

Ischemic Bowel Disease

Etiology • Perfusion pathogenesis • Morphology • Clinical correlation

Pathology of the small intestine and colon

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraisakhri.com

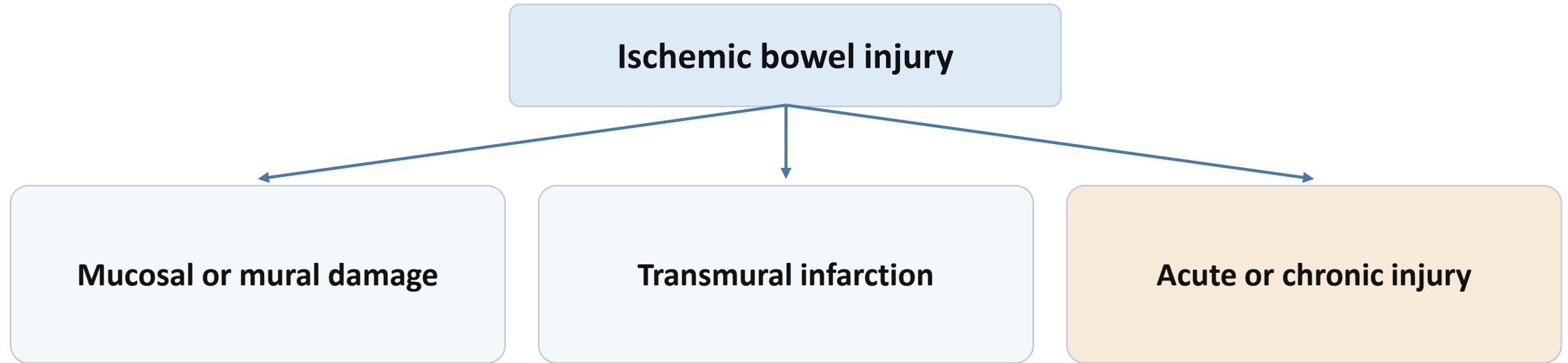
Learning objectives

- Classify ischemic injury by vessel mechanism and wall depth.
- Explain why watershed sites and surface epithelium are vulnerable.
- Recognize acute and chronic gross/microscopic patterns.
- Connect morphology to clinical risk, mimics, and complications.

**Perfusion mechanism →
tissue pattern → clinical
consequence**

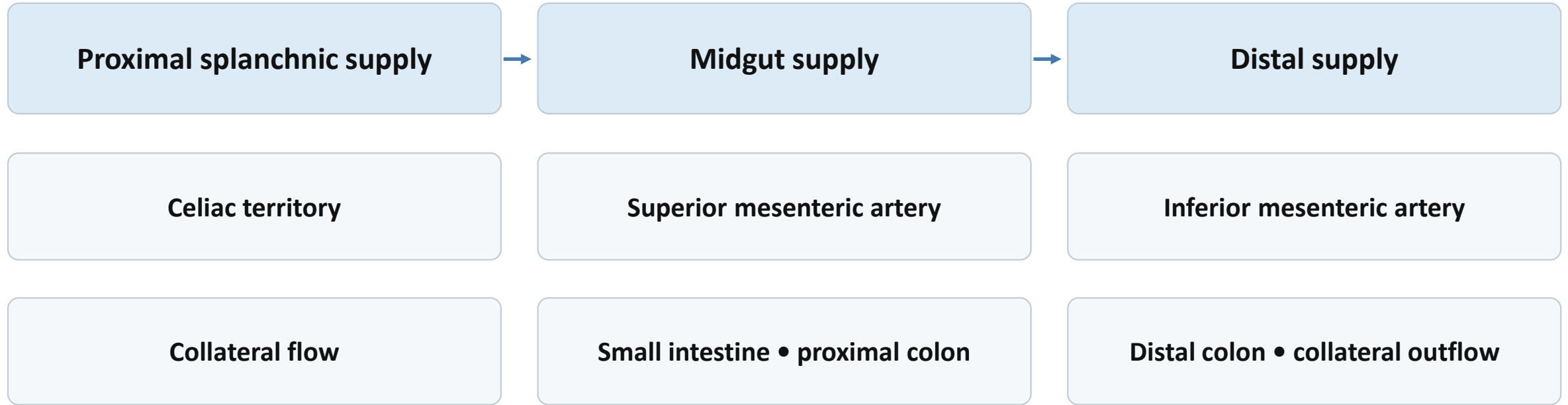
Pathology-led lecture

A perfusion disorder with several phenotypes



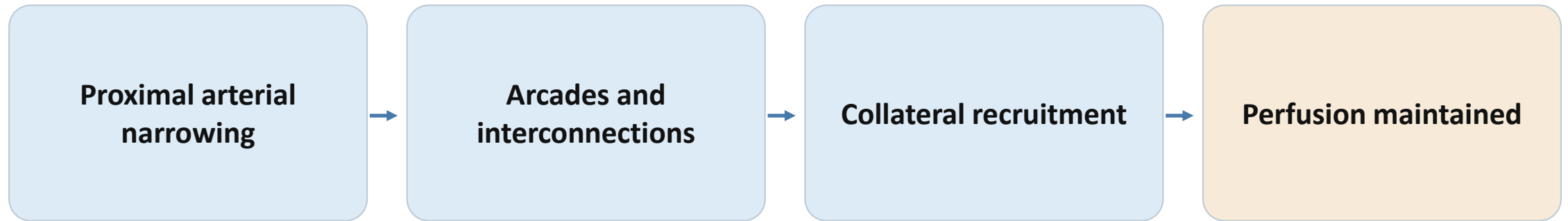
- Vascular anatomy, severity, and duration shape the injury.
- The same core mechanism can produce a range of presentations.

Three major arterial inflows



Celiac, superior mesenteric, and inferior mesenteric systems supply most of the GI tract.

Mesenteric arcades buffer slow vascular loss



Progressive loss of one supply can be compensated by established collateral routes.

Abrupt compromise exceeds collateral capacity

Slow, progressive hypoperfusion

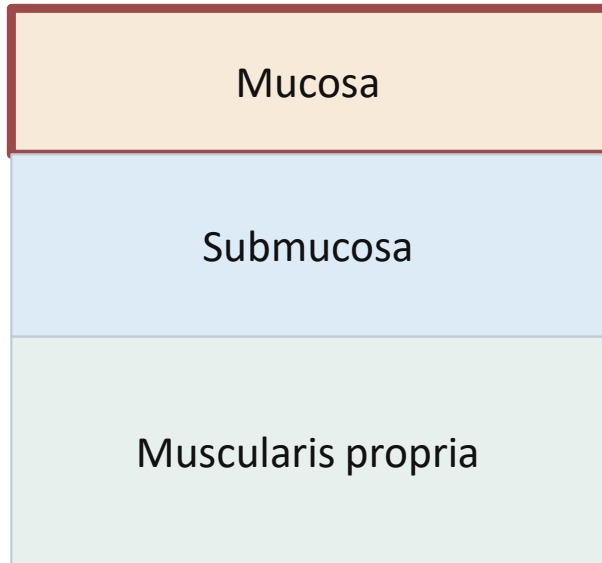
- Collateral channels have time to compensate.
- Mucosal injury may be limited or delayed.
- Anatomic reserve is clinically important.

