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CHAPTER 3

GASTROINTESTINAL BLEEDING

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Dedication

In loving memory of my mother,

who recently returned to the mercy of Allah.

Her love, prayers, patience, and sacrifices were among the greatest blessings of my life. Whatever good I achieve carries something of her guidance and support.

May Allah forgive her, have mercy upon her, illuminate her grave, and grant her the highest place in **Jannat al-Firdaws**.

This work is dedicated to her memory.

أسأل الله أن يرحم أمي رحمة واسعه وأن يدخلها الفردوس الأعلى من الجنة، ورجاءً أسألكم لها خالص الدعاء..

Chapter 3: Gastrointestinal Bleeding

Upper GI Bleeding • Lower GI Bleeding • Small Bowel Bleeding

50 original consultant-level mechanism-based MCQs with randomized correct answer positions and colored high-yield keywords.

Diagnosis / lesion	Mechanism / biology	Therapy / procedure	Biomarker / decision point

Mechanism-Based Questions

1. Nonvariceal UGIB and clot biology

A 68-year-old man presents with **melena** and syncope. Endoscopy reveals a duodenal ulcer with a nonbleeding **visible vessel** that is treated with **clips** and **thermal coagulation**. A high-dose intravenous **PPI** is continued for 72 hours.

Which mechanism best explains the post-endoscopic PPI strategy?

A. It directly activates platelet glycoprotein IIb/IIIa at the ulcer base
B. It reduces **portal pressure** by **splanchnic vasoconstriction**
C. It maintains intragastric **pH greater than 6**, improving **clot stability** and limiting **fibrinolysis**
D. It chelates intraluminal iron and reduces oxidative mucosal injury
E. It blocks H2 receptors and prevents **tachyphylaxis**

Answer: C

Mechanism: Peptic ulcer bleeding rebleeding is promoted by acid-mediated platelet dysfunction and clot lysis. High-dose **PPI** therapy stabilizes the treated ulcer base by maintaining **pH >6**; it is **not** a vasoactive portal-pressure drug.

2. Erythromycin before urgent endoscopy

A patient arrives with large-volume **hematemesis**. After **resuscitation**, the team plans **urgent** EGD. The stomach is likely full of blood and clot, and **erythromycin** is given intravenously before endoscopy.

What is the most accurate mechanism-based rationale?

A. It inhibits gastric **H+/K+-ATPase** and prevents **recurrent** bleeding
B. It activates the **motilin receptor**, promoting gastric emptying and improving visualization
C. It antagonizes dopamine D2 receptors to increase lower esophageal sphincter pressure
D. It neutralizes thrombin at the ulcer base
E. It induces **splanchnic vasoconstriction** like **octreotide**

Answer: B

Mechanism: Pre-endoscopic **erythromycin** is used as a prokinetic. **Motilin receptor** stimulation empties retained blood and clot, improving diagnostic and therapeutic yield during EGD.

3. Restrictive transfusion and portal-pressure physiology

A cirrhotic man with suspected **variceal bleeding** is resuscitated. His **hemoglobin** is 7.2 g/dL, blood pressure has stabilized, and the junior doctor wants to transfuse rapidly to **hemoglobin** 11 g/dL.

Which pathophysiologic concern supports a restrictive transfusion strategy in this setting?

A. Red cells directly inhibit platelet aggregation at the variceal plug
B. **Transfusion** irreversibly blocks endogenous vasopressin release
C. Higher **hemoglobin** always lowers **portal pressure** by increasing viscosity
D. **Overt**transfusion may increase **portal pressure** and disrupt **early** hemostasis
E. Packed red cells worsen bleeding only through citrate-induced alkalosis

Answer: D

Mechanism: In portal hypertensive bleeding, excessive volume expansion can raise **portal pressure** and increase rebleeding. **Resuscitation** must restore perfusion without driving portal venous pressure upward.

4. Aspirin after ulcer bleeding

A 72-year-old man with coronary stents presents with **peptic ulcer bleeding** while taking low-dose aspirin. Endoscopic hemostasis is successful. The team debates stopping aspirin for several weeks.

Which mechanism best frames the risk-benefit decision?

A. Aspirin-induced bleeding is mediated only by reversible platelet inhibition
B. Stopping aspirin immediately restores platelet function in all circulating **platelets**
C. Aspirin protects ulcers by increasing **prostaglandin** synthesis

D. Low-dose aspirin irreversibly inhibits platelet **COX-1**, but prolonged interruption increases thrombotic risk in secondary prevention
E. Aspirin rebleeding risk is unrelated to platelet thromboxane biology

Answer: D

Mechanism: Aspirin causes irreversible platelet **COX-1** inhibition. After hemostasis, secondary cardiovascular prevention often requires **early** resumption because thrombotic harm may outweigh incremental rebleeding risk.

5. NSAID ulcer bleeding

A 64-year-old woman taking naproxen for osteoarthritis presents with **hematemesis**. Endoscopy shows a bleeding gastric ulcer. H. pylori testing is **negative**.

Which mechanism most directly explains NSAID-related ulcer bleeding?

