

Hemostasis Coagulation Practice Test (Sample)

Study Guide



Everything you need from our exam experts!

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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. When there is a disparity between fibrinogen antigen and activity, the most likely diagnosis is which?**
 - A. Dysfibrinogenemia**
 - B. Hypofibrinogenemia**
 - C. Hyperfibrinogenemia**
 - D. Afibrinogenemia**

- 2. Which laboratory pattern is most consistent with Factor XIII deficiency?**
 - A. Prolonged PT, prolonged aPTT**
 - B. Normal PT, normal aPTT, normal fibrinogen, normal platelets**
 - C. Elevated fibrin degradation products**
 - D. Thrombocytopenia**

- 3. Which finding is characteristic of acute DIC?**
 - A. Thrombocytosis**
 - B. Negative D-dimer**
 - C. Hypofibrinogenemia**
 - D. Shortened thrombin time**

- 4. In the Clauss fibrinogen method, the time to clot formation in plasma is measured after the addition of:**
 - A. Calcium**
 - B. Thrombin**
 - C. Phospholipids**
 - D. Kaolin**

- 5. What does the D-Dimer assay detect?**
 - A. Fibrin Degradation Products**
 - B. Fibrin Monomer**
 - C. Prothrombin Fragments**
 - D. Platelet-Derived Factors**

- 6. A 70-year-old female with metastatic ovarian carcinoma shows elevated PT and aPTT, platelets 15,000/mm³ and an elevated D-dimer. The differential diagnosis should include:**
- A. TTP**
 - B. APS**
 - C. DIC**
 - D. HUS**
- 7. Which vWD type shows enhanced ristocetin-induced aggregation activity?**
- A. type 2A**
 - B. type 2B**
 - C. type 2M**
 - D. type 2N**
- 8. The key enzyme of the fibrinolytic system is:**
- A. Thrombin**
 - B. Urokinase**
 - C. Plasmin**
 - D. Streptokinase**
- 9. Which statement correctly describes aspirin's mechanism of action?**
- A. Inactivating cyclooxygenase which blocks thromboxane A₂**
 - B. Antagonizes platelet P₂Y₁₂ receptors**
 - C. Inhibits GPIb receptor binding**
 - D. Inhibits fibrinogen binding to GPIIb/IIIa**
- 10. The major serine protease responsible for clot breakdown is:**
- A. TPA**
 - B. Alpha 2 Antiplasmin**
 - C. Streptokinase**
 - D. PAI-1**

Answers

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

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Explanations

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1. When there is a disparity between fibrinogen antigen and activity, the most likely diagnosis is which?

- A. Dysfibrinogenemia**
- B. Hypofibrinogenemia**
- C. Hyperfibrinogenemia**
- D. Afibrinogenemia**

Disparity between the amount of fibrinogen present (antigen) and how well it functions (activity) points to a qualitative fibrinogen defect. In dysfibrinogenemia, the fibrinogen molecule is structurally abnormal, so its ability to convert to fibrin is impaired even if the protein level is normal or near normal. That mismatch between normal or high antigen and reduced functional activity is the hallmark. By contrast, quantitative disorders like hypofibrinogenemia or afibrinogenemia show proportional changes in both antigen and activity (reduced or absent), and hyperfibrinogenemia shows increased antigen with normal or proportionally normal activity. So the mismatch specifically indicates dysfibrinogenemia.

2. Which laboratory pattern is most consistent with Factor XIII deficiency?

- A. Prolonged PT, prolonged aPTT**
- B. Normal PT, normal aPTT, normal fibrinogen, normal platelets**
- C. Elevated fibrin degradation products**
- D. Thrombocytopenia**

