

Inflammatory Polyps

Mechanisms, morphology and clinicopathologic interpretation

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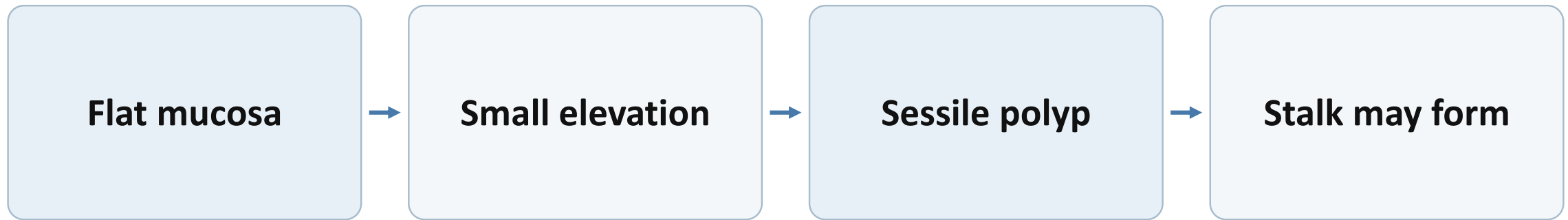
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Learning objectives

- Explain why repeated mucosal injury and healing produce inflammatory polyps.
- Recognize post-inflammatory pseudopolyps and mucosal bridges in colitis.
- Correlate solitary rectal ulcer syndrome with anorectal dysfunction and prolapse.
- Distinguish inflammatory morphology from dysplastic or hamartomatous polyps.

A polyp is a mucosal elevation



- Polyp describes a gross configuration; It is not a histologic diagnosis.
- A pedunculated shape can emerge as an enlarging lesion is pulled into the lumen.

Sessile versus pedunculated configuration

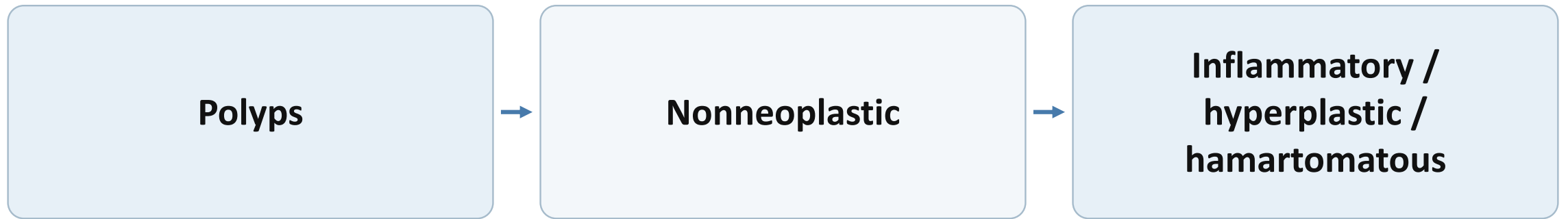
Sessile

- Elevated mucosa without a stalk
- Base contiguous with adjacent mucosa
- Appearance does not establish histologic type

Pedunculated

- Polypoid head connected by a stalk
- Stalk can develop with growth and traction
- Both forms require classification by pathology

The intestinal polyp classification

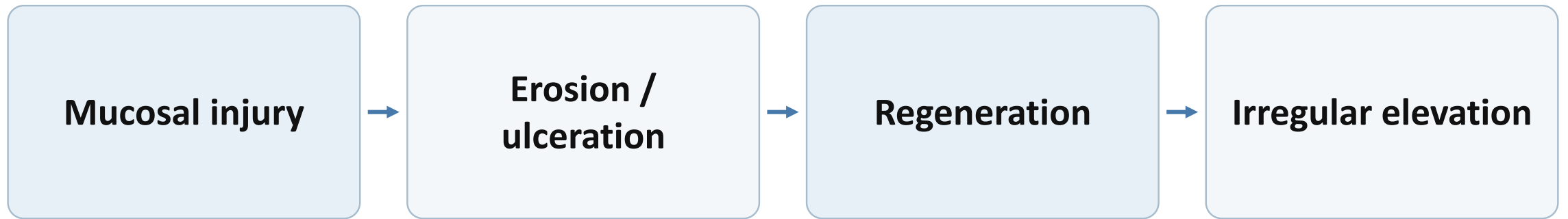


- Neoplastic epithelial polyps are represented most commonly by adenomas.
- A gross polyp may represent a reactive response or a neoplastic process.