A. Direct activation of gastric proton pumps with severe acid hypersecretion
B. Inhibition of COX-derived **prostaglandin**s, reducing **mucus**, **bicarbonate**, and **mucosal blood flow**
C. IgE-mediated mast-cell injury of the gastric mucosa
D. Portal-pressure elevation causing mucosal venous e**cta**sia
E. Bacterial beta-glucuronidase deconjugation of bilirubin in the stomach

Answer: B

Mechanism: NSAIDs injure mucosa through topical effects and systemic COX inhibition. Loss of **prostaglandin**-mediated **mucus**, **bicarbonate** secretion, and **mucosal blood flow** predisposes to ulceration and bleeding.

6. H. pylori eradication after bleeding ulcer

A 39-year-old man with duodenal ulcer bleeding is found to have H. pylori. He receives endoscopic hemostasis and **PPI** therapy. Eradication therapy is planned after stabilization.

Which mechanism best explains why eradication reduces future ulcer bleeding?

A. It directly seals the ulcer vessel by polymerization
B. It produces acute **splanchnic vasoconstriction**
C. It permanently inhibits all parietal-cell proton pumps
D. It blocks platelet **COX-1** inhibition
E. It removes chronic gastritis-driven acid-peptic and mucosal injury that promotes **recurrent** ulceration

Answer: E

Mechanism: H. pylori eradication addresses the causal inflammatory driver of **recurrent peptic ulcer** disease. **PPI** stabilizes acute clot biology, but eradication prevents recurrence.

7. Adherent clot at ulcer base

Endoscopy for **melena** reveals a gastric ulcer with an **adherent clot**. Vigorous irrigation does **not** fully detach it. The fellow suggests leaving it untreated because there is no active spurting.

What is the best mechanism-based response?

A. An **adherent clot** is always equivalent to a clean-base ulcer
B. Only **epinephrine** injection is needed because it creates permanent hemostasis
C. The clot may cover a **high-risk** artery; clot-directed therapy aims to expose or secure the underlying vessel
D. Thermal therapy is **contraindicated** for all gastric ulcers
E. **PPI** therapy alone instantly thromboses the **visible vessel**

Answer: C

Mechanism: An **adherent clot** may conceal a **visible vessel**. Injection alone is temporary; definitive hemostasis usually requires mechanical or thermal treatment when **high-risk** stigmata are present.

8. Hemostatic powder as bridge therapy

A patient with diffuse malignant gastric bleeding has oozing over a broad friable tumor surface. **Hemostatic powder** is applied with immediate reduction in bleeding.

Which statement best describes the mechanism and limitation?

A. It creates a topical barrier and concentrates clotting factors but may be temporary when definitive vessel therapy is impossible
B. It selectively lowers **portal pressure** like **TIPS**
C. It permanently ligates submucosal arteries like an **over-the-scope clip**
D. It reverses anticoagulation by degrading factor Xa inhibitors
E. It requires acid activation in parietal-cell canaliculi

Answer: A

Mechanism: **Hemostatic powder** is useful for broad oozing lesions or as rescue/bridging therapy. Its action is topical and barrier-like rather than precise durable arterial closure.

9. Over-the-scope clip in recurrent ulcer bleeding

A 76-year-old man has **recurrent** bleeding from a fibrotic posterior duodenal ulcer despite prior standard **clips** and injection therapy. **OTSC** is selected during repeat endoscopy.

Why is OTSC mechanistically attractive here?

A. It acidifies the stomach to promote **clot stability**
B. It induces motilin-mediated gastric emptying
C. It blocks vasopressin V1 receptors
D. It provides stronger full-thickness mechanical compression of a fibrotic ulcer base and underlying vessel
E. It selectively treats **portal hypertensive gastropathy** by reducing **portal pressure**

Answer: D

Mechanism: Recurrent peptic ulcer bleeding may arise from a large artery in a fibrotic base. **OTSC** provides robust mechanical compression when standard **clips** or thermal therapy fail.

10. Forrest low-risk stigmata

A 55-year-old woman undergoes EGD for **melena**. A duodenal ulcer has a **clean base** with no **visible vessel**, no clot, and no active bleeding.

Which strategy best follows from the mechanism of rebleeding risk?

A. Urgent angiographic **embolization** because all ulcers have equal rebleeding risk
B. No endoscopic hemostasis; standard acid suppression and **early** discharge can be considered if clinically stable
C. Cyanoacrylate injection into the ulcer base
D. TIPS because clean-base ulcers reflect portal hypertension
E. Hemostatic powder followed by mandatory **surgery**

Answer: B

Mechanism: Clean-base and flat pigmented ulcers have **low-risk** stigmata. **Endoscopic therapy** is reserved for active bleeding, **visible vessel**, or selected **adherent clot** lesions.

11. Dieulafoy lesion

A 61-year-old man has sudden **massive hematemesis**. EGD shows a tiny mucosal defect in the proximal stomach with pulsatile arterial bleeding but no surrounding ulcer crater.

Which mechanism best explains the lesion?

A. Portal hypertension causing diffuse mucosal mosaic congestion
B. Bacterial deconjugation of bilirubin causing pigment precipitation
C. A **large-caliber submucosal artery** eroding through a minute mucosal defect
D. TGF-beta-driven antral vascular ectasia
E. Acid-induced mucosal laceration after retching

Answer: C

Mechanism: A **Dieulafoy lesion** is a **large-caliber submucosal artery** that bleeds through a small mucosal defect, often causing dramatic bleeding with minimal surrounding mucosal disease.

