

NBEO NEUROSCIENCE PRACTICE TEST (SAMPLE)

STUDY GUIDE



SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

VISIT US ONLINE FOR
THE FULL VERSION STUDY GUIDE

<https://nbeoneuroscience.examzify.com>



Copyright © 2026 by Examzify - A Kaluba Technologies Inc. product.

ALL RIGHTS RESERVED.

No part of this book may be reproduced or transferred in any form or by any means, graphic, electronic, or mechanical, including photocopying, recording, web distribution, taping, or by any information storage retrieval system, without the written permission of the author.

Notice: Examzify makes every reasonable effort to obtain accurate, complete, and timely information about this product from reliable sources.

SAMPLE

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

SAMPLE

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.

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!

Questions

SAMPLE

- 1. An elevated ESR and CRP in a patient with unilateral disc edema is most suspicious of which systemic disease?**
 - A. Diabetes
 - B. Giant Cell Arteritis
 - C. Hypertension
 - D. Multiple Sclerosis

- 2. Which Light Near Dissociation is primarily caused by a lesion of the ciliary ganglion?**
 - A. Tonic Pupil
 - B. Argyll Robertson Pupil
 - C. Adie's Pupil
 - D. Horner's Syndrome

- 3. AAION stands for which condition?**
 - A. Arteritic Anterior Ischemic Optic Neuropathy
 - B. Aneurysmal Anterior Ischemic Optic Neuropathy
 - C. Acute Anterior Ischemic Optic Neuropathy
 - D. Arterial Anterior Ischemic Optic Neuropathy

- 4. Which statement is true regarding Thyroid Eye Disease?**
 - A. It is associated with proptosis and upper eyelid retraction
 - B. It always preserves visual acuity
 - C. It never causes diplopia
 - D. It is unrelated to thyroid function

- 5. Which statement about papilledema is true?**
 - A. It is typically bilateral.
 - B. It always causes severe visual loss early.
 - C. It never affects the blind spot.
 - D. Paton's lines are never seen

- 6. Which phenomenon is the sensation of an electric shock that quickly travels down the back and into the limbs with flexure of the neck and is associated with Multiple Sclerosis?**
- A. Lhermitte's phenomenon
 - B. Uhthoff's phenomenon
 - C. Romberg sign
 - D. Nystagmus
- 7. Foster Kennedy syndrome is a rare condition caused by a frontal lobe tumor and presents with simultaneous optic disc edema in one eye and optic disc atrophy in the fellow eye.**
- A. Foster Kennedy syndrome
 - B. Horner syndrome
 - C. Leber's optic neuropathy
 - D. Optic neuritis
- 8. In visual evoked potentials, what does P100 latency reflect?**
- A. The timing of conduction from retina to occipital cortex; delayed P100 latency indicates demyelination or conduction slowing
 - B. It measures retinal nerve fiber layer thickness
 - C. It indicates damage to optic disc
 - D. It measures acuity
- 9. Which of the following is NOT a known risk factor for NAION?**
- A. Hypertension
 - B. Diabetes mellitus
 - C. Giant Cell Arteritis
 - D. Hyperlipidemia
- 10. In Myasthenia Gravis, autoantibodies are directed toward which component at the NMJ?**
- A. Dopamine receptor
 - B. Acetylcholine receptor
 - C. GABA receptor
 - D. NMDA receptor

Answers

SAMPLE

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

SAMPLE

Explanations

SAMPLE

1. An elevated ESR and CRP in a patient with unilateral disc edema is most suspicious of which systemic disease?

- A. Diabetes
- B. Giant Cell Arteritis**
- C. Hypertension
- D. Multiple Sclerosis

Elevated ESR and CRP point to an active inflammatory process. When this is seen alongside unilateral optic disc edema in an older adult, it strongly suggests Giant Cell Arteritis, a systemic vasculitis that can involve the arteries supplying the optic nerve. The inflammatory involvement can lead to arteritic anterior ischemic optic neuropathy, causing sudden disc swelling and a risk of rapid, permanent vision loss if not treated promptly. The other options don't typically produce a systemic inflammatory marker rise in this setting: diabetes and hypertension relate more to non-arteritic optic nerve issues and don't inherently elevate ESR/CRP, while multiple sclerosis can cause optic neuritis but usually without the inflammatory marker pattern seen here.

