

Angiodysplasia

Vascular architecture, bleeding and clinicopathologic interpretation

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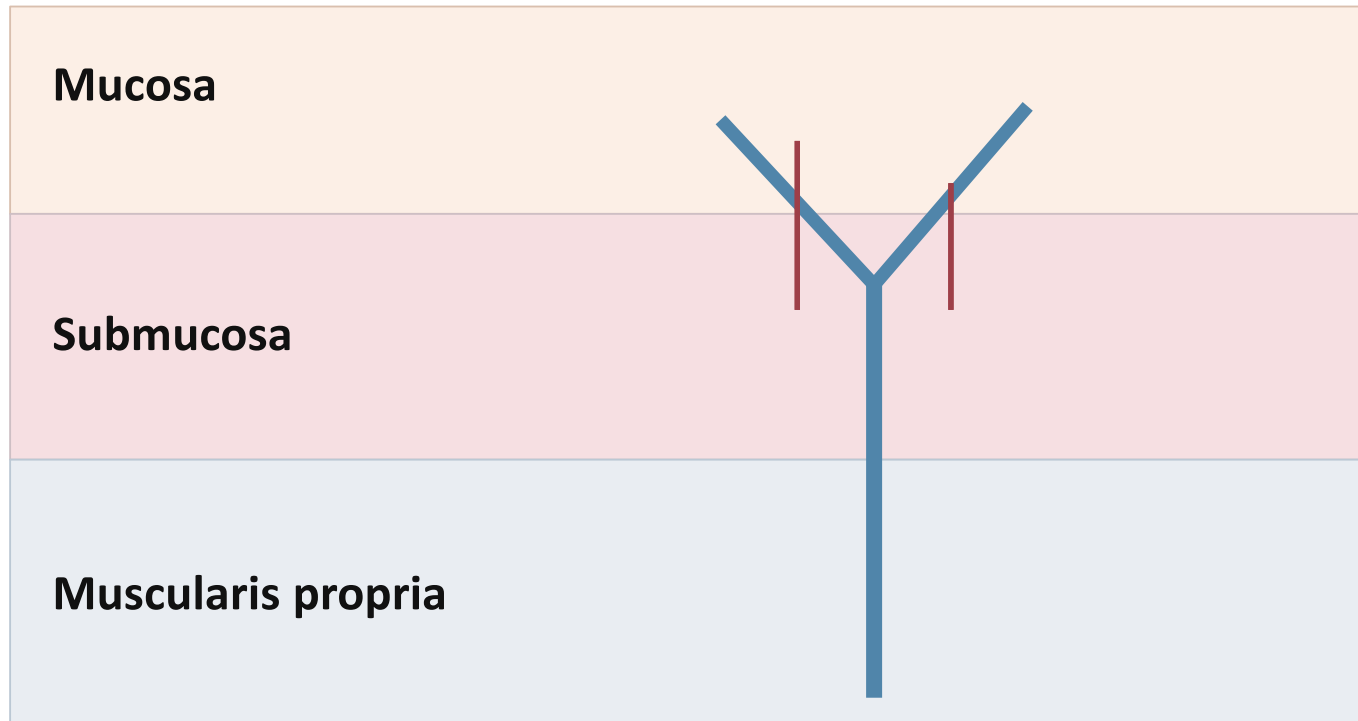
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Learning outcomes

- Identify angiodysplasia as a vascular malformation of intestinal mucosa and submucosa.
- Explain the proposed venous-outflow mechanism without presenting it as proven.
- Relate vessel structure to chronic or major intestinal hemorrhage.
- Distinguish pathology from clinical diagnosis and management.

The intestinal wall provides the anatomic context

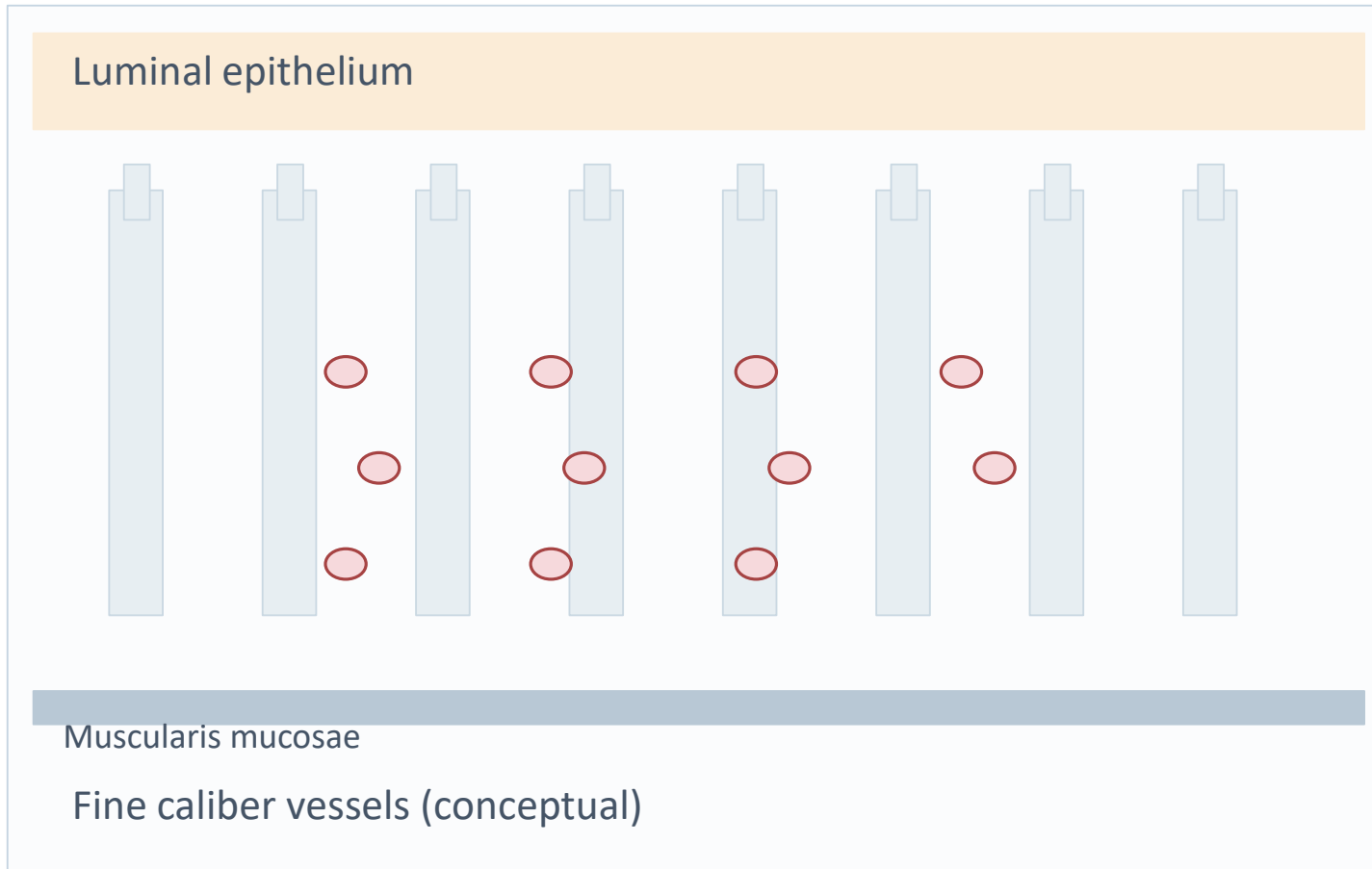


The mucosa faces the lumen and includes epithelium and lamina propria.

The submucosa contains vessels that communicate with the mucosal microcirculation.

Vessels pass through muscularis propria to reach the submucosal plexus.

