and does not include a written explanation for this item.

QUESTION

A woman suffered from sunburn after enjoying a vacation on a beach. Now while taking a shower lukewarm water (40-degree Celsius) touches her back causing her to feel pain. What type of receptors are activated by lukewarm water and what causes her to feel pain?

A Noxious thermal receptor - hyperalgesia

B Innocuous thermal receptor - allodynia

CORRECT

C Noxious thermal receptor - allodynia

D Innocuous thermal receptor - hyperalgesia

RIGHT ANSWER

OK

B. Innocuous thermal receptor - allodynia

WHY THIS IS RIGHT

Lukewarm water (40 °C) normally activates innocuous thermal receptors, not nociceptors. After sunburn, peripheral sensitization lowers the pain threshold of sensory pathways. As a result, a normally non-painful stimulus is perceived as pain, termed allodynia.

Sunburn -> inflammation

↓

Release of mediators (prostaglandins, bradykinin)

↓

↓ Threshold of nociceptors

↓

Non-painful stimuli now activate pain pathways

↓

Allodynia

| Feature | Allodynia | Hyperalgesia |

| ----- | ----- | ----- |

| Stimulus | Non-painful | Painful |

| Response | Pain | Exaggerated pain |

| Example | Sunburn + warm water | Pinprick -> severe pain |

Hyperalgesia refers to exaggerated pain to a painful stimulus, which is not the case here.

| Term | Meaning |

| --- | ----- |

| Allodynia | Pain from non-painful stimulus |

| Hyperalgesia | Increased pain from painful stimulus |

| Analgesia | No pain |

QUESTION

After a right limb amputation, the patient is experiencing severe pain (phantom limb). What is the mechanism behind this?

A Projection of adjacent fibers to overlap to right sensory cortex

B Projection of adjacent fibers to overlap to left sensory cortex

CORRECT

C Expansion of right sensory cortex

D Expansion of left sensory cortex

RIGHT ANSWER

OK

B. Projection of adjacent fibers to overlap to left sensory cortex

WHY THIS IS RIGHT

Phantom limb pain occurs due to cortical reorganization after amputation.

Adjacent sensory areas invade the contralateral (left) primary somatosensory cortex representing the amputated right limb.

Stimulation of these neighboring regions is misinterpreted as pain from the missing limb.

- Pain perceived in an amputated limb
- Due to central (cortical) mechanisms, not peripheral input

Thus, projection of adjacent fibers overlapping the left sensory cortex explains the phenomenon.

Limb amputated

↓

No sensory input from that region

↓

Adjacent cortical areas expand

↓

They “invade” the vacant area

↓

Misinterpretation of signals

↓

Phantom sensation / pain

| Concept | Phantom Limb |

| ----- | ----- |

| Side affected | Contralateral cortex |

| Mechanism | Cortical reorganization |

| Key event | Adjacent area overlap |

| Type | Central phenomenon |

QUESTION

In the below question mechanical receptors are given with their functions. Choose the correctly matched pair.

A Pacinian Corpuscle - Fast Vibration

CORRECT

B Ruffini - Fine touch

C Meissner - Stretch

D Merkel - Slow Vibration

RIGHT ANSWER

OK A. Pacinian Corpuscle - Fast Vibration

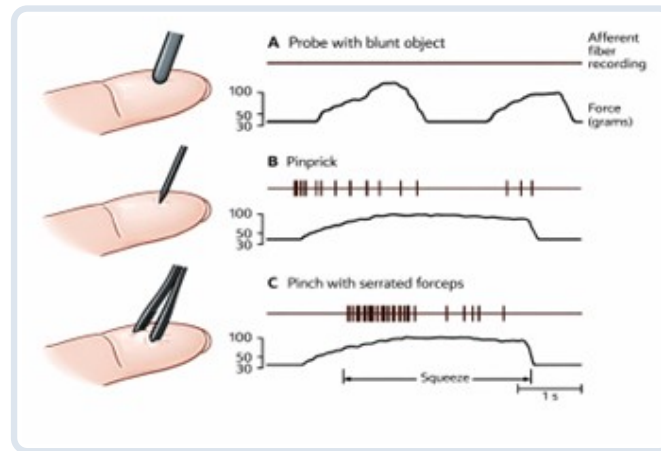
WHY THIS IS RIGHT

Pacinian corpuscles are rapidly adapting mechanoreceptors that detect high-frequency (fast) vibration. They have large receptive fields and respond best to sudden pressure changes. Ruffini endings sense stretch, Meissner corpuscles mediate light touch, and Merkel discs detect form and texture.

Receptor	Adaptation	Function	Receptive Field
-----	-----	-----	-----
Pacinian	Rapid	□ Vibration	Large
Meissner	Rapid	Light touch	Small
Merkel	Slow	Fine touch	Small
Ruffini	Slow	Stretch	Large

QUESTION

Mechanical nociceptors and its response to a probe with blunt object, pin prick and tooth forceps with serration is recorded as a output of a force transducer coupled to the stimulator. What will you infer from a given diagram?



A Blunt object with high force doesn't stimulate nociceptors

CORRECT

B Sharp object doesn't stimulate nociceptors

C Pinching with serrated object stimulate mechanoreceptors but doesn't cause pain

D Sharp pain sensation is carried by type C nerve fiber

RIGHT ANSWER

OK

A. Blunt object with high force doesn't stimulate nociceptors

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1b" here and does not include a written explanation for this item.

QUESTION

| Which of the following is involved in Static Two-point discrimination?

A Meissner's corpuscles

B **Merkel disk**

CORRECT

C Pacinian corpuscles

D Ruffini ending

RIGHT ANSWER

OK **B. Merkel disk**

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1c" here and does not include a written explanation for this item.

QUESTION

Which of the following features are typically associated with delirium?

1. Impaired consciousness
2. Altered sleep-wake cycle
3. Normal EEG
4. Carphologia
5. Autonomic Dysfunction

A 1, 2, 3, 4, 5

B 2, 3, 5

C 1, 3, 5

D 1, 2, 4, 5

CORRECT

RIGHT ANSWER

OK D. 1, 2, 4, 5

WHY THIS IS RIGHT

Delirium is an acute confusional state characterized by fluctuating disturbance in attention, awareness, and cognition due to an underlying medical condition.

Typical features:

- Impaired consciousness/attention
- Altered sleep-wake cycle (day-night reversal)
- Carphologia (picking at bedclothes; floccillation)
- Autonomic dysfunction (tachycardia, sweating, hypertension)
- Fluctuating course and disorientation

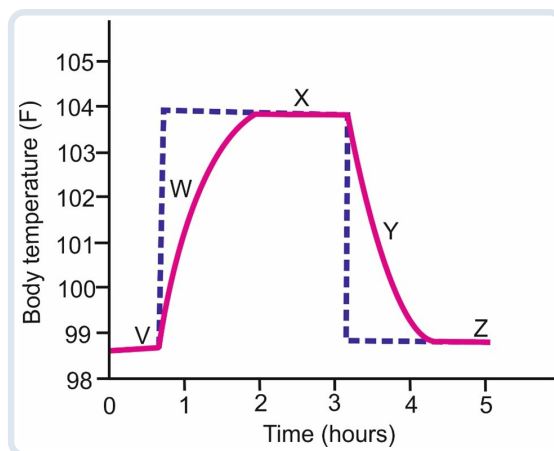
EEG finding:

- Diffuse slowing on EEG (not normal)

*

QUESTION

In a patient, the hypothalamic thermostat was reset from point V to point X as shown below. Which of the following happens in stage W compared to stage Y?



CORRECT

- A** Shivering
- B** Sweating
- C** Increased blood flow to skin
- D** Inhibition of chemical thermogenesis

RIGHT ANSWER

A. Shivering

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$19" here and does not include a written explanation for this item.

QUESTION

| Which centre in the brain is responsible for producing fever during infection?

A Paraventricular nucleus of hypothalamus

B Dorsomedial nucleus

C **Preoptic Nucleus**

CORRECT

D Cerebellum

RIGHT ANSWER

OK **C. Preoptic Nucleus**

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1a" here and does not include a written explanation for this item.

QUESTION

Which of the following brain regions is primarily associated with the processing of explicit memory?

A Hippocampus

CORRECT

B Amygdala

C Insular cortex

D Cerebellum

RIGHT ANSWER

OK A. Hippocampus

WHY THIS IS RIGHT

Type	Example	Brain Region
Explicit	Facts, events	Hippocampus
Implicit (procedural)	Skills	Cerebellum, basal ganglia

Declarative / Explicit Memory:

- Semantic (factual):
 - Prefrontal cortex
 - Temporal cortex (lateral and anterior)
- Episodic (events):
 - Hippocampus
 - Medial temporal lobe
 - Neocortex

Nondeclarative / Implicit Memory:

- Procedural (skills, habits):
 - Striatum
 - Cerebellum
 - Motor cortex
- Priming and perceptual:
 - Neocortex
- Associative learning (classical conditioning):
 - Amygdala
 - Cerebellum

QUESTION

Which of the following areas of the brain are correctly matched with the neurotransmitters they release?

1. Substantia nigra - Dopamine
2. Locus ceruleus - Acetylcholine
3. Raphe nucleus - Serotonin

A 1

B 1, 2 and 3

C 1 and 3

CORRECT

D 2 and 3

RIGHT ANSWER

OK C. 1 and 3

WHY THIS IS RIGHT

Nucleus	Neurotransmitter	Function	Clinical Correlation
Substantia nigra	Dopamine	Movement	Parkinson disease
Locus ceruleus	Norepinephrine	Arousal, attention	Anxiety, stress
Raphe nuclei	Serotonin	Mood, sleep	Depression
Basal nucleus of Meynert	Acetylcholine	Memory	Alzheimer disease

QUESTION

| What is the role of caffeine in wakefulness?

CORRECT

- A** Blocks adenosine action and causes wakefulness
- B** Activates locus coeruleus and cause wakefulness
- C** No role in maintaining wakefulness if taken 1hr before sleep
- D** Activates histamine release and prevents sleep

RIGHT ANSWER

- OK** **A. Blocks adenosine action and causes wakefulness**

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1b” here and does not include a written explanation for this item.

QUESTION

| Which of the following hypothalamic nuclei are responsible for leptin resistance?

A Paraventricular nucleus

B Lateral nucleus

C Arcuate nucleus

CORRECT

D Ventromedial nucleus

RIGHT ANSWER

OK C. Arcuate nucleus

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1c" here and does not include a written explanation for this item.

QUESTION

A motorcyclist suffered a head injury 6 months back and presents with persistent memory deficits following injury. Choose the most appropriately matched option related to memory?

A Hippocampus and implicit memory

B Neocortex and associative learning

C Medial temporal lobe- Declarative memory

CORRECT

D Angular gyrus- procedural memory

RIGHT ANSWER

OK

C. Medial temporal lobe- Declarative memory

WHY THIS IS RIGHT

The medial temporal lobe, especially the hippocampus, is essential for formation of declarative (explicit) memory - facts and events. Types of memory & localization:

- Declarative (explicit) memory: facts, events -> hippocampus & medial temporal lobe
- Procedural (implicit) memory: skills, habits -> basal ganglia & cerebellum
- Associative/semantic memory storage: neocortex
- Emotional memory: amygdala

> Clinical correlation:

> Damage to medial temporal lobe (e.g., head injury) -> anterograde amnesia with impaired formation of new declarative memories.

QUESTION

If you see the word "yellow," you'll likely recognize "banana" faster than "television" because yellow and banana are more closely linked in memory. Which of the following structures is this concept dependent on?

- A Amygdala
- B Hippocampus
- C Neocortex**
- D Striatum

CORRECT

RIGHT ANSWER

OK **C. Neocortex**

WHY THIS IS RIGHT

Faster recognition of related concepts (e.g., "yellow" -> "banana") demonstrates semantic memory and associative priming, which depend primarily on the neocortex which stores long-term semantic knowledge, language, and conceptual associations.

Responsible for linking related concepts and meaning-based memory retrieval

Differentiation:

- Hippocampus: Formation of new episodic memories
- Amygdala: Emotional memory/fear processing
- Striatum: Procedural memory and habit learning

QUESTION

A 67-year-old woman develops a stroke involving the right side of the brain. Weeks later, she presents with severe, persistent burning and aching pain involving the left side of her body, accompanied by hyperalgesia and allodynia. Sensory examination reveals impaired pain and temperature sensation initially, followed by distressing spontaneous pain. Motor power is relatively preserved. Which of the following is the most likely location of her initial infarct?

A Right ventral posterolateral (VPL) nucleus

CORRECT

B Right pulvinar

C Right lateral geniculate nucleus

D Right ventral posteromedial (VPM) nucleus

RIGHT ANSWER

OK A. Right ventral posterolateral (VPL) nucleus

WHY THIS IS RIGHT

Central post-stroke pain (Dejerine-Roussy syndrome) classically occurs after infarction of the ventral posterolateral (VPL) nucleus of the thalamus, which relays pain and temperature from the contralateral body.

Anatomy correlation:

- VPL nucleus: Sensory input from body (spinothalamic + medial lemniscus)
- VPM nucleus: Sensory input from face
- Lateral geniculate body: Vision
- Pulvinar: Visual attention/integration

QUESTION

A patient with hypothalamic damage develops loss of pulsatile secretion of a hormone, leading to hypogonadotropic hypogonadism, while other pituitary axes are relatively preserved. Which neuronal population is most likely affected?

- A** Orexin-secreting neurons
- B** Kisspeptin-neurokinin B-dynorphin neurons CORRECT
- C** Vasopressin-secreting magnocellular neurons
- D** CRH-secreting parvocellular neurons

RIGHT ANSWER

- B. Kisspeptin-neurokinin B-dynorphin neurons**

WHY THIS IS RIGHT

Pulsatile secretion of GnRH from the hypothalamus is essential for normal LH and FSH release and reproductive function. This pulsatility is generated by Kisspeptin-Neurokinin B-Dynorphin (KNDy) neurons located in the arcuate nucleus.

- Kisspeptin: Stimulates GnRH neuron activity
- Neurokinin B: Promotes synchronized pulsatile firing
- Dynorphin: Provides inhibitory feedback to regulate pulse frequency

Damage to KNDy neurons -> loss of GnRH pulsatility -> hypogonadotropic hypogonadism with relatively preserved other pituitary axes.

QUESTION

Which of the following neurotransmitter-peptide combinations originates predominantly from neurons of the arcuate nucleus?

A Orexin - glutamate

B POMC - α -MSH

CORRECT

C Vasopressin - neurophysin II

D TRH - glutamate

RIGHT ANSWER

OK B. POMC - α -MSH

WHY THIS IS RIGHT

The arcuate nucleus of the hypothalamus plays a central role in appetite and energy homeostasis and contains POMC (pro-opiomelanocortin) neurons.

- POMC neurons produce α -MSH (alpha-melanocyte-stimulating hormone)
- α -MSH acts on melanocortin receptors (MC4) in the hypothalamus to suppress appetite and increase satiety

QUESTION

A patient with chronic alcoholism develops memory impairment related to hypothalamic damage. Which structure is classically involved?

A Mammillary bodies

CORRECT

B Suprachiasmatic nucleus

C Ventromedial nucleus

D Paraventricular nucleus

RIGHT ANSWER

OK A. Mammillary bodies

WHY THIS IS RIGHT

Chronic alcoholism can cause thiamine (vitamin B1) deficiency, leading to Wernicke-Korsakoff syndrome, which primarily damages the mammillary bodies and medial thalamus.

Damage results in Korsakoff psychosis characterized by:

- Severe anterograde amnesia
- Confabulation
- Memory impairment

QUESTION

A 35-year-old female patient presents with progressive limb weakness for the past 1 year. On examination, muscle fasciculations and hypertonia are noted, and the sensory system is intact. What is the diagnosis?

A Becker muscular dystrophy

B Multifocal motor neuropathy

C Spinal muscular atrophy

D Amyotrophic Lateral Sclerosis

CORRECT

RIGHT ANSWER

OK

D. Amyotrophic Lateral Sclerosis

WHY THIS IS RIGHT

Feature	Interpretation
-----	-----
Progressive weakness	Neurodegenerative
Fasciculations	LMN involvement
Hypertonia	UMN involvement
Intact sensation	Motor-only disease

Feature	ALS	SMA	MMN	Becker MD
-----	---	---	---	-----
UMN signs	☒	☐	☐	☐
LMN signs	☒	☒	☐	☐
Fasciculations	☒	☒	☐	☐
Sensory loss	☐	☐	☐	☐
Nature	Neurodegenerative	Genetic	Autoimmune	Myopathy

ALS Hallmark Triad

- Mixed UMN + LMN signs
- No sensory loss
- Progressive course

QUESTION

An elderly man presents with rigidity and tremors. On examination, he has blank facial expressions. Which of the following drugs can be used to manage this condition?

A Clozapine

B Donepezil

C Selegiline

CORRECT

D Haloperidol

RIGHT ANSWER

OK C. Selegiline

WHY THIS IS RIGHT

↓ Dopamine
↓
Indirect pathway becomes overactive
↓
↑ Inhibition of thalamus
↓
↓ Motor cortex stimulation
↓
Bradykinesia + rigidity

Parkinson = dopamine deficiency -> treat by increasing dopamine

Selegiline

- Selectively inhibits MAO-B
- Prevents dopamine breakdown
- -> ↑ Dopamine availability in brain

Drug	Mechanism	Use	Effect in Parkinson
Selegiline	MAO-B inhibitor	Parkinson	Improves symptoms
Clozapine	D2 blockade (weak)	Schizophrenia	Limited role
Donepezil	AChE inhibitor	Alzheimer	No benefit
Haloperidol	Strong D2 blocker	Psychosis	Worsens symptoms

QUESTION

A 65-year-old man suffered from a stroke 2 days ago. He now presents with involuntary, violent, and flinging movements of the limbs on one side. What is the likely site of lesion in this patient?

A Subthalamic nuclei

CORRECT

B Globus pallidus

C Putamen

D Caudate nucleus

RIGHT ANSWER

OK **A. Subthalamic nuclei**

WHY THIS IS RIGHT

Subthalamic nucleus lesion

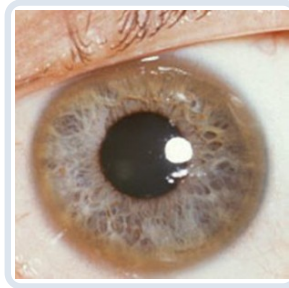
↓
 ↓ Excitation of Globus Pallidus Interna (GPi)
 ↓
 ↓ Inhibition of Thalamus
 ↓
 ↑ Thalamic activity
 ↓
 ↑ Cortical motor output
 ↓
 Violent, excessive movements (Hemiballismus)

Structure	Lesion Effect	Movement Type	Classic Disease
Subthalamic nucleus	↓ Inhibition	Hemiballismus (flinging)	Stroke
Globus pallidus	Motor modulation	Rigidity / dystonia	Parkinsonism variants
Putamen	Motor planning	Chorea / athetosis	Huntington
Caudate nucleus	Cognitive + motor	Chorea + psychiatric	Huntington

Type	Example	Pathology
Hyperkinetic	Hemiballismus, chorea	↓ Inhibition
Hypokinetic	Parkinson disease	↑ Inhibition

QUESTION

A patient had a history of hepatic dysfunction, and the physical examination finding of the eye is shown below. Which of the following is the best way for confirmation of the diagnosis in this patient?



- A** Serum copper
- B** Urinary copper
- C** Liver copper levels
- D** Serum ceruloplasmin

CORRECT

RIGHT ANSWER

- OK** C. Liver copper levels

WHY THIS IS RIGHT

ATP7B mutation
↓
↓ Copper excretion in bile
↓
Copper accumulates in liver
↓
Spills into blood
↓
Deposits in brain + cornea
↓
KF ring + neurological signs

The eye finding is Kayser-Fleischer ring, suggesting Wilson disease with hepatic involvement. The gold standard for confirmation is quantitative liver copper estimation on biopsy. Serum ceruloplasmin may be low but is not definitive, and serum copper can be misleading.

Feature	Interpretation
-----	-----
Hepatic dysfunction	Liver involvement
Eye finding	Kayser-Fleischer (KF) ring
Young patient	Typical age group

Urinary copper is supportive, but liver copper levels provide definitive diagnosis.

Drug	Action
----	-----
Penicillamine	Copper chelation
Zinc	↓ absorption

QUESTION

A patient is presented with a staggering gait and nystagmus after a road traffic accident. Which lobe of cerebellum is affected?

A Flocculonodular

CORRECT

B Dentate

C Anterior lobe

D Vermis

RIGHT ANSWER

OK A. Flocculonodular

WHY THIS IS RIGHT

Staggering gait with nystagmus indicates involvement of the vestibulocerebellum.
The flocculonodular lobe controls balance, posture, and eye movements via vestibular connections.

Lesions cause truncal ataxia, vertigo, and nystagmus with a positive Romberg sign.
Anterior lobe causes limb ataxia, and vermis affects axial posture without prominent eye signs.

Structure	Function	Lesion Feature
-----	-----	-----
Flocculonodular	Balance + eye movement	☒ Ataxia + nystagmus
Anterior lobe	Posture	Gait disturbance
Posterior lobe	Fine movement	Limb ataxia
Vermis	Trunk control	Truncal instability

QUESTION

Which of the following drugs are used in treatment of acute mania?

1. Haloperidol
2. Valproate
3. Lithium
4. Amitriptyline

A Only 3

B 1, 2 & 3

CORRECT

C 1, 2, 3 & 4

D 2, 3 & 4

RIGHT ANSWER

OK

B. 1, 2 & 3

WHY THIS IS RIGHT

Mania is a state of:

- ↑ Dopamine
- ↑ Norepinephrine
- ↓ inhibitory control

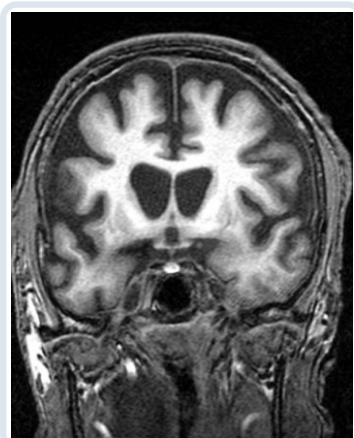
Treatment Strategy

1. Immediate control (hours-days)
-> Antipsychotics (block dopamine)
2. Stabilization (days-weeks)
-> Mood stabilizers (lithium, valproate)

Drug Class	Examples	Role in Mania	Key Feature
Antipsychotics	Haloperidol, Olanzapine	Acute control	Fast acting
Mood stabilizers	Lithium, Valproate	Acute + maintenance	Prevent relapse
Benzodiazepines	Lorazepam	Adjunct	Sedation
Antidepressants	Amitriptyline	□ Avoid	Trigger mania

QUESTION

A 28-year-old man presents with involuntary, purposeless, dance-like movements involving both upper and lower limbs. He has also become aggressive and irritable over the past few months. MRI brain is shown. What is the most likely diagnosis?



A Huntington chorea

CORRECT

B Sydenham chorea

C Tourette syndrome

D Tardive dyskinesia

RIGHT ANSWER

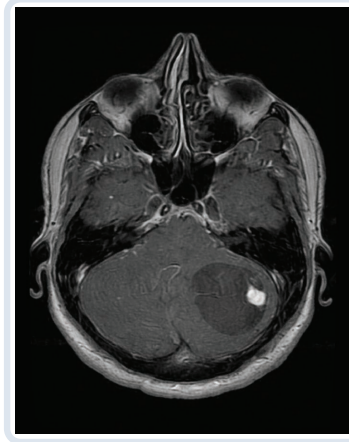
OK A. Huntington chorea

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$19" here and does not include a written explanation for this item.

QUESTION

Which of the following findings is most likely to be seen on physical examination of this patient?



A Left dysdiadochokinesia

CORRECT

B Left Horner syndrome

C Right hemiparesis

D Right hemisensory loss

RIGHT ANSWER

OK A. Left dysdiadochokinesia

WHY THIS IS RIGHT

A lesion in the cerebellar hemisphere produces ipsilateral cerebellar signs due to double crossing of cerebellar pathways.

