

Intestinal Polyps

Morphology • classification • clinicopathologic interpretation

A pathology teaching lecture

Hyperplastic, inflammatory, hamartomatous and neoplastic categories

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Learning objectives

- Describe sessile and pedunculated morphology without confusing appearance with diagnosis.
- Classify intestinal polyps by nonneoplastic and neoplastic pathology.
- Recognize hyperplastic, inflammatory and hamartomatous morphologic signatures.
- Explain why some polyp categories require evaluation for malignant potential.

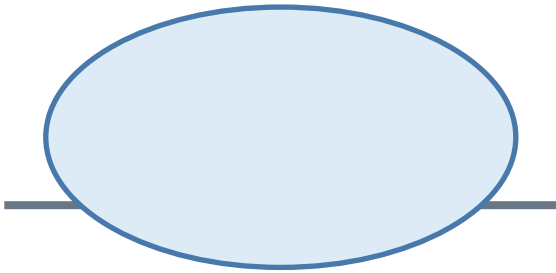
Interpretation: Emphasis: Clinical-pathological reasoning rather than a screening guideline update.

Where intestinal polyps arise

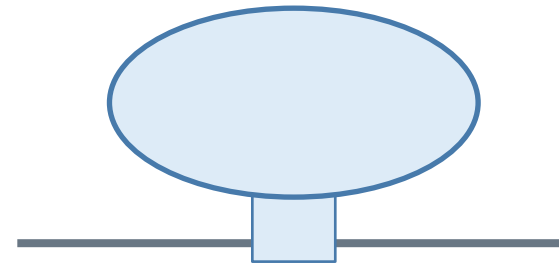
- The colon and rectum are the most common sites of intestinal polyps.
- Polypoid lesions may also occur elsewhere along the gastrointestinal tract.
- A polyp is a mucosal protrusion, not a specific microscopic diagnosis.
- An identical gross contour can be produced by very different lesions.

Interpretation: Location and appearance start the differential; Microscopic architecture establishes type.

A polyp is a gross configuration



Sessile: Broad attachment



Pedunculated: Distinct stalk

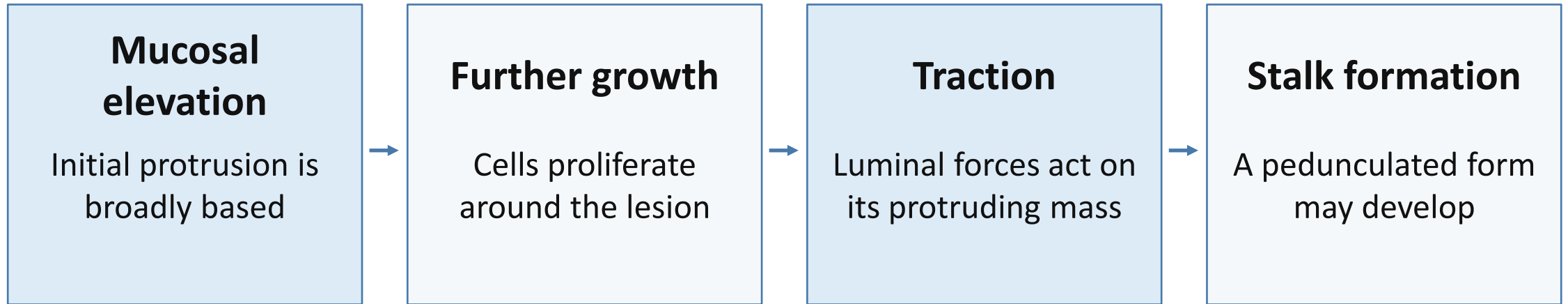
Broad attachment versus a stalk is descriptive—not a malignancy test.

Sessile polyps: Attachment and evolution

- Most polyps begin as small elevated, broad-based mucosal lesions.
- “Sessile” denotes a protrusion without a distinct stalk.
- An enlarging sessile lesion may remain broadly attached.
- Histologic type cannot be inferred from sessile shape alone.

Interpretation: A sessile adenoma and a sessile hyperplastic polyp may have very different biology.

How a stalk can develop



Model: Causal schematic summarizing the source; Not an observed sequential specimen.

Pedunculated polyps: Anatomic concept

- A pedunculated polyp has a distinct stalk joining the head to the mucosa.
- The stalk may contain submucosa-derived vessels and fibromuscular tissue.
- Pedunculation can result from growth and traction over time.
- A stalk is not synonymous with benignity.

Interpretation: Polyp head, stalk and attachment may each matter in specimen interpretation.