Normal blood vessels are structurally supported



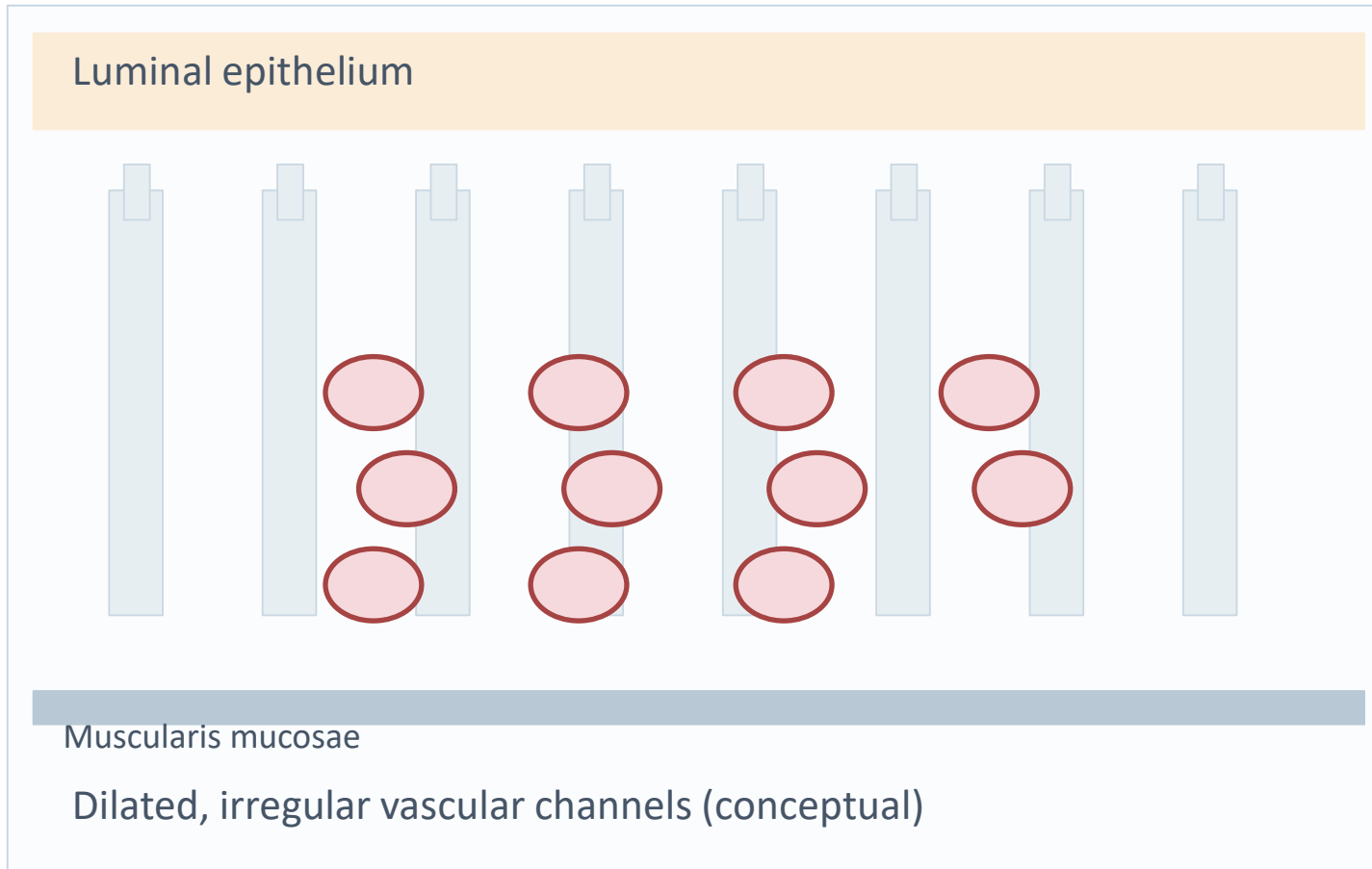
Capillaries lie within connective tissue beneath the epithelium.

Venules and veins carry blood away from mucosal capillary networks.

Vessel caliber and supporting layers influence susceptibility to injury.

Original schematic — not a photomicrograph

Angiodysplasia: The defining structural lesion



Malformed blood vessels occupy mucosa and submucosa.

The affected channels are abnormally dilated and thin walled.

The entity is a vascular malformation, not an epithelial tumor.

Original schematic — not a photomicrograph

Angiodysplasia is a nonneoplastic vascular lesion

Angiodysplasia / ectasia

Dilation and malformation of existing vessels; No true neoplastic mass.

Vascular neoplasm

True vascular tumors show endothelial proliferation; Biologically distinct from ectasia.

Morphologic distinction: Ectatic channels do not imply endothelial malignancy.

Angiodysplasia and angioectasia: Practical vocabulary

- Angiodysplasia is the pathology term used.
- Angioectasia is frequently used in GI endoscopy and bleeding guidance.
- Neither term alone establishes current bleeding or proves lesion causality.

Anatomic predilection: Cecum and right colon

Cecum / ascending

Transverse

Descending

Sigmoid / rectum

Most common location ≠ exclusive location

- This site preference is linked to the mechanical hypothesis.

Age distribution: Predominance after 60 years

- Usual clinical recognition after the sixth decade.
 - Older age increases the relevance of angiodysplasia in lower GI bleeding.
 - Younger age does not categorically exclude a vascular lesion.
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Prevalence: A relatively uncommon adult lesion

<1%

Adult prevalence reported by Robbins

- This denominator is adults overall, not patients undergoing colonoscopy for bleeding.
- A population prevalence must not be confused with a proportion of bleeding etiologies.

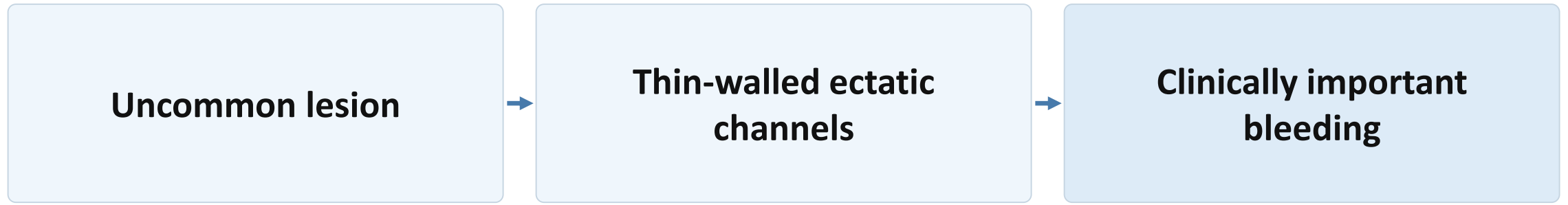
Disproportionate contribution to major bleeding

20%

Major lower intestinal bleeding episodes
in older groups (Robbins)

- The estimate applies to a specified clinical population and severity.
- Do not generalize this number to all patients or all GI bleeding.

Why prevalence and bleeding burden differ

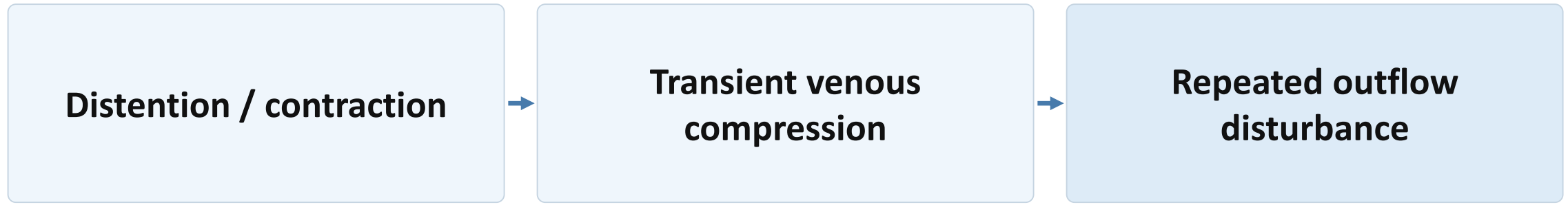


- Population prevalence and cause-of-bleeding proportion answer different questions.
- The malformed, superficially located vessels are vulnerable to disruption.