12. Mallory-Weiss bleeding

A 44-year-old man develops **hematemesis** after repeated vomiting. EGD shows a linear tear at the gastroesophageal junction with mild oozing.

Which mechanism best explains the bleeding?

A. Chronic stasis inflammation from achalasia causing squamous carcinoma
B. Direct **portal pressure** transmission to gastric fundal varices
C. Ectopic gastric mucosa causing acid ulceration in a diverticulum
D. A mucosal laceration from sudden pressure rise during retching at the gastroesophageal junction
E. COX-1 inhibition reducing gastric **prostaglandin**s

Answer: D

Mechanism: Mallory-Weiss tear is a mucosal laceration caused by forceful retching. It is distinct from Boerhaave syndrome, which is transmural rupture.

13. Cameron lesions

A 73-year-old woman with a large hiatal hernia has chronic **iron deficiency anemia** and intermittent **melena**. EGD reveals linear erosions at the diaphragmatic impression.

Which mechanism best explains the lesion?

A. Mechanical trauma and local ischemia where the herniated stomach slides through the diaphragm
B. IL-13-mediated eosinophilic remodeling

C. Aortic stenosis causing acquired **von Willebrand factor** deficiency
D. Pancreatic pseudoaneurysm bleeding into the pancreatic duct
E. Portal hypertensive venous congestion of the entire stomach
Answer: A
Mechanism: Cameron lesions occur in large hiatal hernias due to mechanical rubbing and ischemia at the diaphragmatic pinch, causing **occult** or **overt** upper GI bleeding.

14. GAVE versus portal hypertensive gastropathy

A cirrhotic patient has chronic **transfusion**-dependent **iron deficiency anemia**. Endoscopy shows red stripes radiating from the pylorus in the antrum. The fellow suggests **TIPS** as first-line therapy.
Which correction is most appropriate?
A. GAVE is caused by **esophageal varices** and should be banded
B. GAVE responds primarily to H. pylori eradication
C. GAVE is a proximal gastric disease caused entirely by **portal pressure**
D. GAVE should be treated only with nonselective beta blockers
E. GAVE is an antral vascular ectasia; bleeding is **not** primarily portal-pressure driven and **APC** or other endoscopic ablation is preferred
Answer: E
Mechanism: GAVE can coexist with cirrhosis but is **not** simply **portal hypertensive gastropathy**. Portal decompression is **not** the main therapy; endoscopic ablation such as **APC** is commonly used.

15. Portal hypertensive gastropathy

A man with decompensated cirrhosis has chronic **melena**. EGD shows a snakeskin mosaic pattern in the fundus and body without antral stripes.
Which mechanism best explains the bleeding source?
A. A **large-caliber submucosal artery** crossing a tiny mucosal defect
B. Acid-peptic erosion of a hiatal hernia at the diaphragm
C. Portal-pressure-related mucosal and submucosal vascular congestion
D. NSAID-induced diaphragm disease of the small bowel
E. Ectopic gastric mucosa inside **Meckel diverticulum**
Answer: C
Mechanism: Portal hypertensive gastropathy reflects portal-pressure-driven mucosal congestion, usually in the proximal stomach. Therapy targets **portal pressure** rather than antral ablation.

16. Variceal bleeding and vasoactive therapy

A patient with cirrhosis presents with **hematemesis** and suspected esophageal **variceal bleeding**. **Octreotide** is started immediately before EGD.
Which mechanism explains early treatment?
A. It directly crosslinks fibrin at the variceal rupture site
B. It reduces splanchnic inflow and **portal pressure** through suppression of vasodilatory signaling
C. It neutralizes gastric acid to **pH >6**
D. It causes mechanical compression of varices
E. It increases platelet production within 1 hour
Answer: B
Mechanism: Octreotide and related vasoactive drugs are used **early** because they reduce splanchnic inflow and **portal pressure**, decreasing active **variceal bleeding** before definitive **endoscopic therapy**.

17. Band ligation of esophageal varices

A cirrhotic man undergoes endoscopic **band ligation** for actively bleeding **esophageal varices**.
What is the immediate hemostatic mechanism?
A. Acid suppression and **fibrinolysis** inhibition
B. Argon plasma coagulation of mucosal capillaries
C. V1 receptor-mediated **splanchnic vasoconstriction**
D. Mechanical strangulation and thrombosis of the variceal column
E. Bile acid sequestration in the small bowel
Answer: D
Mechanism: Band ligation achieves hemostasis mechanically by strangulating the varix, leading to thrombosis and later sloughing. It complements, **not** replaces, vasoactive pharmacology.

18. Cyanoacrylate in gastric varices

A patient with cirrhosis has **massive** bleeding from fundal **gastric varices**. **Cyanoacrylate** injection is selected where expertise is available.

Which mechanism best explains the therapy?

A. Rapid polymerization within the varix causing mechanical obturation of the bleeding channel
B. Nonselective beta blockade of portal inflow
C. Activation of **motilin receptor**s to clear blood from the stomach
D. Inhibition of **H+/K+-ATPase** to stabilize ulcer clots
E. Suppression of **gastric antral vascular ectasia**

Answer: A

Mechanism: Cyanoacrylate acts as a tissue adhesive that polymerizes when exposed to blood, obturating **gastric varices**. Embolic complications are a mechanism-based concern.

