

HEMOSTASIS COAGULATION PRACTICE TEST (SAMPLE)

STUDY GUIDE



**SAMPLE STUDY GUIDE
WITH LIMITED QUESTIONS**

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THE FULL VERSION STUDY GUIDE**

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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. A 25-year-old male with celiac disease presents with occult positive stools. What vitamin deficiency should be considered?**
 - A. Vitamin A deficiency
 - B. Vitamin K deficiency
 - C. Vitamin D deficiency
 - D. Vitamin E deficiency
- 2. Which of the following platelet responses is most likely associated with type IIb von Willebrand disease?**
 - A. Decreased aggregation to low-dose ristocetin
 - B. Normal aggregation to low-dose ristocetin
 - C. Increased aggregation to low-dose ristocetin
 - D. Decreased aggregation to high-dose ristocetin
- 3. A patient presents with a low protein S activity and low total and free protein S antigens. The C4b binding protein is normal. This is classified as:**
 - A. type II
 - B. type III
 - C. type I
 - D. deficiency
- 4. Factor XII deficiency is typically associated with which bleeding history?**
 - A. Causes heavy bleeding in most patients
 - B. Has a negative bleeding history
 - C. Leads to low platelet count
 - D. Results in prolonged PT
- 5. The anticoagulant that directly inhibits thrombin is:**
 - A. LMWH
 - B. Argatroban
 - C. Warfarin
 - D. Rivaroxaban

- 6. Dabigatran is the direct oral anticoagulant that should be avoided in patients with renal impairment.**
- A. True
 - B. False
 - C. Only for severe impairment
 - D. Only for mild impairment
- 7. A 60-year-old man presents with a painful right knee and a slightly enlarged spleen. Which diagnosis is most compatible?**
- A. RA with reactive thrombocytosis
 - B. myelofibrosis
 - C. idiopathic thrombocythemia (essential or primary)
 - D. congenital spherocytosis
- 8. The differential diagnosis for a child with mucocutaneous bleeding, normal platelet count, and abnormal platelet function tests should include which condition?**
- A. Bernard-Soulier syndrome
 - B. Glanzmann thrombasthenia
 - C. Von Willebrand disease
 - D. Grey platelet syndrome
- 9. A hemophilic male and a normal female can produce which of the following?**
- A. male carrier
 - B. normal female
 - C. affected male
 - D. female carrier
- 10. Heparin resistance is most likely to occur with a deficiency of which protein?**
- A. Protein C
 - B. Protein S
 - C. Antithrombin
 - D. Factor VIII

Answers

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

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Explanations

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1. A 25-year-old male with celiac disease presents with occult positive stools. What vitamin deficiency should be considered?

- A. Vitamin A deficiency
- B. Vitamin K deficiency**
- C. Vitamin D deficiency
- D. Vitamin E deficiency

Fat malabsorption from celiac disease means fat-soluble vitamins A, D, E, and K can become deficient. Among these, vitamin K deficiency stands out because it directly impairs the body's ability to clot. Vitamin K is needed to activate clotting factors II, VII, IX, and X (and proteins C and S). Without enough vitamin K, the coagulation cascade is weakened, increasing bleeding risk. This can manifest as mucosal or GI bleeding, including occult blood in the stool. So when a patient with celiac disease develops occult positive stools, vitamin K deficiency and related coagulopathy is the most relevant deficiency to consider. Other fat-soluble vitamin deficiencies (A, D, E) cause vision, bone, or neurologic symptoms rather than bleeding tendencies, making them less likely explanations for occult stool positivity.

2. Which of the following platelet responses is most likely associated with type IIb von Willebrand disease?

- A. Decreased aggregation to low-dose ristocetin
- B. Normal aggregation to low-dose ristocetin
- C. Increased aggregation to low-dose ristocetin**
- D. Decreased aggregation to high-dose ristocetin

This question hinges on how a specific vWF abnormality alters its interaction with platelets in a ristocetin test. In type IIb von Willebrand disease, the vWF molecule has a gain-of-function change that makes it bind much more readily to the platelet receptor GPIIb. Ristocetin promotes the vWF-GPIIb interaction; when the vWF is hyperactive, even very low doses of ristocetin can cause platelets to cross-link and clump together. That produces an exaggerated aggregation response at low ristocetin concentrations—the hallmark finding for this type. Thrombocytopenia can accompany this pattern because those vWF-platelet complexes are cleared more rapidly, but the defining lab feature remains increased aggregation with low-dose ristocetin. Other patterns, like decreased aggregation or normal responses, would not reflect this heightened GPIIb-vWF interaction seen in type IIb. Hence, increased aggregation to low-dose ristocetin is the best fit.