Macroscopic versus microscopic diagnosis

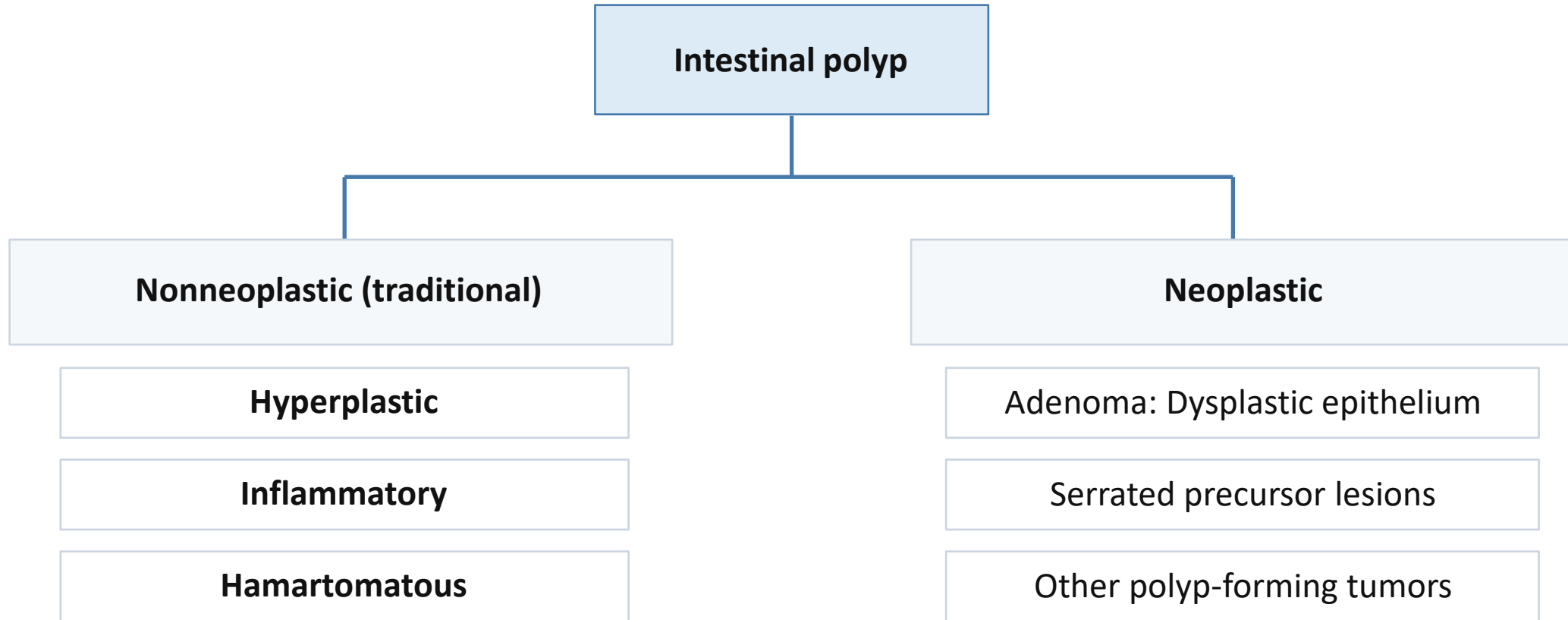
Gross descriptors

Sessile or pedunculated; Smooth, lobulated or velvety; Size, distribution and location.

Pathologic categories

Hyperplastic, inflammatory, hamartomatous or neoplastic; Determined by tissue composition and architecture.

A practical classification of intestinal polyps



Some hamartomatous syndromes confer cancer risk despite this traditional classification.

Nonneoplastic versus neoplastic: Key logic

Nonneoplastic

Growth may reflect delayed shedding, inflammation or disordered tissue organization. The category alone does not specify a single outcome.

Neoplastic

Clonal epithelial lesions may show dysplasia and can be precursors to invasive carcinoma. Some serrated precursor lesions lack conventional dysplasia.

Polyp classification has important exceptions

- Traditional “nonneoplastic” subtypes include hyperplastic, inflammatory and hamartomatous polyps.
- Syndromic hamartomatous polyps can signal inherited cancer susceptibility.
- Some serrated precursors resemble benign hyperplastic polyps.
- Clinical distribution and family history may change interpretation.

Interpretation: The name “polyp” must never replace an assessment of cancer risk.

Hyperplastic polyps: Core definition

- Benign epithelial proliferations of colonic mucosa.
- Thought to reflect delayed surface-cell loss and epithelial accumulation.
- Typically discovered during the sixth or seventh decade.
- Classically distinguished from premalignant sessile serrated polyps.