Acute major-vessel occlusion

- Collateral reserve may be overwhelmed.
- Long bowel segments can infarct.
- The resulting injury may be transmural.

Depth of infarction defines severity

Mucosal infarction



Mural infarction



Transmural infarction



Schematic — red outline marks the depth of injury; Not a histologic specimen.

Mucosal infarction is superficial

- Necrosis extends no deeper than muscularis mucosae.
- The outer bowel wall may remain structurally intact.
- Recovery depends on surviving mucosal regenerative compartments.

**Limited depth ≠ trivial
cause**

Anatomic definition

Mural infarction extends into submucosa

- Includes mucosa and submucosa.
- Edema and hemorrhage can substantially thicken the wall.
- Damage may develop with acute or chronic hypoperfusion.

**Submucosal
involvement increases
mural injury**

Anatomic definition

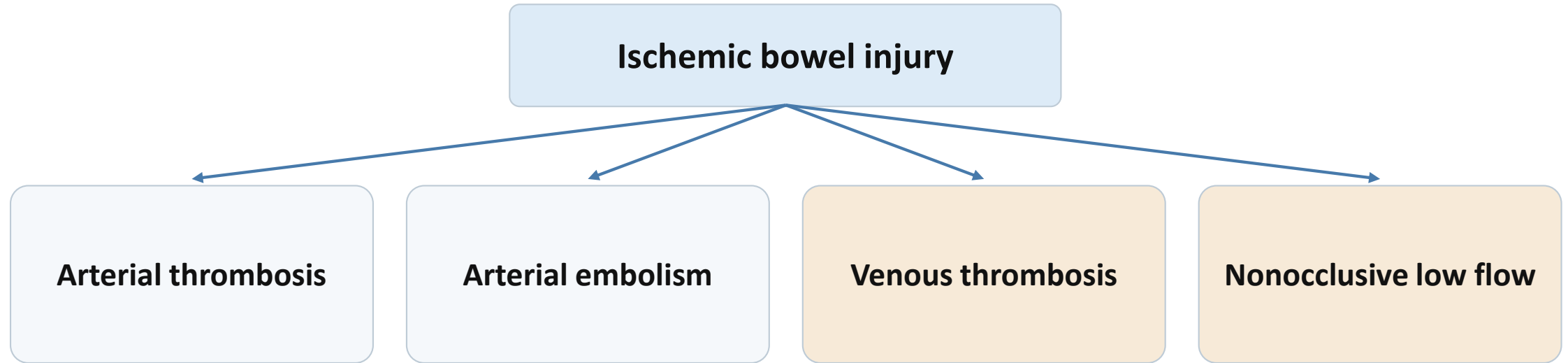
Transmural infarction involves the full wall

- Necrosis extends through the muscularis propria.
- Most typical after acute vascular obstruction.
- Risk includes perforation and serosal inflammation.

**Full-thickness necrosis
threatens bowel
integrity**

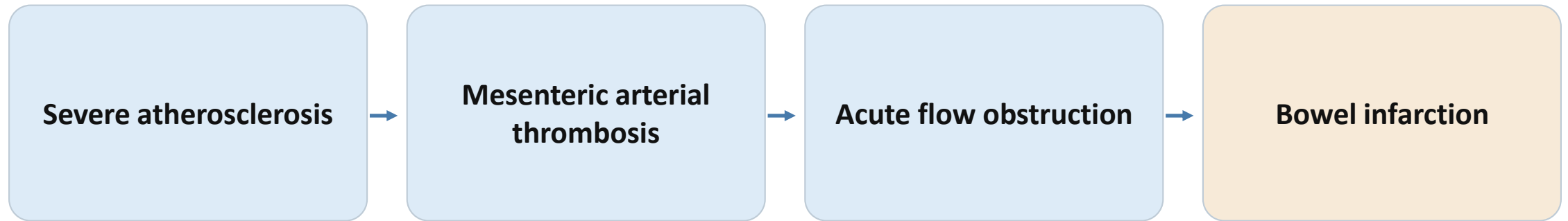
Anatomic definition

Classify the causal perfusion failure



- These mechanisms converge on inadequate intestinal oxygen delivery.

Arterial thrombosis: Atherosclerotic substrate



Severe atherosclerosis, often near vessel origins, as the leading thrombosis risk.

Why mesenteric vessel origins matter

- Atherosclerotic disease often affects the origin of major mesenteric vessels.
- Thrombus superimposed on such disease can acutely obstruct flow.
- Collateral supply may fail to compensate for abrupt interruption.

**Proximal obstruction
can affect long bowel
segments**

Arterial anatomy

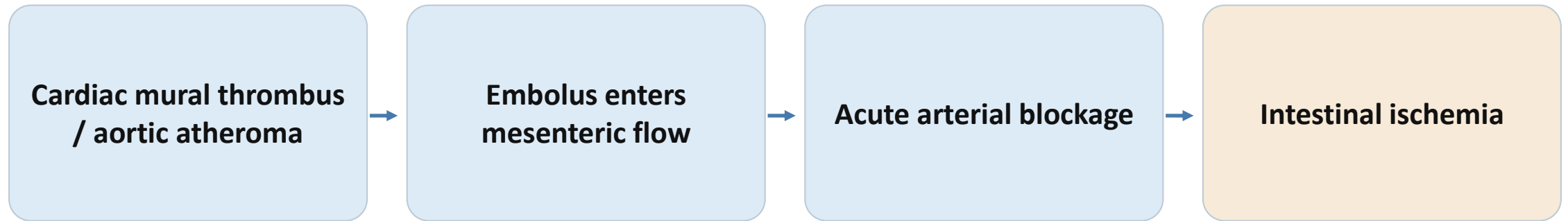
Vasculitis is a less common thrombotic cause

- Systemic vasculitides can compromise intestinal arterial perfusion.
- The vascular lesion may produce local thrombosis and ischemic damage.
- Consider the systemic vascular context when interpreting bowel injury.

**The ischemic pattern
does not specify the
vascular disease**

Etiologic differential

Arterial emboli originate upstream



Cardiac mural thrombi and aortic atheromas as common sources.

Thrombosis versus embolism

Thrombotic obstruction

- Develops at an affected intestinal vessel.
- Severe atherosclerosis is the principal predisposition.
- Systemic vasculitis is a less common setting.

Embolic obstruction

- Material travels from a remote source.
- Cardiac mural thrombi are a source.
- Aortic atheromatous material can embolize.