2. Which Light Near Dissociation is primarily caused by a lesion of the ciliary ganglion?

- A. Tonic Pupil**
- B. Argyll Robertson Pupil
- C. Adie's Pupil
- D. Horner's Syndrome

Light near dissociation arises when parasympathetic input to the iris sphincter is disrupted along the postganglionic pathway to the ciliary muscle. The ciliary ganglion is the relay point for this parasympathetic supply. When it is damaged, the pupillary light reflex is markedly reduced or absent because the sphincter cannot constrict in response to light, while the near response can remain relatively better preserved, producing constriction with accommodation. This pattern is characteristic of a tonic pupil, also known as Adie's pupil, which reflects a ciliary ganglion lesion. Argyll Robertson pupil results from a different problem in the pretectal area (neurosyphilis) and can show light–near dissociation as well, but not due to ciliary ganglion damage. Horner's syndrome involves sympathetic pathway loss, causing miosis with ptosis and an anisocoria that is more pronounced in darkness, rather than the classic light–near dissociation pattern.

3. AAION stands for which condition?

- A. Arteritic Anterior Ischemic Optic Neuropathy**
- B. Aneurysmal Anterior Ischemic Optic Neuropathy
- C. Acute Anterior Ischemic Optic Neuropathy
- D. Arterial Anterior Ischemic Optic Neuropathy

AAION stands for Arteritic Anterior Ischemic Optic Neuropathy. The letters map to Arteritic, Anterior, Ischemic, Optic, Neuropathy, reflecting an inflammatory (vasculitic) cause of anterior optic nerve ischemia—most classically giant cell arteritis. This form is distinct from non-arteritic AION, which is due to microvascular disease rather than an inflammatory arteritis. The other terms don't fit the established terminology: "Aneurysmal" isn't used to describe this acronym, "arterial" would alter the intended meaning, and "acute" describes timing rather than the pathologic etiology captured by the acronym.

4. Which statement is true regarding Thyroid Eye Disease?

- A. It is associated with proptosis and upper eyelid retraction**
- B. It always preserves visual acuity
- C. It never causes diplopia
- D. It is unrelated to thyroid function

Thyroid Eye Disease is an autoimmune process often linked with Graves' disease that inflames and remodels orbital tissues, especially the extraocular muscles and orbital fat. This expansion inside the orbit pushes the eye forward (proptosis) and can cause the upper eyelid to retract due to involvement of the levator muscle and sympathetic changes. The same inflammatory changes can lead to limited eye movements, causing diplopia, and in more severe cases crowding at the orbital apex can threaten vision through optic neuropathy. This means visual acuity is not always preserved, so statements asserting continuous normal vision or never having diplopia are inaccurate. The condition is tied to thyroid dysfunction; most patients have hyperthyroidism, but it can occur with euthyroid or even hypothyroid states as part of the autoimmune process. So the statement that it is associated with proptosis and upper eyelid retraction correctly reflects the hallmark orbital changes of Thyroid Eye Disease.

5. Which statement about papilledema is true?

- A. It is typically bilateral.**
- B. It always causes severe visual loss early.
- C. It never affects the blind spot.
- D. Paton's lines are never seen

Papilledema happens when intracranial pressure rises and is transmitted through the subarachnoid space around both optic nerves. This global pressure disrupts axoplasmic flow at the lamina cribrosa, causing swelling of the optic nerve heads. Because the pressure affects both optic nerves together, swelling is typically bilateral. That's why the statement about papilledema being typically bilateral is the best answer. In contrast, severe visual loss early is not guaranteed—acuity can stay relatively good initially and visual symptoms often appear later or with sustained high pressure. The blind spot can be affected by disc edema, so the idea that it's never affected isn't correct. Paton's lines can be seen with papilledema, so saying they are never seen isn't accurate either.

6. Which phenomenon is the sensation of an electric shock that quickly travels down the back and into the limbs with flexure of the neck and is associated with Multiple Sclerosis?

- A. Lhermitte's phenomenon**
- B. Uhthoff's phenomenon
- C. Romberg sign
- D. Nystagmus

This is Lhermitte's sign. In multiple sclerosis, demyelination of the cervical spinal cord makes the dorsal columns vulnerable; when the neck is flexed, the stretched demyelinated fibers generate a sudden electric-shock sensation that travels down the spine into the limbs. The sensation is brief, reproducible with neck flexion, and serves as a clue to cervical spinal cord involvement in MS. Other options describe different phenomena: heat-related worsening of symptoms (Uhthoff's phenomenon), a balance test reflecting proprioceptive loss (Romberg sign), or involuntary eye movements (nystagmus), none of which capture the characteristic electric shock down the spine with neck flexion.