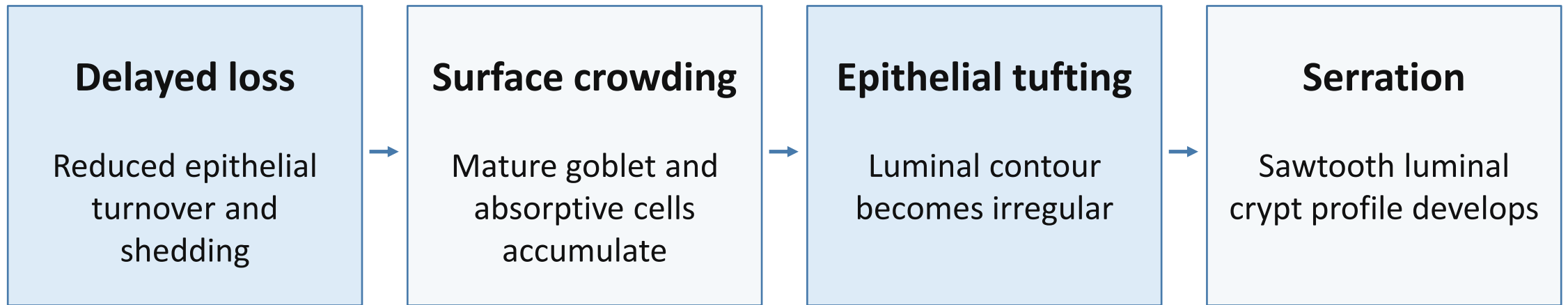
Interpretation: Typical hyperplastic polyps as lacking malignant potential.

Typical location and gross profile

- Usually arise in the left colon, especially sigmoid colon and rectum.
- Frequently measure less than 5 mm in diameter.
- Appear as smooth, nodular mucosal protrusions.
- May be single, but multiple small lesions are common.

Interpretation: These are typical patterns, not absolute diagnostic criteria.

Proposed pathogenesis of a hyperplastic polyp



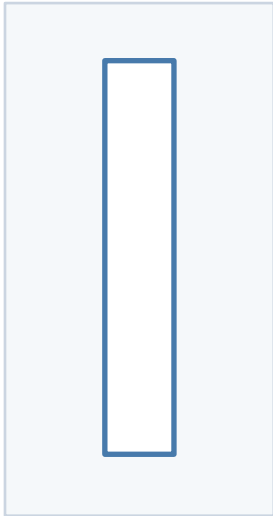
Model: Causal schematic summarizing the source; Not an observed sequential specimen.

Delayed cell shedding is a proposed mechanism

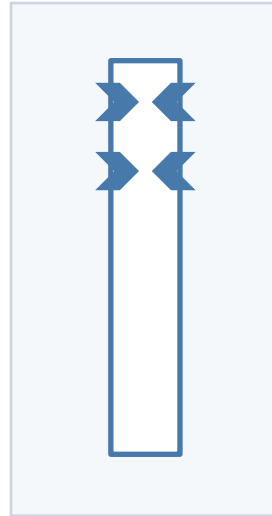
- The full pathogenesis of hyperplastic polyps remains incompletely defined.
- Reduced turnover allows mature surface cells to accumulate.
- Overcrowding creates tufting and epithelial infolding.
- Serration is an architectural result of crowded mature cells.

Interpretation: A suggested biologic mechanism should not be presented as proven in every lesion.

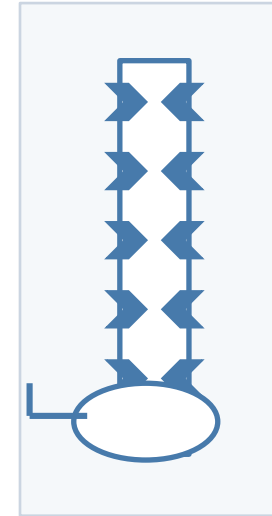
The serrated crypt: A visual concept



Unremarkable crypt



Surface-limited serration



Basal distortion

Schematic crypt architecture: Conceptual only

Hyperplastic polyps contain mature epithelium

- Goblet cells remain part of the crypt epithelium.
- Absorptive epithelial cells are also prominent.
- Irregular surface tufting reflects cell crowding.
- The key abnormality is serrated architecture, not conventional adenomatous dysplasia.

Interpretation: Microscopic architecture is more useful than the smooth gross appearance.

Where the serration occurs matters

- Typical hyperplastic polyps show serration in the upper third of the crypt.
- The lower crypt may retain a regenerative appearance.
- Assessment requires intact, well-oriented crypts when possible.
- Basal crypt changes prompt evaluation for a sessile serrated lesion.