19. TIPS for uncontrolled variceal bleeding

A cirrhotic patient continues to bleed after vasoactive therapy and **band ligation**. Rescue **TIPS** is considered.

Which hemodynamic mechanism is intended?

A. Increase hepatic sinusoidal resistance to reduce shunting
B. Raise portal inflow to tamponade varices
C. Create a low-resistance portosystemic channel that lowers the portal-systemic pressure gradient
D. Destroy **gastric varices** by local polymerization
E. Improve **clot stability** by raising intragastric pH

Answer: C

Mechanism: TIPS treats portal hypertensive bleeding by decompressing the portal system. The tradeoff is increased portosystemic shunting and risk of hepatic encephalopathy.

20. Hemosuccus pancreaticus

A 58-year-old man with chronic pancreatitis has intermittent **melena** and epigastric pain. EGD intermittently shows blood emerging from the major papilla. **CT angiography** demonstrates a splenic artery pseudoaneurysm.

Which diagnosis-mechanism pair is most accurate?

A. Hemobilia due to hepatic artery-bile duct fistula
B. Dieulafoy lesion due to a gastric submucosal artery
C. GAVE due to antral capillary ectasia
D. Diverticular bleeding due to **vasa recta** rupture
E. Hemosuccus pancreaticus due to pseudoaneurysm bleeding into the pancreatic duct

Answer: E

Mechanism: Hemosuccus pancreaticus is bleeding through the pancreatic duct, often from a pseudoaneurysm related to pancreatitis. It classically causes intermittent bleeding from the ampulla.

21. Hemobilia after liver intervention

A patient develops RUQ pain, jaundice, and **melena** one week after percutaneous liver biopsy. Side-viewing endoscopy shows blood from the ampulla.

Which mechanism best explains the presentation?

A. Cameron erosions from a hiatal hernia
B. Arterial-biliary communication causing **hemobilia** with clot-related biliary obstruction
C. Acquired **vWF** deficiency from **aortic stenosis**
D. Diffuse antral vascular ectasia unrelated to bile ducts
E. Ectopic gastric mucosa in **Meckel diverticulum**

Answer: B

Mechanism: Hemobilia occurs when blood enters the biliary tree, often after hepatobiliary trauma or intervention. Clots can cause biliary colic and jaundice.

22. Aortoenteric fistula and herald bleed

A 70-year-old man with prior abdominal aortic graft repair presents with a brief self-limited **hematemesis** episode, followed hours later by shock and **massive** upper GI bleeding. Initial endoscopy is nondiagnostic.

Which mechanism should be feared?

A. NSAID **prostaglandin** depletion causing clean-base ulcer
B. Esophageal varices decompressed by spontaneous shunting
C. Celiac disease-related iron malabsorption

D. Aortoenteric fistula with intermittent sentinel bleeding before catastrophic arterial hemorrhage
E. Hemorrhoids causing **massive hematemesis**

Answer: D
Mechanism: Aortoenteric fistula is a rare, lethal cause of GI bleeding after aortic graft **surgery**. A herald bleed reflects intermittent arterial-enteric communication before exsanguination.

23. Hematochezia with hemodynamic instability

A 66-year-old man presents with bright red **hematochezia**, syncope, hypotension, and **BUN/Cr** ratio >30. He has a history of **peptic ulcer** disease and aspirin use.

Which mechanism-based interpretation is best?

A. Bright red blood always excludes an **upper gastrointestinal bleeding** source
B. Brisk **upper gastrointestinal bleeding** can rapidly transit and present as **hematochezia**; **blood digestion** increases **urea generation**
C. BUN/Cr ratio is irrelevant to **gastrointestinal bleeding** localization
D. Only hemorrhoids cause **hematochezia** with shock
E. Melena is mandatory in all upper GI bleeding

Answer: B
Mechanism: Severe **hematochezia** with instability can arise from brisk UGIB. Digested blood protein increases **BUN**, so a high **BUN/Cr** ratio supports an upper source.

24. Diverticular bleeding anatomy

A 74-year-old man has sudden painless **hematochezia**. Colonoscopy after purge identifies active bleeding from a right-sided diverticulum.

Which mechanism best explains diverticular hemorrhage?

A. Injury to the **vasa recta** at the diverticular neck or dome where penetrating vessels are exposed
B. Autoimmune destruction of colonic crypts by IgA immune complexes
C. Diffuse portal hypertensive congestion of the rectum
D. Ectopic gastric mucosa causing acid ulceration
E. Pancreatic pseudoaneurysm bleeding through the ampulla

Answer: A
Mechanism: Diverticular bleeding is typically painless and arterial. The **vasa recta** drape over the diverticulum and may rupture, especially at the neck or dome.

25. Thermal therapy in diverticular bleeding

During colonoscopy for **diverticular bleeding**, an actively bleeding vessel is seen deep at the dome of a thin-walled diverticulum. The endoscopist avoids aggressive thermal therapy at that site.

What mechanism best explains this caution?