Key cerebellar findings:

- Dysdiadochokinesia (impaired rapid alternating movements)
- Intention tremor
- Ataxia
- Dysmetria
- Hypotonia

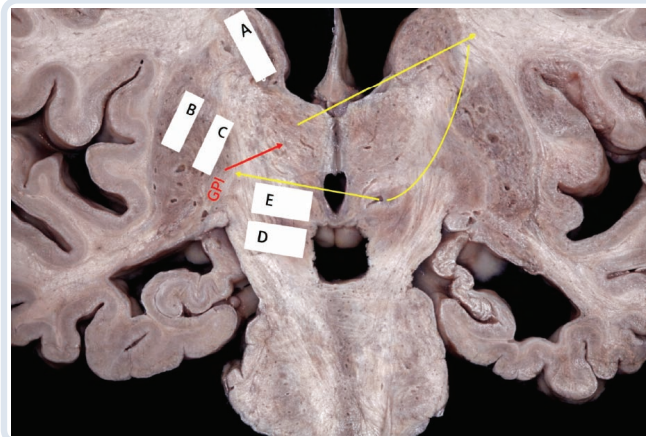
Localization rule:

- Cerebellar hemisphere lesion -> ipsilateral limb incoordination
- Vermis lesion -> truncal ataxia

QUESTION

Match the following:

1. Preferred site for DBS for tremor reduction
2. Hemiballismus results from a lesion at this site
3. Chorea results from a lesion at this site
4. Athetosis is a result of a lesion at this site
5. Parkinsonism occurs due to defect here



A 1-A, 2-D, 3-A, 4-B, 5-E

B 1-E, 2-E, 3-A, 4-C, 5-D

CORRECT

C 1-D, 2-E, 3-A, 4-C, 5-D

D 1-D 2-A, 3-C, 4-B, 5-E

RIGHT ANSWER

OK B. 1-E, 2-E, 3-A, 4-C, 5-D

WHY THIS IS RIGHT

Different movement disorders localize to specific basal ganglia structures, which are frequently tested.

Key correlations:

- Sub-thalamic nucleus lesion -> Hemiballismus
- Caudate nucleus lesion -> Chorea (e.g., Huntington disease)
- Putamen lesion -> Athetosis/dystonia
- Substantia nigra pars compacta degeneration -> Parkinsonism
- Globus pallidus internus/VIM thalamus -> target for deep brain stimulation (DBS) to reduce tremor

QUESTION

A 38-year-old hospitalized woman is evaluated for new-onset confusion. The patient has a prolonged history of Crohn disease. Pupils are equal and reactive to light. Abduction of the right eye is limited and elicits bilateral horizontal nystagmus. Motor strength and deep tendon reflexes are normal throughout. Finger-to-nose and heel-to-shin testing are normal, but the gait is wide based. Which of the following is the most likely cause of this patient's neurologic symptoms?

A Vitamin B12 deficiency

B Thiamine deficiency

CORRECT

C Folate deficiency

D Cerebellar dysfunction

RIGHT ANSWER

OK B. Thiamine deficiency

WHY THIS IS RIGHT

This patient has the classic triad of Wernicke encephalopathy due to thiamine (vitamin B1) deficiency. Triad:

1. Confusion
2. Ophthalmoplegia/nystagmus (e.g., lateral rectus palsy, horizontal nystagmus)
3. Ataxia (wide-based gait)

Risk factors:

- Malnutrition
- Chronic alcoholism
- Malabsorption (e.g., Crohn disease)
- Prolonged hospitalization/TPN without supplementation

Give IV thiamine before glucose to prevent worsening neurologic injury.

QUESTION

The Westphal variant of Huntington's disease is most closely related to which of the following?

A Alzheimer's disease

B Parkinson's disease

CORRECT

C Orthostatic hypotension

D Dementia

RIGHT ANSWER

OK B. Parkinson's disease

WHY THIS IS RIGHT

The Westphal variant is a juvenile form of Huntington's disease characterized predominantly by parkinsonian features rather than chorea. Key features of Westphal variant:

- Early onset (juvenile HD)
- Rigidity and bradykinesia (parkinsonism)
- Dystonia
- Seizures common
- Rapid progression
- Often associated with paternal transmission and larger CAG repeat expansion

Comparison:

- Adult Huntington's: Chorea, behavioral changes, dementia
- Westphal variant: Parkinsonism-dominant -> resembles Parkinson's disease

QUESTION

A 60-year-old woman with metastatic cancer reports moderate bone pain despite taking paracetamol and ibuprofen regularly. What is the next appropriate step according to the WHO analgesic ladder for chronic pain in adults?

CORRECT

A Start tramadol and continue NSAIDs

B Increase NSAID dose

C Start morphine and continue NSAIDs

D Consider non-pharmacological options

RIGHT ANSWER

OK

A. Start tramadol and continue NSAIDs

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$19” here and does not include a written explanation for this item.

QUESTION

| What is the first line therapy for aborting the acute attack of cluster headache?

A 100% oxygen

CORRECT

B Sumatriptan

C Lithium

D Topiramate

RIGHT ANSWER

OK A. 100% oxygen

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1a" here and does not include a written explanation for this item.

QUESTION

A female patient presents to you with a unilateral headache. It is associated with nausea, photophobia, and phonophobia. What is the best drug for acute management?

A Flunarizine

B Sumatriptan

CORRECT

C Propranolol

D Topiramate

RIGHT ANSWER

OK B. Sumatriptan

WHY THIS IS RIGHT

This presentation is classic migraine with unilateral headache, nausea, photophobia, and phonophobia.

Trigger

↓

Trigeminal nerve activation

↓

CGRP release

↓

Vasodilation + inflammation

↓

Migraine pain

Sumatriptan is the drug of choice for acute abortive treatment, acting as a 5-HT_{1B/1D} agonist. It causes cranial vasoconstriction and inhibits trigeminal neuropeptide release.

| Drug | Role | Use |

| --- | --- | --- |

| Sumatriptan | Abortive | Acute migraine |

| Flunarizine | Prophylaxis | Prevention |

| Propranolol | Prophylaxis | Prevention |

| Topiramate | Prophylaxis | Prevention |

QUESTION

An 80-year-old woman presents with a progressive decline in daily activity. She gives a history of staring into space with frequent visual hallucinations. Pathology reveals Lewy bodies in neurons. What is the most likely diagnosis?

A Parkinson's Disease

CORRECT

B Prion's Disease

C Huntington's Disease

D Alzheimer's Disease

RIGHT ANSWER

OK A. Parkinson's Disease

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1b" here and does not include a written explanation for this item.

QUESTION

| Which of the following is a marker of Alzheimer disease?

- A Alpha synuclein
- B Amyloid beta plaque**
- C Glial fibrillary associated protein
- D Desmin

CORRECT

RIGHT ANSWER

OK **B. Amyloid beta plaque**

WHY THIS IS RIGHT

Alzheimer disease is characterized by extracellular amyloid- β plaque deposition in the cerebral cortex. Two pathological signatures:

1. Amyloid beta ($A\beta$) plaques -> extracellular
2. Neurofibrillary tangles (tau protein) -> intracellular

These plaques are derived from abnormal processing of amyloid precursor protein.

Amyloid precursor protein (APP)

↓ (β & γ secretase)

Amyloid β accumulation

↓

Extracellular plaques

↓

Neurotoxicity + synaptic dysfunction

↓

Cognitive decline

α -Synuclein is seen in Parkinson disease, GFAP marks astrocytes, and desmin is a muscle intermediate filament.

QUESTION

A 25-year-old female patient with diagnosed recurrent migraine despite Sumatriptan, has 3-5 episodes/week and presents with features of anxiety and palpitations. There is no clinical neck swelling, no history of coronary artery disease; however, there is a family history of CHD. She reports experiencing chest tightness after taking the medication. What is the most appropriate agent to add for migraine prophylaxis?

A Topiramate

B Propranolol

CORRECT

C Naratriptan

D Ergotamine

RIGHT ANSWER

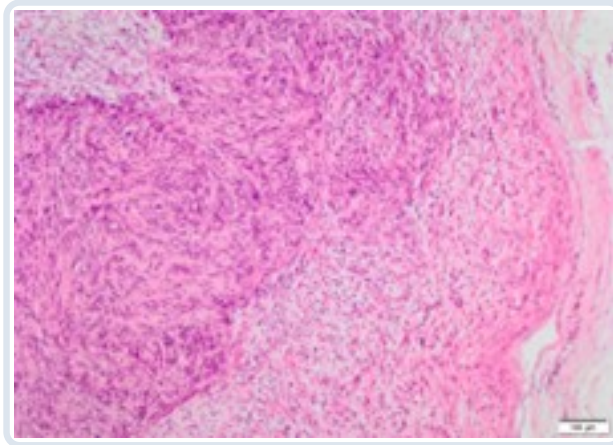
OK B. Propranolol

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1c” here and does not include a written explanation for this item.

QUESTION

A 45-year-old man with abnormal gait and hearing problems presents with a cerebellopontine mass. Histopathological examination is shown below. What is the most likely diagnosis?



A Schwannoma

CORRECT

B Glioblastoma

C Oligodendroglioma

D Medulloblastoma

RIGHT ANSWER

OK A. Schwannoma

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1d" here and does not include a written explanation for this item.

QUESTION

A 72-year-old patient with a confirmed diagnosis of mild to moderate Alzheimer's dementia has shown aversion and non-compliance to oral medications. The patient's caregiver reports difficulty in administering the oral drugs due to the patient's refusal to swallow pills. Considering the need for continued treatment, which of the following medications could be prescribed in a form that does not require oral administration?

A Memantine

B Donepezil

C Rivastigmine

CORRECT

D Galantamine

RIGHT ANSWER

OK C. Rivastigmine

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1e” here and does not include a written explanation for this item.

QUESTION

Which of the following is NOT indicative of underlying serious pathology in headache?

- A Age > 50
- B Temporal tenderness
- C Chronic Cocaine use
- D Subacute progression over 4 weeks

CORRECT

RIGHT ANSWER

OK C. Chronic Cocaine use

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1f" here and does not include a written explanation for this item.

QUESTION

A 20-year-old male presents with sudden onset severe headache and vomiting. On examination, he has neck stiffness. A CT scan of the brain shows hydrocephalus. What is the most likely diagnosis?

A Normal Pressure Hydrocephalus

B Bacterial Meningitis

C Viral Encephalitis

D Subarachnoid Haemorrhage

CORRECT

RIGHT ANSWER

OK

D. Subarachnoid Haemorrhage

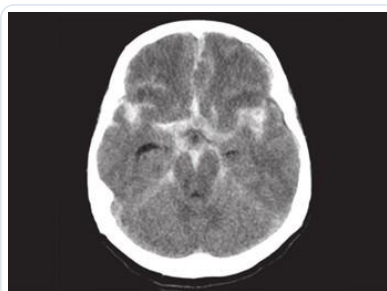
WHY THIS IS RIGHT

Berry aneurysm rupture
↓
Blood enters subarachnoid space
↓
Meningeal irritation -> Neck stiffness
↓
CSF absorption blocked
↓
Hydrocephalus
↓
↑ Intracranial pressure -> Headache + vomiting

Clinical Features of SAH

Feature	Explanation
Thunderclap headache	Sudden ↑ ICP
Vomiting	Brainstem/ICP response
Neck stiffness	Meningeal irritation
Photophobia	Meningeal involvement
Loss of consciousness	Severe cases

Location	%
Anterior communicating artery	Most common
Posterior communicating artery	2nd
MCA bifurcation	Also, common



QUESTION

Which of the following statements regarding Rimegepant is true?

1. It is a partial agonist of the CGRP receptor
2. It causes vasoconstriction
3. It is given via the subcutaneous route
4. It is used in the acute management of migraine

A 1, 2 and 3

B 2 and 3

C 4 only

CORRECT

D 1, 3 and 4

RIGHT ANSWER

OK C. 4 only

WHY THIS IS RIGHT

Rimegepant:

Rimegepant is a “gepant” class drug:

- A CGRP (Calcitonin Gene-Related Peptide) receptor antagonist

During migraine:

- CGRP released from trigeminal nerve endings

-> Causes:

- Vasodilation
- Neurogenic inflammation
- Pain transmission

| Feature | Rimegepant | Triptans (e.g., Sumatriptan) | CGRP mAbs |

| ----- | ----- | ----- | ----- |

| Mechanism | CGRP receptor antagonist | 5-HT_{1B/1D} agonist | CGRP ligand/receptor block |

| Vasoconstriction | ☐ No | ☐ Yes | ☐ No |

| Route | Oral | Oral / SC / nasal | SC / IV |

| Use | Acute ± preventive | Acute | Preventive |

| CV safety | Safe | Contraindicated | Safe |

QUESTION

| **Thalidomide should not be used in which of the following conditions?**

CORRECT

A HIV-associated peripheral neuropathy

B HIV-associated aphthous ulcers

C Behcet syndrome

D Erythema nodosum leprosum

RIGHT ANSWER

OK **A. HIV-associated peripheral neuropathy**

WHY THIS IS RIGHT

Thalidomide is an immunomodulatory drug used for several inflammatory and dermatologic conditions but is contraindicated in peripheral neuropathy because it can cause or worsen neuropathy.

Uses of thalidomide:

- Erythema nodosum leprosum (ENL) - drug of choice
- Behçet syndrome (oral/genital ulcers)
- HIV-associated aphthous ulcers
- Multiple myeloma
- Major adverse effects:
 - Peripheral neuropathy (dose-related, often irreversible)
 - Severe teratogenicity (phocomelia)
 - Sedation, constipation, thrombosis

*

QUESTION

35-year-old woman with a history of migraines is evaluated due to a recent increase in headache frequency and severity. She has had no focal weakness, sensory loss, vision changes, or seizures. Neuroimaging reveals a small aneurysm arising from the segment of right internal carotid artery within the cavernous sinus. If this patient's aneurysm continues to expand, which of the following findings is most likely to be observed?

A Deviation of the tongue to the right when protruded

B Inability to contract the right orbicularis oculi muscle

C Vision defect affecting the left half of the visual field

D Weakness of the right lateral rectus muscle

CORRECT

RIGHT ANSWER

OK

D. Weakness of the right lateral rectus muscle

WHY THIS IS RIGHT

An aneurysm of the internal carotid artery within the cavernous sinus can compress cranial nerves running through the cavernous sinus. Structures in cavernous sinus:

- CN III (oculomotor)
- CN IV (trochlear)
- CN V1 & V2 (trigeminal divisions)
- CN VI (abducens) - runs closest to ICA -> most vulnerable - Causes lateral rectus weakness -> inability to abduct eye -> medial deviation (esotropia) and diplopia
- Internal carotid artery

Why others are wrong:

- Tongue deviation -> CN XII (not in cavernous sinus)
- Orbicularis oculi -> CN VII
- Contralateral visual field defect -> optic tract/chiasm lesion

QUESTION

A 36-year-old woman presents with a unilateral throbbing headache associated with nausea and vomiting. She has had similar episodes in the past where the headache usually lasts for 1-2 days. Bright light increases her discomfort and she prefers sitting in a dark room. Which of the following are used to prevent further episodes of this condition?

1. Sumatriptan
2. Propranolol
3. Naproxen
4. Topiramate

A 2 and 4

CORRECT

B 1, 2 and 3

C 1, 2 and 4

D 1, 2, 3 and 4

RIGHT ANSWER

OK A. 2 and 4

WHY THIS IS RIGHT

The patient has migraine without aura (unilateral throbbing headache with nausea, photophobia, lasting 1-2 days).

Migraine management is divided into:

Acute (abortive) treatment Used during attack:

- Sumatriptan
- NSAIDs (e.g., naproxen)

Prophylaxis (prevention) :Used when attacks are frequent/severe:

- Propranolol (β -blocker; first-line prophylaxis)
- Topiramate (antiepileptic)
- Others: amitriptyline, valproate, CGRP inhibitors

QUESTION

A 22-year-old woman came to the hospital with complaints of excruciating paroxysmal pain on the right side of her lips, gums, and cheek. The episodes last for 2-3 min. Which of the following is incorrect regarding the primary drug used in the treatment of this condition?

- A It can cause agranulocytosis
- B It is a Chloride channel opener
- C It also has anti-epileptic activity against focal seizures
- D It can lead to hyponatremia in the elderly

CORRECT

RIGHT ANSWER

- OK
- B. It is a Chloride channel opener

WHY THIS IS RIGHT

Severe, brief, unilateral facial pain involving lips, gums, and cheek is classic for trigeminal neuralgia.
Drug of choice: Carbamazepine

Mechanism of action:

- Blocks voltage-gated sodium channels -> stabilizes hyperexcitable neuronal membranes
- Not a chloride channel opener (that allows Cl^- influx via GABA-A drugs)

Important adverse effects of carbamazepine:

- Agranulocytosis/aplastic anemia
- Hyponatremia (SIADH), especially in elderly
- Hepatotoxicity
- Also effective for focal seizures and trigeminal neuralgia

QUESTION

A 34-year-old man comes to the emergency department due to severe headache. The patient describes unbearable, throbbing pain around his right eye that awakened him. Over the past week, he has had several similar episodes, which spontaneously resolved after 20-30 minutes. On physical examination, he appears restless and paces in the room. There is mild conjunctival injection and miosis of the right eye. Neurologic examination shows no abnormalities. Which of the following is the most likely cause of this patient's current symptoms?

A Cavernous sinus thrombosis

B Cluster headache

CORRECT

C Migraine without aura

D Trigeminal neuralgia

RIGHT ANSWER

B. Cluster headache

WHY THIS IS RIGHT

Cluster headache is a trigeminal autonomic cephalalgia characterized by severe unilateral periorbital pain with ipsilateral autonomic features like conjunctival injection, lacrimation, nasal congestion and horner syndrome.

- Severe, stabbing/throbbing periorbital unilateral pain
- Short attacks (15-180 min), occurring in clusters over weeks
- Often awakens patient from sleep
- Patient is restless/agitated (unlike migraine where patient prefers dark room)
- Why not other options (short):
- A. Cavernous sinus thrombosis: Fever, toxic patient, ophthalmoplegia & CN palsies -> not brief recurrent attacks.
- C. Migraine: Lasts hours-days, patient lies still in dark room -> here patient is restless.
- D. Trigeminal neuralgia: Seconds-long electric shock pain, no autonomic signs.

QUESTION

A 65-year-old woman is evaluated for worsening urinary urgency and incontinence. The patient has had no abdominal pain, dysuria, or changes in urinary frequency. The patient feels unsteady while ambulating and walks slowly with small steps. Her family also reports that she has become forgetful and has difficulty planning and executing various activities that she used to perform regularly. MRI of the brain reveals chronic white matter changes with no infarction, hemorrhage, or mass lesions. There is diffuse ventriculomegaly with no sulcal enlargement. Which of the following is the most likely underlying cause of this patient's urinary incontinence?

- A Compression of the reticular formation
- B Impaired basal ganglia signaling
- C Spinal cord damage
- D Stretching of descending cortical fibers**

CORRECT

RIGHT ANSWER

- OK D. Stretching of descending cortical fibers**

WHY THIS IS RIGHT

This patient has the classic triad of normal pressure hydrocephalus (NPH):

- Gait disturbance (magnetic/shuffling gait)
- Urinary incontinence/urgency
- Cognitive decline (executive dysfunction)

Pathophysiology of urinary incontinence in NPH:

- Ventriculomegaly with normal sulci causes stretching of periventricular white matter
- Specifically affects descending frontal cortical fibers that normally inhibit the micturition reflex
- Loss of cortical inhibition -> detrusor overactivity -> urgency and incontinence

QUESTION

| Which of the following is not a cause of reversible dementia?

- A B12 deficiency
- B Normal pressure hydrocephalus
- C Lewy body dementia**
- D Hypothyroidism

CORRECT

RIGHT ANSWER

- OK
- C. Lewy body dementia**

WHY THIS IS RIGHT

Reversible (treatable) causes of dementia must always be ruled out before diagnosing primary neurodegenerative dementia.

Common reversible causes:

- Vitamin B12 deficiency -> cognitive decline, neuropathy (improves with replacement)
- Hypothyroidism -> "pseudodementia," improves with thyroxine
- Normal pressure hydrocephalus (NPH) -> triad: gait disturbance, urinary incontinence, dementia; improves with shunting

Irreversible dementia:

- Lewy body dementia -> neurodegenerative disorder with visual hallucinations, parkinsonism, fluctuating cognition; not reversible

QUESTION

| Treatment of choice for an acute attack of cluster headache is:

A Oral sumatriptan

B Subcutaneous sumatriptan

CORRECT

C 100% oxygen at 6 L/minute

D Propranolol

RIGHT ANSWER

OK

B. Subcutaneous sumatriptan

WHY THIS IS RIGHT

Cluster headache presents with severe unilateral periorbital pain with autonomic symptoms (lacrimation, rhinorrhea, ptosis) occurring in clusters.

Treatment of acute attack (abortive):

First-line:

- Subcutaneous sumatriptan -> fastest and most effective
- High-flow 100% oxygen (10-15 L/min) via non-rebreather mask
- Oral triptans are less effective due to slow onset

Prophylaxis (prevention):

- Verapamil (drug of choice)
- Lithium, topiramate (alternatives)

QUESTION

A 55-year-old, previously healthy man is brought to the OPD by his wife after being forced into early retirement due to poor work performance. The patient was a financial planner but began missing important deadlines and mismanaging his client's accounts 6 months ago. He became more irritable during this time and started to curse at and insult his coworkers when they expressed concern about his performance. The patient has also become verbally abusive toward his wife but appears indifferent to the hurt he causes. She has had to take over the finances and grocery shopping. She adds, "My husband has developed quite the sweet tooth. He eats almost two boxes of cookies a day now." Physical examination is unremarkable. This patient is most likely to have which of the following neuropathologic findings?

CORRECT

- A** Aggregations of phosphorylated tau protein
- B** Cytoplasm inclusions with alpha synuclein
- C** Cytosolic vacuolation of neurons and glia with prion inclusions
- D** Extracellular deposition of beta-amyloid

RIGHT ANSWER

- OK** **A. Aggregations of phosphorylated tau protein**

WHY THIS IS RIGHT

Early-onset behavioral changes with personality disturbance, disinhibition, social inappropriateness, apathy, poor judgment, and hyperorality (e.g., increased sweet intake) are classic for frontotemporal dementia (FTD).

Neuropathology:

- Characterized by Pick bodies -> intracellular inclusions of hyperphosphorylated tau protein
- Predominant degeneration of frontal and anterior temporal lobes

Differentiation:

- Alzheimer disease: β -amyloid plaques + tau tangles; early memory loss
- Lewy body dementia: α -synuclein inclusions, hallucinations, parkinsonism
- Prion disease: Rapid dementia + spongiform change.

*

QUESTION

A 24 year old female patient comes with complaints of a bilateral, non-pulsatile headache of tightening quality. She describes it as wearing a tight cap. She has no nausea, photophobia, or phonophobia. She also gives a history that she has had similar headaches for about 10 days in the past month. All the following treatment methods can be used in this patient, except_

A Acetaminophen

B Amitriptyline

CORRECT

C Relaxation techniques

D Aspirin

RIGHT ANSWER

OK B. Amitriptyline

WHY THIS IS RIGHT

The clinical picture describes episodic tension-type headache - bilateral, non-pulsatile, tightening (“band-like” or “tight cap”) headache without nausea, photophobia, or phonophobia. Management:

- Acute treatment:
 - Acetaminophen (paracetamol)
 - NSAIDs (e.g., aspirin, ibuprofen)
- Non-pharmacological:
 - Relaxation therapy
 - Stress management
 - Sleep hygiene

Prophylaxis:

- Amitriptyline is used only for chronic tension-type headache (≥ 15 days/month for >3 months), not for infrequent or episodic attacks.

QUESTION

A 68-year-old man with a long-standing history of poorly controlled hypertension presents with progressive cognitive decline, gait disturbance, and urinary incontinence. Neurological examination reveals upper motor neuron signs. MRI brain shows diffuse periventricular white-matter changes. The most likely diagnosis is:

- A Alzheimer disease
- B Normal pressure hydrocephalus
- C Binswanger disease
- D Dementia with Lewy bodies

CORRECT

RIGHT ANSWER

OK C. Binswanger disease

WHY THIS IS RIGHT

Binswanger disease (subcortical leukoencephalopathy) is a form of vascular dementia caused by chronic hypertension leading to small-vessel ischemic damage of deep white matter.

MRI shows diffuse periventricular and deep white-matter hyperintensities due to chronic ischemia.

- Progressive cognitive decline (subcortical dementia)
- Gait disturbance
- Urinary incontinence
- Upper motor neuron signs (hyperreflexia, spasticity)

Differentiation:

- Binswanger disease: HTN + UMN signs + white-matter lesions
- Normal pressure hydrocephalus: Triad present but ventriculomegaly without marked white-matter ischemia
- Alzheimer disease: Cortical dementia, memory first, no early UMN signs
- Lewy body dementia: Visual hallucinations + parkinsonism + fluctuating cognition

QUESTION

Match the following genes associated with Alzheimer's disease with their chromosomes

List I (Gene)

- A. APOE ($\epsilon 2$ / $\epsilon 4$)
- B. Presenilin-1 (PSEN-1)
- C. Presenilin-2 (PSEN-2)
- D. LRP1
- E. APP (Amyloid precursor protein)

List II (Chromosome)

- 1. Chromosome 1
- 2. Chromosome 12
- 3. Chromosome 14
- 4. Chromosome 19
- 5. Chromosome 21

A A-5,B-1,C-4,D-3,E-2

B A-4,B-3, C-1 D-2 E-5

CORRECT

C A-3,B-2,C-5 ,D-1,E-4

D A-2,B-4, C-3 D-5,E-1

RIGHT ANSWER

OK

B. A-4,B-3, C-1 D-2 E-5

WHY THIS IS RIGHT

Key genes implicated in Alzheimer's disease are frequently tested with their chromosomal locations:

- APP (Amyloid precursor protein) -> Chromosome 21
- Explains early Alzheimer's in Down syndrome (trisomy 21)
- Presenilin-1 (PSEN-1) -> Chromosome 14
- Most common cause of early-onset familial Alzheimer's
- Presenilin-2 (PSEN-2) -> Chromosome 1
- APOE ($\epsilon 4$ risk allele) -> Chromosome 19
- Strongest genetic risk factor for late-onset Alzheimer's
- LRP1 -> Chromosome 12

QUESTION

| All of the following are subcortical dementias, EXCEPT:

A Parkinson's disease dementia

B Huntington disease

C Progressive supranuclear palsy

D Pick's disease

CORRECT

RIGHT ANSWER

OK D. Pick's disease

WHY THIS IS RIGHT

Subcortical dementias primarily affect the basal ganglia, thalamus, brainstem, and white matter, leading to psychomotor slowing, impaired attention, executive dysfunction, and mood changes rather than early aphasia or apraxia.

