

AMERICAN BOARD OF PROFESSIONAL PSYCHOLOGY (ABPP) PRACTICE EXAM (SAMPLE) STUDY GUIDE



SAMPLE STUDY GUIDE WITH LIMITED QUESTIONS

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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.

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

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- 1. Occlusion of both anterior cerebral arteries causing frontal lobe infarction and left limb apraxia is known as?**
 - A. Anterior cerebral artery syndrome
 - B. Posterior circulation
 - C. Carotid artery TIA
 - D. Complex partial sz
- 2. Which disorder is caused by autosomal recessive deficiency in hepatic phenylalanine hydroxylase leading to cognitive and language delays, with newborn Guthrie screening detectable?**
 - A. Down syndrome
 - B. Phenylketonuria (PKU)
 - C. Angelman syndrome
 - D. Fragile X syndrome
- 3. Alien limb syndrome is reported to occur with corticobasal degeneration. Which option reflects this association?**
 - A. Progressive supranuclear palsy
 - B. Alzheimer's disease
 - C. Corticobasal degeneration
 - D. Left PCA stroke
- 4. Which condition features hypoplasia of the cerebellar vermis with expansion into a large cystic structure of the fourth ventricle in the posterior fossa?**
 - A. Arnold-Chiari malformation
 - B. Joubert syndrome
 - C. Dandy-Walker syndrome
 - D. Chiari II malformation
- 5. Which paraneoplastic syndrome is characterized by temporal lobe inflammation, amnesia, irritability, and complex partial seizures and is most commonly associated with gynecologic cancers, lymphoma, or small cell lung cancer?**
 - A. Paraneoplastic limbic encephalitis
 - B. Anti-NMDA receptor encephalitis
 - C. Cerebellar degeneration
 - D. Lambert-Eaton Myasthenic Syndrome

- 6. Anton syndrome is best described as denial of blindness in the setting of lesions to which region?**
- A. Frontal lobes
 - B. Blast injury to both eyes with bilateral occipital lobe strokes
 - C. Temporal lobes
 - D. Parietal lobes
- 7. Which type of FTD is not tau-positive?**
- A. bvFTD
 - B. PPA
 - C. PSP
 - D. FTD-MND
- 8. Huntington's disease is associated with an expanded CAG repeat on which chromosome?**
- A. Chromosome 14
 - B. Chromosome 4
 - C. Chromosome 21
 - D. Chromosome X
- 9. Naming ability is linked to which fiber tract?**
- A. Angular gyrus
 - B. BA 39
 - C. Arcuate fasciculus
 - D. Anterior superior temporal gyri
- 10. Acoustic neuroma is another term for vestibular schwannoma.**
- A. Meningioma
 - B. Schwannomatosis
 - C. Vestibular schwannoma
 - D. Oligodendroglioma

Answers

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

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Explanations

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1. Occlusion of both anterior cerebral arteries causing frontal lobe infarction and left limb apraxia is known as?

- A. Anterior cerebral artery syndrome**
- B. Posterior circulation
- C. Carotid artery TIA
- D. Complex partial sz

The main idea is that loss of blood flow in the anterior cerebral arteries produces a frontal lobe syndrome. When both ACAs are occluded, the medial frontal regions are infarcted, which leads to behavioral and motor planning deficits driven by the frontal lobes. This pattern includes contralateral leg weakness and impairment in coordinating purposeful movements, or apraxia, due to disruption of the motor planning circuits in the dominant hemisphere. So bilateral ACA infarction presenting with frontal lobe signs and limb apraxia fits the anterior cerebral artery syndrome. The other options don't align with this pattern: issues in the posterior circulation point to brainstem or cerebellar signs; a carotid TIA typically causes retinal or hemispheric symptoms depending on embolic flow but not a frontal lobe/apraxia profile; a complex partial seizure would present with episodic, ictal phenomena rather than a persistent infarct-related syndrome.

2. Which disorder is caused by autosomal recessive deficiency in hepatic phenylalanine hydroxylase leading to cognitive and language delays, with newborn Guthrie screening detectable?