Pathogenesis is not established

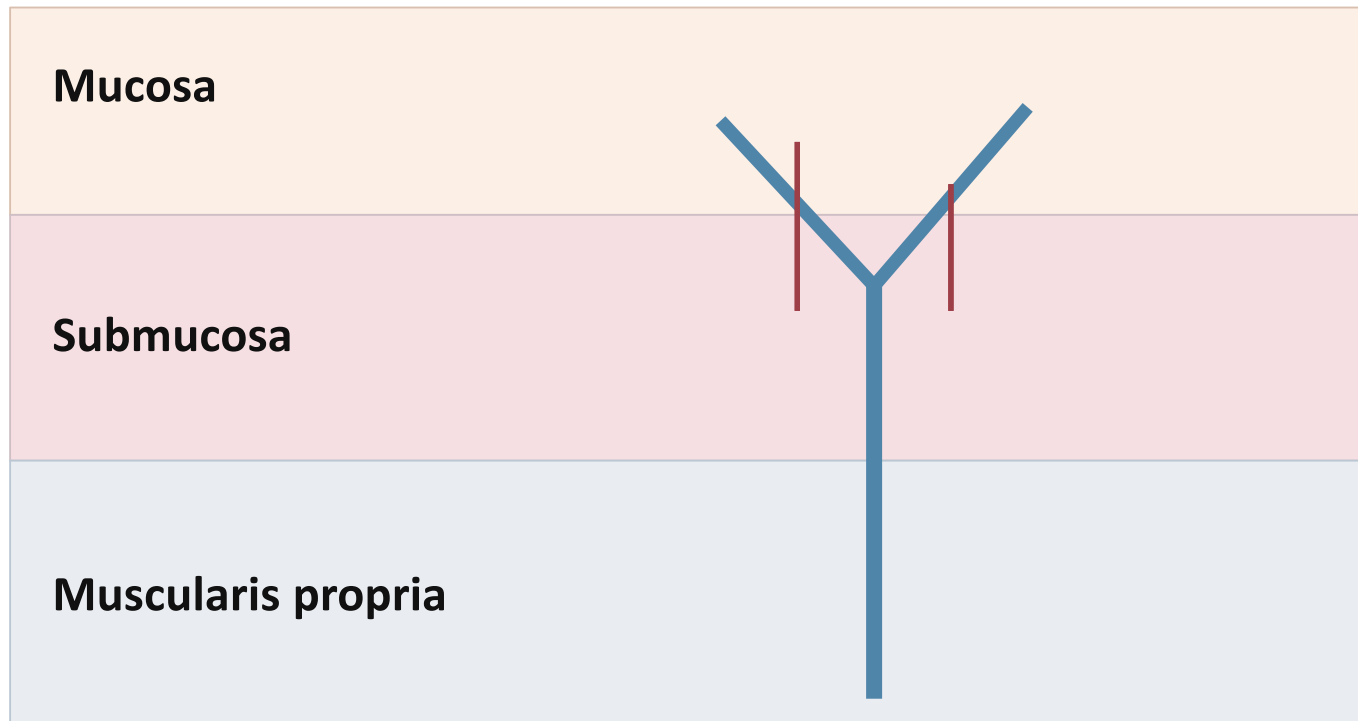
- Robbins explicitly describes the mechanism as undefined.
 - Mechanical and congenital factors are proposed contributors.
 - A plausible model must not be converted into a confirmed causal chain.
-

The mechanical model begins with normal motility



- Distention and contraction repeatedly alter the geometry of the bowel wall.
- Penetrating submucosal veins may be intermittently compressed.

Venous traversal of muscularis propria matters



Submucosal veins traverse the muscularis propria during drainage.

These traversing veins as sites of intermittent occlusion.

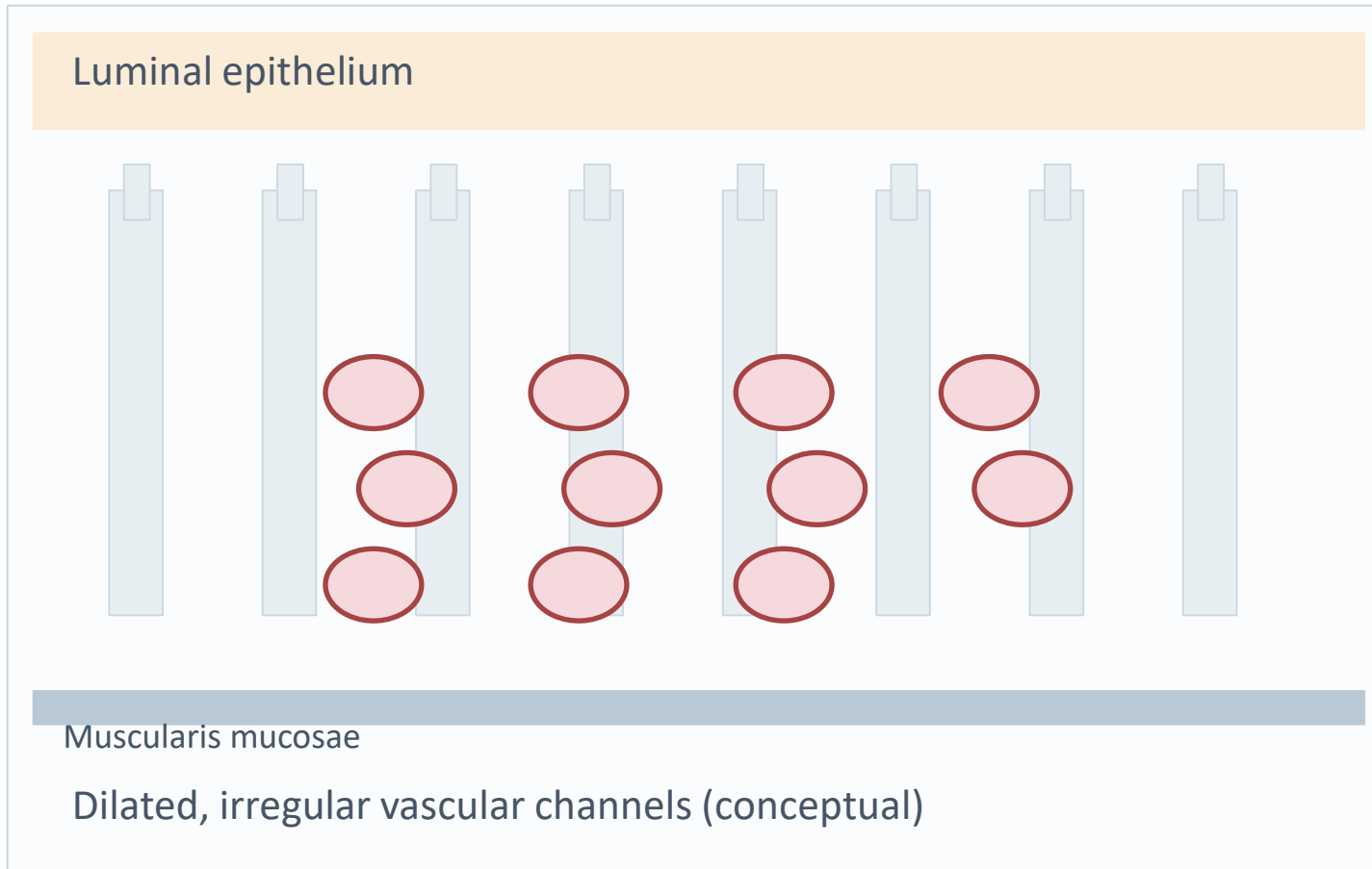
Mechanical location links bowel-wall motion to superficial vascular change.

Intermittent venous obstruction: A proposed trigger



- Temporary obstruction impedes venous drainage from the overlying plexus.
- Repeated episodes could alter vessel diameter and configuration.

Progression toward dilated and tortuous channels



Focal dilation is the proposed consequence of repetitive venous obstruction.

Mucosal and submucosal channels become tortuous.

Tortuosity and ectasia together produce the characteristic vascular nest.

Original schematic — not a photomicrograph

Cecal caliber provides a regional hypothesis

Cecum / ascending

Transverse

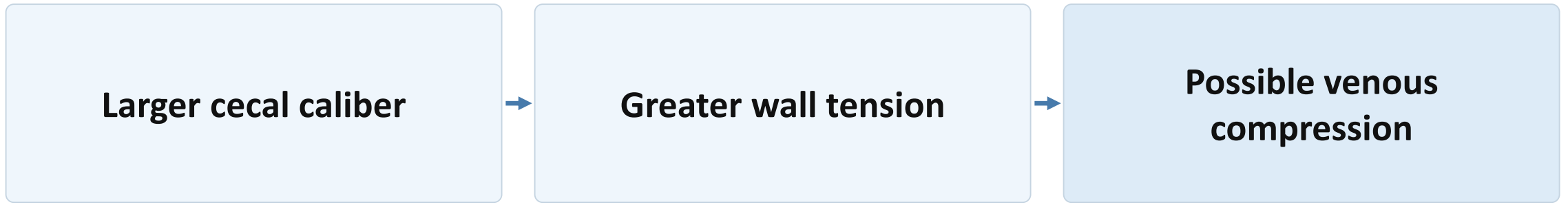
Descending

Sigmoid / rectum

Most common location ≠ exclusive location

- Greater diameter may increase wall tension during distention.

Wall tension is an explanatory link, not proof



- The mechanical model connects wider cecal caliber with greater wall tension.
- Wall tension is a conceptual contributor to transient venous compression.

Integrating the proposed pathogenesis



- Bowel motion may intermittently obstruct penetrating veins.
- Outflow disturbance may yield focal mucosal and submucosal ectasia.