Examples include:

- Parkinson's disease dementia
- Huntington disease
- Progressive supranuclear palsy (PSP)

Pick's disease (Frontotemporal dementia) is a cortical dementia, characterized by early personality changes, behavioral disinhibition, and language impairment due to frontal and temporal lobe degeneration.

*

QUESTION

A 30-year-old female presents with headache, fever, and nuchal rigidity. Lumbar puncture was done, and CSF analysis shows abundant lymphocytes with normal glucose and elevated proteins. What is the diagnosis?

A Acute bacterial meningitis

B Fungal meningitis

C Tubercular meningitis

D Viral meningitis

CORRECT

RIGHT ANSWER

OK

D. Viral meningitis

WHY THIS IS RIGHT

Finding	Interpretation
Fever + headache + neck stiffness	Meningitis
Lymphocytic predominance	Viral / TB / fungal
Normal glucose	Viral
↑ Protein	Mild inflammation

Correct answer is D because viral meningitis classically shows lymphocytic pleocytosis with normal glucose and mildly elevated protein in CSF.

Bacterial meningitis shows neutrophils with low glucose. Tubercular and fungal meningitis usually show lymphocytes but with low glucose and very high protein.

Thus, normal glucose with lymphocyte predominance favors viral meningitis.

Feature	Bacterial	Viral	TB	Fungal
Cells	Neutrophils	Lymphocytes	Lymphocytes	Lymphocytes
Protein	↑↑	↑	↑↑	↑
Glucose	↓	Normal	↓	↓
Onset	Acute	Acute	Subacute	Chronic

QUESTION

A patient presents with high-grade fever, headache, and altered sensorium. Cerebrospinal fluid (CSF) examination reveals low glucose, high protein, and a marked increase in polymorphonuclear leukocytes. Which of the following is the most likely diagnosis?

- A Acute viral meningitis
- B Acute tubercular meningitis
- C Acute bacterial meningitis**
- D Acute subarachnoid haemorrhage

CORRECT

RIGHT ANSWER

- OK
- C. Acute bacterial meningitis**

WHY THIS IS RIGHT

Parameter	Finding	Interpretation
Glucose	↓ Low	Consumed by bacteria + neutrophils
Protein	↑ High	BBB disruption + inflammation
Cells	↑ PMNs (neutrophils)	Acute pyogenic response

Common organisms (age-wise)

- Neonates -> E. coli, GBS
- Young adults -> Neisseria meningitidis
- Elderly -> Streptococcus pneumoniae

Feature	Bacterial	Viral	Tubercular	SAH
Cells	Neutrophils	Lymphocytes	Lymphocytes	RBCs
Glucose	↓↓↓	Normal	↓	Normal
Protein	↑↑	Mild ↑	↑↑↑	Mild ↑
CSF appearance	Turbid	Clear	Cobweb clot	Bloody
Onset	Acute	Subacute	Chronic	Sudden

QUESTION

A 22-year-old college student presents to the emergency department with a 1-day history of high-grade fever, severe headache, neck stiffness, and a petechial rash. Neurological examination reveals positive Kernig and Brudzinski signs. The diagnosis of meningococcal meningitis is suspected. Which of the following medications is the drug of choice for chemoprophylaxis of close contacts of this patient?

A Doxycycline

B Rifampicin

CORRECT

C Amoxicillin

D Ethambutol

RIGHT ANSWER

OK B. Rifampicin

WHY THIS IS RIGHT

Feature	Interpretation
High fever + headache + neck stiffness	Meningitis
Petechial rash	*Neisseria meningitidis*
Kernig & Brudzinski positive	Meningeal irritation

This presentation is classic for meningococcal meningitis (fever, neck stiffness, petechial rash). Close contacts are at high risk due to nasopharyngeal carriage of *Neisseria meningitidis*. Rifampicin is the drug of choice for chemoprophylaxis, as it eradicates nasopharyngeal carriage.

Drug	Role in Meningococcal Prophylaxis
Rifampicin	First-line
Ciprofloxacin	Alternative
Ceftriaxone	Alternative
Others	☐ Not used

QUESTION

| What is the most likely diagnosis?

A Acute bacterial meningitis

B Viral meningitis

C Tubercular meningitis

CORRECT

D Fungal meningitis

RIGHT ANSWER

OK C. Tubercular meningitis

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1a" here and does not include a written explanation for this item.

QUESTION

A child has features of meningitis with following CSF examination: protein=93, glucose=20, Lymphocytes++. Diagnosis?

A Bacterial meningitis

B Viral meningitis

C Tubercular meningitis

CORRECT

D None of the above

RIGHT ANSWER

OK

C. Tubercular meningitis

WHY THIS IS RIGHT

No explanation was provided in the source quiz.

QUESTION

A 40-year-old male patient presented with complaints of drowsiness and was admitted for evaluation. Sodium was found to be 98 mEq/L, which was corrected to 110 mEq/L with 3% saline in 24 hours. The patient recovered for a while and had an altered sensorium later. Now, he developed quadriparesis and bilateral conjugate gaze palsy. Which is the investigation to be done in this case?

- A CSF analysis
- B Brainstem auditory evoked potential
- C MRI brain**
- D EEG

CORRECT

RIGHT ANSWER

C. MRI brain

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1b” here and does not include a written explanation for this item.

QUESTION

Regarding Guillain-Barré syndrome (GBS) in children, which of the following statements is false?

- A** Plasmapheresis is not effective in GBS
- B** IV immunoglobulins are the first line of treatment
- C** Areflexia in the weaker arm is included in diagnostic criteria
- D** Albumino-cytological dissociation is seen

CORRECT

RIGHT ANSWER

- OK** A. Plasmapheresis is not effective in GBS

WHY THIS IS RIGHT

Guillain-Barré Syndrome (GBS)-

- Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)
- Immune-mediated attack on peripheral nerve myelin
- Often follows:
- Infection (e.g., *Campylobacter jejuni*)

Infection (e.g., Campylobacter)

↓

Molecular mimicry

↓

Autoantibodies against myelin

↓

Demyelination of peripheral nerves

↓

↓ Nerve conduction

↓

Ascending weakness + areflexia

A. Plasmapheresis is not effective in GBS

- Plasmapheresis IS effective
- Removes circulating antibodies
- Used in:
- Severe GBS
- Refractory cases

So, statement is FALSE

QUESTION

Which of the following statements about Multiple Sclerosis are incorrect?

- A) 70% of patients with MS develop Optic Neuritis during the course of illness
- B) Oral steroids are first line for acute relapses
- C) Loss of colour and depth perception is proportional to loss of visual acuity
- D) Fundus examination can be normal

A A, B

B B, C

C A, B, C

CORRECT

D D only

RIGHT ANSWER

OK C. A, B, C

WHY THIS IS RIGHT

In multiple sclerosis, optic neuritis occurs in ~50%, not 70%, making statement A incorrect.

Autoimmune attack on optic nerve myelin

↓

Demyelination of optic nerve fibers

↓

↓ Conduction velocity

↓

Visual symptoms:

- Blurring of vision
- Pain on eye movement
- Color vision loss (early)

↓

Fundus initially normal (retrobulbar neuritis)

Feature	Description
Vision loss	Subacute, unilateral
Pain	On eye movement
Color vision	Earliest affected
Pupillary reflex	RAPD (Marcus Gunn pupil)
Fundus	Normal initially

| ----- | ----- |

| Vision loss | Subacute, unilateral |

| Pain | On eye movement |

| Color vision | Earliest affected |

| Pupillary reflex | RAPD (Marcus Gunn pupil) |

| Fundus | Normal initially |

Acute relapses are treated with IV methylprednisolone, not oral steroids, so B is incorrect.

In optic neuritis, loss of color vision (red desaturation) is disproportionately greater than visual acuity loss, making C incorrect.

Fundus can be normal (retrobulbar neuritis), so D is correct; hence A, B, C are incorrect.

QUESTION

Which of the following is the most common presenting symptom in patients with Multiple Sclerosis?

- A

Sensory Disturbances

CORRECT
- B

Vertigo
- C

Ataxia
- D

Facial Palsy

OK

RIGHT ANSWER

A. Sensory Disturbances

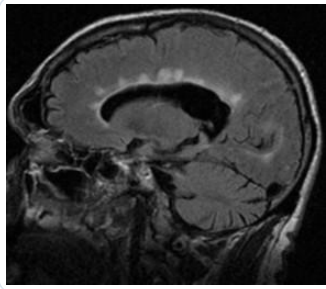
WHY THIS IS RIGHT

Multiple sclerosis most commonly presents initially with sensory disturbances such as numbness, paresthesia, or sensory loss. These symptoms result from demyelination of sensory pathways in the CNS. Motor deficits, ataxia, and cranial nerve involvement usually occur later in the disease course. Thus, sensory symptoms are the earliest and most common presenting feature.

- Classic Early Presentation Triad
- 1. Sensory symptoms (most common)
 - 2. Optic neuritis (painful vision loss)
 - 3. Motor weakness

Initial Symptoms of Multiple Sclerosis (MS)

SYMPTOM	PERCENTAGE OF CASES	SYMPTOM	PERCENTAGE OF CASES
Sensory loss	37	Lhermitte	3
Optic neuritis	36	Pain	3
Weakness	35	Dementia	2
Paresthesias	24	Visual loss	2
Diplopia	15	Facial palsy	1
Ataxia	11	Impotence	1
Vertigo	6	Myokymia	1
Paroxysmal attacks	4	Epilepsy	1
Bladder	4	Falling	1



QUESTION

A male patient presents with fever and nuchal rigidity. His CSF analysis reveals low sugar, elevated WBCs and Cryptococcal antigen positivity. Which of the following is used in the initial management of this condition?

1. Amphotericin B + Flucytosine
2. Amphotericin B + Fluconazole
3. Fluconazole
4. Liposomal Amphotericin B + Itraconazole

A 1 only

CORRECT

B 1 and 2

C 1, 2 and 3

D 2 and 4

RIGHT ANSWER

OK A. 1 only

WHY THIS IS RIGHT

- Fever + neck stiffness + CSF:
- ↓ Glucose
- ↑ WBCs
- Cryptococcal antigen positive

Diagnosis: Cryptococcal meningitis
(most commonly due to *Cryptococcus neoformans* infection)

- Inhaled yeast -> lungs -> hematogenous spread
- > CNS invasion
- > thick polysaccharide capsule prevents immune clearance

Parameter	Finding
Opening pressure	↑↑ (very high)
Glucose	↓↓
Protein	↑↑
Cells	Lymphocytic predominance

Diagnostic Clues

- India ink stain -> encapsulated yeast
- Cryptococcal antigen test -> highly sensitive

Treatment-

Cryptococcal Meningitis

↓

INDUCTION (2 weeks)

-> Amphotericin B + Flucytosine

↓

CONSOLIDATION (8 weeks)

-> Fluconazole (high dose)

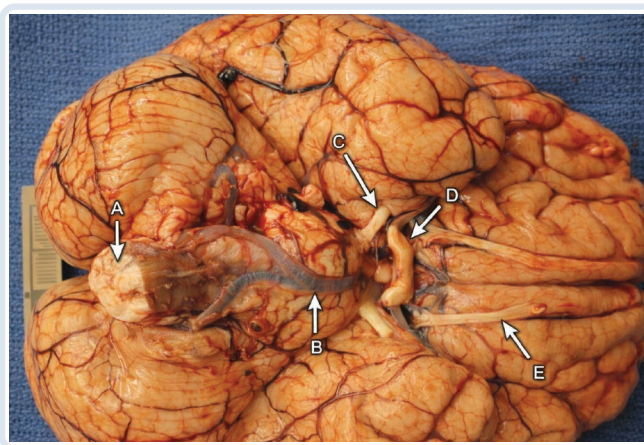
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MAINTENANCE

-> Fluconazole (low dose)

QUESTION

24-year-old man is brought to the emergency department due to seizures. He has had 2 days of worsening fever, headache, and vomiting. Physical examination shows signs of meningeal irritation. The patient rapidly becomes comatose and dies 48 hours later despite aggressive medical care. Autopsy examination shows congested leptomeninges with fibrinopurulent exudate. Microscopy reveals numerous amoeba in the exudate and brain tissue. Which of the following is the most likely portal of entry of this pathogen into the CNS?



A A

B B

C C

D E

CORRECT

RIGHT ANSWER

OK

D. E

WHY THIS IS RIGHT

Rapidly fatal meningoencephalitis in young healthy individuals with freshwater exposure (lakes, swimming pools) with numerous amoebae indicates *Naegleria fowleri* infection causing primary amoebic meningoencephalitis (PAM).

- Organism enters through the nasal mucosa/olfactory epithelium
- Travels via olfactory nerve through cribriform plate
- Reaches brain -> fulminant hemorrhagic meningoencephalitis

Why not other options:

- A: Cerebellar region -> not a portal of entry for infection.
- B: Brainstem/basilar region -> no direct external route for amoeba entry.
- C: Optic pathway -> *Naegleria* does not enter via optic nerve.

QUESTION

A 32-year-old woman comes to the office with several days of tingling and numbness in both hands. She has had occasional headaches, dizziness, fatigue, poor sleep, and blurry vision. She has had no fevers, weight loss, or anorexia. Her medical history is unremarkable. The patient drinks 1 or 2 glasses of wine occasionally but does not use tobacco or illicit drugs. Her blood pressure is 132/70 mm Hg and pulse is 78/min. Sensation to light touch and pain is decreased distally in the bilateral upper extremities. Muscle strength is 5/5 in the upper and lower extremities and deep tendon reflexes are normal. Which of the following is the best next step in management of this patient?

A Carotid Doppler ultrasound

B Cervical spine x-ray

C Lumbar puncture

D MRI of the brain and spine

CORRECT

RIGHT ANSWER

OK D. MRI of the brain and spine

WHY THIS IS RIGHT

Young adult woman with sensory symptoms, visual blurring, fatigue, and dizziness suggests a demyelinating disorder, most notably multiple sclerosis (MS).

Best next step:

- MRI of brain and spine -> detects demyelinating plaques
- Shows lesions disseminated in time and space (periventricular, optic nerve, spinal cord)

Why not others:

- Carotid Doppler -> stroke/TIA evaluation
- Cervical X-ray -> structural bone disease
- Lumbar puncture -> supportive (oligoclonal bands) but done after MRI

QUESTION

42-year-old woman comes to the office due to worsening double vision and gait unsteadiness. She states she had cramping abdominal pain and diarrhoea 2 weeks ago after an outdoor picnic which spontaneously resolved after 3 days. The double vision began 4 days ago and is persistent and progressive. She has had no fever, headache, neck pain, photophobia, or bowel or bladder dysfunction. On physical examination, she is fully alert and oriented with normal memory, speech, and language comprehension. There is mild ptosis of the right eye with weakness of the medial and upward gaze. Left eye movements are normal. Bilateral lower-extremity weakness with loss of deep tendon reflexes is present. Bilateral upper-extremity muscle strength, reflexes, and coordination are normal. Sensation to touch and pinprick is normal throughout. Which of the following is the most likely cause of this patient's current condition?

- A Botulinum toxin ingestion
- B Dietary thiamine deficiency
- C Immune-mediated nerve injury**
- D Neuroinvasive virus infection

CORRECT

RIGHT ANSWER

- OK
- C. Immune-mediated nerve injury**

WHY THIS IS RIGHT

Progressive ascending symmetrical weakness with areflexia following a recent diarrheal illness suggests Guillain-Barré syndrome (GBS), caused by immune-mediated nerve injury.

Pathogenesis:

- Often follows infection (classically *Campylobacter jejuni*)
- Molecular mimicry -> antibodies attack peripheral nerve myelin or axons
- Results in acute inflammatory demyelinating polyradiculoneuropathy

Why not others (short):

- A. Botulism: Descending paralysis, dilated pupils, autonomic signs -> not ascending areflexic weakness.
- B. Thiamine deficiency: Confusion + ataxia (Wernicke), not acute limb weakness with areflexia.
- D. Viral infection: Fever, encephalitis/meningitis signs expected.

*

QUESTION

A 4-year-old boy presents with fever, headache, irritability, nuchal rigidity, and photophobia. CSF analysis shows:

Glucose: 60 mg/dL

Protein: 80 mg/dL

RBCs: 2/mm³

WBCs: 85/mm³ (10% neutrophils, 70% lymphocytes, 20% monocytes)

Which of the following pathogens is most likely responsible?

A Cryptococcus neoformans

B Group B coxsackievirus

CORRECT

C Mycobacterium tuberculosis

D Neisseria meningitidis

RIGHT ANSWER

OK B. Group B coxsackievirus

WHY THIS IS RIGHT

CSF showing normal glucose, mildly elevated protein, and lymphocytic predominance is characteristic of viral (aseptic) meningitis.

Common causes in children:

- Enteroviruses (most common) -> Coxsackievirus, Echovirus
- HSV
- Arboviruses

Differentiation:

- Bacterial meningitis (Neisseria): ↓ glucose, ↑ neutrophils, very high protein
- TB meningitis: ↓ glucose, high protein, lymphocytes
- Cryptococcus: ↓ glucose, high opening pressure (immunocompromised)

QUESTION

| All the following statements are true regarding Miller-Fisher syndrome except?

- A** Associated with ophthalmoplegia
- B** Type of axonal neuropathy
- C** Associated with anti-GQ1b antibodies
- D** Associated with areflexia of the limbs

CORRECT

RIGHT ANSWER

- OK** **B. Type of axonal neuropathy**

WHY THIS IS RIGHT

Miller-Fisher syndrome (MFS) is a rare variant of Guillain-Barré syndrome characterized by the classic triad: Ophthalmoplegia, Ataxia and Areflexia.

Key associations:

- Strongly associated with anti-GQ1b antibodies
- Usually follows a preceding infection
- Considered a demyelinating neuropathy, not primarily axonal

> Why option B is incorrect:

> MFS is classically a demyelinating immune-mediated neuropathy, whereas axonal forms are seen in variants like AMAN/AMSAN of GBS.

QUESTION

A patient presents with fever, neck rigidity and altered sensorium. The resident was instructed to perform a lumbar puncture on this patient. What is the correct order of managing this patient?

1. Place IV cannula and give fluids at 40 mL/h
2. Start injection ceftriaxone
3. Fundoscopy
4. Perform guarded lumbar puncture

A 1, 2, 4, 3

B 1, 4, 3, 2

C 4, 3, 2, 1

D 1, 3, 4, 2

CORRECT

RIGHT ANSWER

OK D. 1, 3, 4, 2

WHY THIS IS RIGHT

In suspected acute bacterial meningitis, lumbar puncture must be performed safely after ruling out raised intracranial pressure to prevent brain herniation.

Correct sequence of management:

- Stabilize patient first -> secure IV access and start fluids
- Fundoscopy -> check for papilledema (sign of raised ICP)
- Guarded lumbar puncture -> if no signs of raised ICP
- Start antibiotics (e.g., ceftriaxone) immediately after CSF collection

Always rule out raised intracranial pressure before LP to avoid herniation.

QUESTION

Which of the following drugs is preferred in the management of primary progressive multiple sclerosis?

A Natalizumab

B Ocrelizumab

CORRECT

C Fingolimod

D Alemtuzumab

RIGHT ANSWER

OK B. Ocrelizumab

WHY THIS IS RIGHT

Ocrelizumab is the drug of choice for primary progressive multiple sclerosis (PPMS) and is the only therapy proven to slow disability progression in this form of MS. Also used in relapsing-remitting MS. Other MS drugs (natalizumab, fingolimod, alemtuzumab) are mainly for relapsing forms, not PPMS.

Mechanism of action:

- Humanized monoclonal antibody against CD20 on B lymphocytes
- Causes B-cell depletion -> reduces demyelination and neuroinflammation

QUESTION

A 35 year old male presented with rapidly progressing, symmetrical muscle weakness, starting in the legs. Which of the following is not a treatment option in this patient?

A Steroids

CORRECT

B IVIG

C Plasmapheresis

D Elective Ventilator support

RIGHT ANSWER

OK

A. Steroids

WHY THIS IS RIGHT

Rapidly progressive, symmetrical ascending weakness suggests Guillain-Barré syndrome (GBS), an acute inflammatory demyelinating polyradiculoneuropathy.

Effective treatments:

- IVIG -> first-line
- Plasmapheresis -> equally effective alternative
- Elective ventilatory support if respiratory muscle weakness or ↓ vital capacity

Corticosteroids have no proven benefit in GBS and are not recommended.

QUESTION

Which of the following is a characteristic feature of an upper motor neuron (UMN) lesion?

A Muscle fasciculations

B Flaccid paralysis

C Hyporeflexia

D Brisk reflexes

CORRECT

RIGHT ANSWER

OK D. Brisk reflexes

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$19" here and does not include a written explanation for this item.

QUESTION

A male patient presents with sensory loss and weakness of limbs for 3 months. He also has angular stomatitis. On examination, there is loss of proprioception, vibration sensations, UMN type of lower limb weakness, and absent ankle reflex. What is the most probable diagnosis?

A Extradural cord compression

B Amyotrophic lateral sclerosis

C Multiple sclerosis

D Subacute combined degeneration of the cord

CORRECT

RIGHT ANSWER

OK

D. Subacute combined degeneration of the cord

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1a” here and does not include a written explanation for this item.

QUESTION

A patient met with a road traffic accident and developed a cervical spine injury. The fracture fragment had pierced the lateral aspect of the dorsal column tract. Which of the following findings is seen in this patient?

- A Absence of ipsilateral lower limb proprioception
- B Absence of fine motor movement of fingers
- C Absence of ipsilateral arm proprioception**
- D Absence of contralateral lower limb proprioception

CORRECT

RIGHT ANSWER

- C. Absence of ipsilateral arm proprioception**

WHY THIS IS RIGHT

Dorsal column carries-

- Fine touch
- Vibration
- Proprioception

And it ascends ipsilaterally (no crossing in spinal cord)

At cervical level:

Region	Tract	Position
Lower limb	Fasciculus gracilis	Medial
Upper limb	Fasciculus cuneatus	Lateral

"Cuneatus = Cervical (upper limb)"

"Gracilis = Ground (lower limb)"

The lateral part of the dorsal column at cervical level corresponds to the cuneate fasciculus, which carries sensory input from the upper limb.

Dorsal column fibers ascend ipsilaterally without crossing in the spinal cord.

Thus, injury causes loss of ipsilateral arm proprioception and vibration sense.

Lower limb proprioception is carried medially by the gracile fasciculus and is spared here.

QUESTION

A 40-year-old man had a RTA and was admitted to a hospital. After a few days, he had an exaggerated right-sided extensor plantar reflex and knee jerk. What other finding would be seen?

- A Atrophy of the right lower limb
- B Loss of proprioception in the left leg
- C Ankle clonus in the right foot**
- D Fasciculations in the right foot

CORRECT

RIGHT ANSWER

- OK
- C. Ankle clonus in the right foot**

WHY THIS IS RIGHT

Exaggerated knee jerk and extensor plantar response indicate an upper motor neuron (UMN) lesion on the right side.

Finding	Interpretation
Exaggerated knee jerk	Hyperreflexia
Extensor plantar reflex	Babinski sign
Same side (right)	Localized lesion

UMN lesions are associated with spasticity and clonus due to loss of inhibitory control.
Sudden stretch (ankle dorsiflexion)

↓
Exaggerated reflex contraction
↓
Repeated oscillations
↓
Clonus

Hence, ankle clonus in the right foot is an expected finding.

Feature	UMN Lesion	LMN Lesion
Reflexes	↑	↓
Babinski	□	□
Clonus	□	□
Fasciculations	□	□
Atrophy	Mild	Severe

Atrophy and fasciculations are LMN signs, and proprioceptive loss suggests dorsal column involvement.

QUESTION

A 30-year-old female notices weakness in her right hand and difficulty while kneading dough. On examination, right arm tone and power are decreased, and reflexes are sluggish. What is the site of the lesion?