Interpretation: Upper-crypt versus full-length serration is a crucial discrimination.

Two causes of a serrated-looking epithelium

Hyperplastic-type pattern

Surface crowding with serration limited mainly to upper crypts; Basal crypt architecture relatively preserved.

Sessile serrated pattern

Serration extends to the base; Dilated and laterally extending crypts may be present. Malignant precursor potential.

Sessile serrated polyps: The important mimic

- May overlap microscopically with hyperplastic polyps.
- More often occur in the right colon, per Robbins.
- Can lack conventional cytologic dysplasia despite malignant potential.
- Basal dilation and lateral crypt growth help resolve the differential.

Interpretation: Conventional dysplasia is not required for a sessile serrated lesion to be clinically significant.

Reactive epithelial hyperplasia is not always a polyp type

- Epithelial hyperplasia may occur overlying other mucosal lesions.
- It also may appear adjacent to an inflammatory lesion.
- A superficially hyperplastic mucosa can mask important underlying pathology.
- Assess the full lesion and relevant adjacent tissue, not isolated surface changes.

Interpretation: Reactive hyperplasia can be a signpost to another lesion.

A lesion may be multiple without being neoplastic

- Hyperplastic polyps frequently occur as multiple left-sided lesions.
- Multiplicity alone does not establish hereditary adenomatous polyposis.
- Distribution, microscopic type and clinical context must be integrated.
- Distinct lesions within one colon may have different histologies.

Interpretation: Polyp counts are interpreted only after classifying the lesions.

Hyperplastic versus adenomatous epithelium

Hyperplastic polyp

Mature goblet and absorptive cells; Prominent upper crypt serration; No conventional epithelial dysplasia.

Conventional adenoma

Elongated hyperchromatic, stratified nuclei; Dysplasia at the mucosal surface; Variable tubular or villous architecture.

Hyperplastic polyp: Diagnostic summary

- Small, often distal colonic, smooth mucosal protrusion.
- Serrated contours most evident in the upper crypt.
- Mature epithelial cell populations predominate.
- Exclude a sessile serrated lesion if the basal crypts are distorted.

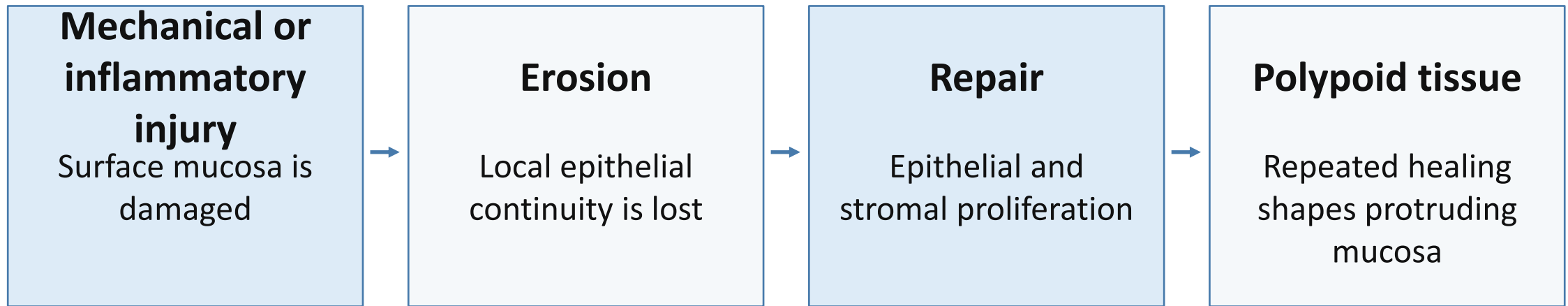
Interpretation: The key significance is correct classification—not the label “serrated” alone.

Inflammatory polyps: A different mechanism

- Can form through repeated episodes of mucosal injury and repair.
- Inflammation, erosion and epithelial regeneration may coexist.
- An inflammatory polyp does not imply a primary epithelial neoplasm.
- Some are manifestations of mucosal prolapse or other injury syndromes.

Interpretation: Tissue injury, not delayed surface shedding, organizes this category.

Repeated injury can create a polyp



Model: Causal schematic summarizing the source; Not an observed sequential specimen.

Why an inflamed mucosa becomes polypoid

- Erosion stimulates regenerative epithelial growth.
- Stromal and vascular responses expand the lamina propria.
- Recurrent injury prevents full restoration of normal architecture.
- Repair tissue can protrude into the intestinal lumen.

Interpretation: The gross protrusion is a consequence of repair and architectural remodeling.