A. Thermal coagulation cannot treat arterial bleeding
B. Diverticula contain thick muscularis propria that prevents injury
C. Thermal therapy worsens **portal pressure**
D. The diverticular wall is thin and deep thermal injury can cause perforation
E. Epinephrine injection permanently closes all diverticular vessels

Answer: D
Mechanism: Colonic diverticula lack the normal muscular wall. Deep thermal injury, especially at the dome, increases perforation risk; mechanical therapy may be safer depending on anatomy.

26. Angioectasia formation

An 82-year-old woman with chronic kidney disease has **recurrent** painless **iron deficiency anemia**. Colonoscopy and capsule endoscopy show multiple flat red vascular lesions in the right colon and jejunum.

Which mechanism best explains acquired angioectasia biology?

A. Ectopic acid secretion from gastric mucosa in a congenital diverticulum
B. IgE-mediated degranulation causing mucosal erosions
C. Age-related intermittent venous obstruction and vascular ectasia leading to dilated mucosal-submucosal vessels
D. Bacterial beta-glucuronidase deconjugating bilirubin
E. COX-2 activation increasing **prostaglandin** cytoprotection

Answer: C
Mechanism: Angioectasia/angiodysplasia is an acquired vascular lesion of aging, with ectatic mucosal and submucosal vessels that bleed intermittently, especially with CKD or antithrombotic exposure.

27. Heyde syndrome

A 78-year-old man with severe **aortic stenosis** has **recurrent** bleeding from small bowel **angioectasia**s. Coagulation studies are otherwise unremarkable.

Which mechanism best links the cardiac lesion to bleeding?

A. Aortic stenosis directly causes gastric acid hypersecretion
B. Calcific valve disease induces portal hypertension
C. Low-flow state causes only **ischemic colitis**
D. Aortic stenosis blocks platelet **COX-1**
E. High **shear stress** promotes loss of high-molecular-weight **von Willebrand factor** multimers, impairing hemostasis at **angioectasia**s

Answer: E

Mechanism: Heyde syndrome links **aortic stenosis**, acquired **vWF** abnormality, and **angioectasia** bleeding. Shear-mediated **vWF** multimer loss impairs platelet adhesion.

28. Ischemic colitis bleeding

A 73-year-old woman with atrial fibrillation and dehydration develops crampy left-sided abdominal pain followed by bloody diarrhea. CT shows segmental left-sided colitis.

Which mechanism best explains the bleeding?

A. Large-caliber gastric submucosal artery erosion
B. Hypoperfusion and reperfusion injury in watershed colonic zones causing **mucosal ischemia** and hemorrhage
C. Portal hypertension causing diffuse **rectal varices**
D. Ectopic gastric mucosa acidifying ileal mucosa
E. Motilin receptor activation causing mucosal sloughing

Answer: B

Mechanism: Ischemic colitis reflects transient hypoperfusion, commonly in watershed regions. Pain plus **hematochezia** differentiates it from many painless vascular sources.

29. Hemorrhoids and diagnostic anchoring

A 59-year-old man with **recurrent** bright red rectal bleeding and anemia is found to have small internal hemorrhoids on anoscopy. No colonoscopy has been performed.

Which reasoning is most appropriate?

A. Hemorrhoids rarely explain significant anemia or shock; proximal colonic or small bowel sources must be excluded
B. Small hemorrhoids prove the bleeding source in any patient with anemia
C. Hemorrhoids cause **melena** by **blood digestion** in the stomach
D. Hemorrhoids are treated by **TIPS** because they are always portal hypertensive
E. Hemorrhoids are the most common cause of **massive** lower GI bleeding

Answer: A

Mechanism: Hemorrhoids are common and can coexist with more important pathology. Significant anemia, **recurrent** bleeding, or risk factors require evaluation beyond anorectal findings.

30. Post-polypectomy delayed bleeding

A 67-year-old man on anticoagulation develops painless **hematochezia** 6 days after hot-snare resection of a 20-mm right colon polyp.

Which mechanism best explains the timing?

A. Immediate perforation causes bleeding only within minutes
B. Portal-pressure rise causes delayed bleeding after all polypectomies
C. Ectopic gastric mucosa secretes acid at the polypectomy site
D. Sloughing of the coagulation eschar exposes a submucosal vessel, with anticoagulation increasing bleeding risk
E. H. pylori causes delayed colonic vessel erosion

Answer: D

Mechanism: Delayed **post-polypectomy bleeding** occurs when a coagulation injury evolves and the eschar separates, exposing vessels. Right colon and antithrombotics increase risk.

31. Timing of colonoscopy in acute LGIB

A patient with acute **hematochezia** is stabilized. The resident argues that unprepped emergency colonoscopy within minutes is always superior to elective colonoscopy.

Which mechanism-based principle is most accurate?

A. Colonoscopy never identifies a bleeding source in lower GI bleeding

B. Bowel preparation is unnecessary because blood improves mucosal contrast
C. Stabilization and adequate purge improve visualization; very **early** colonoscopy has **not** consistently improved hard outcomes
D. All lower GI bleeding should go directly to **surgery**
E. EGD is **contraindicated** when **hematochezia** is present
Answer: C
Mechanism: The colon must be cleared to identify stigmata and deliver therapy. **Early** colonoscopy may increase diagnostic yield but does **not** reliably improve major outcomes compared with stabilized, prepared evaluation.

