DISORDERS OF THE NEUROLOGICAL SYSTEM PRACTICE TEST (SAMPLE)

STUDY GUIDE



SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

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Table of Contents

Copyright 1
Table of Contents 2
Introduction 3
How to Use This Guide 4
Questions 5
Answers 8
Explanations 10
Next Steps 15

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Introduction

Preparing for a certification exam can feel overwhelming, but with the right tools, it becomes an opportunity to build confidence, sharpen your skills, and move one step closer to your goals. At Examzify, we believe that effective exam preparation isn't just about memorization, it's about understanding the material, identifying knowledge gaps, and building the test-taking strategies that lead to success.

This guide was designed to help you do exactly that.

Whether you're preparing for a licensing exam, professional certification, or entry-level qualification, this book offers structured practice to reinforce key concepts. You'll find a wide range of multiple-choice questions, each followed by clear explanations to help you understand not just the right answer, but why it's correct.

The content in this guide is based on real-world exam objectives and aligned with the types of questions and topics commonly found on official tests. It's ideal for learners who want to:

- Practice answering questions under realistic conditions,
- Improve accuracy and speed,
- Review explanations to strengthen weak areas, and
- Approach the exam with greater confidence.

We recommend using this book not as a stand-alone study tool, but alongside other resources like flashcards, textbooks, or hands-on training. For best results, we recommend working through each question, reflecting on the explanation provided, and revisiting the topics that challenge you most.

Remember: successful test preparation isn't about getting every question right the first time, it's about learning from your mistakes and improving over time. Stay focused, trust the process, and know that every page you turn brings you closer to success.

Let's begin.

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How to Use This Guide

This guide is designed to help you study more effectively and approach your exam with confidence. Whether you're reviewing for the first time or doing a final refresh, here's how to get the most out of your Examzify study guide:

1. Start with a Diagnostic Review

Skim through the questions to get a sense of what you know and what you need to focus on. Your goal is to identify knowledge gaps early.

2. Study in Short, Focused Sessions

Break your study time into manageable blocks (e.g. 30 – 45 minutes). Review a handful of questions, reflect on the explanations.

3. Learn from the Explanations

After answering a question, always read the explanation, even if you got it right. It reinforces key points, corrects misunderstandings, and teaches subtle distinctions between similar answers.

4. Track Your Progress

Use bookmarks or notes (if reading digitally) to mark difficult questions. Revisit these regularly and track improvements over time.

5. Simulate the Real Exam

Once you're comfortable, try taking a full set of questions without pausing. Set a timer and simulate test-day conditions to build confidence and time management skills.

6. Repeat and Review

Don't just study once, repetition builds retention. Re-attempt questions after a few days and revisit explanations to reinforce learning. Pair this guide with other Examzify tools like flashcards, and digital practice tests to strengthen your preparation across formats.

There's no single right way to study, but consistent, thoughtful effort always wins. Use this guide flexibly, adapt the tips above to fit your pace and learning style. You've got this!

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Questions

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1. Which statement correctly describes the age of onset for Huntington disease?

A. In infancy

B. In the 30s to 40s

C. In late 70s

D. In adolescence

2. How can nerve conduction studies differentiate demyelinating versus axonal neuropathies?

A. Demyelinating neuropathies show reduced amplitude with preserved velocity; axonal neuropathies show slowed velocity.

B. Demyelinating neuropathies show slowed conduction velocity, prolonged F-wave latency, and conduction block; axonal neuropathies show reduced amplitude with relatively preserved velocity.