Congenital mechanisms remain possible

- Robbins also mentions congenital factors.
 - It does not specify a causal gene or developmental pathway for typical colonic lesions.
 - Avoid assigning hereditary hemorrhagic telangiectasia mechanisms to sporadic angiodysplasia.
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Macroscopic–microscopic distinction

Surface assessment

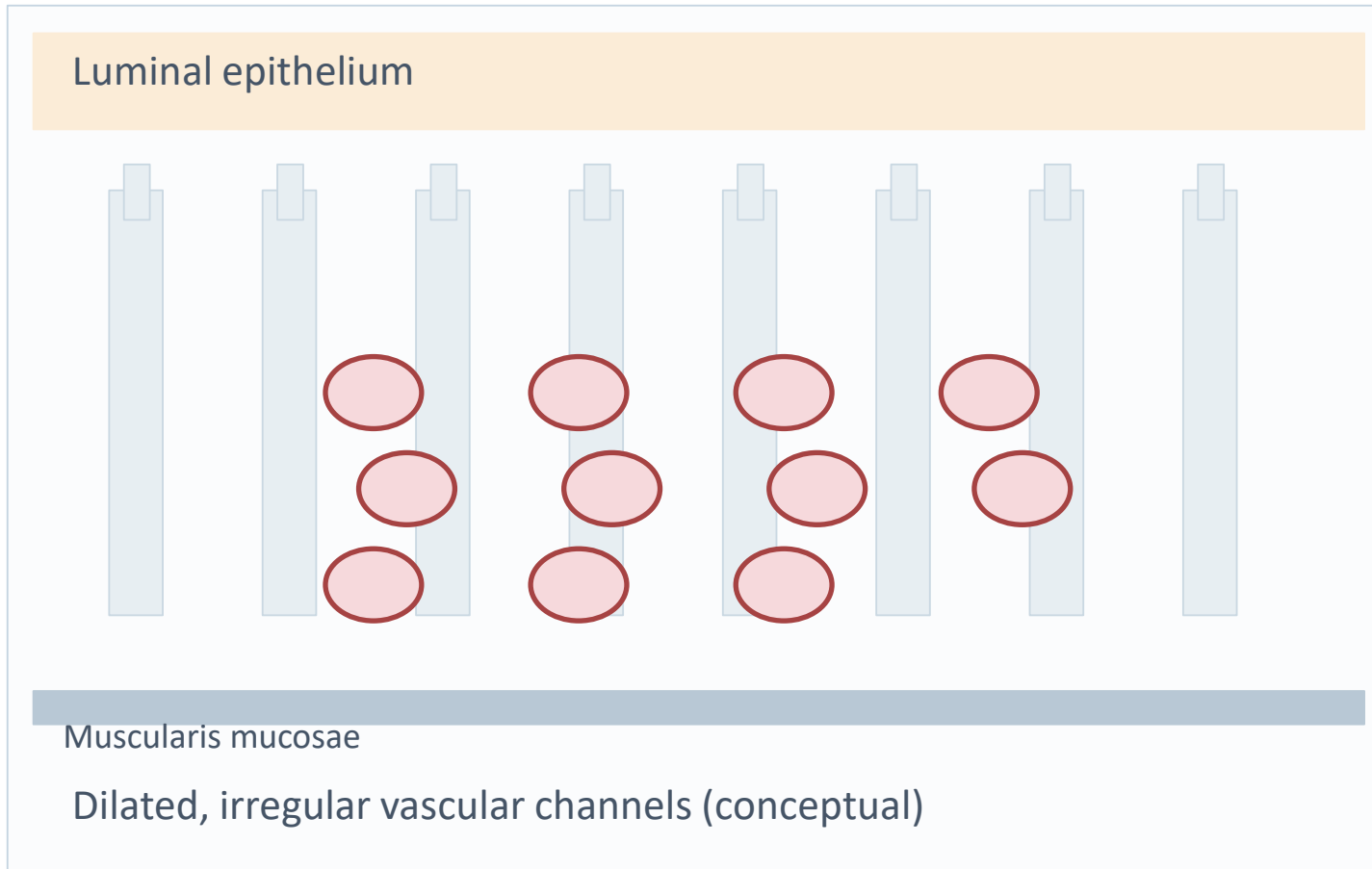
Surface endoscopy reveals ectatic vascular patches and possible current bleeding.

Tissue assessment

Microscopy demonstrates dilated thin-walled vessels within mucosa and submucosa.

The two modalities assess different features of the same clinical problem.

Histology: Ectatic vascular nests



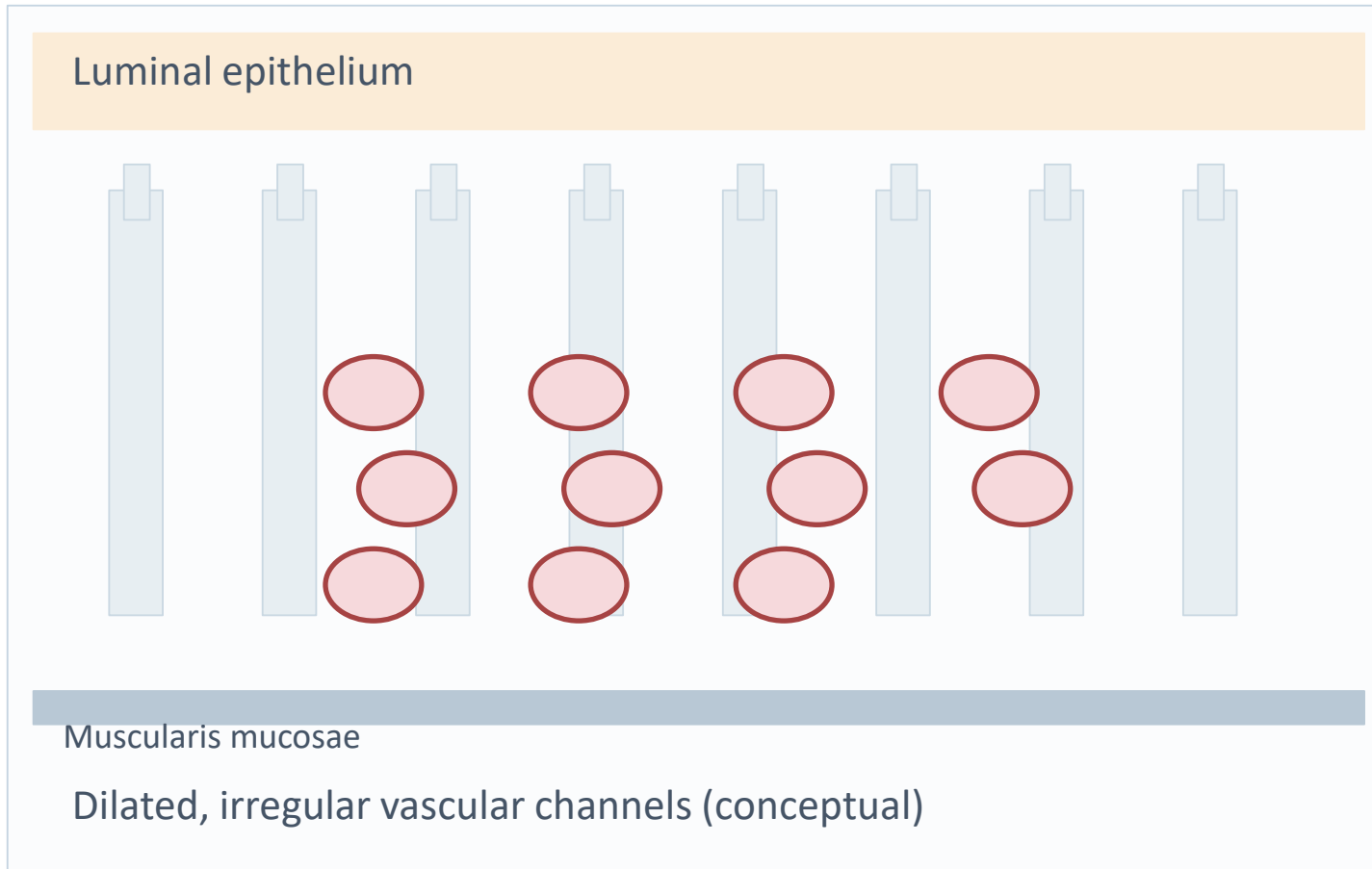
Nests of tortuous vascular channels.

Veins, venules and capillaries contribute to the lesion.

The vessels lie in mucosal and submucosal compartments.