- A. Down syndrome
- B. Phenylketonuria (PKU)**
- C. Angelman syndrome
- D. Fragile X syndrome

Phenylketonuria is an autosomal recessive metabolic disorder caused by a deficiency of hepatic phenylalanine hydroxylase, the enzyme that converts phenylalanine to tyrosine. When this enzyme is missing or defective, phenylalanine builds up in the blood and brain, leading to cognitive and language delays if not treated early. Newborn Guthrie screening detects elevated phenylalanine levels, enabling prompt dietary management to prevent intellectual disability; treatment typically involves a strict low-phenylalanine diet and ensuring adequate tyrosine, which becomes essential. Other conditions like Down syndrome, Angelman syndrome, and Fragile X syndrome arise from chromosomal or gene mechanisms with different inheritance patterns and are not identified by this newborn metabolic screen.

3. Alien limb syndrome is reported to occur with corticobasal degeneration. Which option reflects this association?

- A. Progressive supranuclear palsy
- B. Alzheimer's disease
- C. Corticobasal degeneration**
- D. Left PCA stroke

Alien limb syndrome arises when the brain's networks that plan and monitor movement become disrupted, especially in the parietal-premotor regions that integrate intention, action, and sensory feedback. In corticobasal degeneration, there is asymmetric involvement of those cortical motor-sensory areas, leading to apraxia and an alien limb that acts seemingly on its own and may resist the person's commands. This combination—unilateral, involuntary, purposeful movements with a sense of estrangement from the limb—is a characteristic feature of corticobasal degeneration. The other conditions listed do not typically present with this distinctive limb phenomenon: PSP mainly involves eye movements and axial rigidity, Alzheimer's centers on memory with different atrophy patterns, and a PCA stroke would cause focal deficits rather than the persistent, involuntary limb actions seen in CBD.

4. Which condition features hypoplasia of the cerebellar vermis with expansion into a large cystic structure of the fourth ventricle in the posterior fossa?

- A. Arnold-Chiari malformation
- B. Joubert syndrome
- C. Dandy-Walker syndrome**
- D. Chiari II malformation

This question tests recognition of a classic posterior fossa malformation where the cerebellar vermis is underdeveloped and the fourth ventricle expands into a large cyst. That combination defines Dandy-Walker malformation. In this condition, vermian hypoplasia or agenesis pairs with cystic dilation of the fourth ventricle, enlarging the posterior fossa and often elevating the tentorium. On imaging you'd expect to see a prominent cystic area where the fourth ventricle should be, along with an enlarged posterior fossa. This differentiates it from other posterior fossa anomalies: Arnold-Chiari malformation involves downward displacement of the cerebellar tonsils and brainstem rather than vermian hypoplasia with a fourth-ventricle cyst; Joubert syndrome shows midbrain-hindbrain malformations with the molar tooth sign; Chiari II malformation is typically associated with a myelomeningocele and a beaked tectum.

5. Which paraneoplastic syndrome is characterized by temporal lobe inflammation, amnesia, irritability, and complex partial seizures and is most commonly associated with gynecologic cancers, lymphoma, or small cell lung cancer?

- A. Paraneoplastic limbic encephalitis**
- B. Anti-NMDA receptor encephalitis
- C. Cerebellar degeneration
- D. Lambert-Eaton Myasthenic Syndrome

Paraneoplastic limbic encephalitis is an autoimmune syndrome in which the immune system targets limbic system structures, especially the medial temporal regions like the hippocampus and amygdala, due to cancer-related antigens. This leads to subacute onset of memory impairment (amnesia), mood and personality changes (irritability), and seizures that often have a temporal-lobe or complex partial pattern. The temporal lobe focus explained by limbic involvement accounts for both the memory problems and the seizure type described. This condition is classically associated with cancers such as gynecologic cancers, lymphoma, and small cell lung cancer, which is why it fits the vignette better than other paraneoplastic encephalitides. In contrast, anti-NMDA receptor encephalitis, while it can cause seizures and psychiatric symptoms, typically presents with prominent psychiatric manifestations and movement or autonomic symptoms and is often linked to ovarian teratoma; cerebellar degeneration presents with ataxia rather than temporal-lobe–predominant symptoms; Lambert-Eaton affects the neuromuscular junction with proximal weakness rather than encephalitis.