C. EMG shows denervation in demyelinating neuropathies only.

D. Nerve conduction studies cannot differentiate these conditions.

3. Which pathways primarily rule sensorimotor status below the shoulders?

A. The cerebellar peduncles

B. The corticospinal and spinothalamic tracts

C. The dorsal columns

D. The thalamus

4. Which MRI finding is most characteristic of PRES?

A. Focal restricted diffusion with cytotoxic edema.

B. Symmetric vasogenic edema in the posterior white matter, particularly the parieto-occipital regions.

C. Hippocampal sclerosis.

D. Mass in temporal lobe.

5. Which structure governs sensorimotor status above the shoulders in a neurological examination?

A. The corticospinal tracts

B. The 12 cranial nerves

C. The dorsal columns

D. The peripheral nerves of the limbs

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6. In AMD, central vision loss is described as what?

A. A gradual loss of peripheral vision.

B. Like having a black spot in the center of your vision.

C. Temporary blurring that improves with rest.

D. Complete loss of color vision.

7. Which feature best differentiates central (UMN) facial palsy from peripheral (LMN) facial palsy on examination?

A. Central palsy typically affects the forehead; peripheral palsy spares the forehead.

B. Central palsy spares the forehead; peripheral palsy affects the entire face including the forehead.

C. Central palsy affects the mouth only; peripheral palsy affects the eye.

D. Central palsy affects symmetry of facial expression; peripheral palsy does not.

8. Which reflex may appear in adults with upper motor neuron lesions?

A. Babinski reflex

B. Hoffmann sign

C. Moro reflex

D. Gallant reflex

9. In closed-angle glaucoma, the angle closes how?

A. Opens and closes gradually

B. Closes suddenly and unexpectedly

C. Stays permanently open

D. Does not affect intraocular pressure

10. What are the two main types of brain attacks?

A. Ischemic brain attacks and hemorrhagic brain attacks

B. Traumatic and infectious

C. Metabolic and degenerative

D. Allergic and autoimmune

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Answers

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1. B
2. B
3. B
4. B
5. B
6. B
7. D
8. A
9. B
10. D

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Explanations

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1. Which statement correctly describes the age of onset for Huntington disease?

A. In infancy

B. In the 30s to 40s

C. In late 70s

D. In adolescence

Huntington disease typically begins in midlife, most often showing up in the 30s to 40s. This timing reflects how the disease gradually unfolds as the brain, especially the striatum, loses neurons over years. The genetic basis is a CAG repeat expansion in the HTT gene; longer repeats tend to push the onset earlier, and this can even lead to earlier symptoms in later generations in a phenomenon known as anticipation. Onsets in infancy or adolescence are not typical, and a late onset in the 70s is quite uncommon. So, the statement describing onset in the 30s to 40s matches the usual clinical pattern.

2. How can nerve conduction studies differentiate demyelinating versus axonal neuropathies?

A. Demyelinating neuropathies show reduced amplitude with preserved velocity; axonal neuropathies show slowed velocity.

B. Demyelinating neuropathies show slowed conduction velocity, prolonged F-wave latency, and conduction block; axonal neuropathies show reduced amplitude with relatively preserved velocity.

C. EMG shows denervation in demyelinating neuropathies only.

D. Nerve conduction studies cannot differentiate these conditions.

Nerve conduction studies differentiate these conditions by how the nerve's signal is affected: demyelination slows conduction and disrupts timing, while axonal loss reduces signal strength but largely preserves speed. In demyelinating neuropathies, the myelin sheath is damaged, so conduction velocity drops markedly. Distal and proximal (F-wave) latencies become prolonged, and conduction block or marked temporal dispersion can occur because impulses struggle to traverse the demyelinated segments. Amplitude of the response may stay relatively preserved early on, since many nerves still conduct, but timing and speed are the giveaway. In axonal neuropathies, there is loss of axons, so the overall signal amplitude (CMAP/SNAP) decreases. The remaining fibers conduct at near-normal speed, so conduction velocity is relatively preserved compared to the marked slowing seen with demyelination. F-waves and distal latencies may be affected, but the hallmark is reduced amplitude with retained conduction speed. Thus, the statement that demyelinating neuropathies show slowed conduction velocity, prolonged F-wave latency, and conduction block, while axonal neuropathies show reduced amplitude with relatively preserved velocity, best captures how nerve conduction studies differentiate these conditions.