32. CT angiography in active bleeding

A 71-year-old woman has ongoing brisk **hematochezia** with hypotension despite **resuscitation**. Colonoscopy can**not** be prepared safely. **CT angiography** is ordered.
Which mechanism explains why CTA can guide therapy?
A. It detects healed ulcers by measuring intragastric pH
B. It directly embolizes bleeding vessels during scanning
C. It is **positive** only in chronic **occult** iron deficiency
D. It visualizes active contrast extravasation and localizes the vascular territory for **angiography** or **surgery**
E. It is more sensitive than capsule endoscopy for mucosal aphthae
Answer: D
Mechanism: CTA is useful in active brisk bleeding because intravascular contrast extravasates into the bowel lumen, allowing rapid localization and triage to **angiography**/**embolization**.

33. Tagged red cell scan limitation

A patient has intermittent **hematochezia** every 12 hours but is currently stable and **not** actively bleeding. A **tagged red cell scan** is **positive** in the right abdomen but localization is imprecise.
Which interpretation is best?
A. Tagged scans precisely identify the exact diverticular neck for clipping
B. They can detect slower or intermittent bleeding but often localize less accurately than **CTA** or **angiography**
C. They are therapeutic because radiolabeled cells embolize vessels
D. They diagnose celiac malabsorption
E. A **negative** scan excludes future intermittent bleeding
Answer: B
Mechanism: Tagged red cell scanning can detect intermittent or low-rate bleeding but localization may be broad and timing delays may reduce therapeutic usefulness.

34. Superselective embolization

Angiography shows **active extravasation** from a branch of the ileocolic artery in a patient with ongoing **hematochezia**. Superselective microcoil **embolization** is performed.
What is the main mechanistic advantage of superselective technique?
A. It occludes the bleeding arterial branch while preserving collateral perfusion to reduce bowel ischemia
B. It neutralizes gastric acid to stabilize clot
C. It induces platelet production
D. It treats venous portal hypertension by creating a shunt
E. It removes the need for **resuscitation**
Answer: A
Mechanism: Angiographic **embolization** works by occluding the bleeding arterial supply. Superselective catheterization limits the ischemic territory.

35. Small bowel bleeding terminology

A patient has **recurrent melena**. EGD and colonoscopy, both high quality, are **negative**. The fellow labels this **obscure GI bleeding** and stops further evaluation.
Which terminology and next-step logic is most appropriate?
A. Obscure GI bleeding means bleeding must be from the colon
B. A **negative** EGD and colonoscopy exclude **small bowel bleeding**
C. The next test should always be hemorrhoid banding
D. This is suspected **small bowel bleeding**; **OGIB** is reserved for bleeding with no source after complete evaluation
E. Celiac disease is the most common cause of true **small bowel bleeding**
Answer: D

Mechanism: Modern terminology favors suspected **small bowel bleeding** after **negative** upper and lower endoscopy. **OGIB** is reserved for cases where no source is found anywhere after appropriate evaluation.

36. Video capsule endoscopy as first-line small bowel test

A 62-year-old man has **recurrent melena** and **iron deficiency anemia**. EGD and colonoscopy are complete and **negative**. He has no obstructive symptoms or **Crohn disease** history.

Which test is most mechanism-based as first-line small bowel mucosal evaluation?

A. Immediate laparotomy without localization
B. Video capsule endoscopy because it noninvasively visualizes small bowel mucosa beyond standard endoscopy
C. Routine barium meal because it is most sensitive for **angioectasia**
D. Anoscopy because **melena** is usually hemorrhoidal
E. Gastric pH monitoring

Answer: B

Mechanism: VCE is a first-line test for suspected **small bowel bleeding** when no obstruction is suspected. It visualizes mucosal lesions beyond reach of EGD and colonoscopy.

37. Capsule retention risk

A man with **Crohn disease**, prior small bowel obstruction, and suspected **small bowel bleeding** is referred for capsule endoscopy.

What is the best mechanism-based precaution?

A. Give **erythromycin** because retention is caused by delayed gastric emptying only
B. Evaluate for stricture with **CT enterography** or **patency capsule** before **VCE** to reduce retention risk
C. Proceed directly because capsules can**not** retain in **Crohn disease**
D. Use **tagged red cell scan** to dilate strictures
E. Avoid all small bowel evaluation permanently

Answer: B

Mechanism: Capsule retention occurs when a mechanical narrowing traps the capsule. **Crohn disease**, **NSAID enteropathy**, and tumors are risk settings; cross-sectional imaging or patency testing reduces risk.

38. CT enterography after negative capsule

A 44-year-old man has **overt recurrent** bleeding. Capsule endoscopy is **negative**, but he has weight loss and intermittent obstructive symptoms.

Which mechanism supports CT enterography next?

A. It measures **fibrinolysis** at ulcer bases
B. It replaces all **endoscopic therapy** for **angioectasia**
C. It detects **portal pressure** gradients
D. It is the best test for hemorrhoidal bleeding
E. It can identify mural masses or obstructing lesions that may be missed or incompletely assessed by **VCE**

Answer: E

Mechanism: Capsule is excellent for mucosa, but **CT enterography** is better for mural/extraluminal masses and obstruction. It is especially useful when tumor or stricture is suspected.