3. A patient presents with a low protein S activity and low total and free protein S antigens. The C4b binding protein is normal. This is classified as:

- A. type II
- B. type III
- C. type I**
- D. deficiency

The main idea here is how protein S deficiency is categorized by how much protein S there is (quantity) and how well it works (function), taking into account the two forms it can take in the blood: free S (active) and S bound to C4b-binding protein (C4BP). The total protein S antigen reflects both free and bound S, while the free portion is the active cofactor for anticoagulation. In a type I deficiency, there is a quantitative shortage of protein S: both free and total antigen levels are low, and the functional activity is reduced. In a type II deficiency, the amount of protein S is normal, but its function is impaired (normal antigen, reduced activity). In a type III deficiency, total protein S may be normal, but free protein S is decreased because more S is bound to C4BP, leading to reduced activity as well; this scenario is typically accompanied by elevated C4BP levels that drive the shift toward binding. This patient has low total protein S antigen, low free protein S antigen, and low protein S activity, with normal C4BP. The pattern fits a quantitative deficiency (type I), since both antigen levels are reduced and the activity is low, and the normal C4BP argues against the type III pattern where free S is decreased due to excess binding to C4BP.

4. Factor XII deficiency is typically associated with which bleeding history?

- A. Causes heavy bleeding in most patients
- B. Has a negative bleeding history**
- C. Leads to low platelet count
- D. Results in prolonged PT

Factor XII deficiency prolongs laboratory tests of the intrinsic pathway (aPTT) but does not cause abnormal bleeding in real life. In vivo, hemostasis can proceed normally even without Factor XII, so most patients have a negative bleeding history. This makes the typical answer that there is no history of abnormal bleeding the best choice. Heavy bleeding is not expected because Factor XII is not essential for initial hemostasis. Platelet count isn't affected by this deficiency, so a low platelet count wouldn't be expected. PT is usually normal because Factor XII is part of the intrinsic pathway (measured by aPTT), not the extrinsic pathway (measured by PT).

5. The anticoagulant that directly inhibits thrombin is:

- A. LMWH
- B. Argatroban**
- C. Warfarin
- D. Rivaroxaban

The key idea here is how the drug interacts with thrombin. A direct thrombin inhibitor binds to thrombin itself and blocks its enzymatic activity, preventing it from converting fibrinogen to fibrin. Argatroban does exactly that: it directly inhibits thrombin, affecting both free thrombin and thrombin bound in clots. In contrast, other options act indirectly or target a different factor. Low-molecular-weight heparin enhances the activity of antithrombin to mainly inhibit factor Xa, with only a limited direct effect on thrombin. Warfarin reduces the synthesis of several vitamin K-dependent coagulation factors, not thrombin directly. Rivaroxaban inhibits factor Xa directly, not thrombin. Thus, Argatroban is the direct thrombin inhibitor.

6. Dabigatran is the direct oral anticoagulant that should be avoided in patients with renal impairment.

A. True

B. False

C. Only for severe impairment

D. Only for mild impairment

The main idea here is that dabigatran relies heavily on the kidneys to be cleared from the body. About 80% of dabigatran is excreted renally as the active drug. When kidney function is reduced, the drug stays in the bloodstream longer, raising its levels and heightening the risk of bleeding. Because of this, dabigatran is avoided in renal impairment, especially in more severe cases where the drug can accumulate to dangerous levels. In mild impairment you might still use it with caution or adjust the dose in some guidelines, but the general approach is to avoid it when renal function is decreased. This is why the statement is considered true.

7. A 60-year-old man presents with a painful right knee and a slightly enlarged spleen. Which diagnosis is most compatible?