Solitary rectal ulcer syndrome: A model lesion

- Illustrates the formation of an inflammatory polyp.
- The characteristic lesion involves the anterior rectal wall.
- Abnormal anorectal mechanics produce repetitive mucosal injury.
- The term describes a syndrome, not simply an ulcer seen at endoscopy.

Interpretation: Prolapse-related inflammatory change is the unifying pathologic idea.

Recognize the clinical triad

- Rectal bleeding.
- Mucus discharge.
- An inflammatory lesion of the anterior rectal wall.
- These findings should prompt correlation with mucosal prolapse and defecatory mechanics.

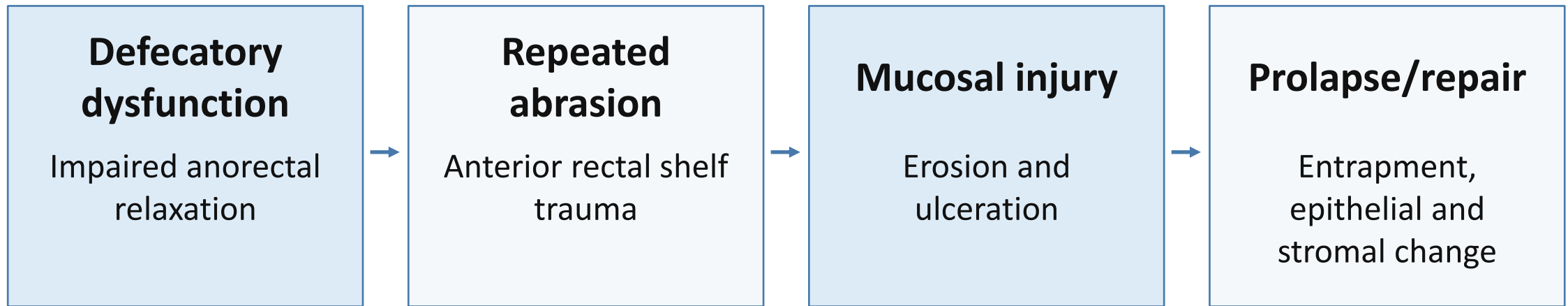
Interpretation: The described triad is from the source, not a universal required diagnostic checklist.

Impaired sphincter relaxation initiates mechanical injury

- Impaired relaxation of the anorectal sphincter changes local anatomy.
- A sharp angle can develop at the anterior rectal shelf.
- Repeated passage of stool abrades the overlying mucosa.
- Continued abrasion leads to erosion and repeated repair.

Interpretation: A local mechanical process explains the anterior-wall distribution.

Solitary rectal ulcer: Injury–prolapse cycle



Model: Causal schematic summarizing the source; Not an observed sequential specimen.

Microscopy of inflammatory polyps

- Mixed inflammatory infiltrates are present.
- Surface erosion may be conspicuous.
- Regenerative epithelial hyperplasia accompanies repair.
- Lamina propria stromal remodeling contributes to the polyp.

Interpretation: Inflammation and epithelial hyperplasia are expected together.

Prolapse produces fibromuscular change

- Smooth-muscle fibers extend into and expand the lamina propria.
- This fibromuscular hyperplasia is characteristic of prolapse injury.
- It can coexist with dilated glands and erosions.
- Recognizing the stromal component helps distinguish reactive prolapse from neoplasia.

Interpretation: The diagnostic clue is in the lamina propria, not only in surface epithelium.

Hyperplastic and inflammatory polyps

Hyperplastic

Mature epithelial cell crowding; Surface serration; Typically distal colon; No recurrent-injury mechanism required.

Inflammatory/prolapse-related

Erosion, inflammation, regeneration and stromal fibromuscular hyperplasia; Associated with repeated mucosal injury.

Reactive atypia is a potential interpretive pitfall

- Active erosion can produce conspicuous epithelial regeneration.
- Prolapse and surface repair can distort crypts and mucosal contours.
- A diagnostic assessment must incorporate the stromal and surface changes together.
- The distinction from neoplasia rests on a coherent morphologic pattern.

Interpretation: Do not diagnose an adenoma from surface irregularity alone.

Inflammatory polyps: Clinicopathologic reading

- History of rectal bleeding or mucus discharge may provide context.
- An anterior rectal wall lesion can fit mucosal prolapse.
- Erosion and mixed inflammation support repeated injury.
- Fibromuscular lamina propria change strengthens the prolapse interpretation.

Interpretation: Correlation is essential when surface appearances are nonspecific.

Hamartomatous polyps: Disordered tissue organization

- May occur as isolated lesions or within a polyposis syndrome.
- Comprise mature tissue components arranged in an abnormal architecture.
- Many associated syndromes involve inherited signaling-gene alterations.
- The clinical significance extends beyond the individual polyp.