Original schematic — not a photomicrograph

Histology: Veins and venules



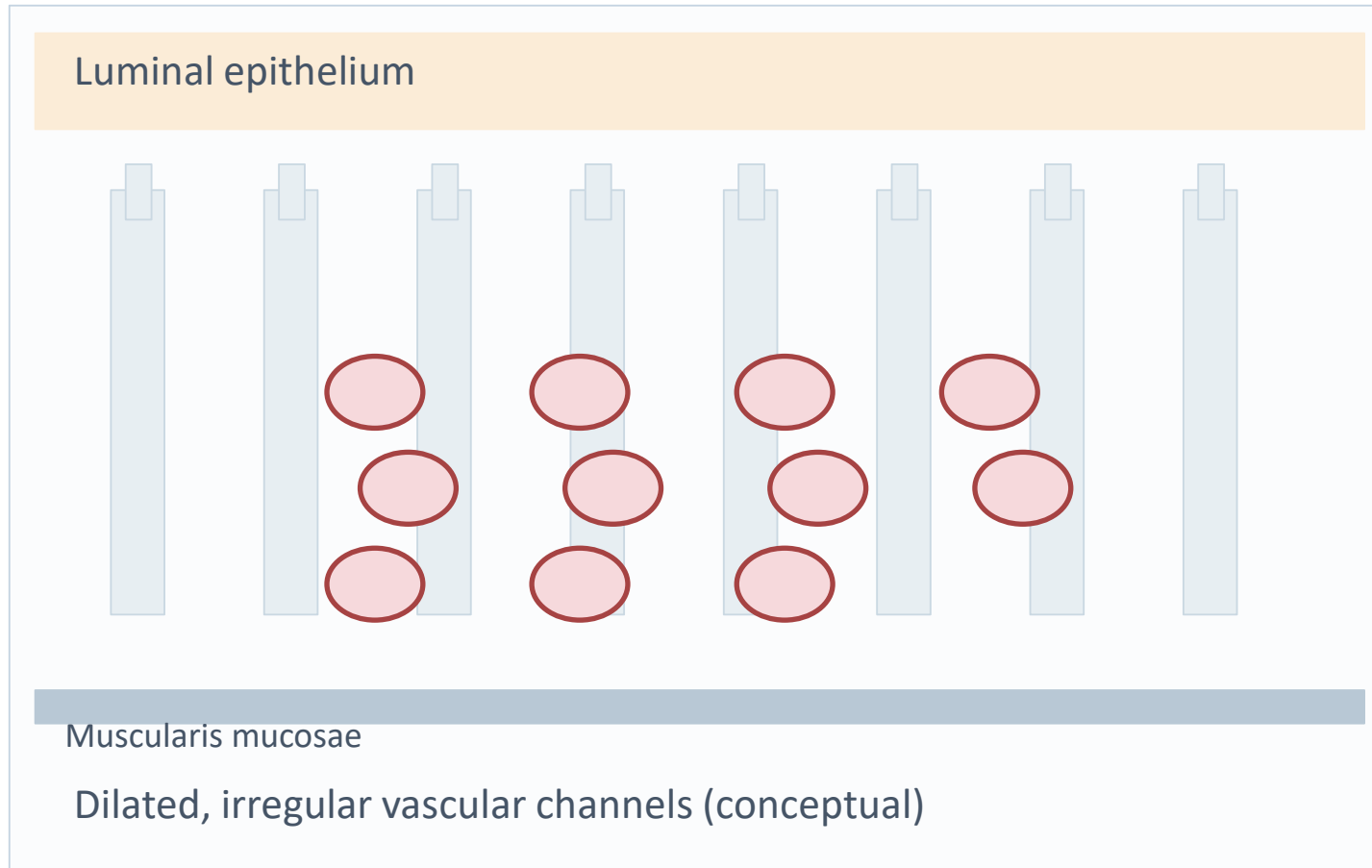
Dilated veins and venules are conspicuous components.

Their thin walls distinguish them from thick-walled muscular arteries.

Venous morphology fits the proposed outflow-disturbance model.

Original schematic — not a photomicrograph

Histology: Capillary ectasia



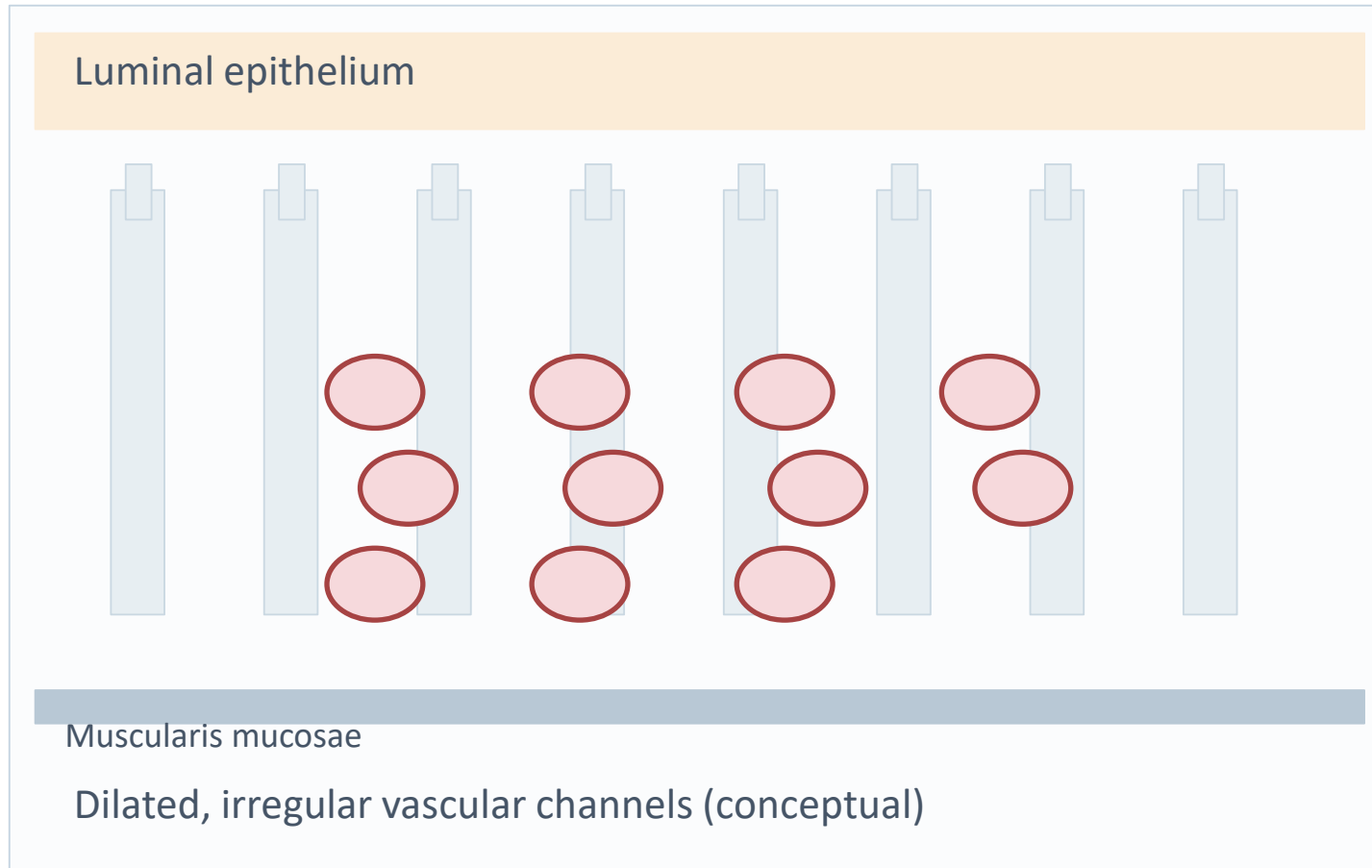
Capillaries are also involved, extending the lesion toward the mucosal surface.

Superficial ectasia places delicate vascular channels near luminal contents.

Location is integral to the risk of hemorrhage.

Original schematic — not a photomicrograph

The epithelial covering may be attenuated



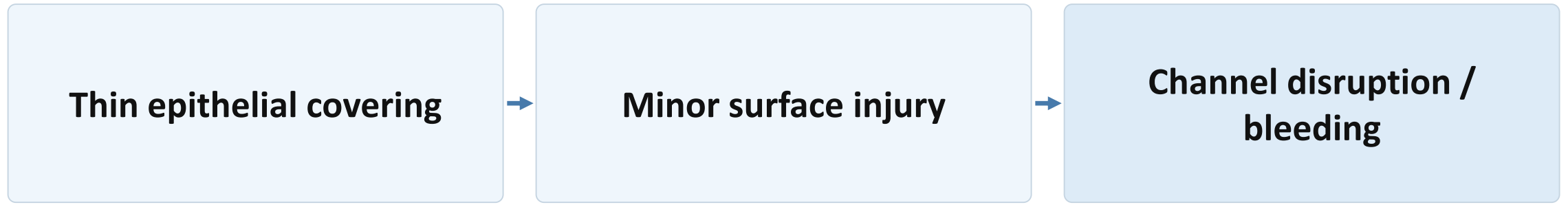
Channels covered only by vessel wall and attenuated epithelium.