Factor XIII deficiency mainly affects how a formed fibrin clot is stabilized, not how the clot is made. PT and aPTT measure the initiation and propagation of coagulation up to fibrin formation, while Factor XIII acts after fibrin is formed to crosslink the fibers and strengthen the clot. Because the early steps of coagulation and the amount of fibrin being produced are normal, the standard tests like PT and aPTT remain normal. Fibrinogen level and platelet count reflect quantity of substrate and cells involved in clot formation, not the stabilization step, so they stay normal as well. A lab test that would hint at Factor XIII deficiency is a clot solubility test, where the clot dissolves in certain solvents due to lack of crosslinking, but that test isn't part of routine panels. Patterns showing prolonged PT or aPTT would point to deficiencies in the coagulation factors that drive clot formation rather than stabilization. Elevated fibrin degradation products suggest excessive fibrinolysis, as seen in conditions like DIC or severe fibrinolytic states. Thrombocytopenia indicates a problem with platelets. Therefore, the presentation with normal PT, normal aPTT, normal fibrinogen, and normal platelets best fits Factor XIII deficiency, which disrupts clot stability without altering the initial formation phase.

3. Which finding is characteristic of acute DIC?

- A. Thrombocytosis
- B. Negative D-dimer
- C. Hypofibrinogenemia**
- D. Shortened thrombin time

In acute DIC, widespread activation of coagulation consumes platelets and coagulation factors, especially fibrinogen, so levels of fibrinogen fall. This consumption of fibrinogen, or hypofibrinogenemia, is a hallmark of acute DIC. You wouldn't expect thrombocytosis; platelets are used up, leading to low platelets. D-dimer would be elevated due to increased fibrin formation and breakdown, not negative. Thrombin time is typically prolonged in this setting because low fibrinogen and fibrin degradation products interfere with clot formation, not shortened.

4. In the Clauss fibrinogen method, the time to clot formation in plasma is measured after the addition of:

- A. Calcium
- B. Thrombin**
- C. Phospholipids
- D. Kaolin

In the Clauss fibrinogen method, the key idea is to trigger the last step of clot formation directly by using thrombin. You add a large excess of thrombin to plasma, and the time it takes for the plasma to clot depends on how much functional fibrinogen is present. Thrombin is the enzyme that cleaves fibrinogen into fibrin, so the rate-limiting step becomes the amount of fibrinogen itself. Therefore, the shorter the clotting time, the higher the fibrinogen; a longer time indicates lower fibrinogen. Calcium alone would initiate many coagulation reactions but does not directly convert fibrinogen to fibrin, so it doesn't make the measurement specifically reflect fibrinogen levels. Phospholipids provide a surface to support coagulation complexes but don't determine fibrinogen concentration. Kaolin activates the contact pathway and would introduce variability from other coagulation factors, masking the specific relation to fibrinogen. By focusing on thrombin-induced conversion, the Clauss method provides a fibrinogen-specific assessment.

5. What does the D-Dimer assay detect?

- A. Fibrin Degradation Products**
- B. Fibrin Monomer
- C. Prothrombin Fragments
- D. Platelet-Derived Factors

D-dimer assay detects fibrin degradation products that arise when a fibrin clot is broken down by plasmin. Specifically, it targets cross-linked fibrin fragments that contain two D domains—the D-dimer. This is a marker of recent clot formation and breakdown, not of fibrin monomer, prothrombin fragments, or platelet-derived factors. Clinically, a negative D-dimer helps rule out active thrombosis in appropriate patients, while elevated levels can be seen in various conditions that cause clotting and fibrinolysis.

6. A 70-year-old female with metastatic ovarian carcinoma shows elevated PT and aPTT, platelets 15,000/mm³ and an elevated D-dimer. The differential diagnosis should include:

- A. TTP
- B. APS
- C. DIC**
- D. HUS

Systemic activation of coagulation with consumption of platelets and clotting factors produces a mixed bleeding-and-thrombosis picture. In DIC, a trigger such as metastatic cancer starts widespread intravascular coagulation, which uses up platelets and coagulation factors, so PT and aPTT become prolonged and platelets drop, while fibrin is formed and then degraded, raising the D-dimer. This combination—prolonged PT and aPTT, severe thrombocytopenia, and elevated D-dimer in a patient with malignancy—is classic for DIC. TTP and HUS involve microangiopathic hemolytic anemia with thrombocytopenia, but coagulation tests like PT and aPTT are typically normal. APS can cause thrombosis and may show a prolonged aPTT because of lupus anticoagulant, but it does not usually present with the consumptive coagulopathy pattern seen in DIC or with such marked thrombocytopenia and D-dimer elevation.