Interpretation: Hamartomatous morphology does not guarantee a benign patient-level risk.

Sporadic versus syndromic hamartomatous polyps

Sporadic setting

An isolated lesion may be limited in clinical significance; Evaluation emphasizes its morphology and symptoms.

Syndromic setting

Numerous polyps, inherited predisposition, extraintestinal findings and risks at other organs may coexist.

Why “hamartomatous” needs clinical context

- Some germline syndromes alter tumor suppressor or growth-control pathways.
- Cancer can arise in associated GI or extra-GI sites.
- Family members may share susceptibility when inheritance is present.
- Clinical distribution and associated manifestations determine syndromic suspicion.

Interpretation: The broader phenotype matters more than isolated appearance.

Juvenile polyps: Terminology

- “Juvenile” names a morphologic polyp type, not a strict patient-age limit.
- Most are detected in young children, but older patients can present.
- Juvenile polyps are epithelial–lamina propria malformations.
- Both sporadic and syndromic settings occur.

Interpretation: Do not use age alone to establish the diagnosis.

Juvenile polyps: Typical presentation

- Rectal bleeding is a prominent presenting complaint.
- Sporadic juvenile polyps are often solitary and rectal.
- Some cause polyp prolapse, intussusception or obstruction.
- Syndromic juvenile polyposis may involve numerous lesions.

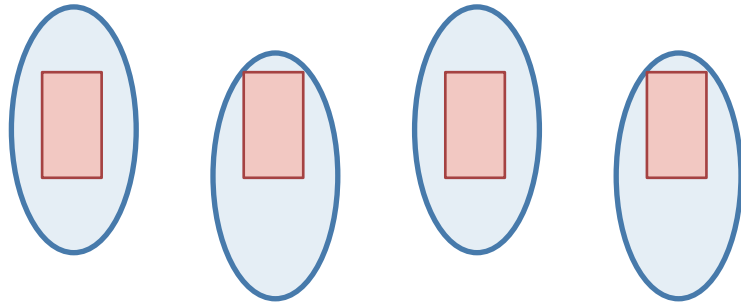
Interpretation: Bleeding reflects mucosal vulnerability and possible surface ulceration.

Juvenile polyp: Gross phenotype

- Typically smooth-surfaced and reddish.
- Most are smaller than 3 cm, as.
- Pedunculated architecture is common.
- Cystic spaces can contribute to the gross cut-surface appearance.

Interpretation: The surface appearance should be reconciled with cystic glands on microscopy.

Juvenile polyp: Cystically dilated glands



Cystically dilated mucin-containing glands

Expanded inflammatory lamina propria surrounds glands

Juvenile polyp: Microscopic signature

- Dilated glands form conspicuous cyst-like spaces.
- Gland lumina contain mucin and inflammatory debris.
- The lamina propria is expanded by mixed inflammatory cells.
- The muscularis mucosae may be normal or attenuated.

Interpretation: Cystically dilated glands in inflamed stroma are the central pattern.

Sporadic and syndromic juvenile polyps differ in risk

- Dysplasia is uncommon in isolated sporadic juvenile polyps.
- Juvenile polyposis syndrome may include dysplasia within or away from polyps.
- Syndromic patients carry increased GI cancer risk.
- A pathology report should not equate one sporadic lesion with a hereditary syndrome.

Interpretation: Polyp histology and the clinical syndrome are distinct levels of diagnosis.

Juvenile polyposis: Growth-signaling pathways

- Autosomal dominant juvenile polyposis is associated with SMAD4 or BMPR1A mutations.
- Both participate in TGF- β -family growth regulation.
- Some patients with SMAD4 variants have hereditary hemorrhagic telangiectasia.
- Known variants do not explain all affected individuals.