39. Deep enteroscopy after positive capsule

Capsule endoscopy identifies multiple jejunal **angioectasia**s in a 79-year-old woman with **recurrent transfusion**-dependent anemia.

Why is deep enteroscopy the rational next step?

A. It lowers **portal pressure** by creating a shunt
B. It detects only gastric ulcers missed by EGD
C. It allows targeted therapy such as **APC** or clipping at the capsule-localized small bowel lesion
D. It prevents capsule retention by dissolving strictures
E. It diagnoses H. pylori infection

Answer: C

Mechanism: VCE localizes **small bowel bleeding**; **deep enteroscopy** converts diagnosis into therapy by enabling ablation, clipping, injection, tattooing, or biopsy.

40. Meckel diverticulum bleeding

A 19-year-old man has painless maroon stools. EGD and colonoscopy are unrevealing. A **Meckel scan** is **positive**.

Which mechanism explains bleeding?

A. Ectopic gastric mucosa secretes acid, causing ulceration of adjacent ileal mucosa

B. Aortic stenosis causes acquired **vWF** loss in ileal villi
C. Portal hypertension causes diffuse jejunal varices
D. Bacterial beta-glucuronidase deconjugates bilirubin
E. PPI therapy causes ileal ischemia
Answer: A
Mechanism: Meckel diverticulum can contain **ectopic gastric mucosa**. Acid secretion ulcerates adjacent ileum, causing painless lower or **small bowel bleeding** in young patients.

41. NSAID enteropathy

A 58-year-old man with chronic ibuprofen use has **iron deficiency anemia**. EGD and colonoscopy are **negative**. Capsule endoscopy shows small bowel erosions and thin **diaphragm-like strictures**.
Which mechanism best explains this entity?
A. Loss of high-molecular-weight **vWF** multimers from **aortic stenosis**
B. NSAID topical injury plus **prostaglandin** depletion causing erosions, ulcers, and **diaphragm-like strictures**
C. Ectopic gastric mucosa in a congenital diverticulum
D. Bile acid malabsorption causing colonic secretion
E. TIPS-induced small bowel venous dilation
Answer: B
Mechanism: NSAID enteropathy involves **topical mitochondrial injury**, permeability changes, and COX/**prostaglandin** inhibition. It can cause erosions, ulcers, bleeding, and diaphragm disease.

42. Portal hypertensive enteropathy

A patient with cirrhosis, **recurrent melena**, and **negative** EGD/colonoscopy undergoes capsule endoscopy, which shows diffuse small bowel edema, red spots, and e**cta**tic vascular lesions.
Which mechanism best fits?
A. IgA deposition against epidermal transglutaminase
B. Meckel diverticulum acid ulceration
C. H. pylori-induced antral gastrin excess
D. Topical NSAID mitochondrial toxicity only
E. Portal hypertension producing small bowel mucosal congestion, vascular e**cta**sia, and enteropathy
Answer: E
Mechanism: Portal hypertensive enteropathy is small bowel mucosal and vascular change driven by portal hypertension, analogous in principle to **portal hypertensive gastropathy**.

43. Blue rubber bleb nevus syndrome

A young adult has **recurrent iron deficiency anemia**, multiple compressible blue skin nodules, and capsule endoscopy showing **venous malformation**s throughout the small bowel.
Which mechanism best explains the bleeding?
A. Aspirin-induced **COX-1** inhibition in normal mucosa
B. Acid-peptic injury from **ectopic gastric mucosa**
C. Congenital **venous malformation**s that bleed from fragile vascular channels
D. Acquired **vWF** loss from **aortic stenosis**
E. Bile acid sequestration failure
Answer: C
Mechanism: Blue rubber bleb nevus syndrome causes multifocal **venous malformation**s in skin and GI tract. Dermatologic clues can identify the bleeding mechanism.

44. Hereditary hemorrhagic telangiectasia

A 46-year-old woman has **recurrent** epistaxis, mucocutaneous telangie**cta**sias, and chronic **iron deficiency anemia**. Small bowel evaluation reveals multiple telangie**cta**sias.
Which mechanism-based explanation is best?
A. Abnormal vascular development in TGF-beta pathway-related signaling causes fragile telangie**cta**sias and arterio**venous malformation**s
B. H. pylori antigenic stimulation causes B-cell lymphoma
C. Portal pressure causes all mucocutaneous telangie**cta**sias
D. NSAIDs induce **diaphragm-like strictures** only
E. PPI-induced hypochlorhydria causes vascular proliferation
Answer: A

Mechanism: Hereditary hemorrhagic telangiectasia is a vascular development disorder causing fragile telangiectasias and AVMs, leading to epistaxis and GI bleeding.

45. Small bowel age-based differential

A 32-year-old man has **recurrent overt bleeding** after **negative** EGD and colonoscopy. The examiner asks why the differential differs from an 82-year-old patient.

Which age-linked reasoning is most accurate?