- A Posterior limb of the internal capsule
- B Right corticospinal tract in cervical cord
- C Anterior horn cell of the cervical spinal cord**
- D Left medullary pyramid

CORRECT

RIGHT ANSWER

- OK
- C. Anterior horn cell of the cervical spinal cord**

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1b" here and does not include a written explanation for this item.

QUESTION

A patient with Type 2 Diabetes Mellitus complains of numbness and burning sensations in the extremities. On examination he has absent ankle reflexes and has no sensation to touch, vibration, or pain in his lower limbs. The loss of sensation in his extremities has a stocking distribution. What type of neuropathy is associated?

- A Autonomic neuropathy
- B Mononeuropathy
- C Symmetric distal polyneuropathy**
- D Proximal motor neuropathy

CORRECT

RIGHT ANSWER

- OK** C. Symmetric distal polyneuropathy

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1c" here and does not include a written explanation for this item.

QUESTION

| Which of the following is seen in UMN (Upper Motor Neuron) weakness?

A Hyper-reflexia

CORRECT

B Atrophy

C Rigidity

D Fasciculations

RIGHT ANSWER

OK A. Hyper-reflexia

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1d” here and does not include a written explanation for this item.

QUESTION

A 65-year-old man presented to you with neuropathy in his bilateral lower limbs and a positive Romberg sign. He has a history of gastrectomy for stomach cancer. He is likely deficient in which of the following?

A Thiamine deficiency

B Folate deficiency

C Vitamin B12 deficiency

CORRECT

D Transferrin deficiency

RIGHT ANSWER

OK C. Vitamin B12 deficiency

WHY THIS IS RIGHT

- Bilateral lower limb neuropathy
- Positive Romberg sign
- History of gastrectomy

These three together form a diagnostic triangle pointing strongly to Vitamin B12 deficiency

Gastrectomy

↓

Loss of parietal cells

↓

↓ Intrinsic factor

↓

↓ B12 absorption (terminal ileum)

↓

Vitamin B12 deficiency

QUESTION

LMN (Lower Motor Neuron) lesion is associated with which one of the following?

1. Reduced muscle tone
2. Spastic paralysis
3. Babinski signs positive
4. Fasciculation

A 1, 2

B 1, 2, 3

C 1, 2, 3, 4

D 1, 4

CORRECT

RIGHT ANSWER

OK

D. 1, 4

WHY THIS IS RIGHT

Lower Motor Neuron (LMN)

LMNs are the final common pathway:

- From anterior horn cell -> peripheral nerve -> muscle

If this pathway is damaged -> muscle loses direct neural input

After LMN injury-

- Muscle becomes denervated
- No trophic support -> atrophy
- Spontaneous discharges -> fasciculations
- Loss of stretch reflex -> hypotonia

| Feature | LMN Lesion | UMN Lesion |

| ----- | ----- | ----- |

| Tone | ↓ Hypotonia | ↑ Hypertonia |

| Paralysis | Flaccid | Spastic |

| Reflexes | ↓ / absent | ↑ exaggerated |

| Babinski | ☐ Absent | ☐ Present |

| Muscle bulk | Atrophy (early) | Mild atrophy (late) |

| Fasciculations | ☐ Present | ☐ Absent |

QUESTION

A patient presented with worsening back pain and weakness in the lower limbs. Which of the following would not support the diagnosis of compressive myelopathy?

- A Positive Babinski sign
- B Normal cremasteric reflex
- C Hypotonia
- D Knee clonus

CORRECT

RIGHT ANSWER

OK
C. Hypotonia

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1e” here and does not include a written explanation for this item.

QUESTION

A 52-year-old male presents with progressive weakness in the lower limbs, difficulty in walking, and tingling sensations in hands and feet. On examination, there is spasticity, increased deep tendon reflexes, along with impaired vibration and position sense. Which of the following best explains this presentation?

A ALS

B Vitamin B12 deficiency

CORRECT

C Vit E deficiency

D Polio

RIGHT ANSWER

OK

B. Vitamin B12 deficiency

WHY THIS IS RIGHT

Vitamin B12 (cobalamin) deficiency causes a combination of hematological and neurological manifestations, making it a key exam diagnosis when both are present.

- B12 is required for:

- o DNA synthesis -> deficiency causes megaloblastic anemia (pallor)
- o Myelin synthesis -> deficiency leads to demyelination of the spinal cord

Neurological features (important clue):

- Subacute combined degeneration of spinal cord
- o Involves posterior columns -> loss of vibration & position sense
- o Involves lateral corticospinal tracts -> UMN signs (spasticity, hyperreflexia)
- Peripheral neuropathy -> paresthesia, numbness

Why other options are incorrect:

- Vitamin B9 (Folate): Causes megaloblastic anemia without neurological symptoms
- Vitamin B3 (Niacin): Pellagra (dermatitis, diarrhea, dementia)
- Vitamin B1 (Thiamine): Beriberi/Wernicke's (neuropathy, cardiac/brain involvement, but no UMN pattern)

QUESTION

A 5-year-old boy is brought to the emergency department because of a 2-day history of lower leg weakness, swallowing difficulty, and drooling of saliva. He has not yet received any childhood vaccinations. Two days after admission, the patient develops shortness of breath. Pulse oximetry shows an oxygen saturation of 64%. Despite resuscitative efforts, the patient dies of respiratory failure. At autopsy, examination of the spinal cord shows destruction of the anterior horn cells. Neurological examination of this patient would have most likely shown which of the following findings?

A Positive Babinski sign

B Clasp knife spasticity

C Hyporeflexia

CORRECT

D Sensory loss

RIGHT ANSWER

OK C. Hyporeflexia

WHY THIS IS RIGHT

Destruction of anterior horn cells (as seen in poliomyelitis) produces a lower motor neuron (LMN) lesion. Poliovirus infects anterior horn cells of spinal cord leading to asymmetric flaccid paralysis and respiratory muscle failure.

Features of LMN lesion:

- Flaccid paralysis
- Hyporeflexia/areflexia
- Hypotonia
- Muscle atrophy and fasciculations
- No sensory loss (pure motor involvement)

Differentiation:

- Babinski sign & spasticity: UMN lesions
- Sensory loss: Not seen in polio (motor neurons only)

QUESTION

| What would be the likely “classical” presentation of this patient?



A Romberg sign with loss of DTR

B UMN + LMN features

C Cape like distribution

CORRECT

D Only preserved dorsal column function

RIGHT ANSWER

OK C. Cape like distribution

WHY THIS IS RIGHT

The MRI suggests syringomyelia, a cystic dilation of the central spinal cord (commonly cervical region).

Classic clinical feature:

- “Cape-like” bilateral loss of pain and temperature over shoulders and upper limbs
- Due to damage of crossing spinothalamic fibers in the anterior white commissure

Key points:

- Dorsal column sensations (vibration, proprioception) initially preserved
- LMN weakness at level (hand muscles) later
- UMN signs below lesion in advanced disease
- Associated with Chiari I malformation

QUESTION

A 5 year old patient presents to the OPD with dysphagia, dysarthria, and dysphonia. On examination, brisk jaw jerk is seen. What is the possible localisation in this patient?

- A Corticospinal tracts
- B Corticobulbar tracts**
- C Mesencephalic trigeminal nucleus
- D V3 nerve

CORRECT

RIGHT ANSWER

B. Corticobulbar tracts

WHY THIS IS RIGHT

Dysphagia, dysarthria, dysphonia with a brisk jaw jerk indicate an upper motor neuron (UMN) lesion of bulbar muscles, localizing to the corticobulbar tracts (from motor cortex to cranial nerve nuclei (V, VII, IX, X, XII)).

Features of corticobulbar (UMN) lesion:

- Dysarthria, dysphagia, dysphonia
- Brisk jaw jerk
- Spastic tongue
- Emotional lability (pseudobulbar affect)

Differentiation:

- LMN bulbar palsy -> absent jaw jerk, fasciculations
- Corticospinal tract lesion -> limb weakness, not isolated bulbar signs
- V3 nerve lesion -> weak mastication, reduced jaw jerk

QUESTION

A 44-year-old man comes to the OPD due to several weeks of difficulty walking and frequent falls. He also reports episodes of sharp, stabbing pain in his extremities. Deep-tendon reflexes are absent at the knee and ankle bilaterally. Proprioception and vibration sensation are reduced throughout the lower extremities. He has a wide-based gait and a positive Romberg sign. Which of the following diagnostic findings is most likely associated with this patient's current symptoms?

A Cerebrospinal fluid (CSF) culture growing acid-fast bacilli

B CSF PCR positive for herpes virus

C Encapsulated yeasts on CSF India ink preparation

D Reactive VDRL tests on CSF samples

CORRECT

RIGHT ANSWER

OK

D. Reactive VDRL tests on CSF samples

WHY THIS IS RIGHT

Loss of proprioception and vibration, areflexia, sensory ataxia, lightning-like pains, and positive Romberg sign, areflexia in lower limbs, possible Argyll Robertson pupil are classic for tabes dorsalis, a late manifestation of neurosyphilis.

Pathophysiology:

- Degeneration of posterior columns and dorsal roots
- Leads to sensory ataxia and neuropathic pain

Diagnostic finding:

- Reactive VDRL in CSF confirms neurosyphilis

QUESTION

What would be seen if the central part of the cord was involved?

1. LMN lesion of trunk and UMN of lower limbs.
2. UMN lesion of trunk and LMN of lower limbs.
3. Bladder and bowel involvement.
4. Posterior tract involvement.

A 1 and 4

B 2 and 3

C 2 and 4

D 1 and 3

CORRECT

RIGHT ANSWER

OK

D. 1 and 3

WHY THIS IS RIGHT

Lesions involving the central part of the spinal cord (e.g., central cord syndrome, syringomyelia) affect structures in a characteristic pattern.

Key features:

- LMN signs at level of lesion (anterior horn cells) -> flaccid weakness of trunk/segmental muscles
- UMN signs below lesion (corticospinal tract) -> spasticity in lower limbs
- Autonomic involvement -> early bladder and bowel dysfunction

Posterior columns are located posteriorly -> often preserved initially

*

QUESTION

| Which of the following does not help in the localization of lesions in the spinal cord?

A Contralateral hemiplegia

CORRECT

B Fasciculation at the level of lesion

C Babinski positive

D Bladder involvement

RIGHT ANSWER

OK

A. Contralateral hemiplegia

WHY THIS IS RIGHT

Localization of spinal cord lesions depends on identifying upper motor neuron (UMN), lower motor neuron (LMN), and autonomic signs below or at the level of the lesion. Findings that help localize spinal cord lesions:

- Fasciculations at level of lesion: LMN involvement at segmental level
- Babinski sign: UMN involvement below lesion
- Bladder/bowel dysfunction: Autonomic tract involvement -> indicates cord pathology

Finding that does NOT help:

- Contralateral hemiplegia suggests a brain (supratentorial) lesion, not a spinal cord lesion, because spinal cord lesions typically cause ipsilateral motor deficits below the lesion.

QUESTION

A 19-year-old man is brought to the emergency department after being stabbed in the back. Neurological examination demonstrates the absence of motor activity in all muscle groups of the right lower extremity as well as decreased muscle tone. Left leg motor function is normal. Right patellar reflex, Achilles reflex, and Babinski sign are absent. There is loss of light touch and proprioception below the right costal margin. Pinprick sensation is absent below the level of umbilicus on the left side. Which of the following is the most likely location of this patient's injury?

A Anterior spinal artery injury

B Posterior spinal artery injury

C Left spinal hemisection

D Right spinal hemisection

CORRECT

RIGHT ANSWER

OK

D. Right spinal hemisection

WHY THIS IS RIGHT

This presentation is classic for Brown-Séquard syndrome due to spinal cord hemisection. Key neurological pattern in hemisection:

Ipsilateral (same side as lesion):

- LMN weakness at level -> flaccidity, ↓ reflexes
- UMN weakness below lesion (later spastic)
- Loss of proprioception, vibration, fine touch (dorsal column)
- Loss of motor function (corticospinal tract)

Contralateral (opposite side):

- Loss of pain and temperature beginning ~1-2 levels below lesion (due to crossing of spinothalamic tract)

QUESTION

Ataxia due to vitamin E deficiency most closely resembles which of the following neurological conditions?

A Friedreich's ataxia

CORRECT

B Tabes dorsalis

C Subacute combined degeneration of the spinal cord (SACD)

D Multiple system atrophy

RIGHT ANSWER

OK **A. Friedreich's ataxia**

WHY THIS IS RIGHT

Vitamin E deficiency-related ataxia produces a neurological picture that closely mimics Friedreich's ataxia due to degeneration of the posterior columns, spinocerebellar tracts, and peripheral nerves.

Common features with Friedreich's ataxia:

- Progressive cerebellar ataxia
- Loss of proprioception and vibration sense
- Areflexia
- Peripheral neuropathy
- Dysarthria

*

QUESTION

A 55-year-old man presents with progressive weakness of both upper and lower limbs. Examination reveals muscle wasting with fasciculations, hyperreflexia, and extensor plantar responses. Sensory examination is normal. Cranial nerve examination later shows dysarthria.

Which of the following is the most likely diagnosis?

- A** Cervical spondylotic myelopathy
- B** Multiple sclerosis
- C** Amyotrophic lateral sclerosis
- D** Chronic inflammatory demyelinating polyneuropathy

CORRECT

RIGHT ANSWER

OK C. Amyotrophic lateral sclerosis

WHY THIS IS RIGHT

Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disorder characterized by simultaneous upper and lower motor neuron degeneration with no sensory loss.

- Lower motor neuron signs: Muscle wasting, fasciculations, weakness
- Upper motor neuron signs: Hyperreflexia, spasticity, extensor plantar (Babinski)
- Bulbar involvement: Dysarthria, dysphagia (late feature)
- Sensory system: Typically normal

Differentiation:

- Cervical spondylotic myelopathy: UMN signs + sensory deficits/radicular pain
- Multiple sclerosis: CNS lesions with sensory/visual involvement
- CIDP: Pure LMN-type peripheral neuropathy with sensory loss, areflexia

QUESTION

A female patient complains of chest pain after waking up in the morning. Pain is more prevalent in the winter months. Which of the following best describes this patient?

- A** Fixed plaque obstruction
- B** Ach-induced coronary vasoconstriction
- C** Subendocardial ischemia due to increased demand of myocardium
- D** Increased sympathomimetic drive in morning hours

CORRECT

RIGHT ANSWER

- OK**
- B. Ach-induced coronary vasoconstriction**

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$19” here and does not include a written explanation for this item.

QUESTION

A 46-year-old male presents to the ER with the acute onset of ataxia. Neurological examination shows loss of pain and temperature sensation from the face on the left side and loss of pain and temperature sensation from the right half of the body. He also has dysarthria, dysphagia, and diminished gag reflex with ptosis, miosis, and anhidrosis on the left. What is the most likely diagnosis?

A Lateral pontine syndrome

B Medial medullary syndrome

C Lateral medullary syndrome

CORRECT

D Locked-In Syndrome

RIGHT ANSWER

OK C. Lateral medullary syndrome

WHY THIS IS RIGHT

Finding	Side	Structure involved
Face pain/temp loss	Left	Spinal trigeminal nucleus
Body pain/temp loss	Right	Spinothalamic tract
Dysphagia, dysarthria, ↓ gag	Left	Nucleus ambiguus (CN IX, X)
Ataxia	Left	Inferior cerebellar peduncle
Ptosis, miosis, anhidrosis	Left	Sympathetic fibers (Horner's)

Ipsilateral face + contralateral body pain loss + Cranial nerve IX, X involvement + Horner's syndrome
This combination = Wallenberg syndrome

PICA infarction

↓

Lateral medulla damage

↓

Multiple structure involvement:

↓

Spinal trigeminal nucleus -> Ipsilateral face pain loss

Spinothalamic tract -> Contralateral body pain loss

Nucleus ambiguus -> Dysphagia, dysarthria

Sympathetic fibers -> Horner's syndrome

Cerebellar peduncle -> Ataxia

QUESTION

A 62-year-old patient presents with left-sided arm and leg weakness, right-sided facial paralysis, and horizontal gaze palsy. Based on the clinical presentation, which of the following is most consistent with the diagnosis?

A Foville syndrome

CORRECT

B Locked In syndrome

C Millard-Gubler syndrome

D Wallenberg syndrome

RIGHT ANSWER

OK
A. Foville syndrome

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1a” here and does not include a written explanation for this item.

QUESTION

I Interventional technique used for the management of cerebral aneurysm:**A Coiling**

CORRECT

B Embolization**C Basal stent placement****D Balloon Angioplasty**

RIGHT ANSWER

OK A. Coiling

WHY THIS IS RIGHT

Cerebral Aneurysm-

- Localized dilatation of an artery (usually at branch points in Circle of Willis)
- Most common: Berry (saccular) aneurysm
- Risk: Subarachnoid hemorrhage (SAH) -> sudden “worst headache of life”

Treatment Goal- Exclude aneurysm from circulation WITHOUT blocking parent artery

Cerebral aneurysm -> Endovascular coiling is the standard minimally invasive treatment to prevent rupture/rebleed

Feature	Coiling	Surgical Clipping
-----	-----	-----
Approach	Endovascular	Open surgery
Invasiveness	Minimal	High
Recovery	Faster	Slower
Recurrence	Slightly higher	Lower

Clinical Insight

- After SAH:
- Secure aneurysm ASAP -> coiling/clipping
- Prevents:
- Rebleeding (highest risk in first 24 hrs)

QUESTION

A patient presented with altered sensorium and his BP was 220/110 mmHg. He also had one episode of vomiting and on examination has weakness in the left upper and lower limbs. Which of the following is incorrect?

- A** BP should be checked multiple times before administering anti-hypertensives
- B** In haemorrhagic stroke, you should aggressively treat with intravenous anti-hypertensives
- C** In haemorrhagic stroke, BP should be reduced to 160/100 mmHg with anti-hypertensives
- D** BP should be reduced immediately to less than 140/90 mmHg in ischemic stroke CORRECT

RIGHT ANSWER

- OK** D. BP should be reduced immediately to less than 140/90 mmHg in ischemic stroke

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1b” here and does not include a written explanation for this item.

QUESTION

The most common site of a berry aneurysm in the circle of Willis is:

A Anterior cerebral artery

CORRECT

B Vertebral artery

C Posterior cerebral artery

D Basilar artery

RIGHT ANSWER

OK

A. Anterior cerebral artery

WHY THIS IS RIGHT

Most common berry aneurysm site = Anterior communicating artery (ACoA)
(which connects the anterior cerebral arteries)

Berry (Saccular) Aneurysm-

- Small, rounded outpouching at arterial bifurcations
- Caused by:
 - Congenital weakness of vessel wall (tunica media defect)
- Strongly associated with:
 - Hypertension
 - Polycystic kidney disease

B) Vertebral artery

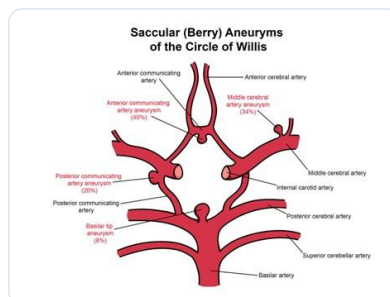
- Rare site for berry aneurysm
- More associated with:
 - Dissections
 - Posterior circulation strokes

C) Posterior cerebral artery

- Much less common
- PCA aneurysms are rare compared to ACoA/PCoM

D) Basilar artery

- Can have aneurysms (especially tip of basilar)
- But not the most common



QUESTION

A highly agitated 54-year-old man is brought to the emergency department by his family because he is unable to effectively communicate. He speaks clearly and with conviction but his sentences are incomprehensible. He does not appear to understand the doctor's questions, does not follow oral or written instructions, and cannot repeat simple phrases. Branch occlusion of which of the following arteries is most likely responsible for this patient's condition?

A Right Middle cerebral artery-superior division

B Right Middle cerebral artery-inferior division

C Left Middle cerebral artery-superior division

D Left Middle cerebral artery-inferior division

CORRECT

RIGHT ANSWER

OK

D. Left Middle cerebral artery-inferior division

WHY THIS IS RIGHT

Fluent but meaningless speech with poor comprehension and impaired repetition indicates Wernicke aphasia.

Patient often unaware of deficit.

- Wernicke area -> posterior superior temporal gyrus
- Dominant hemisphere (usually left)
- Vascular supply: Inferior division of left middle cerebral artery (MCA)

Why not other options:

- A. Right MCA - superior division: Non-dominant side -> neglect, visuospatial deficits; superior division -> Broca area, not Wernicke.
- B. Right MCA - inferior division: Causes neglect and visuospatial problems (non-dominant hemisphere), not aphasia.
- C. Left MCA - superior division: Affects Broca area -> nonfluent aphasia with intact comprehension, opposite of this case.

*

QUESTION

54-year-old man is brought to the emergency department by his wife after he develops difficulty speaking. When asked about the onset of his symptoms, the patient slowly responds with "weak... morning..." and becomes very frustrated. On examination, he is able to state his first name but with difficulty, and correctly points to different body parts on command. This patient's speech difficulties are most likely caused by a lesion affecting which of the following vessels?



A A

B E

C C

D D

CORRECT

RIGHT ANSWER

OK

D. D

WHY THIS IS RIGHT

Nonfluent, effortful broken speech with preserved comprehension with difficulty naming and repeating indicates Broca aphasia, caused by a lesion in the dominant inferior frontal gyrus.

- Vascular supply: Superior division of the middle cerebral artery (MCA) supplies Broca area. Lesion in dominant (usually left) MCA → Broca aphasia

Why not other vessels:

- A (Anterior cerebral artery): Supplies medial frontal/parietal lobes → leg weakness, behavioral changes, not aphasia.
- E (Basilar/vertebrobasilar): Brainstem signs (cranial nerve deficits, coma), not isolated speech problem.
- C (Right MCA): Non-dominant hemisphere → neglect, visuospatial deficits, not Broca aphasia.

QUESTION

Cortical homunculus representation of the body in the cerebrum is:

A Vertical

CORRECT

B Horizontal

C Oblique

D Sagittal

RIGHT ANSWER

OK

A. Vertical

WHY THIS IS RIGHT

The cortical homunculus represents somatotopic organization of the body on the primary motor and sensory cortex (precentral and postcentral gyri).

Orientation:

- Arranged vertically along the central sulcus
- From medial -> lateral:
- Lower limb (paracentral lobule)
- Trunk
- Upper limb
- Face and tongue (most lateral)
- Areas needing fine control (hands, lips) have larger cortical representation

QUESTION

A 70-year-old right-handed man is brought to the emergency department due to sudden onset of right-sided weakness and urinary incontinence that began about 10 hours ago. On examination, there is 4/5 strength in the right upper extremity, 1/5 strength in the right lower extremity, and a Babinski sign on the right side. Sensation is decreased throughout the right foot and leg. Which of the following is the most likely diagnosis of this patient?

A Left Anterior cerebral artery stroke

CORRECT

B Lacunar stroke

C Left middle cerebral artery stroke

D Right ACA stroke

RIGHT ANSWER

OK

A. Left Anterior cerebral artery stroke

WHY THIS IS RIGHT

The anterior cerebral artery (ACA) supplies the medial surface of frontal and parietal lobes, which control lower limb motor and sensory function and bladder control.

Key features of ACA stroke:

- Contralateral lower limb weakness > upper limb weakness
- Contralateral sensory loss in leg
- Urinary incontinence (medial frontal micturition center involvement)
- Behavioral changes/abulia (frontal lobe)

Differentiation:

- MCA stroke: Face/arm > leg weakness + aphasia/neglect
- Lacunar stroke: Pure motor/sensory without cortical signs
- Right ACA stroke: Would affect left side

QUESTION

A 65 year old patient has normal fluency and repetition of speech but impaired naming and comprehension. What is the likely diagnosis?

A Transcortical sensory aphasia

CORRECT

B Transcortical motor aphasia

C Conduction aphasia

D Wernicke aphasia

RIGHT ANSWER

OK A. Transcortical sensory aphasia

WHY THIS IS RIGHT

Transcortical sensory aphasia presents with:

- Fluent speech
- Impaired comprehension
- Impaired naming
- Preserved repetition (key distinguishing feature)

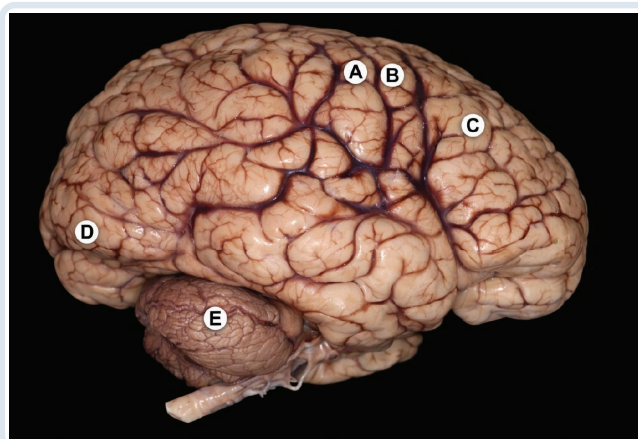
Lesion in temporoparietal junction or watershed area around Wernicke's area.