Inflammatory polyp: Defining principle

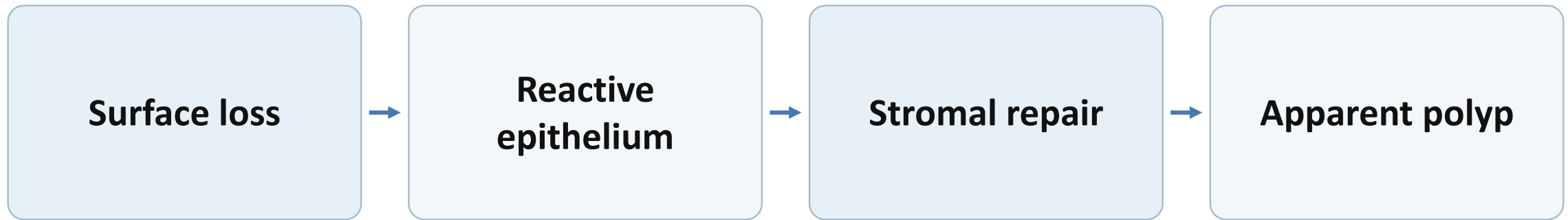
- The lesion is associated with repeated mucosal injury followed by repair.
- Regenerating epithelium and a reactive stromal response may project into the lumen.
- The term describes pathogenesis rather than a unique endoscopic shape.
- Clinical context determines whether colitis or local mucosal prolapse is implicated.

Repeated injury and healing form a polyp



- Surviving epithelium proliferates while previously damaged regions heal.
- Repeated unequal repair can accentuate a polypoid mucosal contour.

Tissue repair explains the gross protrusion



- Epithelial hyperplasia reflects regenerative activity after injury.
- Capillary-rich granulation-like tissue is compatible with repeated surface erosion.

Reactive growth is not synonymous with dysplasia

Reactive / inflammatory

- Related to injury and regeneration
- Often accompanied by inflammation or erosion
- Clinical and architectural context is crucial

Dysplastic / neoplastic

- Abnormal epithelial neoplastic proliferation
- Adenomas are defined by cytologic dysplasia
- Requires independent histologic evaluation

Interpretation requires two complementary views

- Gross and endoscopic appearance: Number, contour and relation to ulcerated mucosa.
- Microscopy: Surface integrity, epithelial regenerative changes and stromal alterations.
- Clinical setting: Chronic colitis versus localized rectal prolapse-related injury.
- Neither morphology nor clinical history should be interpreted in isolation.

Two instructive inflammatory patterns

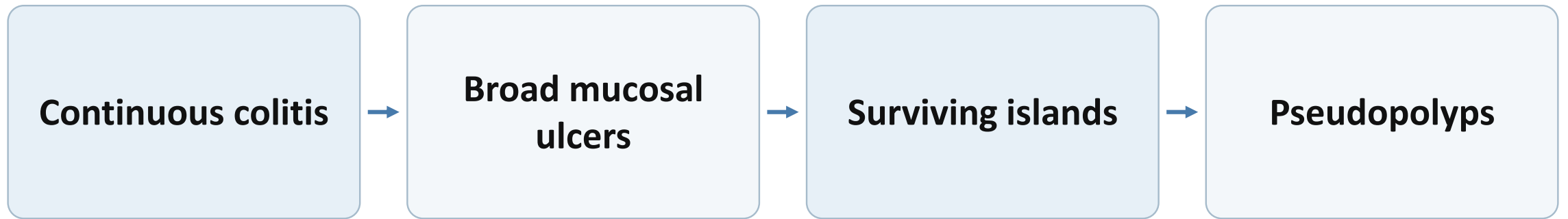
**Colitis-associated
pseudopolyps**

**Solitary rectal ulcer
syndrome**

**Shared repair
response**

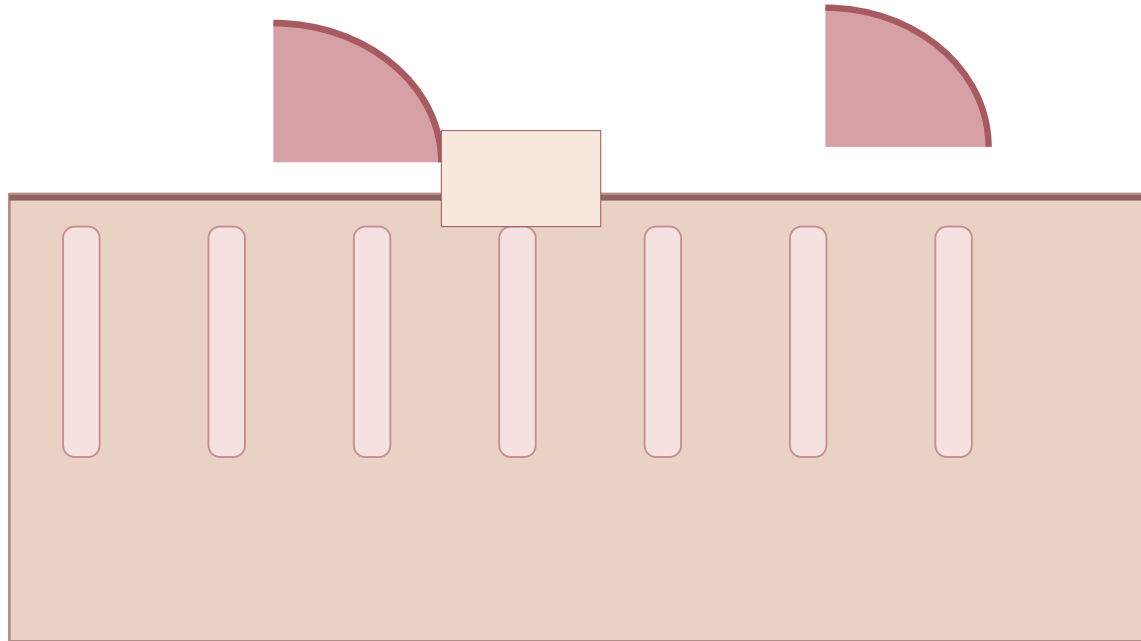
Same broad class — different injury mechanisms