A. RA with reactive thrombocytosis

B. myelofibrosis

C. idiopathic thrombocythemia (essential or primary)

D. congenital spherocytosis

Splenomegaly points to processes that involve sequestration or extramedullary hematopoiesis. In hereditary (congenital) spherocytosis, a defect in red blood cell membrane makes the cells spherical and fragile. These abnormal cells are destroyed primarily in the spleen, leading to chronic extravascular hemolysis and a noticeably enlarged spleen. Even in older adults, this splenic sequestration of destroyed red cells can be the dominant clue, without requiring dramatic other findings. The other options fit less well. Reactive thrombocytosis from rheumatoid arthritis would emphasize inflammatory changes and high platelets, but splenomegaly isn't a defining feature. Myelofibrosis does cause splenomegaly and bone/bone-marrow-related pain, but usually comes with more characteristic blood smear findings (like teardrop red cells) and a broader systemic picture. Idiopathic (essential) thrombocytosis often has prominent thrombocytosis and splenomegaly too, but the presentation here centers on splenic sequestration of fragile red cells rather than a primary platelet disorder. Congenital spherocytosis best explains splenomegaly as a central finding in the vignette, making it the most compatible diagnosis.

8. The differential diagnosis for a child with mucocutaneous bleeding, normal platelet count, and abnormal platelet function tests should include which condition?

- A. Bernard-Soulier syndrome
- B. Glanzmann thrombasthenia**
- C. Von Willebrand disease
- D. Grey platelet syndrome

A qualitative platelet function defect is suggested when mucocutaneous bleeding occurs but the platelet count is normal. In this scenario, the problem isn't lacking platelets but platelets that can't clump together effectively. Glanzmann thrombasthenia fits this pattern because it is caused by a deficiency of the platelet fibrinogen receptor, GPIIb/IIIa. Without this receptor, platelets cannot cross-link via fibrinogen, so they fail to aggregate in response to most agonists (such as ADP, collagen, and epinephrine). The result is markedly reduced or absent platelet aggregation on testing, yet the ristocetin-induced aggregation is preserved because that pathway relies on the GPIb-vWF interaction, not GPIIb/IIIa. Clinically, this presents as mucocutaneous bleeding with a normal platelet count. Bernard-Soulier would more likely show a low or abnormal platelet count with large platelets and defective ristocetin aggregation, which doesn't fit the normal-count scenario. Von Willebrand disease can also cause mucocutaneous bleeding with a normal platelet count and abnormal ristocetin testing, but the broader pattern of impaired aggregation to multiple agonists in Glanzmann helps distinguish it. Grey platelet syndrome involves a qualitative granule defect and has its own distinct lab features.

9. A hemophilic male and a normal female can produce which of the following?

- A. male carrier
- B. normal female
- C. affected male
- D. female carrier**

X-linked recessive inheritance drives this outcome. An affected male has X^hY . A normal female has X^NX^N . Fathers pass their X to daughters and their Y to sons. So all daughters receive the mutated X from the father and a normal X from the mother, giving X^hX^N —female carriers who are usually not affected. Sons receive the normal X from the mother and the Y from the father, giving X^NY , which is normal. Therefore, with a hemophilic male and a normal female, the offspring can be female carriers only. If the mother were a carrier as well, additional possibilities like affected daughters or affected sons could occur.

10. Heparin resistance is most likely to occur with a deficiency of which protein?

- A. Protein C
- B. Protein S
- C. Antithrombin**
- D. Factor VIII

Heparin works by binding to antithrombin and greatly boosting its ability to inactivate clotting factors, especially thrombin and Factor Xa. If antithrombin is deficient, there's less heparin–antithrombin complex formed, so the anticoagulant effect is blunted and you need higher heparin doses to reach the target effect—this is heparin resistance. Antithrombin deficiency can be inherited or acquired (e.g., nephrotic syndrome, liver disease). The other proteins mentioned are anticoagulants (protein C and protein S) or a coagulation factor (Factor VIII) whose deficiencies or elevations don't specifically explain reduced responsiveness to heparin in the same way. So the deficiency most likely to cause heparin resistance is antithrombin.

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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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