Venous thrombosis is an uncommon cause

- Venous outflow obstruction reduces effective intestinal perfusion.
- Arterial inflow initially continues, causing congestion.
- Propagation can involve a larger splanchnic drainage bed.

**Venous obstruction can
eventually limit arterial
inflow**

Distinct hemodynamic mechanism

Risk settings for mesenteric venous thrombosis

Risk group	Source-listed examples
Hypercoagulability	Inherited or acquired disorders
Portal/visceral disease	Cirrhosis; Invasive neoplasms
Mechanical/physical	Trauma; Masses compressing portal drainage

Venous obstruction produces a different border

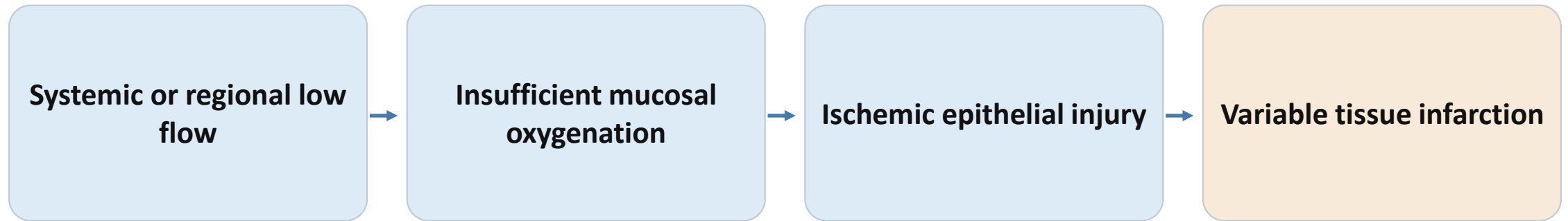
Acute arterial obstruction

- Abrupt reduction in incoming blood.
- Often sharply demarcated infarction.
- May involve extensive intestine.

Mesenteric venous thrombosis

- Arterial inflow continues transiently.
- Congestion develops before full perfusion failure.
- Transition to normal bowel is less abrupt.

Nonocclusive ischemia occurs without a clot



Absence of arterial or venous obstruction does not exclude intestinal ischemia.

Four nonocclusive settings

Setting	Perfusion consequence
Cardiac failure	Reduced effective circulatory output
Shock	Systemic tissue hypoperfusion
Dehydration	Reduced circulating fluid reserve
Vasoconstrictive drugs	Reduced regional intestinal blood flow

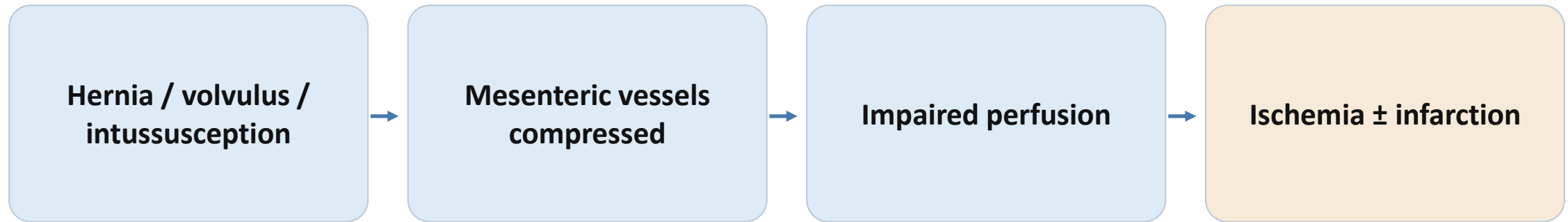
Vasoconstrictors may trigger focal injury

- Drugs capable of intense vasoconstriction can restrict bowel perfusion.
- The textbook explicitly identifies cocaine as an example.
- Watershed zones are susceptible during additional perfusion stress.

**Clinical exposure can
explain a focal ischemic
pattern**

Mechanism, not histologic diagnosis

Mechanical obstruction can compromise vessels



These are parent-context mechanisms; The obstructing process and perfusion injury can coexist.

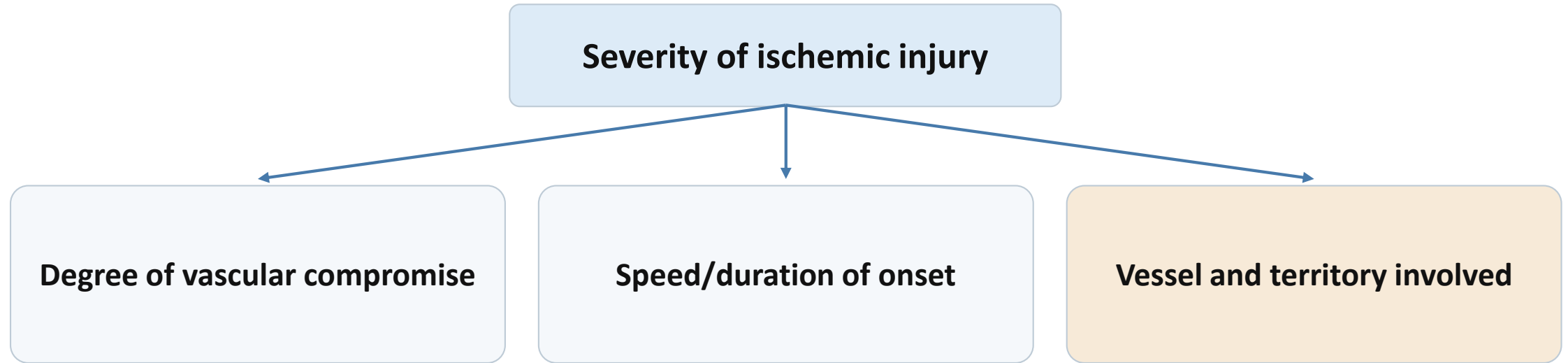
Bowel strangulation: A clinicopathologic link

- Incarcerated hernia may show dusky serosa and associated hemorrhage.
- Volvulus rotates bowel around its mesenteric attachment.
- Intussusception drags mesentery into the telescoped segment.
- Untreated vascular compression can culminate in infarction.

**Mechanical lesion →
compromised vascular
supply**

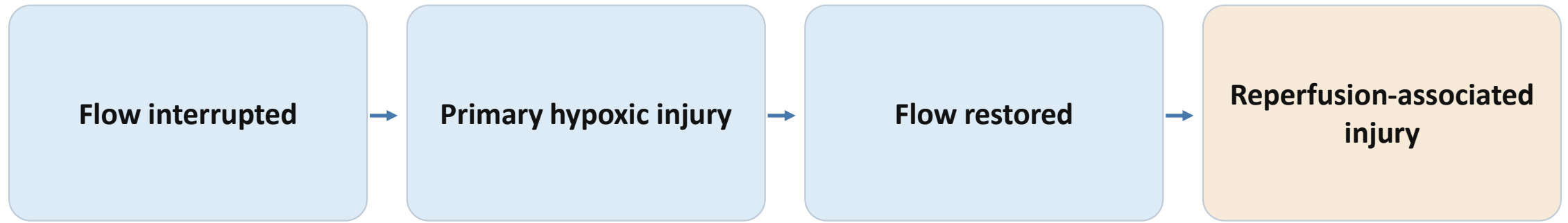
Adjacent Robbins context

Three variables determine injury severity



- Distribution and depth are the integrated consequence of these variables.

