

CAMLPR EVALUATION AND INTERPRETATION COMPETENCY PRACTICE TEST (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. In the G6PD deficiency case, which statement about the direct antiglobulin test (DAT) is true?**
 - A. The DAT is positive at RT
 - B. The DAT is negative
 - C. The DAT is positive at 37C
 - D. The DAT result is not reported

- 2. Lab tests on a patient with bone pain, recurrent kidney stones, abdominal discomfort and fatigue show Serum Mg 2.20 mmol/L, Ca 2.80 mmol/L, Phosphorus 0.70 mmol/L; Urine Ca INC, Urine Phos INC. What is the cause?**
 - A. Hyperparathyroidism
 - B. Hypoparathyroidism
 - C. Secondary hyperparathyroidism
 - D. Pseudohyperparathyroidism

- 3. Hashimoto's disease presents with which pattern of TSH and FT4?**
 - A. TSH markedly elevated with low FT4 and FT3
 - B. TSH suppressed with high FT4
 - C. Normal TSH with normal FT4
 - D. Elevated TSH with high FT4

- 4. A platelet transfusion recipient a week later presents with mucous membrane bleeding and epistaxis. What is the likely transfusion reaction and its treatment?**
 - A. Transfusion-associated graft-versus-host disease; irradiation.
 - B. Post-transfusion purpura; corticosteroids or IVIG.
 - C. Delayed hemolytic transfusion reaction; supportive care.
 - D. FNHTR; acetaminophen.

- 5. In cold agglutinin disease, the DAT is most commonly positive for which component?**
 - A. DAT positive for C3d only
 - B. DAT negative
 - C. DAT positive for IgG only
 - D. DAT positive for IgM

6. Which antibody group is destroyed by enzymes?
- A. MNSs
 - B. Duffy
 - C. Duffy, MNSs
 - D. Duffy, Fya
7. CBC shows WBC $14 \times 10^9/L$ with monoblasts, MPO POS, nonspecific esterase 90% POS, and cytogenetics with t(9;11) positive; flow cytometry shows CD14 and CD64. What is the presumptive diagnosis?
- A. Acute Erythroleukemia (M6)
 - B. Acute Monocytic Leukemia (M5)
 - C. Acute Lymphoblastic Leukemia (ALL L2)
 - D. Acute Promyelocytic Leukemia (APL)
8. A CBC is performed with results: Hgb 74 g/L; Hct 0.220; RBC $2.5 \times 10^{12}/L$; WBC $14 \times 10^9/L$; Platelets $480 \times 10^9/L$; MCV 92 fL; MCH 28 pg; MCHC 329 g/L; Retic 15.0%. Peripheral smear shows sickle cells and polychromasia. Additional testing shows TBIL 48 $\mu\text{mol}/L$; uBIL 40 $\mu\text{mol}/L$; LDH 1500 U/L. Sickle cell test is positive. Hb electrophoresis: HbS 88%; HbF 10%; HbA2 2%; HbA 0%. What is the presumptive diagnosis?
- A. Sickle cell disease
 - B. Sickle cell trait
 - C. Beta-thalassemia major
 - D. Hereditary spherocytosis
9. A child with sickle cell disease receives a transfusion and develops hypertension and edema with rapid pulse. What transfusion reaction is most likely and how should it be managed?
- A. Stop transfusion and give diuretics.
 - B. TACO; sit the patient up and slow the rate of transfusion.
 - C. TRALI; provide respiratory support.
 - D. PTP; corticosteroids or IVIG.

10. A diabetic patient presents with signs of severe metabolic acidosis and ketonuria; which diagnosis is most likely?

- A. Lactic acidosis due to sepsis
- B. Hyperosmolar hyperglycemic nonketotic syndrome
- C. Alcoholic ketoacidosis
- D. Diabetic ketoacidosis with Kussmaul respiration

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Answers

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

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Explanations

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1. In the G6PD deficiency case, which statement about the direct antiglobulin test (DAT) is true?

- A. The DAT is positive at RT
- B. The DAT is negative**
- C. The DAT is positive at 37°C
- D. The DAT result is not reported

The direct antiglobulin test detects antibodies or complement on the surface of red blood cells, helping differentiate immune from non-immune hemolysis. In G6PD deficiency, the hemolysis arises from oxidative injury to red cells, not from immune coating, so red cells are not marked by antibodies or complement. That's why the test is typically negative in this scenario. If the cause were immune-mediated, you'd expect a positive DAT, and the temperature at which it reacts (positive at 37°C for IgG, or at room temperature for certain cold antibodies) would reflect the type of immune hemolysis. A negative result here supports a non-immune mechanism of hemolysis, which fits G6PD deficiency.

2. Lab tests on a patient with bone pain, recurrent kidney stones, abdominal discomfort and fatigue show Serum Mg 2.20 mmol/L, Ca 2.80 mmol/L, Phosphorus 0.70 mmol/L; Urine Ca INC, Urine Phos INC. What is the cause?