The remaining interface can be extremely thin.

This configuration makes minor mucosal trauma consequential.

Original schematic — not a photomicrograph

Bleeding vulnerability follows from structure



- The luminal surface may be separated from blood by little protective tissue.
- Even minor injury can disrupt a dilated, thin-walled channel.

Why angiodysplasia can bleed intermittently

- The clinical course may include symptom-free intervals.
- Minor injury can produce episodes separated by periods without visible loss.
- Absence of hemorrhage at a single examination does not exclude past bleeding.

Clinical spectrum: Chronic to massive hemorrhage

Chronic / intermittent

Long symptom-free intervals or repeated blood loss may occur.

Acute / major

A minority may present with an acute major lower intestinal hemorrhage.

A chronic clinical history does not exclude clinically important bleeding.

Chronic blood loss: Anemia is a clinical consequence

- Repeated blood loss can lead to iron deficiency and falling hemoglobin.
- Anemia requires a bleeding-source evaluation; Lesion presence alone is insufficient.
- Trend, stool history and endoscopic findings must be integrated.

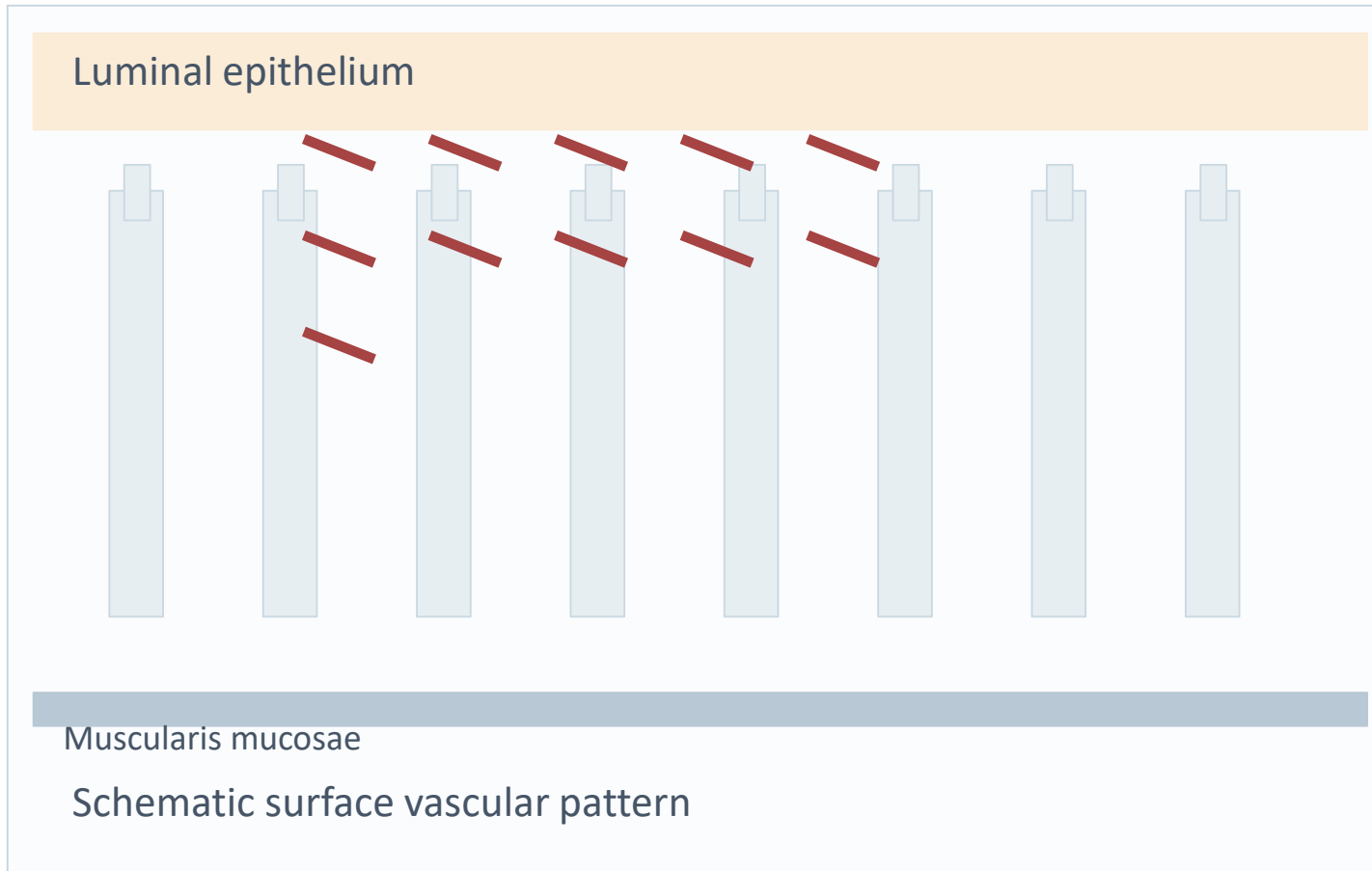
Acute blood loss: Recognize the higher-risk phenotype

- Major lower GI hemorrhage may present with overt hematochezia.
- Assess hemodynamic consequences before concentrating on lesion classification.
- Large-volume bleeding is possible even from a nonneoplastic lesion.

Lesion presence is not identical to lesion culpability

- Angioectasia may be an incidental endoscopic finding.
- Link the lesion to the bleeding phenotype and exclude competing sources.
- Current bleeding stigmata provide stronger attribution than lesion morphology alone.

Endoscopic appearance: Recognize the vascular pattern



Colonic angioectasia typically appears as a superficial erythematous vascular patch.

Branching or fern-like ectatic vessels may be visible.

An endoscopic pattern is not the same as histologic proof of etiology.

Original schematic — not a photomicrograph

Two complementary diagnostic views

Endoscopy

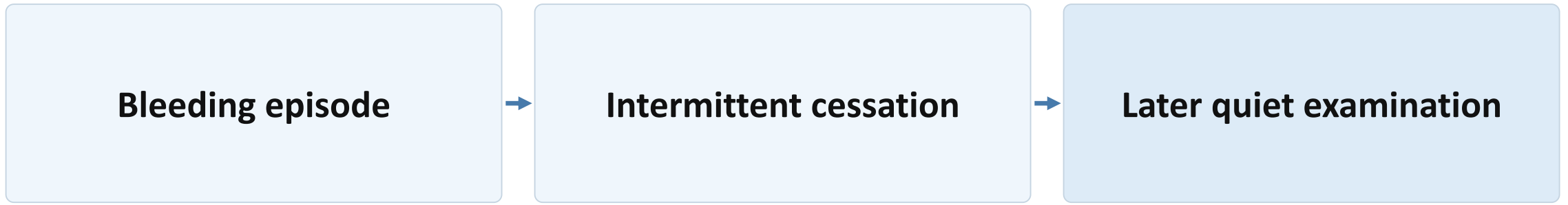
Surface inspection can identify vascular lesions and bleeding stigmata.

Histopathology

Tissue examination, when available, characterizes abnormal vessel architecture.

Both findings require linkage to the patient's actual bleeding phenotype.

A negative snapshot may miss intermittent bleeding



- Bleeding may have stopped before a diagnostic examination.
- The underlying malformation may persist despite a quiet clinical interval.

Lower GI bleeding: Keep a differential diagnosis

- Diverticular hemorrhage, ischemic colitis and neoplasia also require consideration.
- The patient's pain, bowel symptoms and examination modify pretest probability.
- Do not close the diagnostic process after finding a single vascular lesion.

Ischemic colitis has a different injury mechanism

Ischemic colitis

Impaired intestinal perfusion with epithelial damage; Often cramps and bloody diarrhea.

Angiodysplasia

Superficial ectatic vessels can bleed without primary ischemic tissue injury.

Acute pain and mucosal injury shift suspicion toward colonic ischemia.

Hereditary hemorrhagic telangiectasia is distinct

HHT

Multisite inherited disease; Autosomal dominant, endothelial TGF- β pathway defects.

Sporadic colonic lesion

Usually recognized in older adults; Typical colonic lesion has no defined inherited mechanism.

Do not treat a single colonic angioectasia as proof of a systemic genetic disorder.

Gastric antral vascular ectasia is a related differential

GAVE

Antral longitudinal red stripes; Reactive gastropathy, ectatic capillaries and fibrin thrombi.