Differentiation:

- Wernicke aphasia: Poor comprehension + poor repetition
- Conduction aphasia: Poor repetition with relatively intact comprehension
- Transcortical motor aphasia: Nonfluent speech but preserved repetition

QUESTION

65-year-old man with a history of atrial fibrillation comes to the office due to numbness of his left hand for the past 3 weeks. When the eyes are closed, he is unable to recognize the letters written on his left hand with a stylus. Muscle strength is normal in all extremities. Deep tendon reflexes are 2+. Gait is normal. This patient most likely has a lesion in which of the following locations of the brain?



A A

CORRECT

B B

C C

D D

RIGHT ANSWER

OK A. A

WHY THIS IS RIGHT

Inability to recognize letters traced on the skin (agraphesthesia) with preserved strength and reflexes indicates a lesion in the contralateral parietal association cortex, specifically the primary somatosensory cortex/posterior parietal lobe.

Cortical sensory loss:

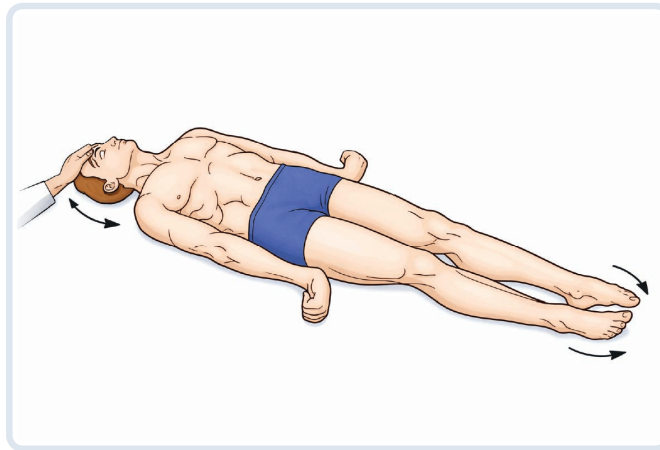
- Loss of higher sensory functions:
- Graphesthesia
- Stereognosis
- Two-point discrimination
- Basic sensations (pain, temperature, crude touch) may be preserved

Why not other options:

- B (Motor cortex - precentral gyrus): Would cause contralateral weakness or UMN signs. Motor exam here is normal -> ruled out.
- C (More posterior/occipital region): Causes visual deficits (hemianopia), not graphesthesia loss.
- D (Temporal lobe): Causes memory or language comprehension problems, not cortical sensory loss.

QUESTION

| The posture shown in image is seen in:



- A Upper pons hyperactivity
- B Upper midbrain hyperactivity
- C Upper pons damage**
- D Upper midbrain damage

CORRECT

RIGHT ANSWER

- OK
- C. Upper pons damage**

WHY THIS IS RIGHT

The posture shown is decerebrate rigidity (extension of upper and lower limbs, plantar flexion), which indicates a lesion at or below the level of the red nucleus, typically in the upper pons or lower midbrain.

Mechanism:

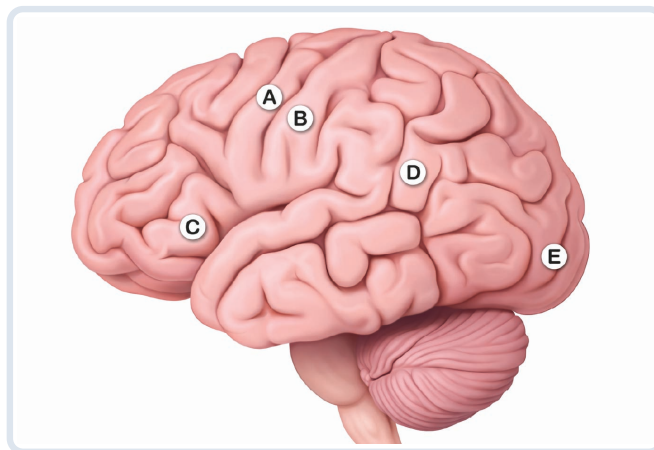
- Damage to corticospinal and rubrospinal tracts
- Unopposed activity of vestibulospinal and reticulospinal tracts -> extensor rigidity

Localization:

- Decerebrate posture: lesion in upper pons/lower midbrain
- Decorticate posture (flexed arms): lesion above red nucleus (cerebral hemispheres/internal capsule)

QUESTION

A 65-year-old man is brought to the emergency department due to an acute change in mental status. On physical examination, the patient is well oriented, answers questions appropriately, and has no motor, sensory, or visual deficits. He is unable to add simple numbers such as 12 plus 9; he is also unable to recognize his fingers. Which of the following brain areas is most likely affected in this patient?



A A

B B

C C

D D

CORRECT

RIGHT ANSWER

OK

D. D

WHY THIS IS RIGHT

Inability to perform calculations (acalculia), agraphia and inability to recognize fingers (finger agnosia) are classic features of Gerstmann syndrome, which localizes to the dominant inferior parietal lobule (angular gyrus) usually left. Patient may have normal motor, sensory, and vision but shows higher cortical dysfunction involving calculation and body schema.

Why not other options:

- A (Frontal lobe): Causes personality/executive dysfunction, not acalculia or finger agnosia.
- B (Motor cortex): Would cause contralateral weakness -> motor exam here is normal.
- C (Temporal lobe): Causes memory or language comprehension problems, not calculation/finger recognition defects.

QUESTION

A 62-year-old, right-handed man is evaluated for an episode of left leg weakness that spontaneously resolved within 30 minutes of onset. The patient also has had transient vision loss in the right eye. Medical history is significant for hypertension and diabetes mellitus. Evaluation reveals an atherosclerotic plaque in the extracranial portion of the supplying artery. During percutaneous stenting, the vascular catheter is inserted into the right common femoral artery and gradually advanced to the level of the aortic arch. Which of the following is the most likely path of the catheter before stenting of the culprit lesion can be performed?

- A** Aorta - brachiocephalic artery - common carotid artery - external carotid artery
- B** Aorta - brachiocephalic artery - common carotid artery - internal carotid artery **CORRECT**
- C** Aorta - common carotid artery - external carotid artery
- D** Aorta - common carotid artery - internal carotid artery

RIGHT ANSWER

- OK** **B. Aorta - brachiocephalic artery - common carotid artery - internal carotid artery**

WHY THIS IS RIGHT

Transient monocular vision loss (amaurosis fugax) and contralateral limb weakness suggest atherosclerotic disease of the internal carotid artery (ICA) supplying the retina and anterior cerebral circulation.

Anatomical pathway for right ICA stenting from femoral artery:

1. Femoral artery -> abdominal aorta -> thoracic aorta -> aortic arch
2. From arch -> brachiocephalic trunk (on right side)
3. Brachiocephalic trunk -> right common carotid artery
4. Common carotid -> right internal carotid artery (culprit vessel)

*

QUESTION

A patient presents with vertigo, diplopia, hoarseness, dysphagia and left Horner's syndrome associated with numbness of the left face and right-side limbs. Which artery is affected in this patient?

A Posterior inferior cerebellar artery

CORRECT

B Anterior inferior cerebellar artery

C Superior cerebellar artery

D Basilar artery

RIGHT ANSWER

OK

A. Posterior inferior cerebellar artery

WHY THIS IS RIGHT

This presentation describes lateral medullary (Wallenberg) syndrome, most commonly caused by infarction of the posterior inferior cerebellar artery (PICA).

- Vertigo, nystagmus -> vestibular nuclei
- Dysphagia, hoarseness -> nucleus ambiguus involvement
- Ipsilateral facial pain/temp loss -> spinal trigeminal nucleus
- Contralateral body pain/temp loss -> spinothalamic tract
- Ipsilateral Horner syndrome -> descending sympathetic fibers
- Ataxia -> inferior cerebellar peduncle

Nucleus ambiguus involvement (dysphagia, hoarseness) is hallmark of PICA (Wallenberg) syndrome and helps differentiate from AICA infarct.

QUESTION

All of the factors would suggest a higher risk for a patient with TIA developing stroke in the future according to ABCD2 scoring except?

A Age 65 years

B Duration of symptoms 5 mins

CORRECT

C BP 140/90mm Hg

D Diabetes

RIGHT ANSWER

OK

B. Duration of symptoms 5 mins

WHY THIS IS RIGHT

The ABCD₂ score is used to predict early stroke risk after a transient ischemic attack (TIA).

ABCD₂ components:

- A - Age ≥ 60 years -> 1 point
- B - Blood pressure $\geq 140/90$ mmHg -> 1 point

• C - Clinical features:

Unilateral weakness -> 2 points

Speech impairment (without weakness) -> 1 point

• D - Duration of symptoms:

≥ 60 min -> 2 points

10-59 min -> 1 point

< 10 min -> 0 points

• D - Diabetes mellitus -> 1 point

A duration of only 5 minutes does not increase ABCD₂ score, hence lower predicted stroke risk.

QUESTION

A patient presents with left-sided facial paralysis and weakness for the past 1 hour. Her blood pressure is 160/100 mmHg and CT is shown below. What would be your next step?



- A CT angiogram
- B Start on aspirin + clopidogrel
- C Intravenous thrombolysis**
- D IV Labetalol

CORRECT

RIGHT ANSWER

- OK
- C. Intravenous thrombolysis**

WHY THIS IS RIGHT

In acute ischemic stroke, if the patient presents within the therapeutic window (≤ 4.5 hours) and non-contrast CT shows no hemorrhage and BP $< 185/110$ mmHg, the next step is intravenous thrombolysis (IV alteplase/tenecteplase).

Why not other options:

- CT angiogram: Done after initiating thrombolysis or if planning thrombectomy
- Aspirin + clopidogrel: Given after thrombolysis or if not eligible
- IV labetalol: Only if BP $> 185/110$ before thrombolysis

QUESTION

| In isolated Gerstman's syndrome, The damage is in :

- A** Superior parietal lobule of the dominant hemisphere
- B** **Inferior parietal lobule of the dominant hemisphere** CORRECT
- C** Superior parietal lobule of the non-dominant hemisphere
- D** Inferior parietal lobule of the non-dominant hemisphere

RIGHT ANSWER

- OK** **B. Inferior parietal lobule of the dominant hemisphere**

WHY THIS IS RIGHT

Gerstmann syndrome results from a lesion in the inferior parietal lobule (angular gyrus) of the dominant hemisphere (usually left).

Classic tetrad:

- Agraphia
- Acalculia
- Finger agnosia
- Left-right disorientation

Exam tip:

- Dominant inferior parietal lesion -> Gerstmann syndrome
- Non-dominant parietal lesion -> hemineglect, constructional apraxia

QUESTION

A patient with a posterior cortical lesion is unable to perceive more than one object at a time, has difficulty accurately reaching for objects under visual guidance, and shows impaired voluntary eye movements despite normal ocular motility. This constellation of findings is best described as:

A Gerstmann syndrome

B Anton syndrome

C **Balint syndrome**

CORRECT

D Visual agnosia

RIGHT ANSWER

OK C. **Balint syndrome**

WHY THIS IS RIGHT

Balint syndrome results from bilateral parieto-occipital (posterior cortical) lesions and is characterized by a classic triad:

1. Simultanagnosia: Inability to perceive more than one object at a time
2. Optic ataxia: Difficulty reaching for objects using visual guidance
3. Oculomotor (ocular) apraxia: Impaired voluntary eye movements despite normal ocular motility

Exam differentiation:

- Gerstmann syndrome: Dominant parietal lesion -> acalculia, agraphia, finger agnosia, L-R disorientation
- Anton syndrome: Cortical blindness with denial of blindness
- Visual agnosia: Cannot recognize objects but vision intact; no optic ataxia or gaze apraxia

QUESTION

| Ablation of Brodmann area 1 leads to loss of:

A Pain and temperature sensation

B Crude touch sensation

C Proprioception

D Tactile discrimination and two-point discrimination

CORRECT

RIGHT ANSWER

OK D. Tactile discrimination and two-point discrimination

WHY THIS IS RIGHT

Brodmann areas 3, 1, and 2 form the primary somatosensory cortex in the postcentral gyrus, each with specialized sensory functions:

- Area 3: Main receiving area for pain, temperature, and crude touch
- Area 1: Responsible for tactile localization, texture perception, and two-point discrimination
- Area 2: Integrates size, shape, and proprioceptive information

QUESTION

A 9-month-old infant is brought to the pediatric clinic with a history of sudden, jerky movements of the limbs. EEG shows a chaotic high-amplitude background with multifocal spikes, consistent with hypsarrhythmia. What is the treatment of choice in this condition?

A Ethosuximide

B Valproate

C ACTH

CORRECT

D Lorazepam

RIGHT ANSWER

OK

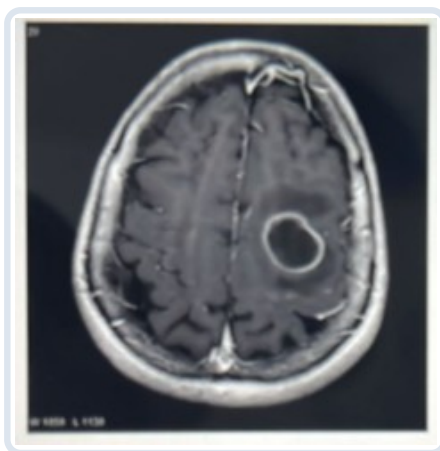
C. ACTH

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$19” here and does not include a written explanation for this item.

QUESTION

A 30-year-old patient presented with GTCS and was admitted. CECT is shown below. Which of the following can be useful in establishing the diagnosis?



A CBNAAT of CSF

B PET scan

C NMR spectroscopy

CORRECT

D B scan of both the eyes

RIGHT ANSWER

OK C. NMR spectroscopy

WHY THIS IS RIGHT

The CECT appearance with seizures suggests a space-occupying lesion where etiology is unclear (e.g., tumor vs infection).

NMR (MR) spectroscopy helps by analyzing metabolite patterns (↑ choline, ↓ NAA, lactate, lipid peaks).

Ring lesion detected

↓

Perform MR spectroscopy

↓

Analyze peaks:

-> Lipid/lactate ↑ -> TB / abscess

-> Amino acids ↑ -> Abscess

-> Choline ↑ -> Tumor

↓

Narrow diagnosis

This aids in characterizing lesion type and grade non-invasively.

| Condition | Key Peaks |

| ----- | ----- |

| Tuberculoma | Lipid peak ↑ |

| Abscess | Amino acids + lactate |

| Tumor | ↑ Choline, ↓ NAA |

| NCC | Variable (often less lipid than TB) |

CSF CBNAAT is for TB meningitis, PET is adjunctive, and B-scan eye exam is unrelated.

QUESTION

A 6-year-old boy is brought to the emergency department with a generalized tonic-clonic seizure after high grade fever. What is the most appropriate immediate treatment for this child?

A Diazepam

CORRECT

B Phenytoin

C Valproate

D Ethosuximide

RIGHT ANSWER

OK
A. Diazepam

WHY THIS IS RIGHT

This is a febrile seizure presenting as a generalized tonic-clonic seizure in a child with high-grade fever. The immediate management is benzodiazepine administration to abort the seizure.

Diazepam (IV/rectal) is the drug of choice for acute control.

Diazepam given

↓

Binds GABA-A receptor

↓

↑ Cl⁻ influx into neuron

↓

Hyperpolarization

↓

↓ Neuronal firing

↓

Seizure stops

Phenytoin, valproate, and ethosuximide are not used for immediate termination of febrile seizures.

| Drug | Role | Use |

| --- | --- | --- |

| Diazepam | □ First-line | Acute seizure |

| Phenytoin | Second-line | Status epilepticus |

| Valproate | Maintenance | Chronic seizures |

| Ethosuximide | Specific | Absence seizures |

Febrile Seizure-

| Feature | Finding |

| ----- | ----- |

| Age | 6 months-5 years |

| Type | Generalized |

| Prognosis | Good |

| Recurrence | Possible |

QUESTION

A 45-year-old patient with a history of depression was started on amitriptyline, a tricyclic antidepressant (TCA). The patient was later found unconscious at home and brought to the emergency department. On examination, the patient shows signs of arrhythmias, confusion, and pupillary dilation. Which of the following does NOT suggest TCA poisoning?

A Arrhythmias

B Confusion

C Hypothermia

CORRECT

D Pupillary dilation

RIGHT ANSWER

OK C. Hypothermia

WHY THIS IS RIGHT

TCA poisoning causes anticholinergic toxicity with confusion, mydriasis, and tachyarrhythmias due to sodium channel blockade.

Excess TCA

↓
Cardiac Na⁺ channel block

↓
Wide QRS → arrhythmias

↓
CNS toxicity

↓
Confusion / coma / seizures

↓
Anticholinergic effects

↓
Dilated pupils, dry skin, tachycardia

| Feature | TCA Poisoning |

| ----- | ----- |

| Cardiac | Arrhythmias (wide QRS) |

| CNS | Confusion, seizures |

| Pupils | Dilated |

| Temperature | Hyperthermia |

| Skin | Dry |

Cardiac effects include QRS prolongation and ventricular arrhythmias.

Patients typically develop hyperthermia, not hypothermia, due to anticholinergic effects.

Hence, hypothermia does not suggest TCA poisoning.

QUESTION

When comparing oxcarbazepine and carbamazepine, which of the following adverse effects is more commonly associated with oxcarbazepine?

- A Skin rash
- B Hepatitis with raised GGT
- C Hyponatremia**
- D Blood dyscrasias

CORRECT

RIGHT ANSWER

OK **C. Hyponatremia**

WHY THIS IS RIGHT

Oxcarbazepine is more commonly associated with hyponatremia due to SIADH-like effect.

Oxcarbazepine vs Carbamazepine

Both:

- Block voltage-gated Na⁺ channels
- Used in focal seizures

BUT they differ in side-effect.

Oxcarbazepine

↓

↑ ADH effect (SIADH-like)

↓

↑ Water reabsorption in kidney

↓

Dilution of sodium

↓

Hyponatremia

Clinical Consequence Flow-

↓ Serum sodium

↓

Cellular swelling (brain)

↓

Headache, confusion

↓

Seizures (paradoxical!)

| Advantage | Reason |

| ----- | ----- |

| Less enzyme induction | Fewer drug interactions |

| Safer hematologically | Less bone marrow suppression |

QUESTION

A patient with a mood disorder has been on medication and presents with new-onset liver dysfunction and hyperammonaemia. Which of the following medications is most likely responsible for these findings?

A Lamotrigine

B Carbamazepine

C Valproate

CORRECT

D Topiramate

RIGHT ANSWER

OK C. Valproate

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1a” here and does not include a written explanation for this item.

QUESTION

| Which of the following statements is true regarding fosphenytoin?

- A** At high doses in elderly patients, it causes cardiotoxicity and in younger patients' nephrotoxicity
- B** Adverse effects can be avoided if the dose is kept less than 250 mg phenytoin sodium equivalents
- C** It is given to all seizure patients in emergency irrespective of CNS adverse effects and hypotension
- D** High oral dose can result in cerebellar degeneration CORRECT

RIGHT ANSWER

- OK** **D. High oral dose can result in cerebellar degeneration**

WHY THIS IS RIGHT

Fosphenytoin is a water-soluble prodrug of phenytoin given IV or IM (no oral form). It is used acutely for status epilepticus or neurosurgical seizure prophylaxis.

Doses are expressed in "phenytoin equivalents" (PE): e.g. 75 mg fosphenytoin = 50 mg phenytoin. Typical IV loading is 15-20 mg PE/kg (e.g. 1000-1500 mg PE in adults) at a max infusion rate of 150 mg PE/min. Hence, Option B is wrong.

Option A and C: Its adverse effect profile mirrors phenytoin's: CNS (nystagmus, ataxia, sedation), and serious cardiovascular toxicity if infused too rapidly. Fosphenytoin can indeed cause serious cardiotoxicity in high doses, especially in elderly patients. The claim about nephrotoxicity in younger patients lacks evidence and is not a recognized phenomenon.

Cerebellar degeneration is associated with chronic phenytoin use, so that seems to be the best answer.

QUESTION

Which antiepileptic drug specifically enhances the inactivation phase of voltage-gated slow sodium channels, helping to stabilize hyperexcitable neurons?

A Phenytoin

B Lamotrigine

C Felbamate

D Lacosamide

CORRECT

RIGHT ANSWER

OK D. Lacosamide

WHY THIS IS RIGHT

Voltage-gated Na⁺ channels have 3 states:

1. Resting (closed)
2. Open (activated)
3. Inactivated

There are TWO types of inactivation:

- Fast inactivation (milliseconds)
- Slow inactivation (longer, stabilizing phase)

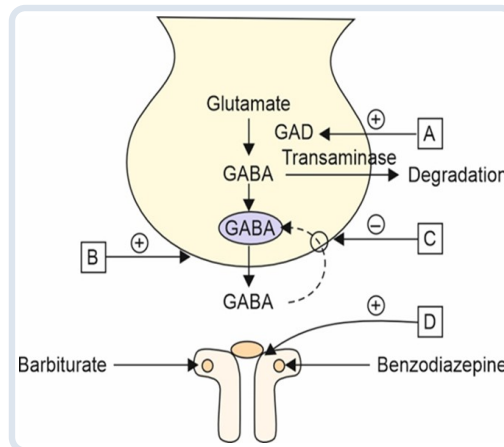
Lacosamide selectively enhances SLOW inactivation

- Keeps neurons in a non-excitabile state longer
- Prevents repetitive firing
- Stabilizes hyperexcitable neuronal membranes

Drug	Mechanism	Na ⁺ Channel Effect	
---	-----	-----	---
Lacosamide	Enhances slow inactivation	Keeps channels inactive longer	
Phenytoin	Blocks fast inactivation	Prevents rapid firing	
Lamotrigine	Fast inactivation + ↓ glutamate	Dual action	
Felbamate	NMDA antagonist	No Na ⁺ effect	

QUESTION

In the figure shown below, mechanism of action of valproate is best represented by:



A A

CORRECT

B B

C C

D D

RIGHT ANSWER

OK A. A

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1b" here and does not include a written explanation for this item.

QUESTION

Which of the following statements about barbiturates and benzodiazepines is correct?

A Both act on GABA chloride channels

CORRECT

B Flumazenil is antagonist of barbiturates

C Benzodiazepines increase the duration of chloride channel opening

D Barbiturates are used to treat acute anxiety

RIGHT ANSWER

OK A. Both act on GABA chloride channels

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1c" here and does not include a written explanation for this item.

QUESTION

A 28-year-old male with a known history of generalized tonic-clonic seizures is on long-term carbamazepine therapy. He recently developed a sore throat and was prescribed erythromycin by a local physician. Two days later, he presents to the emergency department with complaints of dizziness, diplopia, ataxia, and confusion. What is the most likely explanation for his symptoms?

- A** Refractory seizures due to pharmacodynamic resistance
- B** Carbamazepine toxicity due to drug interaction CORRECT
- C** Erythromycin toxicity due to enzyme induction
- D** Decreased absorption of carbamazepine leading to subtherapeutic levels

RIGHT ANSWER

- OK** **B. Carbamazepine toxicity due to drug interaction**

WHY THIS IS RIGHT

- Carbamazepine is metabolized in the liver by CYP3A4
- Erythromycin = strong CYP3A4 inhibitor

So, when erythromycin is added:

- ↓ metabolism of carbamazepine
- ↑ plasma levels
- -> toxicity

Features of Carbamazepine Toxicity

- Dizziness
- Diplopia
- Ataxia
- Nystagmus
- Confusion

| Option | Mechanism | Expected Effect | Why Correct/Incorrect | Key Exam Point |

| ----- | ----- | ----- | ----- | ----- |

| A | Resistance | ↑ seizures | ☐ No seizures here | Wrong clinical picture |

| B | CYP inhibition | ↑ CBZ levels | ☒ Correct | Classic interaction |

| C | Enzyme induction | ↓ drug levels | ☐ Wrong mechanism | Erythromycin = inhibitor |

| D | ↓ absorption | ↓ CBZ levels | ☐ Opposite effect | Would cause seizures |

QUESTION

Which of the following drugs is not given in status epilepticus?