Two phases of ischemic tissue injury



That injury can increase after blood supply is restored.

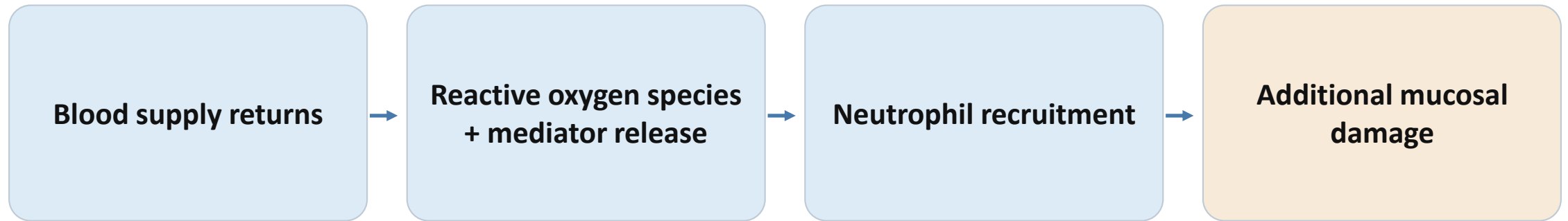
Phase I: Hypoxic epithelial damage

- Impaired perfusion reduces oxygen delivery at the outset.
- Surface epithelial cells are especially vulnerable.
- Early surface atrophy and sloughing can appear before florid inflammation.

**Initial ischemia need
not be neutrophil-rich**

Chronology of injury

Phase II: Reperfusion amplifies injury

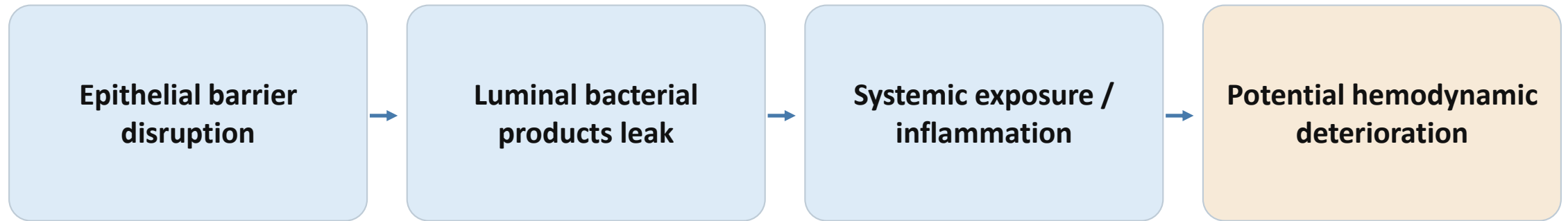


The mechanistic list is proposed and not portrayed as fully resolved.

Potential amplifiers during reperfusion

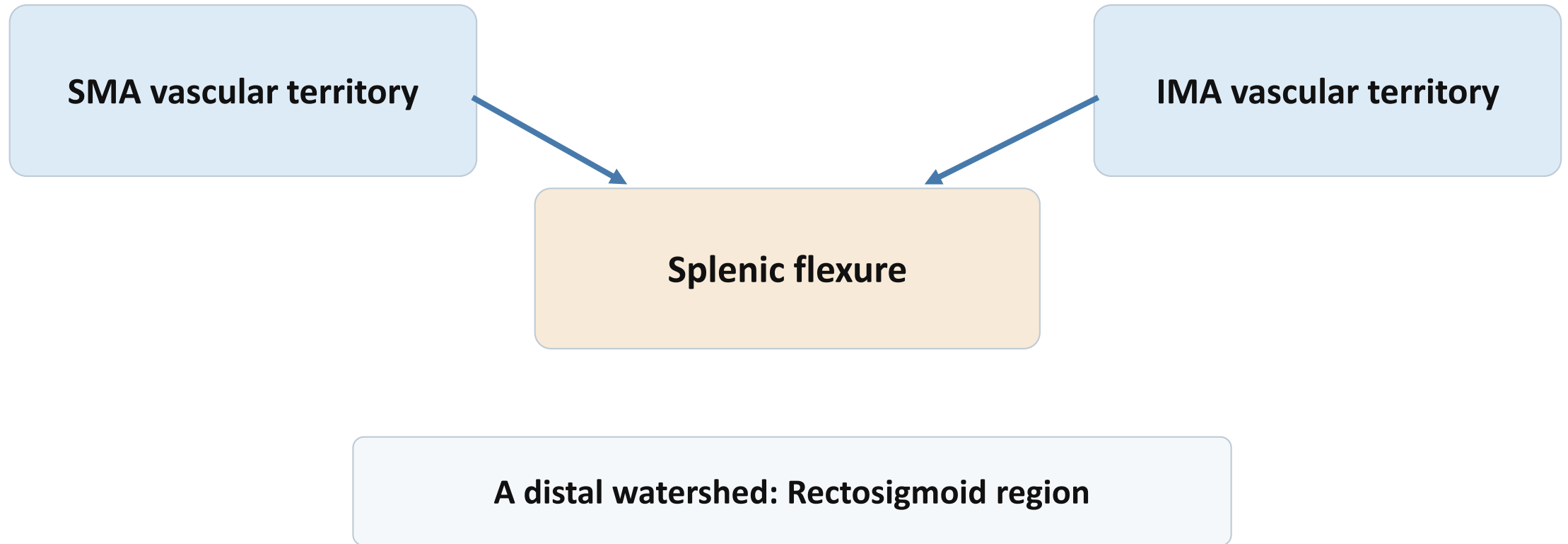
Contributor	Potential effect
Reactive oxygen species	Oxidative tissue damage
Neutrophil influx	Inflammatory tissue injury
Inflammatory mediators	Propagation of local damage
Luminal bacterial products	Systemic exposure after barrier failure

Bacterial products connect mucosa and systemic effects



Robbins specifically names lipopolysaccharide as an example of luminal bacterial material.

Vascular geography predicts vulnerable sites



Schematic: Boundary zones are disproportionately affected during systemic low flow.

Splenic flexure: A classic watershed zone

- Lies near the termination of superior and inferior mesenteric arterial territories.
- Generalized hypotension can cause segmental injury here.
- Focal splenic-flexure colitis warrants consideration of ischemia.