Right-colonic angiodysplasia

Cecal/right-colonic predominance; Ectatic tortuous mucosal–submucosal veins and capillaries.

Location and accompanying mucosal changes are discriminating features.

Dieulafoy lesion: A different vascular source

Dieulafoy lesion

Abnormal submucosal arteriole,
classically along the gastric lesser
curvature.

Angiodysplasia

Nests of dilated veins, venules and
capillaries, classically in right colon.

The two vascular bleeding sources should not be conflated.

The clinical priority is bleeding severity

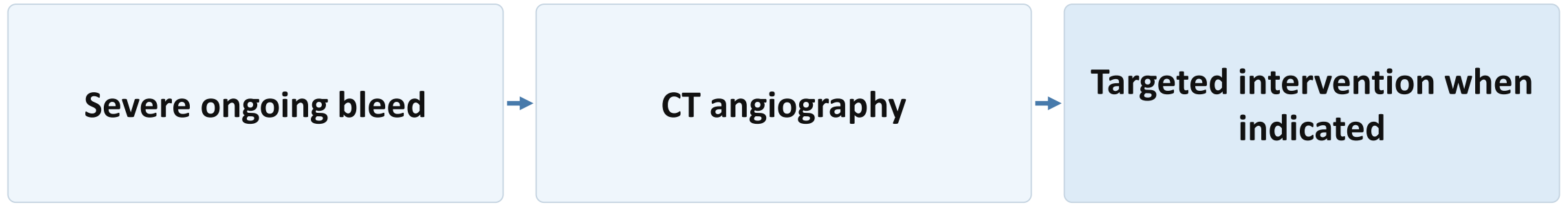


- Unstable patients require immediate assessment and resuscitative care.
- Definitive lesion characterization follows stabilization as appropriate.

Colonoscopy evaluates suspected colonic bleeding

- Colonoscopy permits direct inspection of mucosa for angioectasia.
- A suspected lesion should be interpreted with blood distribution and other findings.
- The 2023 ACG guideline generally favors nonurgent inpatient colonoscopy over routine colonoscopy within 24 hours.

CT angiography in significant ongoing hemorrhage



- CTA has an increasing role in severe lower GI bleeding.
- It is principally a bleeding-localization strategy during active hemorrhage.

Why colonoscopy can miss small-bowel disease

- Angiodysplasia can involve sites beyond the accessible colon.
- A negative colonic examination does not exclude a small-intestinal source.
- The next test follows the pattern of persistent or recurrent bleeding.

Capsule endoscopy: First-line small-bowel survey

- The ESGE 2022 update recommends capsule endoscopy first in suspected small-bowel bleeding.
 - It offers mucosal visualization beyond conventional upper/lower endoscopy.
 - The capsule identifies lesions but cannot deliver hemostatic therapy.
-

Timing matters for overt small-bowel bleeding

≤ 48 h

Ideal timing after overt suspected small-bowel bleeding (ESGE)

- The guideline specifies ideally within 48 hours when feasible.
- This recommendation concerns diagnostic yield, not the pathogenesis of lesions.

Device-assisted enteroscopy enables treatment

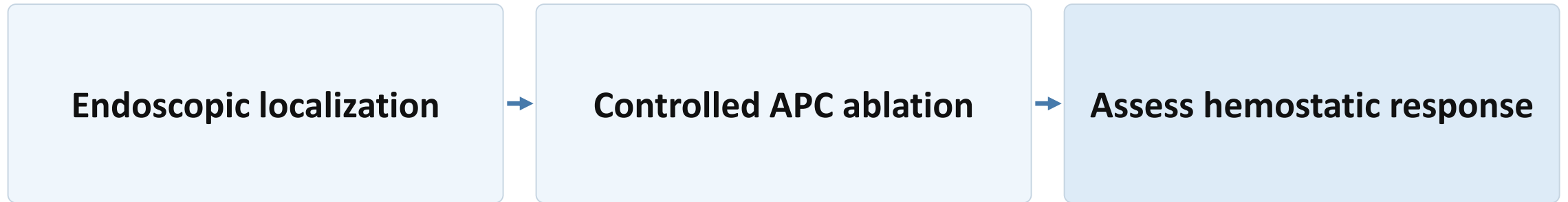


- Deep enteroscopy can investigate a lesion localized by prior testing.
- Unlike capsule endoscopy, it can facilitate endoscopic therapy.

Therapy targets bleeding, not merely lesion visibility

- A visible ectatic area without clinical blood loss may not need ablation.
- When attributable hemorrhage is present, endoscopic hemostasis is a principal option.
- The therapeutic decision is individualized to bleeding burden.

Argon plasma coagulation: Noncontact ablation



- APC delivers thermal energy to superficial bleeding vascular lesions.
- It is a commonly used endoscopic approach for colonic angioectasia.

Rebleeding remains an important outcome

- Successful local therapy does not necessarily eliminate all lesions.
- Additional or previously unrecognized foci may contribute to recurrence.
- Follow-up is guided by symptoms, hemoglobin and treatment requirements.

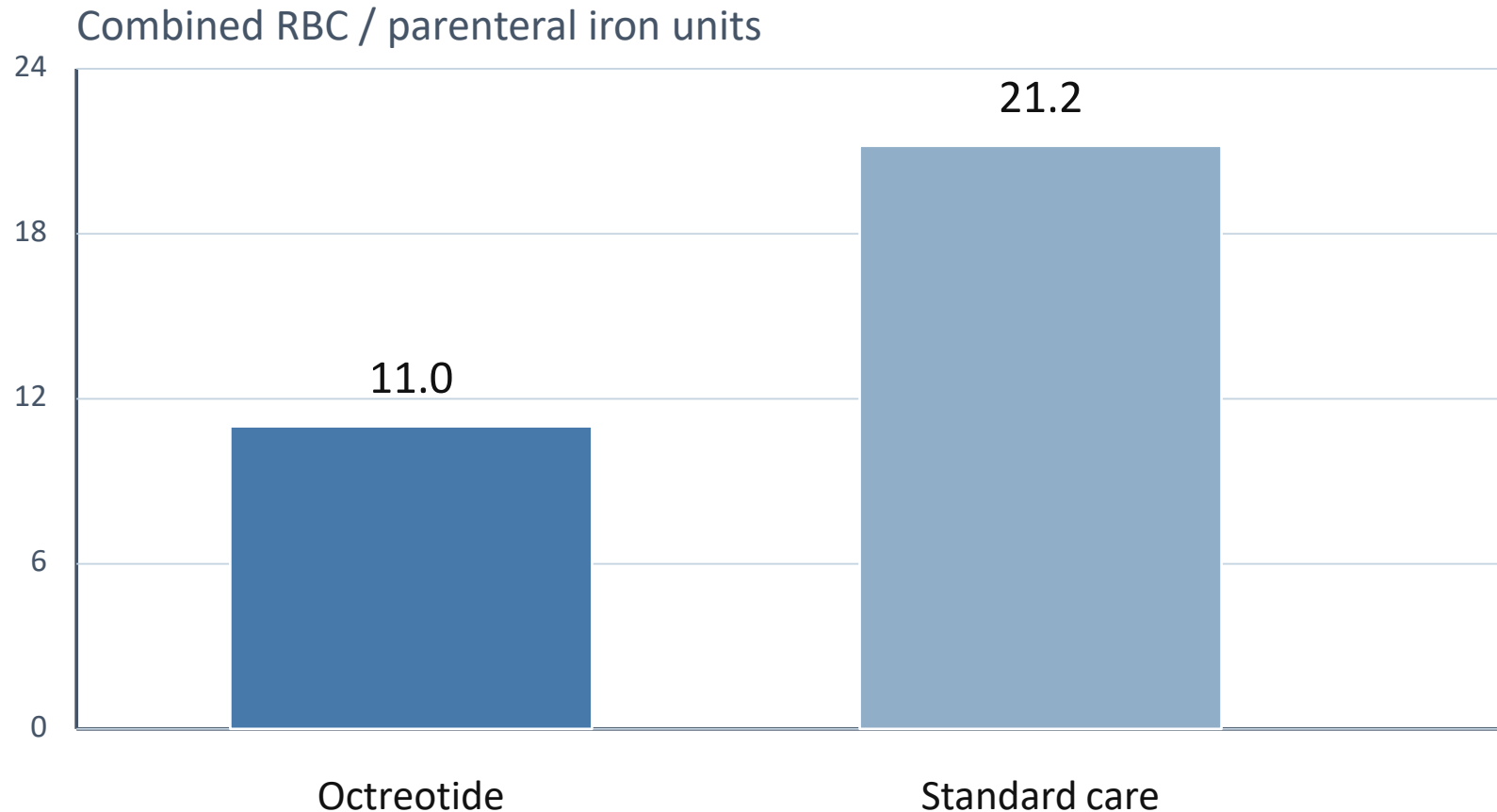
Refractory disease changes the management question

- Persistent transfusion or parenteral iron needs define clinically important burden.
- Repeated procedures may be insufficient for some patients.
- Systemic therapies have trial evidence in selected severe/recurrent bleeding populations.