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3. Which pathways primarily rule sensorimotor status below the shoulders?

A. The cerebellar peduncles

B. The corticospinal and spinothalamic tracts

C. The dorsal columns

D. The thalamus

The key idea is that sensorimotor status below the shoulders is governed by the major pathways that carry voluntary motor commands and basic sensory signals from the body to the brain. The corticospinal tract is the primary descending motor pathway; it originates in the motor cortex and, after traveling through the brainstem, reaches the spinal cord to control voluntary movement of the limbs and trunk below the neck. The spinothalamic tracts are the main ascending pathways for pain, temperature, and crude touch, carrying these sensations from the body up to the thalamus. Together, they largely determine how motor output and basic sensory information are expressed below the shoulders. While the dorsal columns carry fine touch, vibration, and proprioception, those modalities are additional and not the primary routes described for general sensorimotor status in this context. The thalamus is a relay center, not a direct motor or sensory pathway.

4. Which MRI finding is most characteristic of PRES?

A. Focal restricted diffusion with cytotoxic edema.

B. Symmetric vasogenic edema in the posterior white matter, particularly the parieto-occipital regions.

C. Hippocampal sclerosis.

D. Mass in temporal lobe.

PRES is driven by a disruption of cerebral autoregulation that leads to vasogenic edema. The most characteristic MRI finding is symmetric edema in the posterior white matter, especially in the parieto-occipital regions. On MRI this appears as T2/FLAIR hyperintensities in those areas, and the edema is vasogenic, so diffusion imaging typically shows no marked restriction (often increased ADC), distinguishing it from cytotoxic edema seen in infarcts. Other choices suggest different conditions—cytotoxic edema points to acute infarction; hippocampal sclerosis and a temporal lobe mass indicate chronic or neoplastic processes, not PRES.

5. Which structure governs sensorimotor status above the shoulders in a neurological examination?

A. The corticospinal tracts

B. The 12 cranial nerves

C. The dorsal columns

D. The peripheral nerves of the limbs

Sensorimotor status above the shoulders is governed by the cranial nerves because they innervate the head and neck, providing both sensory input and motor control for facial muscles, eye movements, swallowing, facial sensation, and other functions in that region. In a neurological exam, assessing the twelve cranial nerves directly tests the integrity of sensorimotor function in the head and upper face. The corticospinal tracts mainly control voluntary movement of the limbs and trunk, so they're more about the body below the neck. The dorsal columns carry fine touch and proprioception from the body below the neck, not the face. Peripheral nerves of the limbs supply only the limbs, not the head and neck. So the structure that best explains sensorimotor status above the shoulders is the cranial nerves.

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6. In AMD, central vision loss is described as what?

A. A gradual loss of peripheral vision.

B. Like having a black spot in the center of your vision.

C. Temporary blurring that improves with rest.

D. Complete loss of color vision.

Central vision loss in AMD happens because the macula—the part of the retina responsible for sharp, straight-ahead vision—degenerates. As a result, a central blind spot or distortion appears, so people notice a dark or blank spot in the center of their vision while the edges remain clear. This central deficit is the hallmark of AMD, with peripheral vision usually preserved in the early stages. The description of a black spot in the center matches what patients commonly report, making it the best fit. The other options don't fit the typical pattern: peripheral vision loss would point to problems outside the macula; temporary blurring that improves with rest isn't characteristic of AMD; and complete color vision loss isn't how AMD presents, though color perception can be subtly affected.