A. **Angioectasia** is equally the dominant cause at every age
B. Young patients more often have **Meckel diverticulum**, **Crohn disease**, or **small bowel tumor**; older patients more often have **angioectasia** or NSAID ulcers
C. Young patients can**not** bleed from the small bowel
D. Older patients only bleed from hemorrhoids
E. Age has no effect on **small bowel bleeding** etiology

Answer: B

Mechanism: Small bowel bleeding causes vary with age. Mechanism-based pattern recognition prevents reflexively treating every **negative** EGD/colonoscopy case as **angioectasia**.

46. Celiac disease and iron deficiency

A woman with **iron deficiency anemia** has **positive** tTG IgA and villous atrophy. Capsule endoscopy shows no bleeding lesions. The resident labels this **small bowel bleeding** from celiac disease.

Which mechanism-based correction is best?

A. Celiac disease classically causes iron deficiency by malabsorption rather than **occult bleeding**, unless complications such as ulcerative jejunitis or malignancy are present
B. Celiac disease causes bleeding by acquired **vWF** deficiency
C. Celiac disease always produces visible capsule **angioectasia**
D. Celiac disease bleeding is treated by **TIPS**
E. Celiac disease is the most common cause of **massive hematochezia**

Answer: A

Mechanism: Uncomplicated celiac disease usually causes iron deficiency through impaired absorption. Bleeding suggests a**not**her lesion or celiac complications such as lymphoma, ulcerative jejunitis, or adenocarcinoma.

47. Anticoagulation and angioectasia recurrence

An 84-year-old man with chronic kidney disease and atrial fibrillation has **recurrent** small bowel **angioectasia** bleeding despite **APC** therapy. He remains on anticoagulation.

Which mechanism best explains recurrence risk?

A. **APC** converts **angioectasia** into varices
B. Anticoagulation creates new Meckel diverticula
C. CKD eliminates all mucosal hemostasis permanently
D. **PPI** therapy causes small bowel **angioectasia**
E. Multiple fragile ectatic vessels plus impaired coagulation increase the probability of **recurrent** bleeding

Answer: E

Mechanism: Angioectasia bleeding often recurs because lesions are multiple and biologically fragile. Advanced age, comorbidity, lesion burden, and anticoagulants increase recurrence.

48. Glasgow-Blatchford risk score biology

A man presents with coffee-ground vomiting. He is normotensive, has **hemoglobin** 15 g/dL, normal **BUN**, no **melena**, no syncope, and no liver or heart failure. The team calculates a very low **Glasgow-Blatchford** score.

What does the score mechanistically estimate?

A. The probability of H. pylori infection
B. The presence of Barrett metaplasia
C. Need for clinical intervention such as **transfusion**, **endoscopic therapy**, or hospital-based management
D. **Portal pressure** gradient in cirrhosis
E. The number of small bowel **angioectasia**s

Answer: C

Mechanism: The **Glasgow-Blatchford** score integrates hemodynamic, bleeding, comorbidity, **hemoglobin**, and **BUN** data to identify UGIB patients likely to need intervention.

49. Hemoglobin lag in acute bleeding

A patient arrives 30 minutes after **massive hematemesis**. Initial **hemoglobin** is 12.8 g/dL despite hypotension and tachycardia. A trainee concludes bleeding was minor.

Which mechanism-based interpretation is best?

A. Initial **hemoglobin** may be misleading before plasma equilibration and fluid **resuscitation** reveal dilutional anemia
B. **Hemoglobin** always falls instantly in proportion to blood loss
C. Normal **hemoglobin** excludes shock from bleeding
D. **BUN** is never affected by **blood digestion**
E. Tachycardia excludes hemorrhage

Answer: A

Mechanism: In acute hemorrhage, whole blood is lost initially, so **hemoglobin** may remain near baseline until intravascular refilling or IV fluids dilute the circulation. Hemodynamics matter **early**.

50. Missed lesions after negative EGD and colonoscopy

A patient is referred for **recurrent melena** after **negative** EGD and colonoscopy done during active bleeding with poor visualization. A small bowel evaluation is requested immediately.

Which reasoning is most appropriate before committing to small bowel-only evaluation?

A. Prior **negative** endoscopy always excludes missed upper and lower lesions
B. **Melena** is never caused by lesions within reach of standard endoscopy
C. Repeat targeted EGD, **push enteroscopy**, or colonoscopy may be warranted because **Cameron lesions**, **Dieulafoy lesion**, **angioectasia**, or obscured lesions can be missed
D. Only **tagged red cell scan** can diagnose a **missed lesion**
E. Capsule endoscopy treats bleeding and therefore replaces repeat endoscopy

Answer: C

Mechanism: A substantial fraction of suspected **small bowel bleeding** is ultimately due to missed or hard-to-see lesions within reach of standard endoscopes. Quality, completeness, and visualization of prior exams determine next steps.

Answer Key

1C, 2B, 3D, 4D, 5B, 6E, 7C, 8A, 9D, 10B, 11C, 12D, 13A, 14E, 15C, 16B, 17D, 18A, 19C, 20E, 21B, 22D, 23B, 24A, 25D, 26C, 27E, 28B, 29A, 30D, 31C, 32D, 33B, 34A, 35D, 36B, 37B, 38E, 39C, 40A, 41B, 42E, 43C, 44A, 45B, 46A, 47E, 48C, 49A, 50C