OCEAN trial: The studied population

- A 2024 open-label multicenter RCT enrolled 62 patients with angiodysplasia-related anemia.
- Patients had substantial prior RBC transfusion or parenteral iron needs.
- Octreotide long-acting release was compared with standard care over one year.

OCEAN trial: Transfusion requirement decreased



Adjusted between-group difference: 10.2 composite units (95% CI 2.4–18.1).

The result concerns severe angiodysplasia-related anemia, not all incidental ectasias.

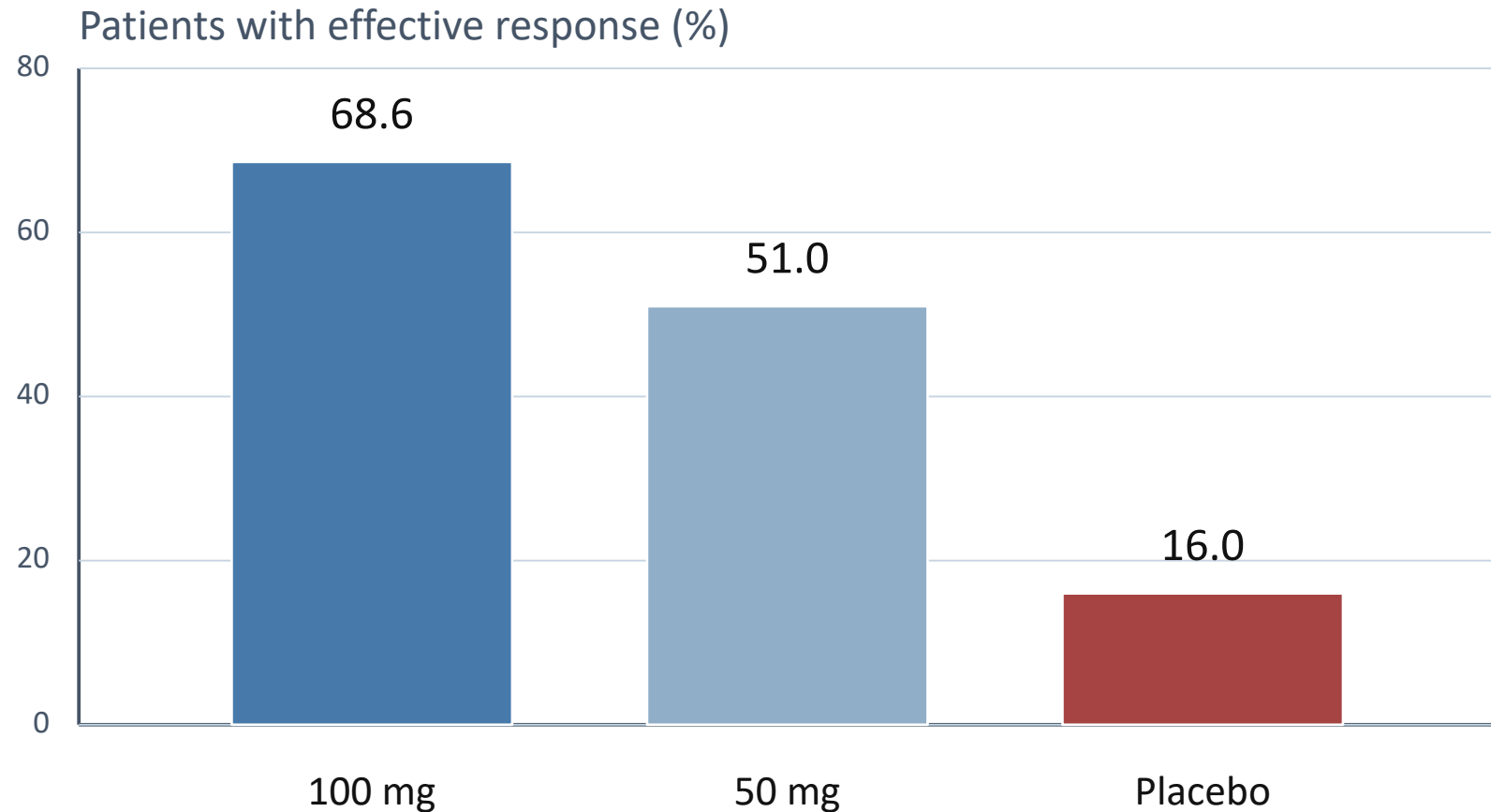
OCEAN trial: Implications and constraints

- The intervention group required fewer endoscopic procedures.
- The trial was open label and small, with selected transfusion-dependent patients.
- This is supplementary management evidence, not a Robbins morphologic criterion.

Thalidomide RCT: A separate clinical phenotype

- A 2023 placebo-controlled RCT enrolled 150 patients with recurrent small-intestinal bleeding.
- Patients were assigned to 100 mg/day, 50 mg/day or placebo for four months.
- The primary endpoint assessed at least 50% fewer bleeding episodes during follow-up.

Thalidomide RCT: Primary response



The trial showed a reduction in recurrent bleeding in its selected population.

It does not establish routine use for all patients with colonic angiodysplasia.

Benefits must be interpreted alongside harms

- Thalidomide adverse events included constipation, somnolence and peripheral symptoms.
 - Teratogenic risk and neurotoxicity demand specialist safeguards and careful selection.
 - Evidence of efficacy is not equivalent to unrestricted treatment recommendation.
-

Case study: Intermittent occult blood loss

An older adult has recurrent iron-deficiency anemia without dramatic hematochezia.

Colonoscopy reveals right-colonic ectatic vascular patches.

Interpretation: Lesion location fits Robbins, but competing causes of anemia must be assessed.

Case study: Brisk overt lower GI bleeding

An older adult presents with repeated hematochezia and hemodynamic compromise.

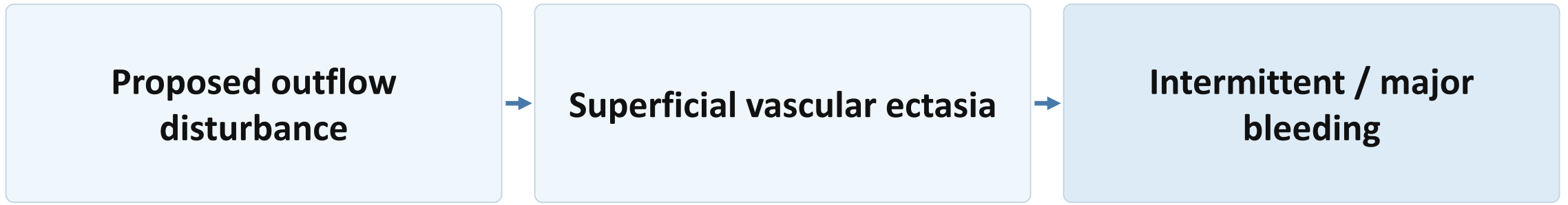
Immediate priorities are severity assessment and stabilization.
Localize active hemorrhage before attributing it to a suspected angioectasia.

Case study: Persistent anemia after endoscopic care

A patient with repeated treatment still requires transfusions or intravenous iron.

Reassess bleeding sources and cumulative burden rather than assuming cure.
Selected patients may merit specialized discussion of evidence-supported systemic options.

Mechanism–morphology–clinical correlation



- Proposed venous compression explains a plausible route to vascular ectasia.
- Dilated, tortuous superficial channels explain bleeding susceptibility.

Five diagnostic distinctions that prevent error

- Malformation is not a vascular tumor.
- Right-colonic preference is not right-colon exclusivity.
- Proposed pathogenesis is not experimentally established causation.
- Incidental angioectasia is not always the active bleeding source.
- Small-bowel disease requires a different diagnostic route.

References and source boundaries

- Core morphology, distribution and mechanism: Robbins, Cotran & Kumar, 11th edition, Ch. 17, pp. 712–713.
- Vascular ectasias and mimics: Robbins Ch. 11 and Ch. 17.
- Clinical investigation: ACG lower GI bleeding guideline (2023); ESGE small-bowel guideline (2022 update).
- Therapy trial context: OCEAN (Gastroenterology, 2024); Chen et al. (NEJM, 2023).