- A. Hyperparathyroidism**
- B. Hypoparathyroidism
- C. Secondary hyperparathyroidism
- D. Pseudohyperparathyroidism

Elevated calcium with low phosphate and urinary calcium and phosphate loss points to excess parathyroid hormone activity on bone and kidney. In primary hyperparathyroidism, one gland overproduces PTH, often from an adenoma. PTH drives bone resorption and increases calcium reabsorption in the kidney while causing phosphate wasting. The result is high serum calcium, low serum phosphate, and hypercalciuria, which explains the kidney stones and bone pain. The other conditions don't fit this pattern: hypoparathyroidism would lower calcium and raise phosphate; secondary hyperparathyroidism usually has low or normal calcium with high PTH in CKD; pseudohyperparathyroidism has high PTH with low calcium due to PTH resistance. Thus, the cause is primary hyperparathyroidism.

3. Hashimoto's disease presents with which pattern of TSH and FT4?

- A. TSH markedly elevated with low FT4 and FT3**
- B. TSH suppressed with high FT4
- C. Normal TSH with normal FT4
- D. Elevated TSH with high FT4

Hashimoto's disease destroys thyroid tissue, leading to reduced thyroid hormone production. The pituitary senses the low hormones and elevates TSH in an attempt to stimulate the thyroid. So the common pattern is a markedly elevated TSH with low free T4 (and often low free T3). This is why the correct choice shows high TSH with low FT4. The other patterns point to different states: a hyperthyroid pattern would have suppressed TSH with high FT4, euthyroid would have normal TSH and FT4, and high TSH with high FT4 isn't typical for Hashimoto's.

4. A platelet transfusion recipient a week later presents with mucous membrane bleeding and epistaxis. What is the likely transfusion reaction and its treatment?

A. Transfusion-associated graft-versus-host disease; irradiation.

B. Post-transfusion purpura; corticosteroids or IVIG.

C. Delayed hemolytic transfusion reaction; supportive care.

D. FNHTR; acetaminophen.

Late-onset thrombocytopenia after a transfusion with mucosal bleeding points to post-transfusion purpura. This condition usually occurs about a week after transfusion and is driven by alloantibodies against platelet antigens (often HPA-1a) that cause the rapid destruction of platelets by the spleen and other reticuloendothelial tissues. The key clinical clue is a sudden, severe drop in platelets with mucous membrane bleeding, while coagulation tests remain normal, since the problem is platelet destruction rather than a coagulation factor defect. The treatment aims to stop the immune-mediated platelet destruction. High-dose corticosteroids or intravenous immunoglobulin (IVIG) are effective options: steroids suppress the immune response, and IVIG saturates Fc receptors on macrophages, reducing clearance of platelets. Platelet transfusions are often not helpful on their own because the transfused platelets can be destroyed by the same alloantibodies.

5. In cold agglutinin disease, the DAT is most commonly positive for which component?

A. DAT positive for C3d only

B. DAT negative

C. DAT positive for IgG only

D. DAT positive for IgM

This question hinges on how the direct antiglobulin test (DAT) appears in cold agglutinin disease, where the attack on red cells is driven primarily by IgM antibodies that fix complement. In cold agglutinin disease, IgM binds red cells at cold temperatures and activates the classical complement pathway, leaving complement fragments, especially C3 (C3b/C3d), attached to the red cell surface. The DAT uses reagents that detect either IgG or C3 on the surface of red cells. Because IgM is large and its binding is temperature-dependent, it is not reliably detected in standard DAT procedures, and it often dissociates at the warmer temperatures used in testing. In contrast, the C3 fragments that were deposited on the red cells remain and are detected by the DAT. As a result, the typical DAT pattern is positive for C3d (complement) and negative for IgG. So, the most consistent and characteristic finding is a DAT positive for C3d only, reflecting complement deposition rather than IgG coating.

6. Which antibody group is destroyed by enzymes?

- A. MNSs
- B. Duffy
- C. Duffy, MNSs**
- D. Duffy, Fya

Enzyme treatment of red blood cells alters or destroys certain surface antigens, changing antibody reactivity patterns. Proteolytic enzymes such as ficin, papain, or bromelain can cleave or alter some antigen sites, making them invisible to corresponding antibodies. Duffy antigens (Fy^a and Fy^b) are destroyed by enzyme exposure, so antibodies against Duffy will not react with enzyme-treated cells. In the MNSs system, the M and N antigens are also sensitive to enzymatic destruction, leading to loss of reactivity for anti-M and anti-N when the cells have been treated with enzymes. Because both Duffy and MNSs antigens are affected by enzymatic treatment, antibodies against these groups are described as being destroyed by enzymes. That's why the option listing both Duffy and MNSs is the best choice. Options that include only one of those groups or mix in a Duffy-only or redundant antigen like Fya don't capture the full enzyme-destroyed pattern.