7. Which feature best differentiates central (UMN) facial palsy from peripheral (LMN) facial palsy on examination?

A. Central palsy typically affects the forehead; peripheral palsy spares the forehead.

B. Central palsy spares the forehead; peripheral palsy affects the entire face including the forehead.

C. Central palsy affects the mouth only; peripheral palsy affects the eye.

D. Central palsy affects symmetry of facial expression; peripheral palsy does not.

The key idea is forehead involvement on exam. The muscles of the upper face (forehead) get input from both sides of the brain, so a unilateral upper motor neuron lesion cannot completely paralyze the forehead; the patient can still move it, even though the lower face may be weak. In contrast, a lower motor neuron lesion affecting the facial nerve on one side knocks out all ipsilateral facial muscles, including the forehead, so the entire half of the face cannot move. So, the best differentiating feature is whether the forehead is involved. Central (UMN) palsy spares the forehead; peripheral (LMN) palsy includes forehead weakness. That's why the description that central palsy spares the forehead and peripheral palsy affects the entire face (including the forehead) is the correct way to distinguish them.

8. Which reflex may appear in adults with upper motor neuron lesions?

A. Babinski reflex

B. Hoffmann sign

C. Moro reflex

D. Gallant reflex

The main idea here is that upper motor neuron (UMN) lesions disrupt the corticospinal pathways, leading to abnormal reflexes in adults. The Babinski sign is the classic indicator: when the sole is stimulated, instead of curling downward, the big toe dorsiflexes and the other toes fan out. This upgoing big toe reflex results from loss of inhibition of spinal reflexes by the intact cortex, signaling a corticospinal tract lesion above the level of the brainstem. Moro and Gallant are primitive infant reflexes that normally disappear within the first few months of life; their presence in adults is not a typical marker of a focal UMN lesion. Hoffmann sign is also an upper motor neuron sign, but the Babinski response is the most characteristic and widely recognized example in adults.

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9. In closed-angle glaucoma, the angle closes how?

A. Opens and closes gradually

B. Closes suddenly and unexpectedly

C. Stays permanently open

D. Does not affect intraocular pressure

The key idea is that in angle-closure glaucoma the drainage angle can shut abruptly, not inch by inch. A sudden pupillary block or iris bunching causes the iris to press against the trabecular meshwork, momentarily blocking outflow. When drainage is suddenly blocked, aqueous humor backs up quickly, causing a rapid rise in intraocular pressure. This creates an acute, painful attack that is a true emergency. Think of it as an abrupt lid-closure of the drainage pathway triggered by factors like dim lighting or an anatomy with a shallow anterior chamber. That abrupt closure explains why the correct description is that the angle closes suddenly and unexpectedly. Other scenarios—gradual closure, the angle remaining open, or no impact on pressure—don't fit the abrupt, pressure-spiking nature of this condition.

10. What are the two main types of brain attacks?

A. Ischemic brain attacks and hemorrhagic brain attacks

B. Traumatic and infectious

C. Metabolic and degenerative

D. Allergic and autoimmune

Brain attacks, or strokes, arise from two primary mechanisms. An ischemic brain attack occurs when a blood clot blocks a brain artery, cutting off blood flow and oxygen to a region of the brain. Without quick restoration of circulation, brain tissue begins to die. A hemorrhagic brain attack happens when a blood vessel ruptures, causing bleeding into brain tissue or surrounding spaces, which increases pressure and damages cells. This distinction matters because treatments differ: ischemic strokes are often treated with clot-dissolving therapy or mechanical clot removal to restore flow within a critical time window, while hemorrhagic strokes require stopping the bleed, managing pressure, and sometimes surgical intervention. The other listed categories don't describe the main mechanisms behind most brain attacks, which are blockage-induced or bleeding-induced events.

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Next Steps

Congratulations on reaching the final section of this guide. You've taken a meaningful step toward passing your certification exam and advancing your career.

As you continue preparing, remember that consistent practice, review, and self-reflection are key to success. Make time to revisit difficult topics, simulate exam conditions, and track your progress along the way.

If you need help, have suggestions, or want to share feedback, we'd love to hear from you. Reach out to our team at hello@examzify.com.

Or visit your dedicated course page for more study tools and resources:

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We wish you the very best on your exam journey. You've got this!

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