A Levetiracetam

B Sodium valproate

C Ethosuximide

CORRECT

D Fosphenytoin

RIGHT ANSWER

OK

C. Ethosuximide

WHY THIS IS RIGHT

Status Epilepticus:

- Continuous seizure activity >5 minutes OR
- Recurrent seizures without regaining consciousness

It's a neurological emergency -> risk of:

- Neuronal injury
- Hypoxia
- Death

1\ Immediate suppression (fastest acting)

-> Benzodiazepines (IV Lorazepam)

2\ Stabilization (prevent recurrence)

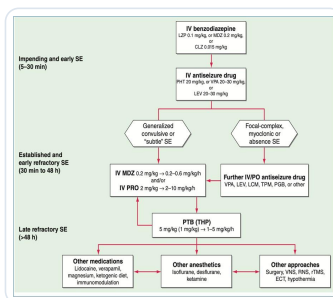
-> Longer-acting anticonvulsants

3\ Refractory phase

-> Anesthetics (propofol, midazolam infusion)

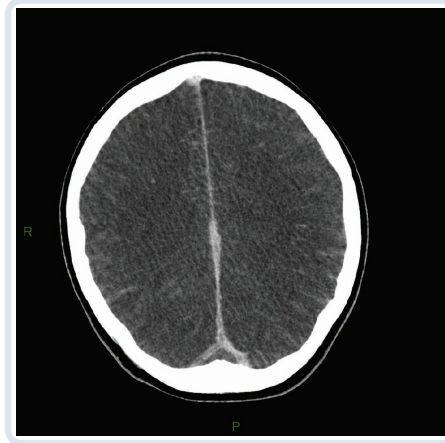
Ethosuximide Fails in SE

- Works on thalamic pacemaker activity
- SE involves diffuse cortical excitation
- Ethosuximide -> *ONLY absence seizures*
- Phenytoin/fosphenytoin -> NOT for absence seizures
- Valproate -> covers almost all seizure types



QUESTION

A 30-year-old lady who is 2 weeks postpartum presents with headache, and acute episode of generalized seizures. The contrast-enhanced CT of the head is shown below. What is the drug of choice for the acute management of her condition?



A IV Labetalol

B IV MgSo₄

C LMWH

CORRECT

D Aspirin

RIGHT ANSWER

OK

C. LMWH

WHY THIS IS RIGHT

Postpartum woman with headache and seizures plus CT showing venous infarction/hemorrhage suggests cerebral venous sinus thrombosis (CVST).

Key risk factors:

- Postpartum state
- Pregnancy
- Hypercoagulable states
- OCP use

Treatment of choice: Immediate anticoagulation with LMWH or heparin, even if hemorrhagic venous infarct is present. Prevents thrombus propagation and improves outcome

Differentiation:

- Eclampsia -> MgSO₄
- Ischemic stroke -> thrombolysis/antiplatelet
- CVST -> anticoagulation

QUESTION

| True about electroencephalogram (EEG) is all except:

A 10% of the normal population can have epileptiform discharges

CORRECT

B Scalp EEG may be helpful in localizing frontal lobe epilepsy

C Doing EEG is not mandatory for the diagnosis of seizures

D Progressive multifocal leukoencephalopathy shows triphasic and slow waves

RIGHT ANSWER

OK

A. 10% of the normal population can have epileptiform discharges

WHY THIS IS RIGHT

Electroencephalogram (EEG) supports seizure diagnosis but is not mandatory, as epilepsy is primarily a clinical diagnosis.

Key EEG facts:

- Only ~1-2% of normal individuals may show epileptiform discharges -> not 10%
- EEG can help localize seizure focus, including frontal lobe epilepsy (though sometimes limited)
- Normal EEG does not exclude epilepsy
- EEG is supportive, not essential, for diagnosis
- Waveform associations:
 - Triphasic waves: Classically seen in metabolic encephalopathy (especially hepatic)
 - PML: Shows nonspecific focal slowing, not typical triphasic waves

*

QUESTION

A 5-year-old boy is brought to the emergency department due to recurrent, generalized tonic-clonic seizures over the past 24 hours. He takes no medications and has no family history of epilepsy. The patient's temperature is 39.4 °C (103 °F), blood pressure is 110/70 mm Hg, pulse is 112/min, and respirations are 10/min. During the examination, the patient suddenly develops sustained, generalized tonic-clonic convulsions without fully regaining consciousness between episodes. Which of the following describes the mechanism of action of the most appropriate initial therapy for his seizures?

A Blockade of presynaptic voltage-gated calcium channels

B Blockade of presynaptic voltage-gated sodium channels

C Enhanced postsynaptic chloride influx

CORRECT

D Inhibition of vesicle fusion and neurotransmitter release

RIGHT ANSWER

OK

C. Enhanced postsynaptic chloride influx

WHY THIS IS RIGHT

This child has status epilepticus (continuous seizures without regaining consciousness). The initial treatment is a benzodiazepine (IV lorazepam/diazepam or IM midazolam).

Mechanism of action of benzodiazepines:

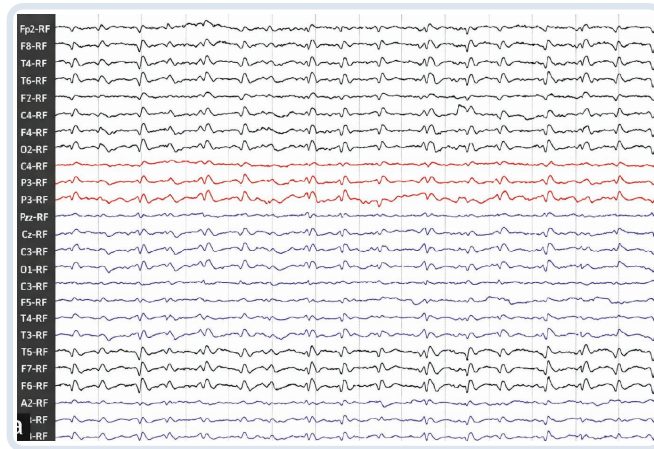
- Bind to GABA-A receptors
- Increase frequency of chloride channel opening
- Cause enhanced postsynaptic Cl^- influx $\rightarrow$ neuronal hyperpolarization $\rightarrow$ rapid seizure termination

Stepwise management of status epilepticus:

1. First-line: Benzodiazepines $\rightarrow$ enhance GABA-mediated Cl^- influx
2. Second-line: Phenytoin/fosphenytoin, valproate, levetiracetam
3. Refractory: Phenobarbital, propofol, midazolam infusion

QUESTION

A 56 year old male presented with rapidly progressive behavioural changes. EEG pattern given below is suggestive of which of the following diseases?



- A** Hepatic encephalopathy
- B** Generalized tonic clonic seizures
- C** Creutzfeldt-Jakob Disease
- D** Herpes simplex encephalitis

CORRECT

RIGHT ANSWER

- OK** C. Creutzfeldt-Jakob Disease

WHY THIS IS RIGHT

Rapidly progressive dementia with behavioral changes, myoclonus, ataxia and characteristic EEG showing periodic biphasic/triphasic sharp wave complexes at regular intervals is highly suggestive of Creutzfeldt-Jakob disease (CJD), a prion-mediated neurodegenerative disorder.

Differentiation:

- Hepatic encephalopathy: Triphasic waves but clinical liver failure
- Generalized seizures: Spike-and-wave discharges
- HSV encephalitis: Temporal lobe periodic discharges, fever, altered sensorium

QUESTION

A patient in convulsive status epilepticus continues to seize despite adequate benzodiazepine therapy.
The next drug of choice is:

A Levetiracetam

B Phenytoin / Fosphenytoin

CORRECT

C Phenobarbital

D Propofol

RIGHT ANSWER

OK

B. Phenytoin / Fosphenytoin

WHY THIS IS RIGHT

Management of convulsive status epilepticus follows a stepwise protocol:

1. First-line: Rapid-acting benzodiazepine
 - IV lorazepam, diazepam, or IM midazolam
2. Second-line (if seizures persist):
 - IV phenytoin or fosphenytoin -> stabilizes neuronal membranes by blocking voltage-gated Na⁺ channels
3. Third-line (refractory status):
 - Phenobarbital, levetiracetam, valproate
4. Super-refractory:
 - General anesthesia (propofol, midazolam infusion)

*

QUESTION

A 62-year-old man with small cell lung carcinoma presents with confusion and generalized tonic-clonic seizures. Laboratory investigations reveal hyponatremia, low serum osmolality, inappropriately concentrated urine, and euvolemic status. He requires long-term antiepileptic therapy.

Which of the following antiepileptic drugs is most appropriate in this patient?

A Carbamazepine

B Oxcarbazepine

C Phenytoin

D Phenobarbital

CORRECT

RIGHT ANSWER

OK

D. Phenobarbital

WHY THIS IS RIGHT

In patients with SIADH-related hyponatremia (classically seen with small cell lung carcinoma), antiepileptic drugs that worsen hyponatremia must be avoided.

- Carbamazepine and oxcarbazepine can induce or worsen SIADH, leading to further hyponatremia -> contraindicated
- Phenytoin may reduce ADH release but is not preferred for long-term control in this setting
- Phenobarbital does not worsen hyponatremia and is safer for long-term seizure control in SIADH

QUESTION

Match List I (Drugs) with List II (Primary mechanism of action)**List I - Drugs**

- A. Ethosuximide
- B. Levetiracetam
- C. Lacosamide
- D. Vigabatrin
- E. Perampanel

List II - Mechanism

- 1. Inhibition of synaptic vesicle protein involved in neurotransmitter release
- 2. Irreversible inhibition of GABA transaminase
- 3. Blockade of slow inactivation of voltage-gated sodium channels
- 4. Non-competitive AMPA receptor antagonism
- 5. Blockade of thalamic pacemaker currents

Choose the correct matching:

A A-5, B-1, C-3, D-2, E-4

CORRECT

B A-3, B-1, C-5, D-2, E-4

C A-5, B-3, C-1, D-2, E-4

D A-1, B-5, C-3, D-4, E-2

RIGHT ANSWER

OK A. A-5, B-1, C-3, D-2, E-4

WHY THIS IS RIGHT

Antiepileptic drugs are frequently tested by their unique primary mechanisms of action, which target ion channels, neurotransmitter release, or excitatory/inhibitory pathways.

- Ethosuximide: Blocks T-type Ca^{2+} (thalamic pacemaker) currents -> drug of choice for absence seizures
- Levetiracetam: Inhibits synaptic vesicle protein SV2A -> ↓ neurotransmitter release
- Lacosamide: Enhances slow inactivation of voltage-gated Na^{+} channels
- Vigabatrin: Irreversibly inhibits GABA transaminase -> ↑ GABA levels
- Perampanel: Non-competitive AMPA receptor antagonist -> ↓ glutamate-mediated excitation

QUESTION

Which of the following is a characteristic feature of an upper motor neuron (UMN) lesion?

A Muscle fasciculations

B Flaccid paralysis

C Hyporeflexia

D Brisk reflexes

CORRECT

RIGHT ANSWER

OK

D. Brisk reflexes

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$19” here and does not include a written explanation for this item.

QUESTION

A male patient presents with sensory loss and weakness of limbs for 3 months. He also has angular stomatitis. On examination, there is loss of proprioception, vibration sensations, UMN type of lower limb weakness, and absent ankle reflex. What is the most probable diagnosis?

A Extradural cord compression

B Amyotrophic lateral sclerosis

C Multiple sclerosis

D Subacute combined degeneration of the cord

CORRECT

RIGHT ANSWER

OK

D. Subacute combined degeneration of the cord

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1a” here and does not include a written explanation for this item.

QUESTION

A patient met with a road traffic accident and developed a cervical spine injury. The fracture fragment had pierced the lateral aspect of the dorsal column tract. Which of the following findings is seen in this patient?

- A** Absence of ipsilateral lower limb proprioception
- B** Absence of fine motor movement of fingers
- C** Absence of ipsilateral arm proprioception
- D** Absence of contralateral lower limb proprioception

CORRECT

RIGHT ANSWER

- OK** **C. Absence of ipsilateral arm proprioception**

WHY THIS IS RIGHT

Dorsal column carries-

- Fine touch
- Vibration
- Proprioception

And it ascends ipsilaterally (no crossing in spinal cord)

At cervical level:

Region	Tract	Position
Lower limb	Fasciculus gracilis	Medial
Upper limb	Fasciculus cuneatus	Lateral

"Cuneatus = Cervical (upper limb)"

"Gracilis = Ground (lower limb)"

The lateral part of the dorsal column at cervical level corresponds to the cuneate fasciculus, which carries sensory input from the upper limb.

Dorsal column fibers ascend ipsilaterally without crossing in the spinal cord.

Thus, injury causes loss of ipsilateral arm proprioception and vibration sense.

Lower limb proprioception is carried medially by the gracile fasciculus and is spared here.

QUESTION

A 40-year-old man had a RTA and was admitted to a hospital. After a few days, he had an exaggerated right-sided extensor plantar reflex and knee jerk. What other finding would be seen?

- A Atrophy of the right lower limb
- B Loss of proprioception in the left leg
- C Ankle clonus in the right foot**
- D Fasciculations in the right foot

CORRECT

RIGHT ANSWER

- OK C. Ankle clonus in the right foot**

WHY THIS IS RIGHT

Exaggerated knee jerk and extensor plantar response indicate an upper motor neuron (UMN) lesion on the right side.

Finding	Interpretation
Exaggerated knee jerk	Hyperreflexia
Extensor plantar reflex	Babinski sign
Same side (right)	Localized lesion

UMN lesions are associated with spasticity and clonus due to loss of inhibitory control.

Sudden stretch (ankle dorsiflexion)

↓

Exaggerated reflex contraction

↓

Repeated oscillations

↓

Clonus

Hence, ankle clonus in the right foot is an expected finding.

Feature	UMN Lesion	LMN Lesion
Reflexes	↑	↓
Babinski	□	□
Clonus	□	□
Fasciculations	□	□
Atrophy	Mild	Severe

Atrophy and fasciculations are LMN signs, and proprioceptive loss suggests dorsal column involvement.

QUESTION

A 30-year-old female notices weakness in her right hand and difficulty while kneading dough. On examination, right arm tone and power are decreased, and reflexes are sluggish. What is the site of the lesion?

- A Posterior limb of the internal capsule
- B Right corticospinal tract in cervical cord
- C Anterior horn cell of the cervical spinal cord**
- D Left medullary pyramid

CORRECT

RIGHT ANSWER

- OK C. Anterior horn cell of the cervical spinal cord**

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1b” here and does not include a written explanation for this item.

QUESTION

A patient with Type 2 Diabetes Mellitus complains of numbness and burning sensations in the extremities. On examination he has absent ankle reflexes and has no sensation to touch, vibration, or pain in his lower limbs. The loss of sensation in his extremities has a stocking distribution. What type of neuropathy is associated?

- A Autonomic neuropathy
- B Mononeuropathy
- C Symmetric distal polyneuropathy**
- D Proximal motor neuropathy

CORRECT

RIGHT ANSWER

- OK C. Symmetric distal polyneuropathy**

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1c” here and does not include a written explanation for this item.

QUESTION

| Which of the following is seen in UMN (Upper Motor Neuron) weakness?

A Hyper-reflexia

CORRECT

B Atrophy

C Rigidity

D Fasciculations

RIGHT ANSWER

OK

A. Hyper-reflexia

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1d" here and does not include a written explanation for this item.

QUESTION

A 65-year-old man presented to you with neuropathy in his bilateral lower limbs and a positive Romberg sign. He has a history of gastrectomy for stomach cancer. He is likely deficient in which of the following?

A Thiamine deficiency

B Folate deficiency

C Vitamin B12 deficiency

CORRECT

D Transferrin deficiency

RIGHT ANSWER

OK

C. Vitamin B12 deficiency

WHY THIS IS RIGHT

- Bilateral lower limb neuropathy
- Positive Romberg sign
- History of gastrectomy

These three together form a diagnostic triangle pointing strongly to Vitamin B12 deficiency

Gastrectomy

↓

Loss of parietal cells

↓

↓ Intrinsic factor

↓

↓ B12 absorption (terminal ileum)

↓

Vitamin B12 deficiency

QUESTION

LMN (Lower Motor Neuron) lesion is associated with which one of the following?

1. Reduced muscle tone
2. Spastic paralysis
3. Babinski signs positive
4. Fasciculation

A 1, 2

B 1, 2, 3

C 1, 2, 3, 4

D 1, 4

CORRECT

RIGHT ANSWER

OK

D. 1, 4

WHY THIS IS RIGHT

Lower Motor Neuron (LMN)

LMNs are the final common pathway:

- From anterior horn cell -> peripheral nerve -> muscle

If this pathway is damaged -> muscle loses direct neural input

After LMN injury-

- Muscle becomes denervated
- No trophic support -> atrophy
- Spontaneous discharges -> fasciculations
- Loss of stretch reflex -> hypotonia

| Feature | LMN Lesion | UMN Lesion |

| ----- | ----- | ----- |

| Tone | ↓ Hypotonia | ↑ Hypertonia |

| Paralysis | Flaccid | Spastic |

| Reflexes | ↓ / absent | ↑ exaggerated |

| Babinski | ☐ Absent | ☐ Present |

| Muscle bulk | Atrophy (early) | Mild atrophy (late) |

| Fasciculations | ☐ Present | ☐ Absent |

QUESTION

A patient presented with worsening back pain and weakness in the lower limbs. Which of the following would not support the diagnosis of compressive myelopathy?

- A** Positive Babinski sign
- B** Normal cremasteric reflex
- C** Hypotonia
- D** Knee clonus

CORRECT

RIGHT ANSWER

C. Hypotonia

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1e” here and does not include a written explanation for this item.

QUESTION

A 52-year-old male presents with progressive weakness in the lower limbs, difficulty in walking, and tingling sensations in hands and feet. On examination, there is spasticity, increased deep tendon reflexes, along with impaired vibration and position sense. Which of the following best explains this presentation?

A ALS

B Vitamin B12 deficiency

CORRECT

C Vit E deficiency

D Polio

RIGHT ANSWER

OK

B. Vitamin B12 deficiency

WHY THIS IS RIGHT

Vitamin B12 (cobalamin) deficiency causes a combination of hematological and neurological manifestations, making it a key exam diagnosis when both are present.

- B12 is required for:

- o DNA synthesis -> deficiency causes megaloblastic anemia (pallor)
- o Myelin synthesis -> deficiency leads to demyelination of the spinal cord

Neurological features (important clue):

- Subacute combined degeneration of spinal cord
- o Involves posterior columns -> loss of vibration & position sense
- o Involves lateral corticospinal tracts -> UMN signs (spasticity, hyperreflexia)
- Peripheral neuropathy -> paresthesia, numbness

Why other options are incorrect:

- Vitamin B9 (Folate): Causes megaloblastic anemia without neurological symptoms
- Vitamin B3 (Niacin): Pellagra (dermatitis, diarrhea, dementia)
- Vitamin B1 (Thiamine): Beriberi/Wernicke's (neuropathy, cardiac/brain involvement, but no UMN pattern)

QUESTION

A 35-year-old female patient presents with progressive limb weakness for the past 1 year. On examination, muscle fasciculations and hypertonia are noted, and the sensory system is intact. What is the diagnosis?

A Becker muscular dystrophy

B Multifocal motor neuropathy

C Spinal muscular atrophy

D Amyotrophic Lateral Sclerosis

CORRECT

RIGHT ANSWER

OK

D. Amyotrophic Lateral Sclerosis

WHY THIS IS RIGHT

Feature	Interpretation
-----	-----
Progressive weakness	Neurodegenerative
Fasciculations	LMN involvement
Hypertonia	UMN involvement
Intact sensation	Motor-only disease

Feature	ALS	SMA	MMN	Becker MD
-----	---	---	---	-----
UMN signs	☐	☐	☐	☐
LMN signs	☐	☐	☐	☐
Fasciculations	☐	☐	☐	☐
Sensory loss	☐	☐	☐	☐
Nature	Neurodegenerative	Genetic	Autoimmune	Myopathy

ALS Hallmark Triad

- Mixed UMN + LMN signs
- No sensory loss
- Progressive course

QUESTION

An elderly man presents with rigidity and tremors. On examination, he has blank facial expressions. Which of the following drugs can be used to manage this condition?

A Clozapine

B Donepezil

C Selegiline

CORRECT

D Haloperidol

RIGHT ANSWER

OK C. Selegiline

WHY THIS IS RIGHT

↓ Dopamine
↓
Indirect pathway becomes overactive
↓
↑ Inhibition of thalamus
↓
↓ Motor cortex stimulation
↓
Bradykinesia + rigidity

Parkinson = dopamine deficiency -> treat by increasing dopamine

Selegiline

- Selectively inhibits MAO-B
- Prevents dopamine breakdown
- -> ↑ Dopamine availability in brain

Drug	Mechanism	Use	Effect in Parkinson
Selegiline	MAO-B inhibitor	Parkinson	Improves symptoms
Clozapine	D2 blockade (weak)	Schizophrenia	Limited role
Donepezil	AChE inhibitor	Alzheimer	No benefit
Haloperidol	Strong D2 blocker	Psychosis	Worsens symptoms

QUESTION

A 65-year-old man suffered from a stroke 2 days ago. He now presents with involuntary, violent, and flinging movements of the limbs on one side. What is the likely site of lesion in this patient?

A Subthalamic nuclei

CORRECT

B Globus pallidus

C Putamen

D Caudate nucleus

RIGHT ANSWER

OK

A. Subthalamic nuclei

WHY THIS IS RIGHT

Subthalamic nucleus lesion

↓

↓ Excitation of Globus Pallidus Interna (GPi)

↓

↓ Inhibition of Thalamus

↓

↑ Thalamic activity

↓

↑ Cortical motor output

↓

Violent, excessive movements (Hemiballismus)

| Structure | Lesion Effect | Movement Type | Classic Disease |

| ----- | ----- | ----- | ----- |

| Subthalamic nucleus | ↓ Inhibition | Hemiballismus (flinging) | Stroke |

| Globus pallidus | Motor modulation | Rigidity / dystonia | Parkinsonism variants |

| Putamen | Motor planning | Chorea / athetosis | Huntington |

| Caudate nucleus | Cognitive + motor | Chorea + psychiatric | Huntington |

| Type | Example | Pathology |

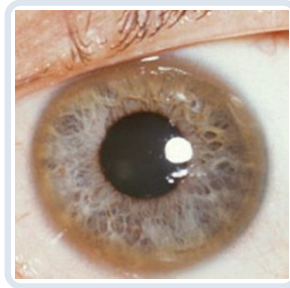
| --- | ----- | ----- |

| Hyperkinetic | Hemiballismus, chorea | ↓ Inhibition |

| Hypokinetic | Parkinson disease | ↑ Inhibition |

QUESTION

A patient had a history of hepatic dysfunction, and the physical examination finding of the eye is shown below. Which of the following is the best way for confirmation of the diagnosis in this patient?



- A Serum copper
- B Urinary copper
- C Liver copper levels**
- D Serum ceruloplasmin

CORRECT

RIGHT ANSWER

- OK
- C. Liver copper levels**

WHY THIS IS RIGHT

ATP7B mutation
↓
↓ Copper excretion in bile
↓
Copper accumulates in liver
↓
Spills into blood
↓
Deposits in brain + cornea
↓
KF ring + neurological signs

The eye finding is Kayser-Fleischer ring, suggesting Wilson disease with hepatic involvement. The gold standard for confirmation is quantitative liver copper estimation on biopsy. Serum ceruloplasmin may be low but is not definitive, and serum copper can be misleading.

Feature	Interpretation
-----	-----
Hepatic dysfunction	Liver involvement
Eye finding	Kayser-Fleischer (KF) ring
Young patient	Typical age group

Urinary copper is supportive, but liver copper levels provide definitive diagnosis.

Drug	Action
----	-----
Penicillamine	Copper chelation
Zinc	↓ absorption

QUESTION

A patient is presented with a staggering gait and nystagmus after a road traffic accident. Which lobe of cerebellum is affected?

A Flocculonodular

CORRECT

B Dentate

C Anterior lobe

D Vermis

RIGHT ANSWER

OK A. Flocculonodular

WHY THIS IS RIGHT

Staggering gait with nystagmus indicates involvement of the vestibulocerebellum.
The flocculonodular lobe controls balance, posture, and eye movements via vestibular connections.

Lesions cause truncal ataxia, vertigo, and nystagmus with a positive Romberg sign.
Anterior lobe causes limb ataxia, and vermis affects axial posture without prominent eye signs.

Structure	Function	Lesion Feature
-----	-----	-----
Flocculonodular	Balance + eye movement	☒ Ataxia + nystagmus
Anterior lobe	Posture	Gait disturbance
Posterior lobe	Fine movement	Limb ataxia
Vermis	Trunk control	Truncal instability

QUESTION

Which of the following drugs are used in treatment of acute mania?

1. Haloperidol
2. Valproate
3. Lithium
4. Amitriptyline

A Only 3

B 1, 2 & 3

CORRECT

C 1, 2, 3 & 4

D 2, 3 & 4

RIGHT ANSWER

OK

B. 1, 2 & 3

WHY THIS IS RIGHT

Mania is a state of:

- ↑ Dopamine
- ↑ Norepinephrine
- ↓ inhibitory control

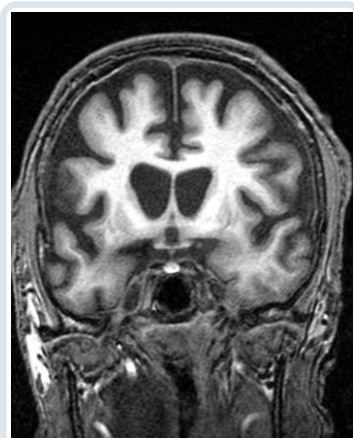
Treatment Strategy

1. Immediate control (hours-days)
-> Antipsychotics (block dopamine)
2. Stabilization (days-weeks)
-> Mood stabilizers (lithium, valproate)

Drug Class	Examples	Role in Mania	Key Feature
Antipsychotics	Haloperidol, Olanzapine	Acute control	Fast acting
Mood stabilizers	Lithium, Valproate	Acute + maintenance	Prevent relapse
Benzodiazepines	Lorazepam	Adjunct	Sedation
Antidepressants	Amitriptyline	□ Avoid	Trigger mania

QUESTION

A 28-year-old man presents with involuntary, purposeless, dance-like movements involving both upper and lower limbs. He has also become aggressive and irritable over the past few months. MRI brain is shown. What is the most likely diagnosis?