6. Anton syndrome is best described as denial of blindness in the setting of lesions to which region?

- A. Frontal lobes
- B. Blast injury to both eyes with bilateral occipital lobe strokes**
- C. Temporal lobes
- D. Parietal lobes

Anton syndrome is a form of cortical blindness with anosognosia for vision—the person is blind because the visual cortex is damaged, yet denies being blind. This pattern arises most clearly when both occipital lobes are injured, so the brain cannot process visual information despite intact eyes. Bilateral occipital damage disrupts visual awareness while leaving some language and confabulation processes intact, which explains the denial of blindness. Frontal, temporal, and parietal damage produce other deficits—frontal lobe issues often affect judgment and insight for nonvisual problems; temporal lobe injury leads to visual recognition problems; parietal lobe damage can cause visuospatial neglect. None of these produce the characteristic denial of blindness seen with occipital cortex damage, which is why bilateral occipital lobe injury best fits Anton syndrome.

7. Which type of FTD is not tau-positive?

- A. bvFTD
- B. PPA
- C. PSP
- D. FTD-MND**

In frontotemporal degeneration, the disease is linked to different protein pathologies. Tau-positive disorders include classic tauopathies like PSP, which show tau inclusions. Language and behavioral variants of FTD can be associated with either tau or TDP-43 pathology, depending on the subtype. The type that is not tau-positive is frontotemporal dementia with motor neuron disease, because this form is typically driven by TDP-43 (or FUS) pathology rather than tau. So, even though other FTD variants can sometimes involve tau, FTD-MND is characteristically non-tau and aligns with TDP-43-type pathology.

8. Huntington's disease is associated with an expanded CAG repeat on which chromosome?

- A. Chromosome 14
- B. Chromosome 4**
- C. Chromosome 21
- D. Chromosome X

Huntington's disease is caused by an expanded CAG trinucleotide repeat in the HTT gene, which sits on chromosome 4. Normal individuals typically have about 10–35 CAG repeats; when the repeats exceed roughly 36, the HTT protein gains a long polyglutamine tract that disrupts neuronal function, especially in the striatum, leading to the movement, cognitive, and behavioral signs of the disease. It's autosomal dominant, so a single mutated copy is enough to cause the disorder, with a 50% risk to offspring. Larger repeat sizes often mean earlier onset due to anticipation, particularly with paternal transmission. Because the HTT gene is on chromosome 4, chromosome 4 is the correct chromosomal location—unlike the other chromosomes listed.

9. Naming ability is linked to which fiber tract?

- A. Angular gyrus
- B. BA 39
- C. Arcuate fasciculus**
- D. Anterior superior temporal gyri

Naming depends on linking what you know about a concept with how you say the word, a process that relies on the dorsal language pathway connecting language comprehension regions to speech production areas. The arcuate fasciculus carries phonological information between Wernicke's area in the posterior temporal lobe and Broca's area in the frontal lobe, enabling phonological encoding and the articulation of lexical items. If this tract is compromised, the bridge between understanding a concept and producing the corresponding name weakens, leading to naming difficulties. The angular gyrus and anterior superior temporal regions are important for broader semantic processing, but naming performance specifically hinges on this dorsal connection between comprehension and production areas—the arcuate fasciculus.

10. Acoustic neuroma is another term for vestibular schwannoma.

- A. Meningioma
- B. Schwannomatosis
- C. Vestibular schwannoma**
- D. Oligodendroglioma

The concept at play is naming a tumor by its tissue of origin and nerve involvement. Acoustic neuroma is the historical or lay term for a tumor that actually arises from Schwann cells of the vestibulocochlear nerve. The correct anatomical name is vestibular schwannoma, since the tumor originates from the vestibular portion of cranial nerve VIII and is a Schwann cell–derived lesion. This distinction matters because it points to the nerve component affected and aligns with medical terminology. In the cerebellopontine angle and often extending into the internal auditory canal, vestibular schwannomas primarily disrupt hearing and balance, causing unilateral sensorineural hearing loss, tinnitus, and vertigo. Other options describe different entities: meningioma comes from the meninges, schwannomatosis refers to a condition with multiple schwannomas rather than this single lesion, and oligodendroglioma arises from oligodendrocytes within the brain parenchyma, not from cranial nerves.

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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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