**Arterial boundary →
vulnerability to low
flow**

Localization matters

Rectosigmoid watershed vulnerability

- Distal arterial circulations meet in the sigmoid and rectal region.
- The inferior mesenteric, pudendal, and iliac supplies contribute.
- This zone as vulnerable to a lesser degree.

**Distribution is
informative, not
diagnostic alone**

Collateral anatomy

Watershed injury can be focal

Generalized insult

- Shock or hypotension reduces perfusion everywhere.
- Watershed territories experience disproportionate injury.
- A localized mucosal lesion can result.

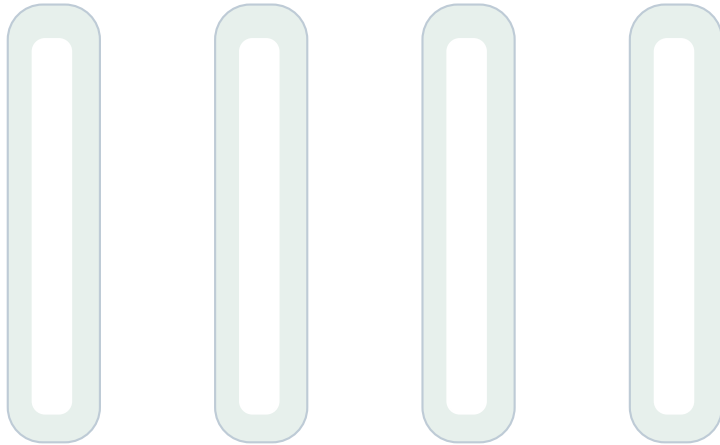
Localized observation

- Splenic-flexure or rectosigmoid colitis.
- Consider ischemia alongside alternative colitides.
- Correlate location with perfusion history.

Mucosal microcirculation creates selective injury

Conceptual mucosal unit

Surface epithelium — vulnerable



Crypt compartment — regeneration

Arterial inflow at crypt base



Capillaries ascend toward surface



Hairpin turn → venular return

Schematic — vessels and crypts are simplified; No scale or actual tissue architecture implied.

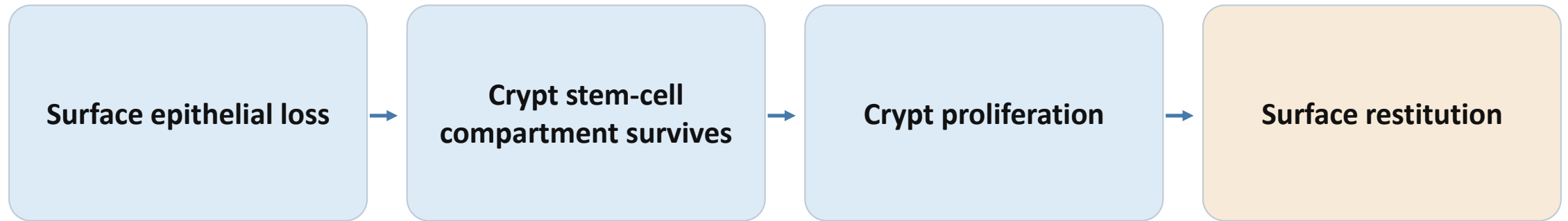
Surface epithelium is a hemodynamic weak point

- Capillaries ascend along intestinal glands to the luminal surface.
- A hairpin-turn pattern precedes venular drainage.
- Surface epithelium is exposed to relative oxygen vulnerability.

**The superficial
compartment fails first**

Microvascular topology

Crypt preservation enables epithelial repair



The ischemic pattern often combines attenuated surface epithelium with normal or proliferating crypts.

Surface atrophy with reactive crypts: Signature

Surface epithelial compartment

- Attenuation or sloughing after ischemia.
- Early mucosal injury may be striking.

Crypt epithelial compartment

- Often relatively preserved.
- Regenerative hyperproliferation may occur.

GI ischemia is often segmental and patchy

- The colon is the most common GI location.
- Mucosal or mural infarction can occur from stomach to anus.
- Lesions may be continuous but are more often segmental or patchy.

Pattern reflects vascular territories and severity

Gross distribution

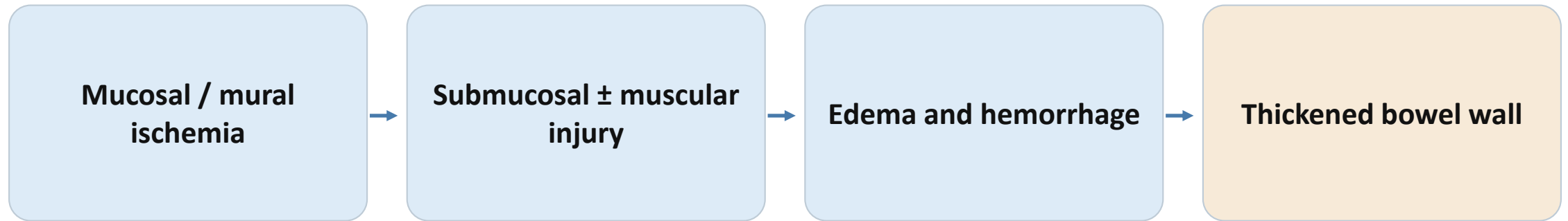
Acute mucosal injury: Hemorrhage and ulceration

- Affected mucosa becomes hemorrhagic.
- Surface injury can produce mucosal ulceration.
- Patchy injury may coexist with apparently spared mucosa.

Hemorrhagic colitis is a pattern, not an etiology

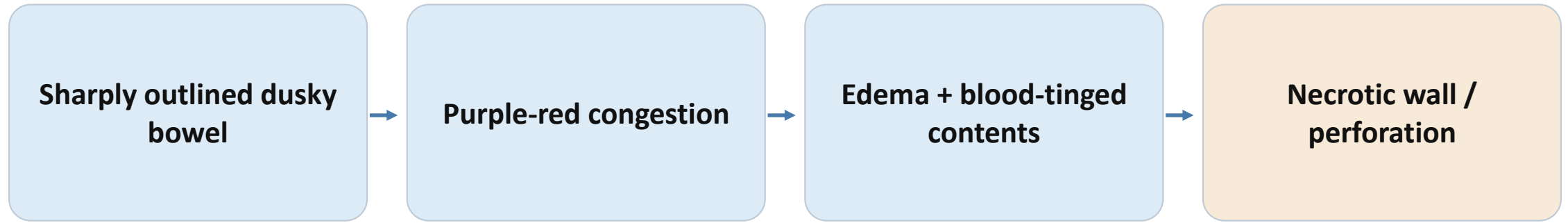
Gross morphology

Edematous wall thickening: Depth-dependent



Gross wall thickening becomes more conspicuous when injury extends below the mucosa.

Acute transmural infarction: Gross sequence



This sequence summarizes the temporal gross evolution given by Robbins.

Arterial infarct borders are often sharp

Major arterial obstruction

- Abrupt inflow failure.
- Distinct interface between viable and infarcted bowel.
- Often substantial segments affected.