Ulcerative colitis creates a field of injury



- Colitis begins in the rectum and extends proximally without skip lesions.
- Preserved islands can stand proud when adjacent mucosa is ulcerated.

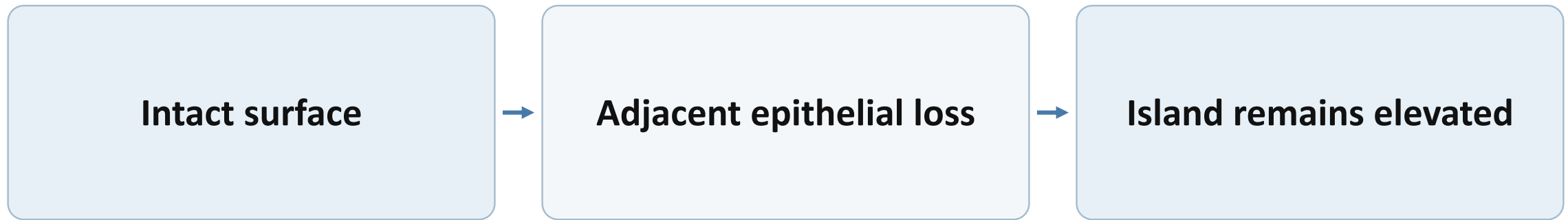
Pseudopolyp: Regenerating mucosal island



Conceptual tissue section — schematic

- Residual mucosa remains between ulcers.
- Surviving mucosa projects into the lumen.
- The elevation can mimic a true neoplasm.
- Interpret the surrounding injured mucosa.

An ulcer becomes a relative depression



- A pseudopolyp is best understood against the depth of nearby injury.
- The elevation can represent preservation and repair, not a new growth clone.

Ulcerative colitis: Distribution is diagnostic context

- The rectum is involved, with continuous proximal spread into the colon.
- The extent ranges from rectal disease to pancolitis.
- Pseudopolyps may coexist with areas of active ulceration and healed mucosal atrophy.
- The distribution supports colitis as the background process, not a standalone polyp category.

Pseudopolyp versus an isolated adenomatous mass

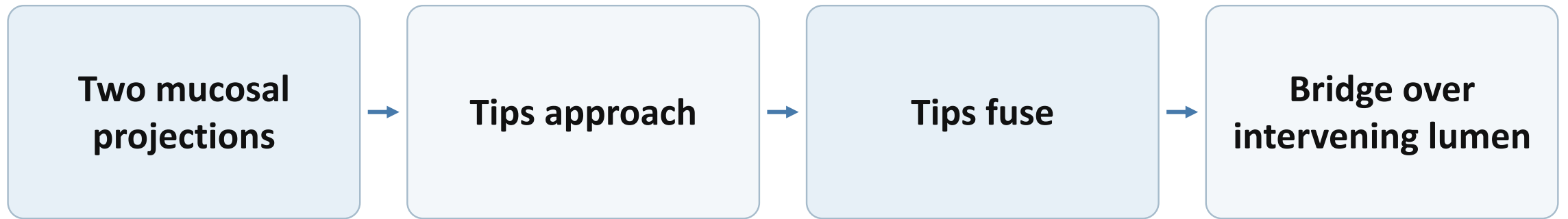
Inflammatory pseudopolyp

- Arises in injured, regenerating mucosa
- May be one of many irregular islands
- Underlying chronic colitis is relevant