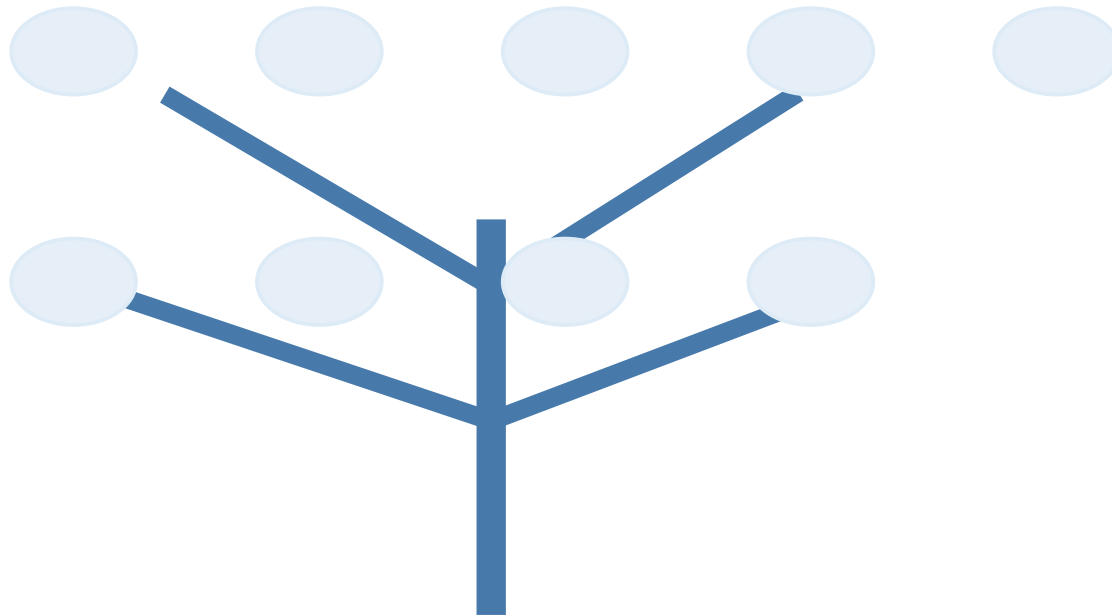
Interpretation: Gene examples are Robbins source-derived; Molecular testing indications are not specified here.

Peutz–Jeghers syndrome: Recognize the phenotype

- Autosomal dominant syndrome with multiple hamartomatous polyps.
- Polyps are particularly prominent in the small intestine.
- Mucocutaneous dark macules include the lips and buccal mucosa.
- Intussusception and increased cancer susceptibility are important consequences.

Interpretation: Pigmentation plus small-intestinal polyposis is a diagnostic combination.

Peutz–Jeghers polyp: Arborizing stroma



Arborizing smooth-muscle core with mucosal glands

Original schematic — not histology

Peutz–Jeghers: Molecular and clinical integration

- The STK11 tumor suppressor gene is implicated in a subset of cases.
- STK11 influences AMPK-related polarity and growth pathways.
- An undetected STK11 variant does not exclude the clinical syndrome.
- Family history and extraintestinal features remain diagnostically important.

Interpretation: Do not equate absence of a detected variant with absence of disease.

Juvenile versus Peutz–Jeghers histology

Juvenile

Cystically dilated, mucus-filled glands in edematous or inflamed lamina propria; Surface erosion may occur.

Peutz–Jeghers

Arborizing smooth-muscle bundles with branching mucosal glands; Often lobulated and pedunculated.

Adenomas: The neoplastic comparison

- Conventional adenomas are epithelial neoplasms.
- They range from pedunculated to large sessile lesions.
- The defining microscopic feature is dysplasia.
- Although they are precursors, most individual adenomas do not progress to carcinoma.

Interpretation: The presence of dysplasia, not stalk status, distinguishes conventional adenoma.

Epithelial dysplasia in an adenoma

- Nuclear hyperchromasia accompanies abnormal epithelial proliferation.
- Nuclei become elongated and stratified.
- Goblet cells may be reduced relative to normal epithelium.
- The surface can be a particularly useful site for appreciating dysplasia.

Interpretation: These are conventional adenomatous findings, not required in every serrated precursor.

Architecture of conventional adenomas

Tubular

Predominantly rounded glandular profiles; Commonly smaller and pedunculated.

Tubulovillous

Combination of tubular glands and finger-like villous projections.

Villous

Slender mucosal villi; Can occur in larger lesions and correlate with higher cancer prevalence.

Conventional adenoma versus sessile serrated polyp

Conventional adenoma

Cytologic dysplasia with elongated, hyperchromatic stratified nuclei; Variable tubular/villous growth.

Sessile serrated polyp

Serration extends through crypts with basal dilation or lateral growth; Conventional dysplasia may be absent.

Adenoma size and dysplasia affect risk

- Polyp size as a strong correlate of invasive cancer.
- Higher-grade dysplasia is linked to cancer within the involved polyp.
- Larger lesions warrant careful pathologic evaluation.
- Adenomatous architecture alone is not a substitute for size and dysplasia assessment.