Venous thrombosis

- Inflow persists before venous backup dominates.
- Less sharply abrupt transition.
- Hemorrhagic congestion may be extensive.

Muscularis propria necrosis takes time

- Coagulative necrosis of the muscularis propria within 1–4 days.
- Depth and duration determine loss of mechanical integrity.
- Progressive necrosis can lead to bowel perforation.

**Timing influences the
observed wall pattern**

Textbook-reported morphologic timing

Serosal findings signal advanced damage

- Severe infarction can produce serositis.
- Fibrin deposition and purulent exudate may be evident.
- Perforation is a critical consequence of full-thickness injury.

**Serosal response
supports advanced
transmural involvement**

Gross/surgical pathology

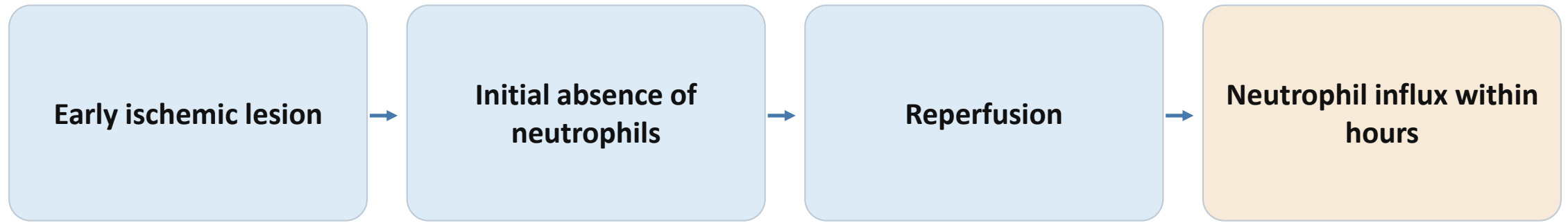
Early microscopy: Surface loss predominates

- Surface epithelial atrophy and sloughing are characteristic.
- Crypt proliferation may persist alongside surface damage.
- Early inflammatory infiltrates can be absent.

**Think ischemia even
without early
neutrophil excess**

Microscopic morphology

Reperfusion recruits neutrophils



Inflammation varies with injury chronology; It is not a fixed morphologic constant.

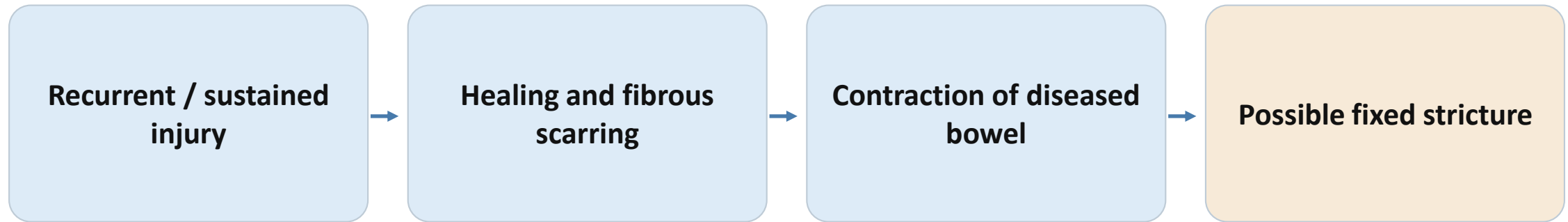
Chronic ischemia remodels the lamina propria

- Surface epithelium may remain atrophic.
- Fibrous scarring affects the lamina propria.
- Repeated damage and healing can alter architecture.

**Lamina propria fibrosis
suggests chronicity**

Histologic context

Ischemic strictures are uncommon sequelae



Stricture formation as uncommon, not an inevitable outcome.

Ischemia may produce pseudomembranes

- Bacterial superinfection and enterotoxin release can cause pseudomembrane formation.
- This complication occurs in both acute and chronic ischemia.
- The pattern can resemble C. Difficile–associated colitis.

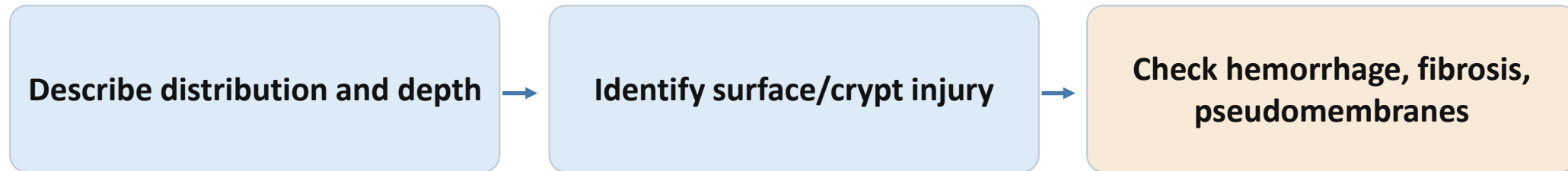
**Pseudomembranes are
not exclusive to
infection**

Morphologic differential

Histologic signatures: Acute versus chronic

Feature	Acute ischemia	Chronic ischemia
Surface	Atrophy / sloughing	Persistent atrophy
Crypts	May be hyperproliferative	Regenerative changes possible
Inflammation	Initially sparse; Later neutrophils	Variable
Stroma	Hemorrhage / edema	Lamina propria fibrosis

A practical morphology interpretation sequence



- Avoid assigning a vascular cause from morphology alone.
- Integrate the bowel segment and clinical vascular setting.

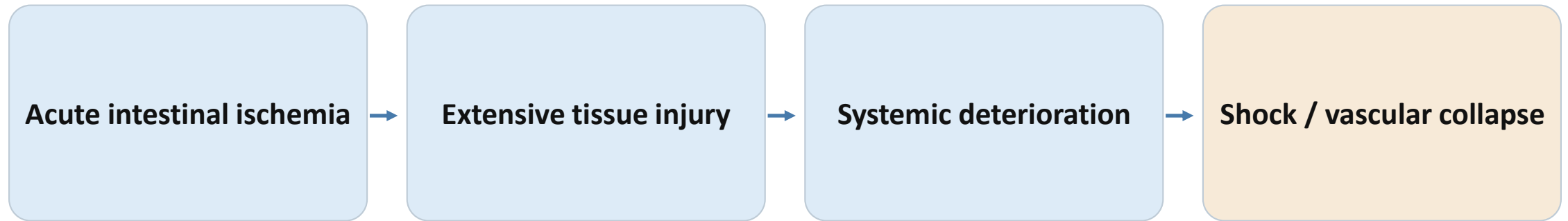
Colonic ischemia: Typical acute presentation

- Sudden cramping abdominal pain, often left lower quadrant.
- Urgency or desire to defecate.
- Blood per rectum or bloody diarrhea.

**Pain and bleeding may
appear abruptly**

Clinical features

Severe injury may rapidly destabilize patients



In severe cases progression to shock may occur within hours.