Adenoma

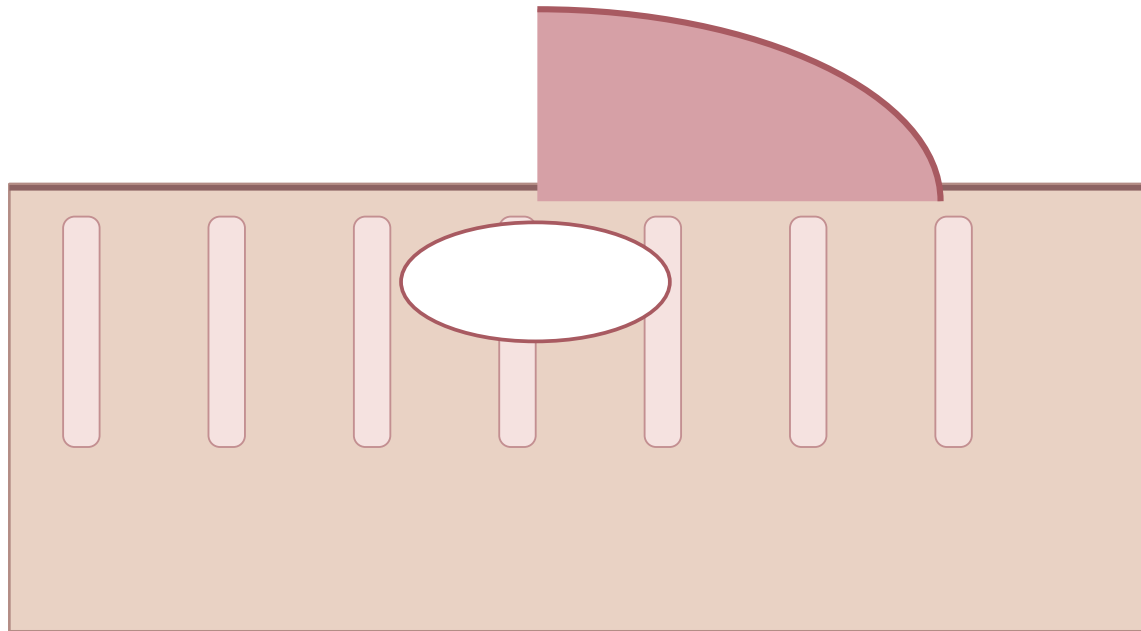
- Neoplastic epithelial proliferation
- Cytologic dysplasia is the hallmark
- Can occur sporadically or in IBD

Mucosal bridges: A reparative configuration



- Fusion of inflammatory polyp tips in ulcerative colitis.
- A mucosal bridge is a gross architectural consequence of repair.

Mucosal bridge: Geometric explanation



Conceptual tissue section — schematic

- A bridge joins adjacent projecting tips.
- Surrounding ulceration makes the contours striking.
- Architecture reflects repeated colitic injury.
- This is a conceptual section, not a micrograph.

Active ulceration and chronic repair coexist

Active surface injury

- Erythematous, granular or ulcerated mucosa
- Surface erosion and inflammatory activity
- Crypt abscesses may be found histologically

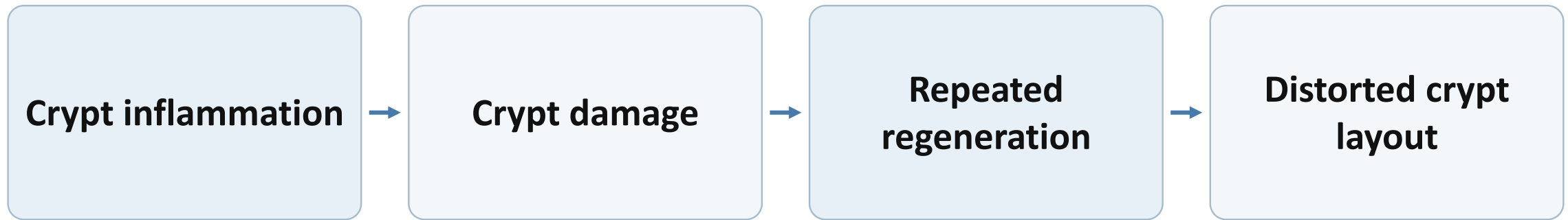
Chronic or healed change

- Regenerating islands or mucosal bridges
- Architectural distortion may persist
- Atrophic smooth mucosa may occur

Chronic ulcerative colitis can flatten the mucosa

- Persistent disease may produce mucosal atrophy and a smooth surface.
- Normal mucosal folds may be lost in chronically affected segments.
- Polypoid regeneration and flattening can appear in different areas of one colon.
- Gross heterogeneity does not necessarily imply different underlying diseases.

Crypt injury produces architectural remodeling



- Crypt abscesses indicate neutrophilic crypt injury in active colitis.
- Architectural disorganization is evidence of repeated injury and repair.

Histologic activity in ulcerative colitis

- Inflammatory infiltrates accompany damaged colonic mucosa.
- Crypt abscesses reflect active neutrophilic injury to glandular epithelium.
- Surface ulceration may be extensive in severe active disease.
- Activity and longstanding architectural distortion are related but distinct findings.