7. Foster Kennedy syndrome is a rare condition caused by a frontal lobe tumor and presents with simultaneous optic disc edema in one eye and optic disc atrophy in the fellow eye.

A. Foster Kennedy syndrome

B. Horner syndrome

C. Leber's optic neuropathy

D. Optic neuritis

Foster Kennedy syndrome is defined by an asymmetric optic neuropathy pattern produced by a frontal lobe mass: the eye on the same side as the tumor shows optic disc edema, while the other eye develops optic disc pallor or atrophy. This happens because the tumor directly irritates or compresses the ipsilateral optic nerve, causing edema from impaired axoplasmic flow and local inflammation. At the same time, the tumor can involve or compress structures near the optic chiasm, leading to degeneration of the crossing fibers and resulting in contralateral optic nerve atrophy over time. Anosmia may accompany the tumor, reinforcing the diagnosis. Other conditions don't produce this distinctive combination. Horner syndrome affects sympathetic pathways and causes ptosis, miosis, and anhidrosis, not a unilateral disc edema with opposite disc atrophy. Leber's optic neuropathy presents with subacute central vision loss, typically bilateral and without a mass lesion causing this asymmetrical disc pattern. Optic neuritis causes acute unilateral vision loss with pain and sometimes disc edema, but it does not create the contralateral atrophy pattern seen in Foster Kennedy syndrome.

8. In visual evoked potentials, what does P100 latency reflect?

A. The timing of conduction from retina to occipital cortex; delayed P100 latency indicates demyelination or conduction slowing

B. It measures retinal nerve fiber layer thickness

C. It indicates damage to optic disc

D. It measures acuity

P100 latency reflects the speed of neural conduction along the entire afferent visual pathway—from the retina through the optic nerve and beyond to the occipital cortex. The P100 is the prominent positive wave that peaks around 100 milliseconds after a visual stimulus, so its timing directly indicates how quickly the visual signal travels through the pathway. If the conduction is slowed, such as from demyelination along the optic nerve or along the optic radiations, the P100 peak occurs later than normal. This makes P100 latency a sensitive measure of conduction integrity in the visual system and a useful indicator of demyelinating processes. This measure differs from retinal nerve fiber layer thickness, which is a structural trait assessed by other imaging methods; from damage to the optic disc, which relates to structural loss at the optic nerve head; and from visual acuity, which assesses the clarity of vision rather than the speed of neural conduction along the pathway.

9. Which of the following is NOT a known risk factor for NAION?

A. Hypertension

B. Diabetes mellitus

C. Giant Cell Arteritis

D. Hyperlipidemia

Non-arteritic ischemic optic neuropathy happens when the optic nerve head is poorly perfused, and vascular risk factors heighten that risk. Hypertension, diabetes mellitus, and hyperlipidemia are all linked to vascular disease and contribute to reduced ocular blood flow, making them established risk factors for NAION. Giant Cell Arteritis, in contrast, causes arteritic AION through inflammatory occlusion of the arteries supplying the optic nerve. It's a different, inflammatory process, not a risk factor for the non-arteritic form. So the option that does not fit as a NAION risk factor is Giant Cell Arteritis.

10. In Myasthenia Gravis, autoantibodies are directed toward which component at the NMJ?

- A. Dopamine receptor
- B. Acetylcholine receptor**
- C. GABA receptor
- D. NMDA receptor

The main concept is that Myasthenia Gravis disrupts communication at the neuromuscular junction by attacking the postsynaptic receptors that respond to acetylcholine. Autoantibodies bind to acetylcholine receptors on the skeletal muscle side of the NMJ, blocking receptor activation, promoting internalization of receptors, and activating complement to damage the postsynaptic membrane. With fewer functional receptors, acetylcholine released from the nerve end cannot produce a sufficient end-plate potential to trigger a strong muscle contraction, leading to fatigable weakness that worsens with use. The other receptors listed (dopamine, GABA, NMDA) are located in the CNS and are not involved at the NMJ, so they aren't targets in MG. (Some patients may have antibodies against MuSK, another component of the postsynaptic apparatus, but the classic, most common target in MG is the acetylcholine receptor.)

SAMPLE

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:

<https://nbeoneuroscience.examzify.com>

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

SAMPLE