When physical signs raise concern for infarction

- Diminished peristaltic sounds can be concerning.
- Guarding and rebound tenderness suggest more advanced abdominal pathology.
- Surgical assessment is considered when infarction is suspected.

**Peritoneal findings
change the urgency of
evaluation**

Textbook clinical discussion

Acute bowel infarction has competing diagnoses

Clinical overlap	Why misclassification matters
Acute appendicitis	Overlapping pain and examination findings
Perforated peptic ulcer	Peritonitis can look similar
Acute cholecystitis	Can resemble an acute surgical abdomen
Other colitides	Blood and diarrhea are not etiology-specific

Epidemiology of colonic ischemia

- Colonic ischemia as most common after age 70.
- A slight female predominance is noted.
- Approximately 10% require surgical intervention series.

**Epidemiology informs
suspicion, not exclusion**

Robbins, 11th ed.; Not a contemporary
population estimate

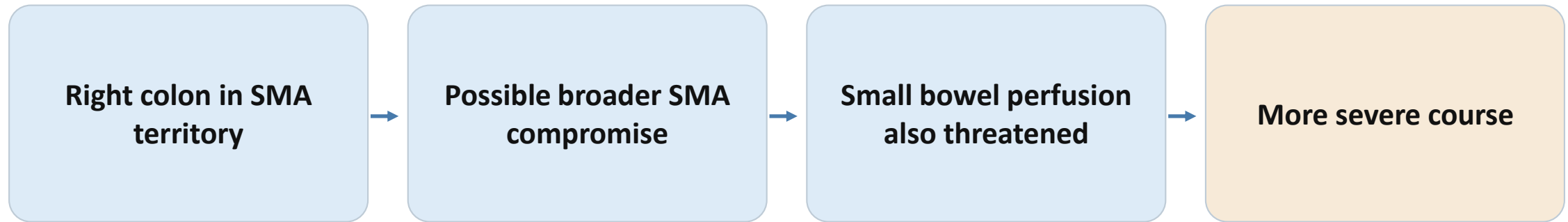
Clinical outcomes reported by Robbins

- With appropriate management, reported 30-day mortality is approximately 10%.
- Right-sided colonic ischemia has approximately twice the mortality.
- These textbook estimates are context dependent, not patient-specific predictions.

**Distribution and
systemic comorbidity
affect prognosis**

Textbook estimates; Not individualized
predictions

Right-sided colonic ischemia may be more severe



This is the textbook explanation for the adverse right-sided pattern; Not a universal causal proof.

Markers of a poorer clinical course

Factor	Why it matters for interpretation
Right-colon involvement	May imply broader compromised SMA perfusion
Coexisting COPD	Reported adverse prognostic association
Symptoms persisting >2 weeks	Reported poor prognostic indicator
Evidence of infarction	Associated with need for urgent surgical assessment

Subacute nonocclusive injury can be missed

- Intermittent bloody diarrhea is not specific for ischemia.
- Obstructive symptoms can coexist.
- The absence of obvious arterial occlusion can complicate diagnosis.

**Nonocclusive disease is
a clinicopathologic
diagnosis**

Subacute/chronic clinical forms

Chronic ischemia can mimic inflammatory bowel disease

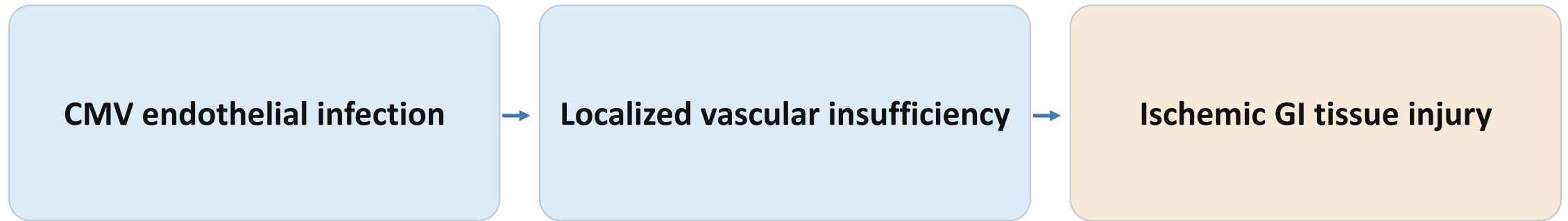
Chronic ischemia

- Episodes of bloody diarrhea may recur.
- Periods of partial healing intervene.
- Fibrosis can accumulate in lamina propria.

Inflammatory bowel disease mimic

- Overlapping chronic colitis manifestations.
- History and ischemic surface/stromal pattern matter.
- Clinical correlation is essential.

CMV infection can cause vascular insufficiency



CMV as a vascular mechanism, particularly relevant with immunosuppression.

Radiation injury combines two targets

Epithelial injury

- Damage to surface and glandular epithelium.
- Acute cramps, anorexia, or malabsorptive diarrhea.

Endothelial / stromal injury

- Contributes to perfusion impairment.
- Chronic enterocolitis can be indolent.
- Atypical radiation fibroblasts may be a clue.

Necrotizing enterocolitis is a neonatal parallel

- An acute disorder of small and large intestines.
- Most prominent among premature or low-birth-weight neonates.
- Often manifests when feeding begins; Can progress to transmural necrosis.
- Ischemia as one contributor, not the sole pathogenesis.

**Similar tissue endpoint;
Distinct neonatal
disease**

Related etiology mentioned by Robbins

Pseudomembranes require an etiologic differential

Morphologic observation	Interpretation
Pseudomembrane	Can arise in ischemia or infection
Surface epithelial loss	Supports mucosal injury; Not cause-specific
Fibrotic lamina propria	Supports a chronic process
Vascular / systemic history	Helps adjudicate etiology

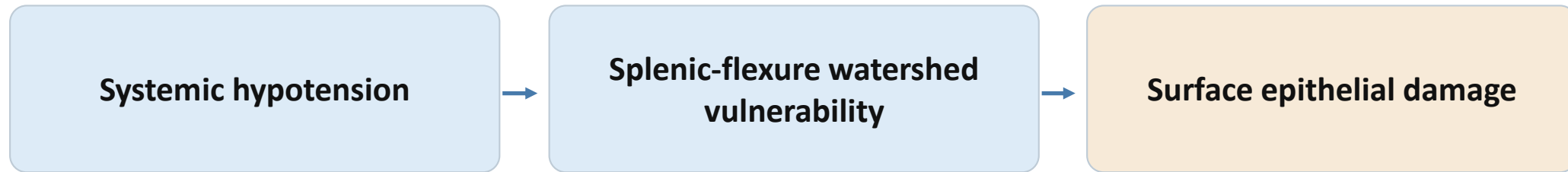
Clinical exercise: Low-flow watershed injury

An older adult develops left-sided cramps and bloody diarrhea after a severe hypotensive episode. Endoscopy identifies a segmental lesion near the splenic flexure.