Histologic chronicity in colitis

- Crypt distortion indicates an altered regenerative architecture.
- Metaplastic changes can include pseudopyloric and Paneth cell metaplasia.
- Atrophy may remain after active inflammatory changes recede.
- The background biopsy contributes evidence that a polyp is post-inflammatory.

Ulcerative colitis versus Crohn disease context

Ulcerative colitis

- Continuous disease from rectum
- Inflammation usually mucosal/superficial submucosal
- Granulomas are absent

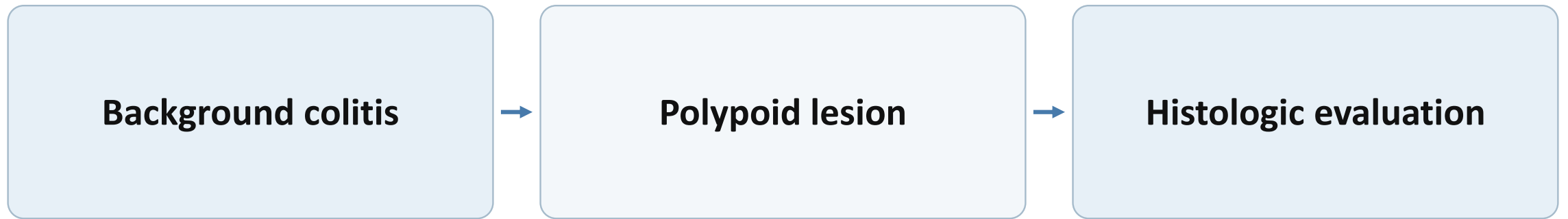
Crohn disease

- Often patchy with skip lesions
- Can involve full bowel-wall thickness
- Noncaseating granulomas can occur

A pseudopolyp does not establish IBD subtype

- A polypoid reaction can accompany chronic mucosal injury.
- Ulcerative colitis is characterized by continuous rectal-based inflammation.
- Crohn disease requires assessment of distribution and other characteristic findings.
- Use the complete clinical, endoscopic and histologic pattern.

Separate two cancer-related questions



- Longstanding colitis can increase the risk of epithelial dysplasia and adenocarcinoma.
- An inflammatory pseudopolyp itself should not be equated with dysplasia.
- A suspicious polypoid focus still needs assessment as a possible neoplastic lesion.

Cancer risk in IBD is a background-disease issue

- Increased epithelial dysplasia and carcinoma risk in longstanding colonic IBD.
- Disease duration and extent contribute to that risk.
- Dysplasia may be present even in flat mucosa that appears inconspicuous grossly.
- Classifying a particular inflammatory polyp does not eliminate broader colitis assessment.

Inflammation versus dysplasia in biopsy interpretation

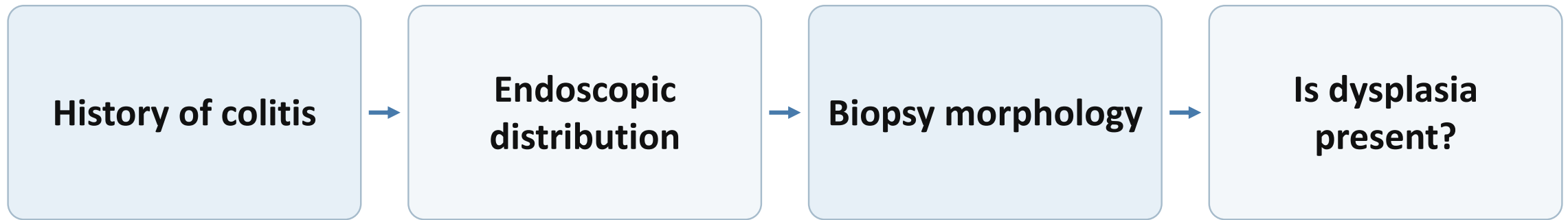
Reactive polypoid epithelium

- Erosion and repair provide a stimulus
- Epithelium may be hyperplastic
- Look for concordant inflammatory stroma

Dysplastic epithelium

- Neoplastic epithelial atypia is pivotal
- May be polypoid or flat in chronic IBD
- Requires diagnostic histologic scrutiny

Contextual workup of a colitic polyp



- Evidence of mucosal injury and repair favors an inflammatory process.
- When atypia or a focal mass is concerning, evaluate that lesion independently.

Solitary rectal ulcer syndrome: Textbook example

- This syndrome as an example of a purely inflammatory lesion.
- A rectal mucosal injury evolves in association with disordered anorectal mechanics.
- The syndrome may generate a polypoid lesion with mucosal prolapse.
- The diagnosis rests on concordance of symptoms, anatomic location and tissue changes.