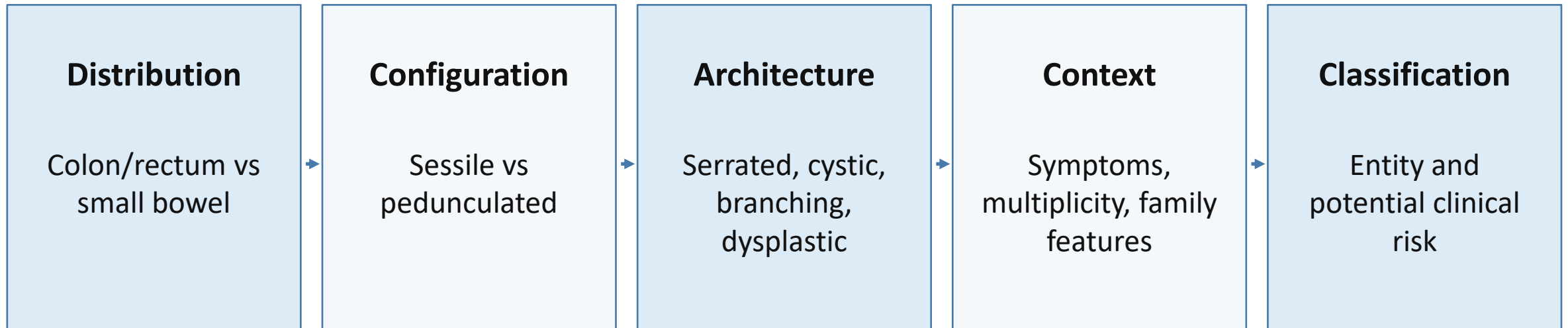
Interpretation: Risk statements apply to adenomas and should not be transferred to hyperplastic polyps.

Multiple adenomas raise the possibility of inherited disease

- Familial adenomatous polyposis (FAP) is associated with APC pathway alterations.
- FAP produces numerous colorectal adenomas.
- FAP polyps resemble sporadic adenomas microscopically.
- The number, age of onset and family history help identify syndromic disease.

Interpretation: Adenomatous polyposis is a different category from juvenile polyposis.

A structured pathology approach to a polyp



Interpret the histologic pattern together with clinical distribution and patient-level risk.

Case 1: A diminutive distal colonic polyp

Clinical vignette

A small, smooth polyp is removed from the sigmoid colon. The crypts show serration mainly in their upper portions, with preserved lower-crypt architecture.

Diagnostic question

Which polyp category best fits this pattern?

Case 2: Anterior rectal polypoid lesion

Clinical vignette

A patient has rectal bleeding and mucus discharge. The lesion shows erosion, epithelial hyperplasia, mixed inflammatory cells and fibromuscular expansion of the lamina propria.

Diagnostic question

What pathogenic mechanism unifies these findings?

Case 3: Cystic glands in a child

Clinical vignette

A child with intermittent rectal bleeding has a solitary pedunculated rectal lesion. Microscopy shows dilated mucus-filled glands and inflamed lamina propria.

Diagnostic question

What is the most likely morphologic diagnosis?

Case 4: Polyps with mucocutaneous pigmentation

Clinical vignette

An adolescent has multiple small-bowel polyps and dark macules involving the lips and buccal mucosa. A polyp contains arborizing smooth-muscle bundles.

Diagnostic question

What syndrome should be considered?

Case 5: A serrated polyp with basal crypt distortion

Clinical vignette

A right-sided colonic lesion shows serration to the base of the crypts, crypt dilation and lateral growth, but little conventional cytologic dysplasia.

Diagnostic question

Why is the distinction from a hyperplastic polyp important?

Morphology-based comparison: Five major patterns

Pattern	Microscopic cue	Clinical implication
Hyperplastic	Upper-crypt serration	Typically no malignant potential
Inflammatory	Erosion + repair + inflammation	Reflects repeated injury
Juvenile	Cystic dilated glands	Syndromic status changes risk
Peutz–Jeghers	Arborizing smooth muscle	Syndromic cancer predisposition
Adenoma / serrated precursor	Dysplasia or basal serrated distortion	Neoplastic precursor potential

Concluding interpretation

- Polyp describes luminal configuration; Histology supplies the diagnosis.
- Upper-crypt serration supports a hyperplastic pattern; Basal distortion suggests a serrated precursor.
- Inflammatory polyps can arise from repeated injury, repair and prolapse.
- Syndromic hamartomatous polyps require attention to family and extraintestinal features.

Interpretation: Integrated interpretation requires morphology plus clinical context.

Primary reference and source scope

Robbins, Cotran & Kumar: Pathologic Basis of Disease

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

Intestinal Polyps, pp. 735–739

The core topic begins at p. 735. Related hamartomatous and neoplastic comparison material is drawn from immediately following chapter sections; those entities also have separate topics in this lecture series.

All illustrative diagrams are newly constructed, editable schematics; no textbook images were reproduced.