Diagnostic reasoning prompt

- Which arterial-territory boundary is involved?
- Which epithelial compartment should be most vulnerable?

Low-flow case: Integrate the evidence



- Anatomic pattern and hemodynamic event support ischemia.
- Do not infer transmural infarction from mucosal hemorrhage alone.

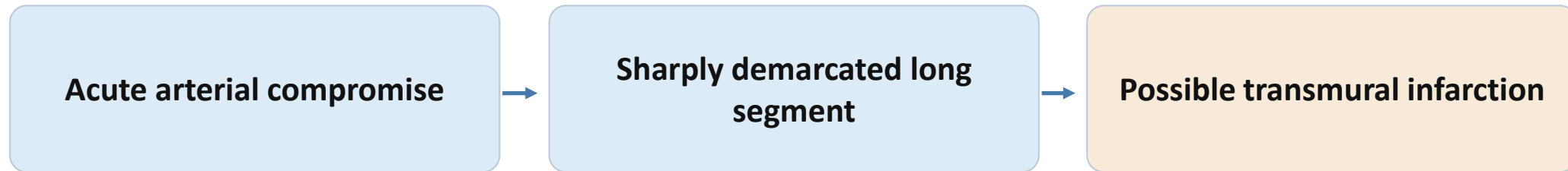
Clinical exercise: Abrupt major-vessel obstruction

A patient with extensive vascular disease develops sudden severe abdominal symptoms. Surgery finds a long sharply demarcated purple-red segment of bowel.

Diagnostic reasoning prompt

- Which vascular mechanism is strongly suggested?
- What wall-depth complication must be evaluated?

Obstructive case: Interpret the gross border



- Severe atherosclerosis favors in situ thrombosis; Embolic sources remain possible.
- Extent of necrosis and perforation risk demand careful examination.

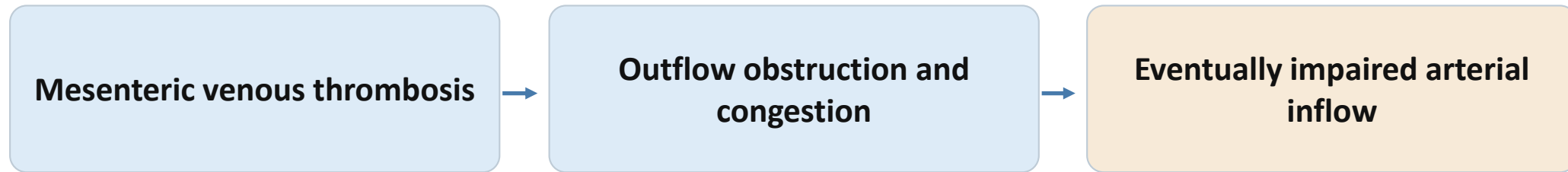
Clinical exercise: Venous outflow failure

A patient with a hypercoagulable condition develops ischemic bowel injury. Affected bowel grades gradually into viable bowel with marked congestion.

Diagnostic reasoning prompt

- Why is the transition less abrupt than arterial occlusion?
- What process ultimately reduces capillary inflow?

Venous case: Follow the hemodynamic sequence



- Initial continuation of arterial inflow alters gross demarcation.
- The endpoint can still resemble severe arterial ischemia.

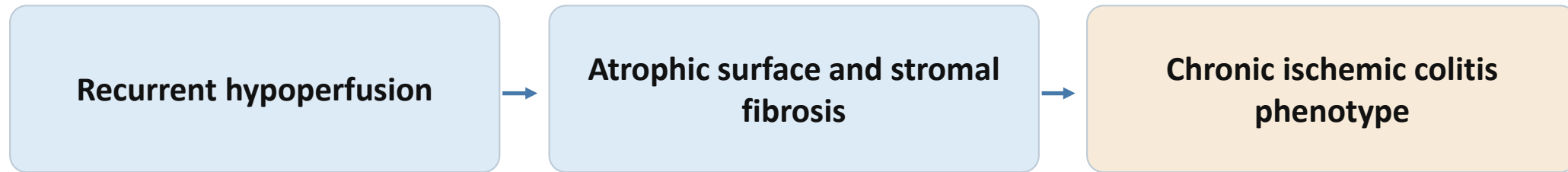
Clinical exercise: Chronic colitis mimic

A patient has recurrent bloody diarrhea with intervals of healing. Biopsy shows surface epithelial attenuation and a fibrotic lamina propria.

Diagnostic reasoning prompt

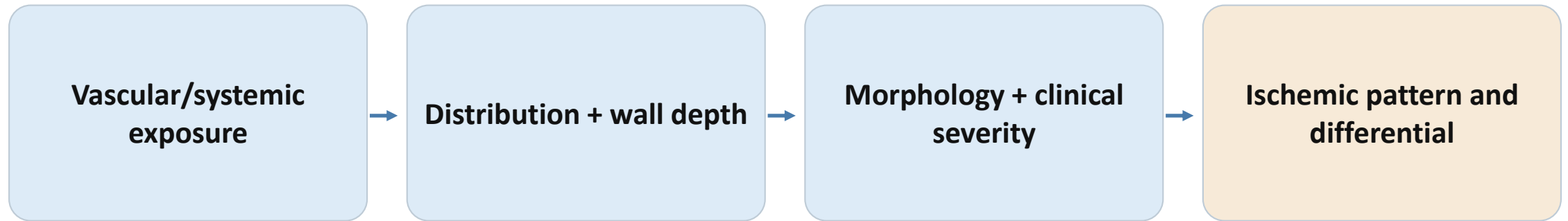
- Which chronic tissue-response pattern is present?
- Why should IBD not be assumed solely from recurrent symptoms?

Chronic case: Morphology and history converge



- Temporal symptoms and ischemic structural changes are concordant.
- The final etiologic diagnosis still requires clinical investigation.

Integrated diagnostic reasoning



The source supports clinicopathologic synthesis, not a morphology-only diagnostic shortcut.

Five core principles of ischemic bowel disease

- Sudden occlusion can overwhelm otherwise protective collateral networks.
- Depth distinguishes mucosal, mural, and transmural infarction.
- Reperfusion can amplify primary hypoxic injury.
- Watershed sites and surface epithelium are selectively vulnerable.
- Chronic injury, CMV, radiation, and pseudomembranes broaden the differential.

**Vascular physiology
explains much of the
pathology**

Final synthesis

Reference and illustration clarification

Robbins, Cotran & Kumar: Pathologic Basis of Disease. 11th edition (2026). Chapter 17, “The Gastrointestinal Tract,” section “Ischemic Bowel Disease,” pp. 711–712.

All teaching schematics are original editable PowerPoint shapes. No reproduced textbook figures or synthetic histopathology are presented.

Conceptual schematics should not be interpreted as actual histopathology or surgical specimens.