The classic presenting triad

Rectal bleeding

Mucus discharge

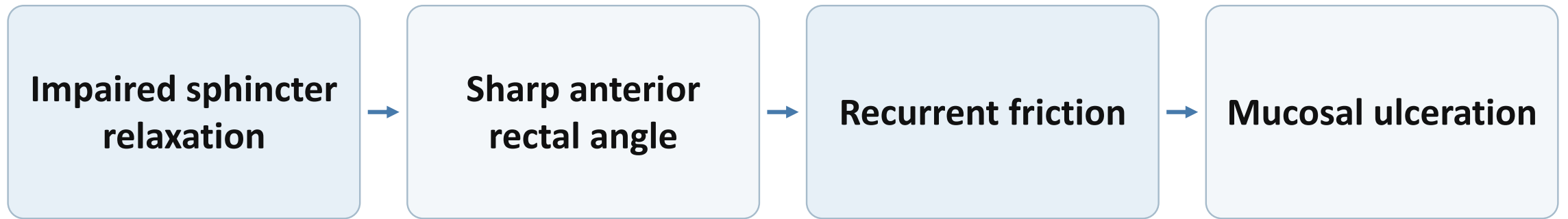
**Anterior rectal wall
lesion**

Think of localized prolapse-related injury

Location is part of the diagnostic pattern

- The source describes an inflammatory lesion on the anterior rectal wall.
- Anorectal geometry can focus recurrent friction onto this site.
- This distribution is different from continuous colonic injury in ulcerative colitis.
- A localized rectal lesion must still undergo morphologic assessment.

The initiating anorectal mechanical problem

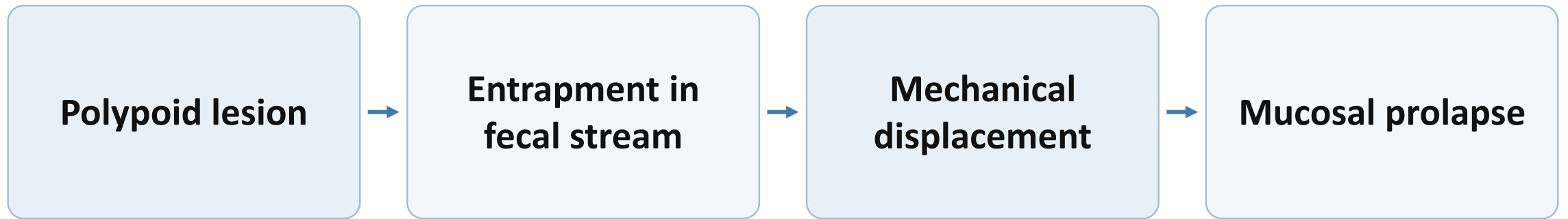


- Abnormal defecatory mechanics expose the rectal mucosa to repeated abrasion.
- This mechanism links a functional disturbance to a structural lesion.

Why a sharp rectal angle matters

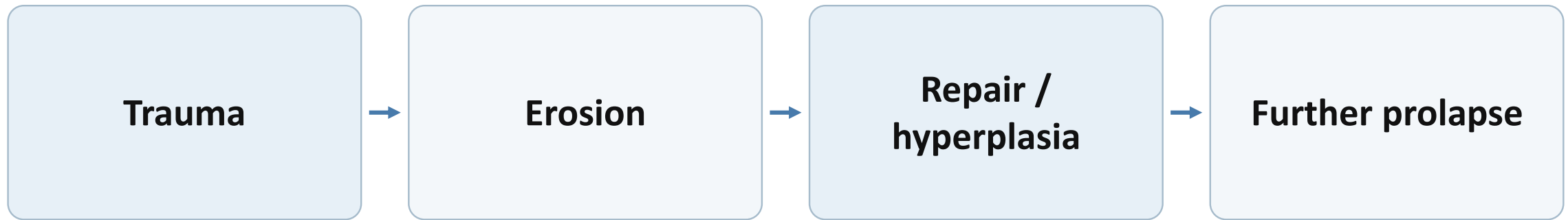
- Impaired anorectal relaxation increases abnormal mechanical stress.
- The anterior rectal shelf develops a sharp angulation according to the textbook.
- Repeated abrasion damages the overlying mucosal surface.
- Ulceration becomes a recurring stimulus for epithelial and stromal repair.