7. Which vWD type shows enhanced ristocetin-induced aggregation activity?

- A. type 2A
- B. type 2B**
- C. type 2M
- D. type 2N

Ristocetin-induced aggregation shows how von Willebrand factor binds to platelet GPIb to link platelets together. In this test, ristocetin promotes the VWF-GPIb interaction that drives platelet clumping. A gain-of-function mutation in von Willebrand factor found in type 2B increases its affinity for GPIb, so platelets aggregate more readily in the presence of ristocetin than normal. This enhanced aggregation is characteristic of type 2B and can be accompanied by thrombocytopenia due to clearance of platelet-VWF complexes. Other vWD types alter ristocetin-induced aggregation differently: type 2A tends to reduce aggregation because high-molecular-weight multimers that best bridge VWF to platelets are lost; type 2M has defective platelet binding without the multimer defect, reducing aggregation; type 2N involves poor binding to FVIII, not platelet adhesion, so it does not cause enhanced RIPA.

8. The key enzyme of the fibrinolytic system is:

- A. Thrombin**
- B. Urokinase**
- C. Plasmin**
- D. Streptokinase**

The fibrinolytic system is driven by plasmin, the enzyme that actually digests fibrin and dissolves clots. Plasmin is produced from its inactive form, plasminogen, when activators such as tissue plasminogen activator, urokinase, or streptokinase stimulate the conversion. Thrombin belongs to coagulation and promotes clot formation rather than breakdown. Urokinase and streptokinase are activators that trigger plasminogen to become plasmin, but they are not the enzymatic agents that degrade fibrin themselves. Therefore, plasmin is the best answer because it directly performs fibrinolysis.

9. Which statement correctly describes aspirin's mechanism of action?

- A. Inactivating cyclooxygenase which blocks thromboxane A2**
- B. Antagonizes platelet P2Y12 receptors**
- C. Inhibits GPIb receptor binding**
- D. Inhibits fibrinogen binding to GPIIb/IIIa**

Aspirin works by irreversibly inhibiting cyclooxygenase-1 in platelets, which blocks the production of thromboxane A₂, a powerful promoter of platelet activation and aggregation. Because platelets lack nuclei, they cannot synthesize new COX-1, so the effect lasts for the platelet's lifetime, giving an lasting antiplatelet effect at low doses. The other mechanisms described belong to different drugs: blocking the ADP receptor (P2Y₁₂) interferes with ADP signaling rather than COX-1; inhibiting GPIb would affect von Willebrand factor binding; and preventing fibrinogen from binding to GPIIb/IIIa targets later steps of aggregation, not the COX-1-thromboxane pathway that aspirin acts on.

10. The major serine protease responsible for clot breakdown is:

- A. tPA**
- B. Alpha 2 Antiplasmin**
- C. Streptokinase**
- D. PAI-1**

Fibrinolysis dissolves clots through plasmin, a serine protease that cleaves fibrin to break down the clot. The body's main trigger for generating plasmin is tissue plasminogen activator, a serine protease released by endothelial cells that converts plasminogen to plasmin directly at the clot site. This makes tissue plasminogen activator the key enzyme initiating clot breakdown. Alpha-2 antiplasmin inhibits plasmin, so it protects clots rather than driving breakdown. PAI-1 inhibits the activators (including tPA and uPA), reducing fibrinolysis rather than acting as the protease. Streptokinase activates plasminogen indirectly but is not a human serine protease; it forms a complex to promote plasmin formation, whereas the principal serine protease driving the process is tPA. So, the best answer is tissue plasminogen activator.

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://hemostasiscoag.examzify.com>

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

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