A Huntington chorea

CORRECT

B Sydenham chorea

C Tourette syndrome

D Tardive dyskinesia

RIGHT ANSWER

OK

A. Huntington chorea

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1f" here and does not include a written explanation for this item.

QUESTION

A 60-year-old woman with metastatic cancer reports moderate bone pain despite taking paracetamol and ibuprofen regularly. What is the next appropriate step according to the WHO analgesic ladder for chronic pain in adults?

CORRECT

A Start tramadol and continue NSAIDs

B Increase NSAID dose

C Start morphine and continue NSAIDs

D Consider non-pharmacological options

RIGHT ANSWER

OK

A. Start tramadol and continue NSAIDs

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$20” here and does not include a written explanation for this item.

QUESTION

| What is the first line therapy for aborting the acute attack of cluster headache?

A 100% oxygen

CORRECT

B Sumatriptan

C Lithium

D Topiramate

RIGHT ANSWER

OK A. 100% oxygen

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$21” here and does not include a written explanation for this item.

QUESTION

A female patient presents to you with a unilateral headache. It is associated with nausea, photophobia, and phonophobia. What is the best drug for acute management?

A Flunarizine

B Sumatriptan

CORRECT

C Propranolol

D Topiramate

RIGHT ANSWER

OK

B. Sumatriptan

WHY THIS IS RIGHT

This presentation is classic migraine with unilateral headache, nausea, photophobia, and phonophobia.

Trigger

↓

Trigeminal nerve activation

↓

CGRP release

↓

Vasodilation + inflammation

↓

Migraine pain

Sumatriptan is the drug of choice for acute abortive treatment, acting as a 5-HT_{1B/1D} agonist. It causes cranial vasoconstriction and inhibits trigeminal neuropeptide release.

| Drug | Role | Use |

| --- | --- | --- |

| Sumatriptan | Abortive | Acute migraine |

| Flunarizine | Prophylaxis | Prevention |

| Propranolol | Prophylaxis | Prevention |

| Topiramate | Prophylaxis | Prevention |

QUESTION

An 80-year-old woman presents with a progressive decline in daily activity. She gives a history of staring into space with frequent visual hallucinations. Pathology reveals Lewy bodies in neurons. What is the most likely diagnosis?

A Parkinson's Disease

CORRECT

B Prion's Disease

C Huntington's Disease

D Alzheimer's Disease

RIGHT ANSWER

OK **A. Parkinson's Disease**

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$22" here and does not include a written explanation for this item.

QUESTION

| Which of the following is a marker of Alzheimer disease?

- A Alpha synuclein
- B Amyloid beta plaque**
- C Glial fibrillary associated protein
- D Desmin

CORRECT

RIGHT ANSWER

- OK **B. Amyloid beta plaque**

WHY THIS IS RIGHT

Alzheimer disease is characterized by extracellular amyloid- β plaque deposition in the cerebral cortex. Two pathological signatures:

1. Amyloid beta ($A\beta$) plaques -> extracellular
2. Neurofibrillary tangles (tau protein) -> intracellular

These plaques are derived from abnormal processing of amyloid precursor protein.

Amyloid precursor protein (APP)

↓ (β & γ secretase)

Amyloid β accumulation

↓

Extracellular plaques

↓

Neurotoxicity + synaptic dysfunction

↓

Cognitive decline

α -Synuclein is seen in Parkinson disease, GFAP marks astrocytes, and desmin is a muscle intermediate filament.

QUESTION

A 25-year-old female patient with diagnosed recurrent migraine despite Sumatriptan, has 3-5 episodes/week and presents with features of anxiety and palpitations. There is no clinical neck swelling, no history of coronary artery disease; however, there is a family history of CHD. She reports experiencing chest tightness after taking the medication. What is the most appropriate agent to add for migraine prophylaxis?

A Topiramate

B Propranolol

CORRECT

C Naratriptan

D Ergotamine

RIGHT ANSWER

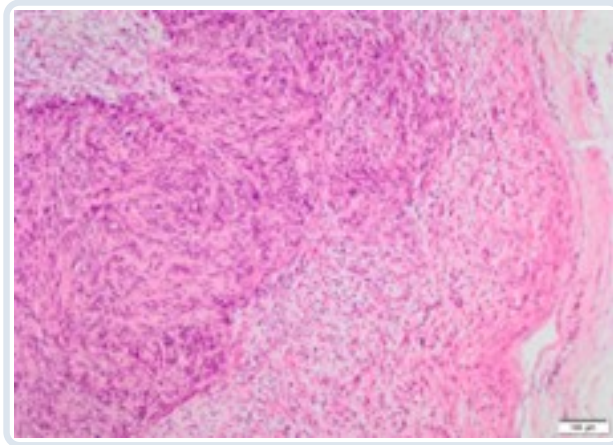
OK B. Propranolol

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$23” here and does not include a written explanation for this item.

QUESTION

A 45-year-old man with abnormal gait and hearing problems presents with a cerebellopontine mass. Histopathological examination is shown below. What is the most likely diagnosis?



A Schwannoma

CORRECT

B Glioblastoma

C Oligodendroglioma

D Medulloblastoma

RIGHT ANSWER

OK A. Schwannoma

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$24" here and does not include a written explanation for this item.

QUESTION

A 72-year-old patient with a confirmed diagnosis of mild to moderate Alzheimer's dementia has shown aversion and non-compliance to oral medications. The patient's caregiver reports difficulty in administering the oral drugs due to the patient's refusal to swallow pills. Considering the need for continued treatment, which of the following medications could be prescribed in a form that does not require oral administration?

A Memantine

B Donepezil

C Rivastigmine

CORRECT

D Galantamine

RIGHT ANSWER

OK C. Rivastigmine

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$25” here and does not include a written explanation for this item.

QUESTION

Which of the following is NOT indicative of underlying serious pathology in headache?

- A Age > 50
- B Temporal tenderness
- C Chronic Cocaine use
- D Subacute progression over 4 weeks

CORRECT

RIGHT ANSWER

OK
C. Chronic Cocaine use

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$26" here and does not include a written explanation for this item.

QUESTION

A 20-year-old male presents with sudden onset severe headache and vomiting. On examination, he has neck stiffness. A CT scan of the brain shows hydrocephalus. What is the most likely diagnosis?

A Normal Pressure Hydrocephalus

B Bacterial Meningitis

C Viral Encephalitis

D Subarachnoid Haemorrhage

CORRECT

RIGHT ANSWER

OK

D. Subarachnoid Haemorrhage

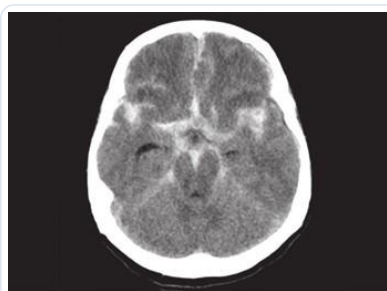
WHY THIS IS RIGHT

Berry aneurysm rupture
↓
Blood enters subarachnoid space
↓
Meningeal irritation -> Neck stiffness
↓
CSF absorption blocked
↓
Hydrocephalus
↓
↑ Intracranial pressure -> Headache + vomiting

Clinical Features of SAH

Feature	Explanation
Thunderclap headache	Sudden ↑ ICP
Vomiting	Brainstem/ICP response
Neck stiffness	Meningeal irritation
Photophobia	Meningeal involvement
Loss of consciousness	Severe cases

Location	%
Anterior communicating artery	Most common
Posterior communicating artery	2nd
MCA bifurcation	Also, common



QUESTION

Which of the following statements regarding Rimegepant is true?

1. It is a partial agonist of the CGRP receptor
2. It causes vasoconstriction
3. It is given via the subcutaneous route
4. It is used in the acute management of migraine

A 1, 2 and 3

B 2 and 3

C 4 only

CORRECT

D 1, 3 and 4

RIGHT ANSWER

OK C. 4 only

WHY THIS IS RIGHT

Rimegepant:

Rimegepant is a “gepant” class drug:

- A CGRP (Calcitonin Gene-Related Peptide) receptor antagonist

During migraine:

- CGRP released from trigeminal nerve endings

-> Causes:

- Vasodilation
- Neurogenic inflammation
- Pain transmission

| Feature | Rimegepant | Triptans (e.g., Sumatriptan) | CGRP mAbs |

| ----- | ----- | ----- | ----- |

| Mechanism | CGRP receptor antagonist | 5-HT_{1B/1D} agonist | CGRP ligand/receptor block |

| Vasoconstriction | ☐ No | ☐ Yes | ☐ No |

| Route | Oral | Oral / SC / nasal | SC / IV |

| Use | Acute ± preventive | Acute | Preventive |

| CV safety | Safe | Contraindicated | Safe |

QUESTION

A 30-year-old female presents with headache, fever, and nuchal rigidity. Lumbar puncture was done, and CSF analysis shows abundant lymphocytes with normal glucose and elevated proteins. What is the diagnosis?

A Acute bacterial meningitis

B Fungal meningitis

C Tubercular meningitis

D Viral meningitis

CORRECT

RIGHT ANSWER

OK

D. Viral meningitis

WHY THIS IS RIGHT

Finding	Interpretation
Fever + headache + neck stiffness	Meningitis
Lymphocytic predominance	Viral / TB / fungal
Normal glucose	Viral
↑ Protein	Mild inflammation

Correct answer is D because viral meningitis classically shows lymphocytic pleocytosis with normal glucose and mildly elevated protein in CSF.

Bacterial meningitis shows neutrophils with low glucose. Tubercular and fungal meningitis usually show lymphocytes but with low glucose and very high protein.

Thus, normal glucose with lymphocyte predominance favors viral meningitis.

Feature	Bacterial	Viral	TB	Fungal
Cells	Neutrophils	Lymphocytes	Lymphocytes	Lymphocytes
Protein	↑↑	↑	↑↑	↑
Glucose	↓	Normal	↓	↓
Onset	Acute	Acute	Subacute	Chronic

QUESTION

A patient presents with high-grade fever, headache, and altered sensorium. Cerebrospinal fluid (CSF) examination reveals low glucose, high protein, and a marked increase in polymorphonuclear leukocytes. Which of the following is the most likely diagnosis?

- A Acute viral meningitis
- B Acute tubercular meningitis
- C Acute bacterial meningitis**
- D Acute subarachnoid haemorrhage

CORRECT

RIGHT ANSWER

- OK
- C. Acute bacterial meningitis**

WHY THIS IS RIGHT

Parameter	Finding	Interpretation
Glucose	↓ Low	Consumed by bacteria + neutrophils
Protein	↑ High	BBB disruption + inflammation
Cells	↑ PMNs (neutrophils)	Acute pyogenic response

Common organisms (age-wise)

- Neonates -> E. coli, GBS
- Young adults -> Neisseria meningitidis
- Elderly -> Streptococcus pneumoniae

Feature	Bacterial	Viral	Tubercular	SAH
Cells	Neutrophils	Lymphocytes	Lymphocytes	RBCs
Glucose	↓↓↓	Normal	↓	Normal
Protein	↑↑	Mild ↑	↑↑↑	Mild ↑
CSF appearance	Turbid	Clear	Cobweb clot	Bloody
Onset	Acute	Subacute	Chronic	Sudden

QUESTION

A 22-year-old college student presents to the emergency department with a 1-day history of high-grade fever, severe headache, neck stiffness, and a petechial rash. Neurological examination reveals positive Kernig and Brudzinski signs. The diagnosis of meningococcal meningitis is suspected. Which of the following medications is the drug of choice for chemoprophylaxis of close contacts of this patient?

A Doxycycline

B Rifampicin

CORRECT

C Amoxicillin

D Ethambutol

RIGHT ANSWER

OK B. Rifampicin

WHY THIS IS RIGHT

Feature	Interpretation
High fever + headache + neck stiffness	Meningitis
Petechial rash	*Neisseria meningitidis*
Kernig & Brudzinski positive	Meningeal irritation

This presentation is classic for meningococcal meningitis (fever, neck stiffness, petechial rash). Close contacts are at high risk due to nasopharyngeal carriage of *Neisseria meningitidis*. Rifampicin is the drug of choice for chemoprophylaxis, as it eradicates nasopharyngeal carriage.

Drug	Role in Meningococcal Prophylaxis
Rifampicin	First-line
Ciprofloxacin	Alternative
Ceftriaxone	Alternative
Others	☐ Not used

QUESTION

| What is the most likely diagnosis?

A Acute bacterial meningitis

B Viral meningitis

C Tubercular meningitis

CORRECT

D Fungal meningitis

RIGHT ANSWER

OK C. Tubercular meningitis

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$27” here and does not include a written explanation for this item.

QUESTION

A child has features of meningitis with following CSF examination: protein=93, glucose=20, Lymphocytes++. Diagnosis?

A Bacterial meningitis

B Viral meningitis

C Tubercular meningitis

CORRECT

D None of the above

RIGHT ANSWER

OK C. Tubercular meningitis

WHY THIS IS RIGHT

No explanation was provided in the source quiz.

QUESTION

A 40-year-old male patient presented with complaints of drowsiness and was admitted for evaluation. Sodium was found to be 98 mEq/L, which was corrected to 110 mEq/L with 3% saline in 24 hours. The patient recovered for a while and had an altered sensorium later. Now, he developed quadriparesis and bilateral conjugate gaze palsy. Which is the investigation to be done in this case?

- A CSF analysis
- B Brainstem auditory evoked potential
- C MRI brain
- D EEG

CORRECT

RIGHT ANSWER

OK
C. MRI brain

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$28” here and does not include a written explanation for this item.

QUESTION

Regarding Guillain-Barré syndrome (GBS) in children, which of the following statements is false?

- A** Plasmapheresis is not effective in GBS
- B** IV immunoglobulins are the first line of treatment
- C** Areflexia in the weaker arm is included in diagnostic criteria
- D** Albumino-cytological dissociation is seen

CORRECT

RIGHT ANSWER

- OK** A. Plasmapheresis is not effective in GBS

WHY THIS IS RIGHT

Guillain-Barré Syndrome (GBS)-

- Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)
- Immune-mediated attack on peripheral nerve myelin
- Often follows:
- Infection (e.g., *Campylobacter jejuni*)

Infection (e.g., Campylobacter)

↓

Molecular mimicry

↓

Autoantibodies against myelin

↓

Demyelination of peripheral nerves

↓

↓ Nerve conduction

↓

Ascending weakness + areflexia

A. Plasmapheresis is not effective in GBS

- Plasmapheresis IS effective
- Removes circulating antibodies
- Used in:
- Severe GBS
- Refractory cases

So, statement is FALSE

QUESTION

Which of the following statements about Multiple Sclerosis are incorrect?

- A) 70% of patients with MS develop Optic Neuritis during the course of illness
- B) Oral steroids are first line for acute relapses
- C) Loss of colour and depth perception is proportional to loss of visual acuity
- D) Fundus examination can be normal

A A, B

B B, C

C A, B, C

CORRECT

D D only

RIGHT ANSWER

OK C. A, B, C

WHY THIS IS RIGHT

In multiple sclerosis, optic neuritis occurs in ~50%, not 70%, making statement A incorrect.

Autoimmune attack on optic nerve myelin

↓

Demyelination of optic nerve fibers

↓

↓ Conduction velocity

↓

Visual symptoms:

- Blurring of vision
- Pain on eye movement
- Color vision loss (early)

↓

Fundus initially normal (retrobulbar neuritis)

Feature	Description
Vision loss	Subacute, unilateral
Pain	On eye movement
Color vision	Earliest affected
Pupillary reflex	RAPD (Marcus Gunn pupil)
Fundus	Normal initially

----- | ----- |

| Vision loss | Subacute, unilateral |

| Pain | On eye movement |

| Color vision | Earliest affected |

| Pupillary reflex | RAPD (Marcus Gunn pupil) |

| Fundus | Normal initially |

Acute relapses are treated with IV methylprednisolone, not oral steroids, so B is incorrect.

In optic neuritis, loss of color vision (red desaturation) is disproportionately greater than visual acuity loss, making C incorrect.

Fundus can be normal (retrobulbar neuritis), so D is correct; hence A, B, C are incorrect.

QUESTION

Which of the following is the most common presenting symptom in patients with Multiple Sclerosis?

- A

Sensory Disturbances

CORRECT
- B

Vertigo
- C

Ataxia
- D

Facial Palsy

RIGHT ANSWER

A. Sensory Disturbances

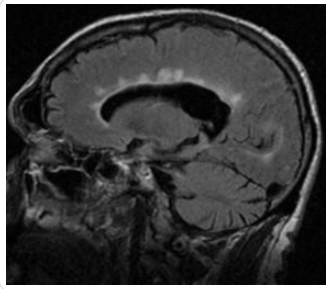
WHY THIS IS RIGHT

Multiple sclerosis most commonly presents initially with sensory disturbances such as numbness, paresthesia, or sensory loss. These symptoms result from demyelination of sensory pathways in the CNS. Motor deficits, ataxia, and cranial nerve involvement usually occur later in the disease course. Thus, sensory symptoms are the earliest and most common presenting feature.

- Classic Early Presentation Triad
1. Sensory symptoms (most common)
 2. Optic neuritis (painful vision loss)
 3. Motor weakness

Initial Symptoms of Multiple Sclerosis (MS)

SYMPTOM	PERCENTAGE OF CASES	SYMPTOM	PERCENTAGE OF CASES
Sensory loss	37	Lhermitte	3
Optic neuritis	36	Pain	3
Weakness	35	Dementia	2
Paresthesias	24	Visual loss	2
Diplopia	15	Facial palsy	1
Ataxia	11	Impotence	1
Vertigo	6	Myokymia	1
Paroxysmal attacks	4	Epilepsy	1
Bladder	4	Falling	1



QUESTION

A male patient presents with fever and nuchal rigidity. His CSF analysis reveals low sugar, elevated WBCs and Cryptococcal antigen positivity. Which of the following is used in the initial management of this condition?

1. Amphotericin B + Flucytosine
2. Amphotericin B + Fluconazole
3. Fluconazole
4. Liposomal Amphotericin B + Itraconazole

A 1 only

CORRECT

B 1 and 2

C 1, 2 and 3

D 2 and 4

RIGHT ANSWER

OK A. 1 only

WHY THIS IS RIGHT

- Fever + neck stiffness + CSF:
- ↓ Glucose
- ↑ WBCs
- Cryptococcal antigen positive

Diagnosis: Cryptococcal meningitis
(most commonly due to *Cryptococcus neoformans* infection)

- Inhaled yeast -> lungs -> hematogenous spread
- > CNS invasion
- > thick polysaccharide capsule prevents immune clearance

Parameter	Finding
Opening pressure	↑↑ (very high)
Glucose	↓↓
Protein	↑↑
Cells	Lymphocytic predominance

Diagnostic Clues

- India ink stain -> encapsulated yeast
- Cryptococcal antigen test -> highly sensitive

Treatment-

Cryptococcal Meningitis

↓

INDUCTION (2 weeks)

-> Amphotericin B + Flucytosine

↓

CONSOLIDATION (8 weeks)

-> Fluconazole (high dose)

↓

MAINTENANCE

-> Fluconazole (low dose)

QUESTION

A female patient complains of chest pain after waking up in the morning. Pain is more prevalent in the winter months. Which of the following best describes this patient?

- A** Fixed plaque obstruction
- B** Ach-induced coronary vasoconstriction
- C** Subendocardial ischemia due to increased demand of myocardium
- D** Increased sympathomimetic drive in morning hours

CORRECT

RIGHT ANSWER

- OK**
- B. Ach-induced coronary vasoconstriction**

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$29” here and does not include a written explanation for this item.

QUESTION

A 46-year-old male presents to the ER with the acute onset of ataxia. Neurological examination shows loss of pain and temperature sensation from the face on the left side and loss of pain and temperature sensation from the right half of the body. He also has dysarthria, dysphagia, and diminished gag reflex with ptosis, miosis, and anhidrosis on the left. What is the most likely diagnosis?

A Lateral pontine syndrome

B Medial medullary syndrome

C Lateral medullary syndrome

CORRECT

D Locked-In Syndrome

RIGHT ANSWER

OK C. Lateral medullary syndrome

WHY THIS IS RIGHT

Finding	Side	Structure involved
Face pain/temp loss	Left	Spinal trigeminal nucleus
Body pain/temp loss	Right	Spinothalamic tract
Dysphagia, dysarthria, ↓ gag	Left	Nucleus ambiguus (CN IX, X)
Ataxia	Left	Inferior cerebellar peduncle
Ptosis, miosis, anhidrosis	Left	Sympathetic fibers (Horner's)

Ipsilateral face + contralateral body pain loss + Cranial nerve IX, X involvement + Horner's syndrome
This combination = Wallenberg syndrome

PICA infarction

↓

Lateral medulla damage

↓

Multiple structure involvement:

↓

Spinal trigeminal nucleus -> Ipsilateral face pain loss

Spinothalamic tract -> Contralateral body pain loss

Nucleus ambiguus -> Dysphagia, dysarthria

Sympathetic fibers -> Horner's syndrome

Cerebellar peduncle -> Ataxia

QUESTION

A 62-year-old patient presents with left-sided arm and leg weakness, right-sided facial paralysis, and horizontal gaze palsy. Based on the clinical presentation, which of the following is most consistent with the diagnosis?

A Foville syndrome

CORRECT

B Locked In syndrome

C Millard-Gubler syndrome

D Wallenberg syndrome

RIGHT ANSWER

OK
A. Foville syndrome

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$2a” here and does not include a written explanation for this item.

QUESTION

| Interventional technique used for the management of cerebral aneurysm:**A Coiling**

CORRECT

B Embolization**C Basal stent placement****D Balloon Angioplasty**

RIGHT ANSWER

OK A. Coiling

WHY THIS IS RIGHT

Cerebral Aneurysm-

- Localized dilatation of an artery (usually at branch points in Circle of Willis)
- Most common: Berry (saccular) aneurysm
- Risk: Subarachnoid hemorrhage (SAH) -> sudden "worst headache of life"

Treatment Goal- Exclude aneurysm from circulation WITHOUT blocking parent artery

Cerebral aneurysm -> Endovascular coiling is the standard minimally invasive treatment to prevent rupture/rebleed

Feature	Coiling	Surgical Clipping
-----	-----	-----
Approach	Endovascular	Open surgery
Invasiveness	Minimal	High
Recovery	Faster	Slower
Recurrence	Slightly higher	Lower

Clinical Insight

- After SAH:
- Secure aneurysm ASAP -> coiling/clipping
- Prevents:
- Rebleeding (highest risk in first 24 hrs)

QUESTION

A patient presented with altered sensorium and his BP was 220/110 mmHg. He also had one episode of vomiting and on examination has weakness in the left upper and lower limbs. Which of the following is incorrect?

- A** BP should be checked multiple times before administering anti-hypertensives
- B** In haemorrhagic stroke, you should aggressively treat with intravenous anti-hypertensives
- C** In haemorrhagic stroke, BP should be reduced to 160/100 mmHg with anti-hypertensives
- D** BP should be reduced immediately to less than 140/90 mmHg in ischemic stroke CORRECT

RIGHT ANSWER

- OK** D. BP should be reduced immediately to less than 140/90 mmHg in ischemic stroke

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$2b” here and does not include a written explanation for this item.

QUESTION

| The most common site of a berry aneurysm in the circle of Willis is:

A Anterior cerebral artery

CORRECT

B Vertebral artery

C Posterior cerebral artery

D Basilar artery

RIGHT ANSWER

OK

A. Anterior cerebral artery

WHY THIS IS RIGHT

Most common berry aneurysm site = Anterior communicating artery (ACoA)
(which connects the anterior cerebral arteries)

Berry (Saccular) Aneurysm-

- Small, rounded outpouching at arterial bifurcations
- Caused by:
 - Congenital weakness of vessel wall (tunica media defect)
- Strongly associated with:
 - Hypertension
 - Polycystic kidney disease

B) Vertebral artery

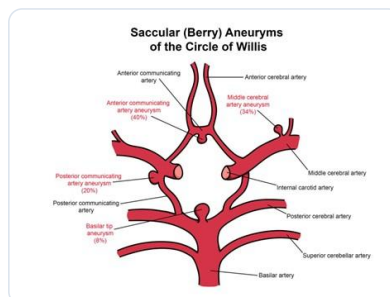
- Rare site for berry aneurysm
- More associated with:
 - Dissections
 - Posterior circulation strokes

C) Posterior cerebral artery

- Much less common
- PCA aneurysms are rare compared to ACoA/PCoM

D) Basilar artery

- Can have aneurysms (especially tip of basilar)
- But not the most common



QUESTION

A 9-month-old infant is brought to the pediatric clinic with a history of sudden, jerky movements of the limbs. EEG shows a chaotic high-amplitude background with multifocal spikes, consistent with hypsarrhythmia. What is the treatment of choice in this condition?