Polyp entrapment creates mucosal prolapse



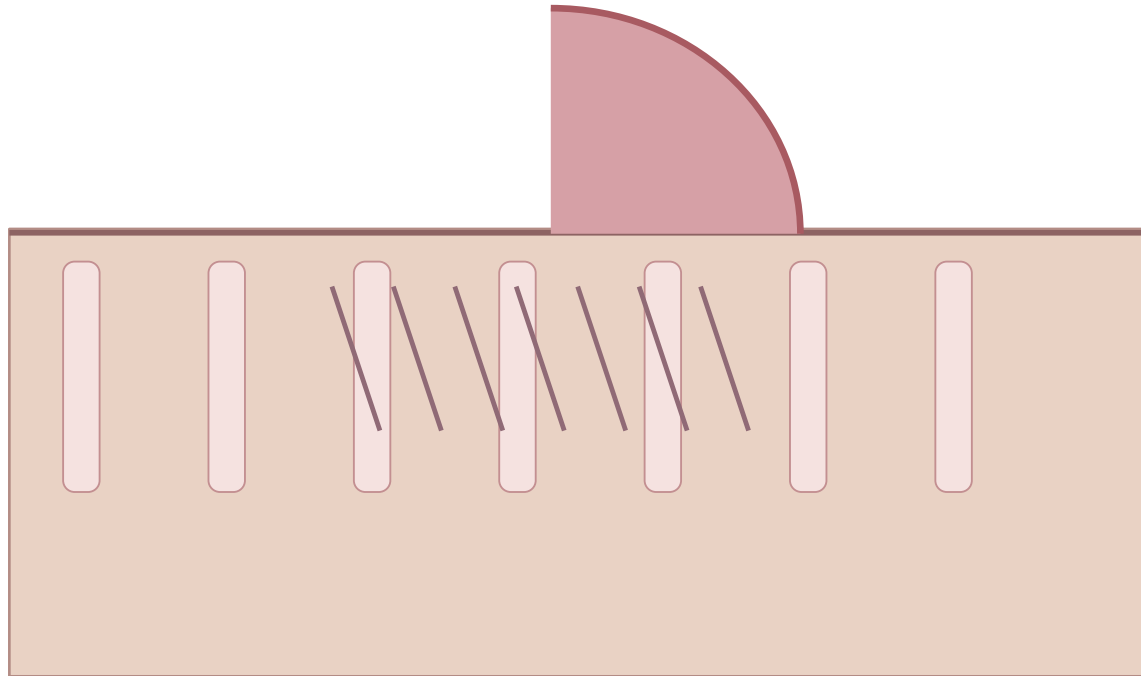
- The source links entrapment of the rectal polyp to prolapse.
- Prolapse provides a second mechanical pathway for continued tissue distortion.

Prolapse establishes a reinforcing cycle



- The same lesion may display fresh injury and longstanding reactive remodeling.
- The tissue architecture preserves evidence of repeated mechanical injury.

Prolapse-associated lesion: Tissue layers



Conceptual tissue section — schematic

- Surface change: Erosion or hyperplasia.
- Stroma: Inflammatory cells and capillaries.
- Lamina propria: Smooth muscle expansion.
- Interpreted together, these support prolapse.

Microscopy I: Mixed inflammatory cells

- An inflammatory infiltrate occupies the lesion stroma.
- Mixed inflammation documents a tissue response rather than a specific infectious cause.
- The infiltrate is interpreted alongside erosion and reactive epithelial changes.
- The textbook specifically describes a mixed infiltrate in this setting.

Microscopy II: Superficial erosion

- Loss of the surface epithelium is evidence of direct mucosal injury.
- Recurrent abrasion explains why erosion and ulceration recur.
- Granulation-like capillary proliferation can form beneath repeatedly eroded mucosa.
- Erosion alone is nonspecific; The full architectural context is essential.

Microscopy III: Reactive epithelial hyperplasia

- Proliferative epithelium is a typical component of an inflammatory polyp.
- The epithelial response reflects repeated repair of damaged mucosa.
- On its own, epithelial hyperplasia is not equivalent to an adenoma.
- Correlate reactive epithelium with surface erosion and underlying stromal change.

Microscopy IV: Fibromuscular hyperplasia

- Prolapse produces fibromuscular hyperplasia within the lamina propria.
- Smooth muscle expansion is a distinctive component of the described lesion.
- Its coexistence with inflammation and erosion favors mechanically induced injury.
- This finding links histologic morphology to the causal mechanism.

How muscle appears within lamina propria



- This fibromuscular response as prolapse induced.
- Do not misinterpret a muscular stromal reaction as proof of invasion.

Microscopy V: Dilated glands

- Figure 17.45 describes dilated glands in the inflammatory rectal lesion.
- Gland dilation occurs alongside epithelial proliferation and erosion.
- Architecture should be reviewed with the lamina propria rather than in isolation.
- The combination is more characteristic than any single gland profile.

Microscopy VI: Capillary-rich repair

- Repeated erosion is associated with granulation tissue–like capillary proliferation.
- Increased capillaries represent a repair response in the lamina propria.
- This vascular component helps explain a fragile, repeatedly injured surface.
- Assess it with accompanying epithelial and fibromuscular changes.