7. CBC shows WBC $14 \times 10^9/L$ with monoblasts, MPO POS, nonspecific esterase 90% POS, and cytogenetics with t(9;11) positive; flow cytometry shows CD14 and CD64. What is the presumptive diagnosis?

- A. Acute Erythroleukemia (M6)
- B. Acute Monocytic Leukemia (M5)**
- C. Acute Lymphoblastic Leukemia (ALL L2)
- D. Acute Promyelocytic Leukemia (APL)

The key idea is recognizing monocytic differentiation in AML. The blasts show strong nonspecific esterase (NSE) activity, and flow cytometry confirms monocytic lineage with CD14 and CD64. NSE positivity is a classic hint for monocytes, while CD14 and CD64 are textbook monocyte markers. The t(9;11) cytogenetic abnormality is also commonly associated with AML with monocytic differentiation, reinforcing this origin. In contrast, ALL would predominantly show lymphoid markers (and TdT), APL is defined by the PML-RARA fusion t(15;17) and often Auer rods, and erythroleukemia would feature erythroid precursors with different marker patterns. Putting it together, these findings are most consistent with acute monocytic leukemia (AML M5).

8. A CBC is performed with results: Hgb 74 g/L; Hct 0.220; RBC $2.5 \times 10^{12}/L$; WBC $14 \times 10^9/L$; Platelets $480 \times 10^9/L$; MCV 92 fL; MCH 28 pg; MCHC 329 g/L; Retic 15.0%. Peripheral smear shows sickle cells and polychromasia. Additional testing shows TBIL 48 $\mu\text{mol}/L$; uBIL 40 $\mu\text{mol}/L$; LDH 1500 U/L. Sickle cell test is positive. Hb electrophoresis: HbS 88%; HbF 10%; HbA2 2%; HbA 0%. What is the presumptive diagnosis?

A. Sickle cell disease

B. Sickle cell trait

C. Beta-thalassemia major

D. Hereditary spherocytosis

The pattern fits homozygous sickle cell disease (HbSS). The electrophoresis shows predominant HbS at about 88%, with no HbA detected and a modest HbF elevation. In sickle cell trait you would expect a mix of HbA and HbS (HbA present), not virtually no HbA. The clinical picture supports chronic hemolysis: marked anemia with reticulocytosis, elevated bilirubin (especially unconjugated) and LDH, and a smear showing sickled cells with polychromasia. The normal MCV argues against beta-thalassemia major (which often has high HbF and altered indices) and the presence of sickled cells on smear and HbS predominance argues against hereditary spherocytosis (which would not show HbS). So this constellation points to sickle cell disease.

9. A child with sickle cell disease receives a transfusion and develops hypertension and edema with rapid pulse. What transfusion reaction is most likely and how should it be managed?

A. Stop transfusion and give diuretics.

B. TACO; sit the patient up and slow the rate of transfusion.

C. TRALI; provide respiratory support.

D. PTP; corticosteroids or IVIG.

Transfusion-associated circulatory overload (TACO) is the likely reaction here. It happens when the transfused volume overwhelms the heart's ability to manage it, leading to fluid overload with high blood pressure, edema, and a fast pulse after the transfusion. Management aims to reduce circulating volume and support breathing. Stop or slow the transfusion, place the patient upright to improve breathing, and give a diuretic to remove excess fluid. Monitor vitals closely and provide additional supportive care (oxygen, fluids management) as needed. This approach targets the excess volume causing the symptoms. This scenario differs from TRALI, which presents with acute respiratory distress and hypoxemia from noncardiogenic pulmonary edema, and from posttransfusion purpura, which involves immune-mediated platelet destruction with bleeding risks.

10. A diabetic patient presents with signs of severe metabolic acidosis and ketonuria; which diagnosis is most likely?

- A. Lactic acidosis due to sepsis
- B. Hyperosmolar hyperglycemic nonketotic syndrome
- C. Alcoholic ketoacidosis
- D. Diabetic ketoacidosis with Kussmaul respiration**

The key idea is that in a diabetic patient, severe metabolic acidosis with ketonuria points to diabetic ketoacidosis. Insulin deficiency leads to increased lipolysis and ketone production, causing metabolic acidosis with ketones detectable in urine. Kussmaul respiration shows the body's effort to compensate for this acidemia by blowing off CO₂, a hallmark of severe DKA. Other possibilities don't fit as well. Lactic acidosis from sepsis can cause acidosis but typically lacks significant ketone production. Hyperosmolar hyperglycemic nonketotic syndrome involves very high glucose with little or no ketones. Alcoholic ketoacidosis presents with ketoacidosis but usually in the setting of recent alcohol use and not in a known diabetic. Taken together, the scenario most consistent with the findings is diabetic ketoacidosis with Kussmaul respiration.

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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.

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

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