A Ethosuximide

B Valproate

C ACTH

CORRECT

D Lorazepam

RIGHT ANSWER

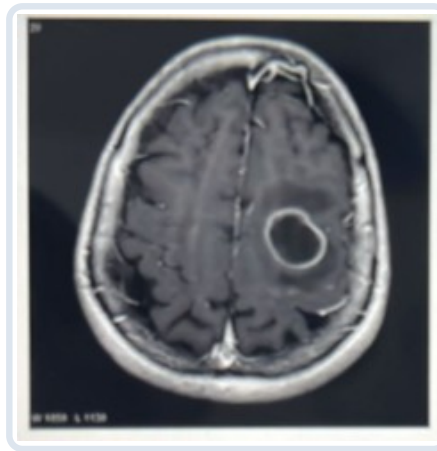
OK
C. ACTH

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$2c” here and does not include a written explanation for this item.

QUESTION

A 30-year-old patient presented with GTCS and was admitted. CECT is shown below. Which of the following can be useful in establishing the diagnosis?



A CBNAAT of CSF

B PET scan

C NMR spectroscopy

CORRECT

D B scan of both the eyes

RIGHT ANSWER

OK C. NMR spectroscopy

WHY THIS IS RIGHT

The CECT appearance with seizures suggests a space-occupying lesion where etiology is unclear (e.g., tumor vs infection).

NMR (MR) spectroscopy helps by analyzing metabolite patterns (↑ choline, ↓ NAA, lactate, lipid peaks).

Ring lesion detected

↓

Perform MR spectroscopy

↓

Analyze peaks:

-> Lipid/lactate ↑ -> TB / abscess

-> Amino acids ↑ -> Abscess

-> Choline ↑ -> Tumor

↓

Narrow diagnosis

This aids in characterizing lesion type and grade non-invasively.

| Condition | Key Peaks |

| ----- | ----- |

| Tuberculoma | Lipid peak ↑ |

| Abscess | Amino acids + lactate |

| Tumor | ↑ Choline, ↓ NAA |

| NCC | Variable (often less lipid than TB) |

CSF CBNAAT is for TB meningitis, PET is adjunctive, and B-scan eye exam is unrelated.

QUESTION

A 6-year-old boy is brought to the emergency department with a generalized tonic-clonic seizure after high grade fever. What is the most appropriate immediate treatment for this child?

A Diazepam

CORRECT

B Phenytoin

C Valproate

D Ethosuximide

RIGHT ANSWER

OK
A. Diazepam

WHY THIS IS RIGHT

This is a febrile seizure presenting as a generalized tonic-clonic seizure in a child with high-grade fever. The immediate management is benzodiazepine administration to abort the seizure.

Diazepam (IV/rectal) is the drug of choice for acute control.

Diazepam given

↓

Binds GABA-A receptor

↓

↑ Cl⁻ influx into neuron

↓

Hyperpolarization

↓

↓ Neuronal firing

↓

Seizure stops

Phenytoin, valproate, and ethosuximide are not used for immediate termination of febrile seizures.

| Drug | Role | Use |

| --- | --- | --- |

| Diazepam | □ First-line | Acute seizure |

| Phenytoin | Second-line | Status epilepticus |

| Valproate | Maintenance | Chronic seizures |

| Ethosuximide | Specific | Absence seizures |

Febrile Seizure-

| Feature | Finding |

| ----- | ----- |

| Age | 6 months-5 years |

| Type | Generalized |

| Prognosis | Good |

| Recurrence | Possible |

QUESTION

A 45-year-old patient with a history of depression was started on amitriptyline, a tricyclic antidepressant (TCA). The patient was later found unconscious at home and brought to the emergency department. On examination, the patient shows signs of arrhythmias, confusion, and pupillary dilation. Which of the following does NOT suggest TCA poisoning?

A Arrhythmias

B Confusion

C Hypothermia

CORRECT

D Pupillary dilation

RIGHT ANSWER

OK C. Hypothermia

WHY THIS IS RIGHT

TCA poisoning causes anticholinergic toxicity with confusion, mydriasis, and tachyarrhythmias due to sodium channel blockade.

Excess TCA

↓
Cardiac Na⁺ channel block

↓
Wide QRS -> arrhythmias

↓
CNS toxicity

↓
Confusion / coma / seizures

↓
Anticholinergic effects

↓
Dilated pupils, dry skin, tachycardia

| Feature | TCA Poisoning |

| ----- | ----- |

| Cardiac | Arrhythmias (wide QRS) |

| CNS | Confusion, seizures |

| Pupils | Dilated |

| Temperature | Hyperthermia |

| Skin | Dry |

Cardiac effects include QRS prolongation and ventricular arrhythmias.

Patients typically develop hyperthermia, not hypothermia, due to anticholinergic effects.

Hence, hypothermia does not suggest TCA poisoning.

QUESTION

When comparing oxcarbazepine and carbamazepine, which of the following adverse effects is more commonly associated with oxcarbazepine?

- A Skin rash
- B Hepatitis with raised GGT
- C Hyponatremia**
- D Blood dyscrasias

CORRECT

RIGHT ANSWER

OK **C. Hyponatremia**

WHY THIS IS RIGHT

Oxcarbazepine is more commonly associated with hyponatremia due to SIADH-like effect.

Oxcarbazepine vs Carbamazepine

Both:

- Block voltage-gated Na^+ channels
- Used in focal seizures

BUT they differ in side-effect.

Oxcarbazepine

↓

↑ ADH effect (SIADH-like)

↓

↑ Water reabsorption in kidney

↓

Dilution of sodium

↓

Hyponatremia

Clinical Consequence Flow-

↓ Serum sodium

↓

Cellular swelling (brain)

↓

Headache, confusion

↓

Seizures (paradoxical!)

| Advantage | Reason |

| ----- | ----- |

| Less enzyme induction | Fewer drug interactions |

| Safer hematologically | Less bone marrow suppression |

QUESTION

A patient with a mood disorder has been on medication and presents with new-onset liver dysfunction and hyperammonaemia. Which of the following medications is most likely responsible for these findings?

A Lamotrigine

B Carbamazepine

C Valproate

CORRECT

D Topiramate

RIGHT ANSWER

OK C. Valproate

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$19” here and does not include a written explanation for this item.

QUESTION

| Which of the following statements is true regarding fosphenytoin?

- A** At high doses in elderly patients, it causes cardiotoxicity and in younger patients' nephrotoxicity
- B** Adverse effects can be avoided if the dose is kept less than 250 mg phenytoin sodium equivalents
- C** It is given to all seizure patients in emergency irrespective of CNS adverse effects and hypotension
- D** High oral dose can result in cerebellar degeneration CORRECT

RIGHT ANSWER

- OK** **D. High oral dose can result in cerebellar degeneration**

WHY THIS IS RIGHT

Fosphenytoin is a water-soluble prodrug of phenytoin given IV or IM (no oral form). It is used acutely for status epilepticus or neurosurgical seizure prophylaxis.

Doses are expressed in "phenytoin equivalents" (PE): e.g. 75 mg fosphenytoin = 50 mg phenytoin. Typical IV loading is 15-20 mg PE/kg (e.g. 1000-1500 mg PE in adults) at a max infusion rate of 150 mg PE/min. Hence, Option B is wrong.

Option A and C: Its adverse effect profile mirrors phenytoin's: CNS (nystagmus, ataxia, sedation), and serious cardiovascular toxicity if infused too rapidly. Fosphenytoin can indeed cause serious cardiotoxicity in high doses, especially in elderly patients. The claim about nephrotoxicity in younger patients lacks evidence and is not a recognized phenomenon.

Cerebellar degeneration is associated with chronic phenytoin use, so that seems to be the best answer.

QUESTION

Which antiepileptic drug specifically enhances the inactivation phase of voltage-gated slow sodium channels, helping to stabilize hyperexcitable neurons?

A Phenytoin

B Lamotrigine

C Felbamate

D Lacosamide

CORRECT

RIGHT ANSWER

OK D. Lacosamide

WHY THIS IS RIGHT

Voltage-gated Na⁺ channels have 3 states:

1. Resting (closed)
2. Open (activated)
3. Inactivated

There are TWO types of inactivation:

- Fast inactivation (milliseconds)
- Slow inactivation (longer, stabilizing phase)

Lacosamide selectively enhances SLOW inactivation

- Keeps neurons in a non-excitabile state longer
- Prevents repetitive firing
- Stabilizes hyperexcitable neuronal membranes

| Drug | Mechanism | Na⁺ Channel Effect | |

| --- | --- | --- | --- |

| Lacosamide | Enhances slow inactivation | Keeps channels inactive longer | |

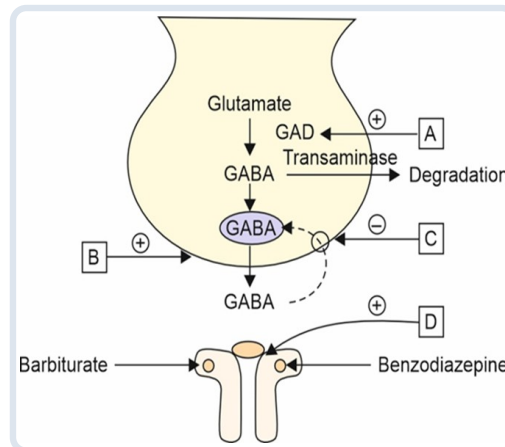
| Phenytoin | Blocks fast inactivation | Prevents rapid firing | |

| Lamotrigine | Fast inactivation + ↓ glutamate | Dual action | |

| Felbamate | NMDA antagonist | No Na⁺ effect | |

QUESTION

In the figure shown below, mechanism of action of valproate is best represented by:



A A

CORRECT

B B

C C

D D

RIGHT ANSWER

OK

A. A

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1a" here and does not include a written explanation for this item.

QUESTION

Which of the following statements about barbiturates and benzodiazepines is correct?

A Both act on GABA chloride channels

CORRECT

B Flumazenil is antagonist of barbiturates

C Benzodiazepines increase the duration of chloride channel opening

D Barbiturates are used to treat acute anxiety

RIGHT ANSWER

OK A. Both act on GABA chloride channels

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1b" here and does not include a written explanation for this item.

QUESTION

A 28-year-old male with a known history of generalized tonic-clonic seizures is on long-term carbamazepine therapy. He recently developed a sore throat and was prescribed erythromycin by a local physician. Two days later, he presents to the emergency department with complaints of dizziness, diplopia, ataxia, and confusion. What is the most likely explanation for his symptoms?

- A** Refractory seizures due to pharmacodynamic resistance
- B** Carbamazepine toxicity due to drug interaction **CORRECT**
- C** Erythromycin toxicity due to enzyme induction
- D** Decreased absorption of carbamazepine leading to subtherapeutic levels

RIGHT ANSWER

- OK** **B. Carbamazepine toxicity due to drug interaction**

WHY THIS IS RIGHT

- Carbamazepine is metabolized in the liver by CYP3A4
- Erythromycin = strong CYP3A4 inhibitor

So, when erythromycin is added:

- ↓ metabolism of carbamazepine
- ↑ plasma levels
- → toxicity

Features of Carbamazepine Toxicity

- Dizziness
- Diplopia
- Ataxia
- Nystagmus
- Confusion

| Option | Mechanism | Expected Effect | Why Correct/Incorrect | Key Exam Point |

| ----- | ----- | ----- | ----- | ----- |

| A | Resistance | ↑ seizures | ☐ No seizures here | Wrong clinical picture |

| B | CYP inhibition | ↑ CBZ levels | ☒ Correct | Classic interaction |

| C | Enzyme induction | ↓ drug levels | ☐ Wrong mechanism | Erythromycin = inhibitor |

| D | ↓ absorption | ↓ CBZ levels | ☐ Opposite effect | Would cause seizures |

QUESTION

Which of the following drugs is not given in status epilepticus?

A Levetiracetam

B Sodium valproate

C Ethosuximide

CORRECT

D Fosphenytoin

RIGHT ANSWER

OK

C. Ethosuximide

WHY THIS IS RIGHT

Status Epilepticus:

- Continuous seizure activity >5 minutes OR
- Recurrent seizures without regaining consciousness

It's a neurological emergency -> risk of:

- Neuronal injury
- Hypoxia
- Death

1\ Immediate suppression (fastest acting)

-> Benzodiazepines (IV Lorazepam)

2\ Stabilization (prevent recurrence)

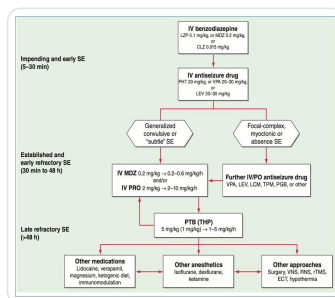
-> Longer-acting anticonvulsants

3\ Refractory phase

-> Anesthetics (propofol, midazolam infusion)

Ethosuximide Fails in SE

- Works on thalamic pacemaker activity
- SE involves diffuse cortical excitation
- Ethosuximide -> *ONLY absence seizures*
- Phenytoin/fosphenytoin -> NOT for absence seizures
- Valproate -> covers almost all seizure types



QUESTION

A 28-year-old man reports arm and hand weakness and numbness after sleeping with his arm over a chair post heavy drinking. What is the correct order of susceptibility of different nerve Fibers to pressure?

A A Fiber is less susceptible than B Fibers

B B Fiber is more susceptible than A Fibers

C C Fiber is less susceptible than B Fibers

CORRECT

D C Fiber is more susceptible than A Fibers

RIGHT ANSWER

OK

C. C Fiber is less susceptible than B Fibers

WHY THIS IS RIGHT

Pressure neuropathy preferentially affects larger, myelinated Fibers.

B Fibers (preganglionic autonomic, myelinated) are more susceptible to pressure than C Fibers (unmyelinated).

C Fibers are relatively resistant to compression and hypoxia.

Fiber	Myelination	Size	Function
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A Fibers	Heavy	Large	Motor, proprioception
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B Fibers	Light	Small	Autonomic
----------	-------	-------	-----------

C Fibers	None	Very small	Pain, temperature
----------	------	------------	-------------------

QUESTION

Which autoimmune disorder involves antibodies against post-synaptic ACh receptors?

- A Multiple sclerosis
- B Myasthenia gravis
- C Lambert-Eaton syndrome
- D Guillain-Barré syndrome

CORRECT

RIGHT ANSWER

- OK
- B. Myasthenia gravis

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1c” here and does not include a written explanation for this item.

QUESTION

A person after sleeping overnight with the arm under his head now experiences paresis but no numbness in the morning. Which of the following is the best explanation for it?

- A** C Fibers are more sensitive to pressure than A Fibers
- B** A Fibers are more sensitive to hypoxia than B Fibers
- C** A Fibers are more susceptible to pressure changes than C Fibers CORRECT
- D** A Fibers are more susceptible to hypoxia than C Fibers

RIGHT ANSWER

- OK** C. A Fibers are more susceptible to pressure changes than C Fibers

WHY THIS IS RIGHT

The source quiz contains the placeholder “\$1d” here and does not include a written explanation for this item.

QUESTION

A woman suffered from sunburn after enjoying a vacation on a beach. Now while taking a shower lukewarm water (40-degree Celsius) touches her back causing her to feel pain. What type of receptors are activated by lukewarm water and what causes her to feel pain?

A Noxious thermal receptor - hyperalgesia

B Innocuous thermal receptor - allodynia

CORRECT

C Noxious thermal receptor - allodynia

D Innocuous thermal receptor - hyperalgesia

RIGHT ANSWER

OK

B. Innocuous thermal receptor - allodynia

WHY THIS IS RIGHT

Lukewarm water (40 °C) normally activates innocuous thermal receptors, not nociceptors. After sunburn, peripheral sensitization lowers the pain threshold of sensory pathways. As a result, a normally non-painful stimulus is perceived as pain, termed allodynia.

Sunburn -> inflammation

↓

Release of mediators (prostaglandins, bradykinin)

↓

↓ Threshold of nociceptors

↓

Non-painful stimuli now activate pain pathways

↓

Allodynia

| Feature | Allodynia | Hyperalgesia |

| ----- | ----- | ----- |

| Stimulus | Non-painful | Painful |

| Response | Pain | Exaggerated pain |

| Example | Sunburn + warm water | Pinprick -> severe pain |

Hyperalgesia refers to exaggerated pain to a painful stimulus, which is not the case here.

| Term | Meaning |

| --- | ----- |

| Allodynia | Pain from non-painful stimulus |

| Hyperalgesia | Increased pain from painful stimulus |

| Analgesia | No pain |

QUESTION

After a right limb amputation, the patient is experiencing severe pain (phantom limb). What is the mechanism behind this?

A Projection of adjacent fibers to overlap to right sensory cortex

B Projection of adjacent fibers to overlap to left sensory cortex

CORRECT

C Expansion of right sensory cortex

D Expansion of left sensory cortex

RIGHT ANSWER

OK

B. Projection of adjacent fibers to overlap to left sensory cortex

WHY THIS IS RIGHT

Phantom limb pain occurs due to cortical reorganization after amputation.

Adjacent sensory areas invade the contralateral (left) primary somatosensory cortex representing the amputated right limb.

Stimulation of these neighboring regions is misinterpreted as pain from the missing limb.

- Pain perceived in an amputated limb
- Due to central (cortical) mechanisms, not peripheral input

Thus, projection of adjacent fibers overlapping the left sensory cortex explains the phenomenon.

Limb amputated

↓

No sensory input from that region

↓

Adjacent cortical areas expand

↓

They “invade” the vacant area

↓

Misinterpretation of signals

↓

Phantom sensation / pain

| Concept | Phantom Limb |

| ----- | ----- |

| Side affected | Contralateral cortex |

| Mechanism | Cortical reorganization |

| Key event | Adjacent area overlap |

| Type | Central phenomenon |

QUESTION

In the below question mechanical receptors are given with their functions. Choose the correctly matched pair.

A Pacinian Corpuscle - Fast Vibration

CORRECT

B Ruffini - Fine touch

C Meissner - Stretch

D Merkel - Slow Vibration

RIGHT ANSWER

OK A. Pacinian Corpuscle - Fast Vibration

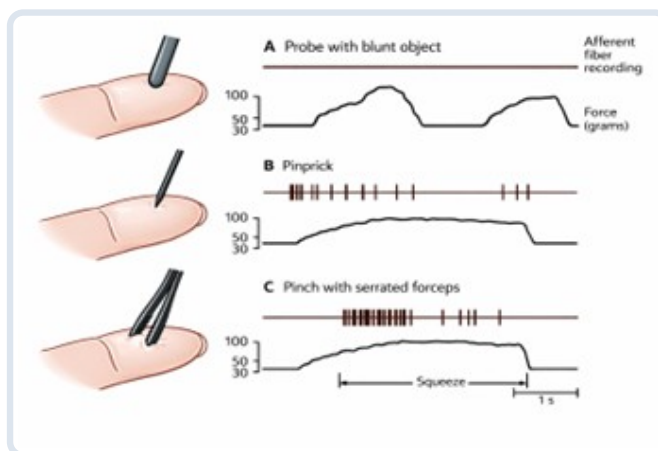
WHY THIS IS RIGHT

Pacinian corpuscles are rapidly adapting mechanoreceptors that detect high-frequency (fast) vibration. They have large receptive fields and respond best to sudden pressure changes. Ruffini endings sense stretch, Meissner corpuscles mediate light touch, and Merkel discs detect form and texture.

Receptor	Adaptation	Function	Receptive Field
Pacinian	Rapid	□ Vibration	Large
Meissner	Rapid	Light touch	Small
Merkel	Slow	Fine touch	Small
Ruffini	Slow	Stretch	Large

QUESTION

Mechanical nociceptors and its response to a probe with blunt object, pin prick and tooth forceps with serration is recorded as a output of a force transducer coupled to the stimulator. What will you infer from a given diagram?



A Blunt object with high force doesn't stimulate nociceptors

CORRECT

B Sharp object doesn't stimulate nociceptors

C Pinching with serrated object stimulate mechanoreceptors but doesn't cause pain

D Sharp pain sensation is carried by type C nerve fiber

RIGHT ANSWER

OK

A. Blunt object with high force doesn't stimulate nociceptors

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1e" here and does not include a written explanation for this item.

QUESTION

| Which of the following is involved in Static Two-point discrimination?

A Meissner's corpuscles

B **Merkel disk**

CORRECT

C Pacinian corpuscles

D Ruffini ending

RIGHT ANSWER

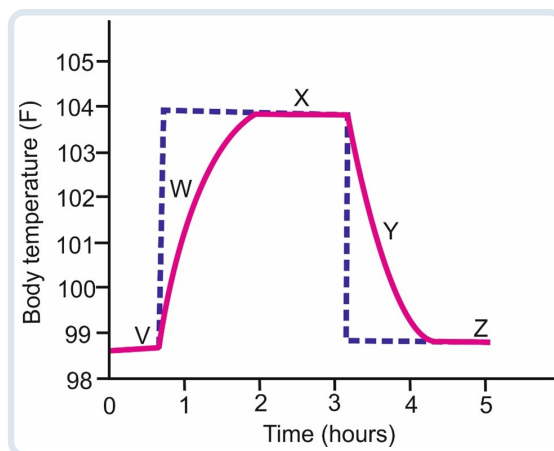
OK **B. Merkel disk**

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$1f" here and does not include a written explanation for this item.

QUESTION

In a patient, the hypothalamic thermostat was reset from point V to point X as shown below. Which of the following happens in stage W compared to stage Y?



A Shivering

CORRECT

B Sweating

C Increased blood flow to skin

D Inhibition of chemical thermogenesis

RIGHT ANSWER

OK **A. Shivering**

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$20" here and does not include a written explanation for this item.

QUESTION

| Which centre in the brain is responsible for producing fever during infection?

A Paraventricular nucleus of hypothalamus

B Dorsomedial nucleus

C **Preoptic Nucleus**

CORRECT

D Cerebellum

RIGHT ANSWER

OK **C. Preoptic Nucleus**

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$21" here and does not include a written explanation for this item.

QUESTION

Which of the following brain regions is primarily associated with the processing of explicit memory?

A Hippocampus

CORRECT

B Amygdala

C Insular cortex

D Cerebellum

RIGHT ANSWER

OK A. Hippocampus

WHY THIS IS RIGHT

Type	Example	Brain Region
Explicit	Facts, events	Hippocampus
Implicit (procedural)	Skills	Cerebellum, basal ganglia

Declarative / Explicit Memory:

- Semantic (factual):
 - Prefrontal cortex
 - Temporal cortex (lateral and anterior)
- Episodic (events):
 - Hippocampus
 - Medial temporal lobe
 - Neocortex

Nondeclarative / Implicit Memory:

- Procedural (skills, habits):
 - Striatum
 - Cerebellum
 - Motor cortex
- Priming and perceptual:
 - Neocortex
- Associative learning (classical conditioning):
 - Amygdala
 - Cerebellum

QUESTION

Which of the following areas of the brain are correctly matched with the neurotransmitters they release?

1. Substantia nigra - Dopamine
2. Locus ceruleus - Acetylcholine
3. Raphe nucleus - Serotonin

A 1

B 1, 2 and 3

C 1 and 3

CORRECT

D 2 and 3

RIGHT ANSWER

OK C. 1 and 3

WHY THIS IS RIGHT

Nucleus	Neurotransmitter	Function	Clinical Correlation
Substantia nigra	Dopamine	Movement	Parkinson disease
Locus ceruleus	Norepinephrine	Arousal, attention	Anxiety, stress
Raphe nuclei	Serotonin	Mood, sleep	Depression
Basal nucleus of Meynert	Acetylcholine	Memory	Alzheimer disease

QUESTION

| What is the role of caffeine in wakefulness?

CORRECT

- A** Blocks adenosine action and causes wakefulness
- B** Activates locus coeruleus and cause wakefulness
- C** No role in maintaining wakefulness if taken 1hr before sleep
- D** Activates histamine release and prevents sleep

RIGHT ANSWER

- OK** **A. Blocks adenosine action and causes wakefulness**

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$22" here and does not include a written explanation for this item.

QUESTION

| Which of the following hypothalamic nuclei are responsible for leptin resistance?

A Paraventricular nucleus

B Lateral nucleus

C Arcuate nucleus

CORRECT

D Ventromedial nucleus

RIGHT ANSWER

OK C. Arcuate nucleus

WHY THIS IS RIGHT

The source quiz contains the placeholder "\$23" here and does not include a written explanation for this item.