Integrated pathology of the prolapse lesion

Component	What is observed	Pathogenic interpretation
Surface	Erosion / ulceration	Repetitive abrasion
Epithelium	Hyperplasia; Dilated glands	Regenerative repair
Lamina propria	Mixed inflammatory cells	Response to injury
Stroma	Fibromuscular hyperplasia	Mucosal prolapse

Inflammatory versus hyperplastic polyps

Inflammatory

- Injury-and-healing mechanism
- Erosion, inflammation or prolapse-associated stroma
- May accompany IBD or rectal prolapse

Hyperplastic

- Benign epithelial proliferation
- Surface serration from cell crowding
- Common in left colon / rectum

Inflammatory versus juvenile polyps

Inflammatory

- Reactive injury with epithelial / stromal repair
- Rectal prolapse subtype shows fibromuscular hyperplasia
- Consider local mechanical or colitic history

Juvenile / hamartomatous

- Focal epithelial and stromal malformation
- Cystically dilated mucin/debris-filled glands
- Can be sporadic or associated with a syndrome

Inflammatory versus adenomatous polyps

Inflammatory

- Nonneoplastic injury-and-repair phenomenon
- Reactive epithelial hyperplasia may be present
- Inflammatory stroma is a key clue

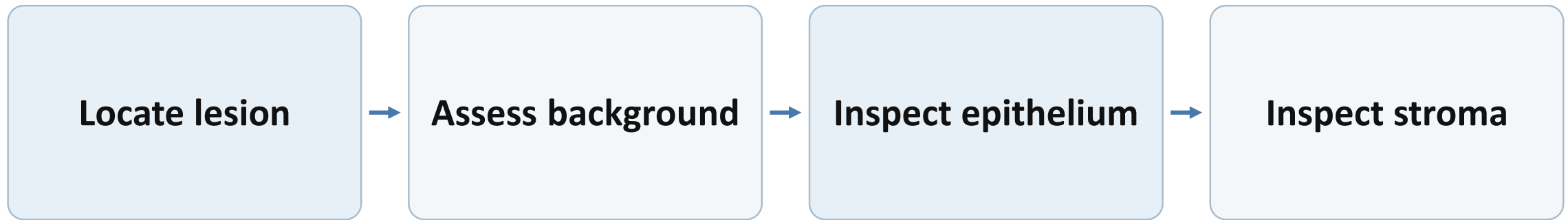
Adenoma

- Neoplastic epithelial lesion
- Cytologic dysplasia is the defining hallmark
- A precursor lesion for colorectal carcinoma

Three settings that may produce a raised lesion

Setting	Driving process	Major interpretation clue
Ulcerative colitis	Patchy mucosal survival in ulcers	Pseudopolyps, mucosal bridges
Rectal prolapse	Repetitive local mechanical injury	Lamina propria fibromuscular hyperplasia
Adenoma	Epithelial neoplastic proliferation	Cytologic dysplasia

A practical diagnostic reasoning sequence



- Differentiate diffuse colitic damage from localized prolapse-associated injury.
- Seek dysplasia separately from purely reactive epithelial change.

Case study: Multiple raised lesions in colitis

A patient with established ulcerative colitis has broad areas of colonic mucosal ulceration. Multiple islands of intact mucosa rise between ulcers; Several tips appear joined.

Interpretation task

- Explain the polypoid elevations and connecting bridges.
- State which background features should still be reviewed histologically.

Case study: Rectal bleeding and mucus

A patient reports rectal bleeding and mucus discharge. Examination reveals a localized inflammatory lesion along the anterior rectal wall; Biopsy shows superficial erosion and smooth muscle expansion in the lamina propria.

Interpretation task

- Identify the syndrome suggested by this combination.
- Explain how disordered defecatory mechanics produce the lesion.

Case study: Epithelial crowding in a rectal polyp

A polypoid rectal biopsy shows hyperplastic glands, superficial erosion, mixed inflammation and fibromuscular lamina propria. The patient also reports mucus discharge.

Interpretation task

- Which findings favor an injury-and-prolapse process?
- Why should epithelial proliferation not immediately be labeled adenomatous?

Core diagnostic synthesis

- Inflammatory polyps express mucosal injury followed by regeneration.
- Colitic pseudopolyps are islands of regenerating mucosa among ulcers.
- Mucosal bridges may form when adjacent inflammatory polyp tips fuse.
- Rectal prolapse lesions combine erosion, inflammation, epithelial hyperplasia and fibromuscular stroma.
- Dysplasia must be considered separately from reactive polyp formation.

Selected source readings

Robbins, Cotran & Kumar: Pathologic Basis of Disease

11th edition (2026) — Chapter 17, The Gastrointestinal Tract

- Intestinal polyps and inflammatory polyps: pages 735–736.
- Ulcerative colitis morphology: pages 730–732.
- Colitis-associated neoplasia: page 733.
- Comparative hyperplastic and juvenile polyp sections: pages 735–737.