

# Gastropathy and Acute Gastritis

Mechanisms of gastric mucosal injury and morphologic recognition

Pathology teaching lecture

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## Learning objectives

- Distinguish reactive gastropathy from neutrophil-defined gastritis.
- Relate gastric mucosal defenses to the mechanisms by which they fail.
- Recognize the continuum from mild injury to erosion and hemorrhage.
- Interpret characteristic histologic and clinical consequences.

# The stomach contains functionally distinct regions

|                        |                                                                |
|------------------------|----------------------------------------------------------------|
| <b>Cardia</b>          | Predominantly mucin-secreting foveolar cells and small glands. |
| <b>Antrum</b>          | Mucin-secreting glands with endocrine G cells.                 |
| <b>Body and fundus</b> | Well-developed glands with parietal and chief cells.           |

## Functional relevance

- Antral gastrin stimulates acid secretion.
- Body/fundus supply acid and digestive enzymes.
- Epithelial defenses are required in every region.

# Gastric epithelial populations are specialized

## Antrum / cardia

- Foveolar cells contribute mucin to the surface barrier.
- Antral G cells release gastrin.
- Protective secretion is central to mucosal integrity.

## Body / fundus

- Parietal cells secrete gastric acid.
- Chief cells produce digestive enzymes.
- Strong chemical digestion coexists with protective defenses.

## The gastric lumen is a potentially injurious environment

- Gastric luminal pH may approach 1.
- The lumen is more than one million times more acidic than blood.
- Acid and peptic enzymes support digestion but threaten surface cells.
- Normal tissue integrity depends on multiple coordinated defenses.

# Gastropathy and gastritis are not synonyms

## Gastropathy

- Reactive epithelial injury with rare or absent inflammatory cells.
- Typically associated with irritants and barrier dysfunction.
- Foveolar hyperplasia may be conspicuous.

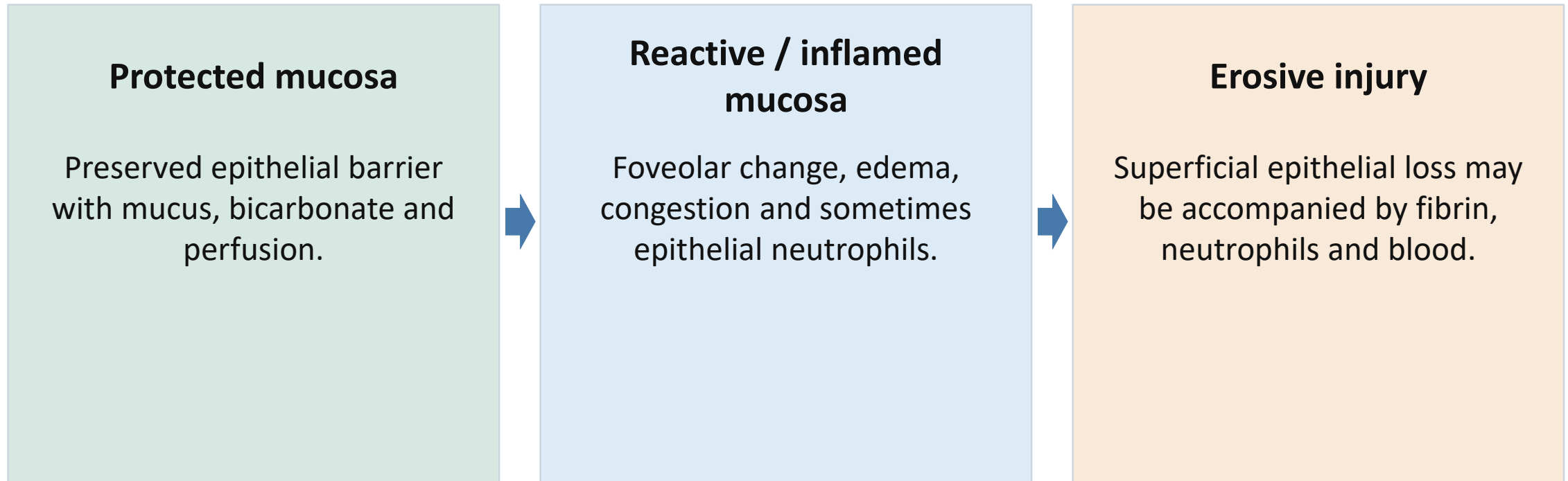
## Gastritis

- Gastric mucosal inflammation with neutrophils.
- Neutrophils in epithelium or gland lumina signal activity.
- May also show injury, erosions and hemorrhage.

## The term “acute” describes time, not exclusively cell type

- Neutrophils can occur in both acute and chronic gastric disease.
- “Active inflammation” describes a neutrophilic inflammatory pattern.
- “Acute gastritis” refers to an acute-duration process.
- Avoid inferring clinical duration solely from neutrophil presence.

# Gastric injury spans a morphologic continuum



- The extent of damage depends on the balance between injury and defense.

## Clinical expression is variable

- Both gastropathy and gastritis may be clinically silent.
- Symptoms can include epigastric discomfort, nausea and vomiting.
- Severe injury may produce erosions, ulceration or gastrointestinal bleeding.
- The histologic distinction cannot be made from symptoms alone.

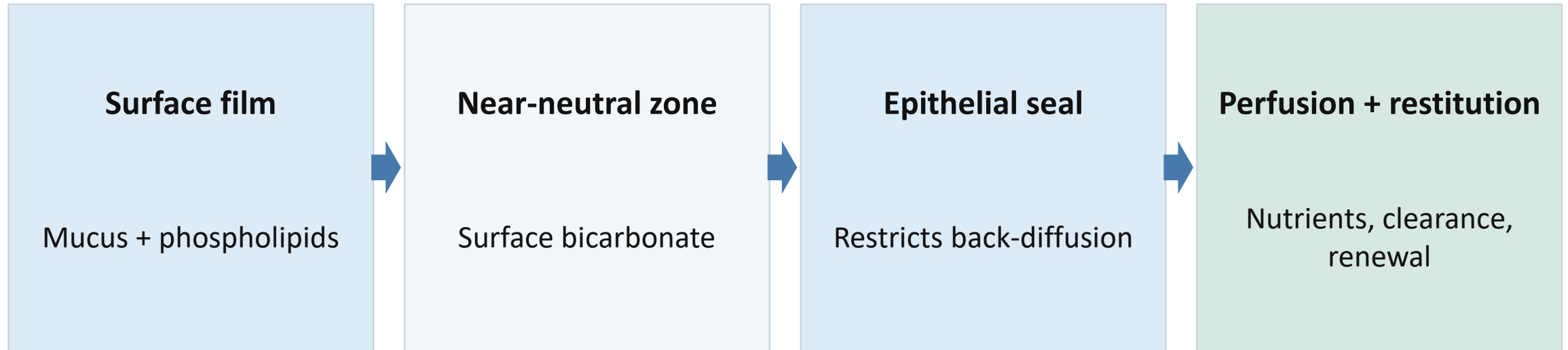
# Gastric defense is multilayered

|                           |                                                                    |
|---------------------------|--------------------------------------------------------------------|
| <b>Luminal interface</b>  | Mucus, phospholipids and bicarbonate maintain a protected surface. |
| <b>Epithelial barrier</b> | Intercellular integrity restricts acid and enzyme back-diffusion.  |
| <b>Lamina propria</b>     | Mucosal blood flow brings oxygen and clears back-diffused acid.    |
| <b>Renewal</b>            | Surface foveolar cells are replaced rapidly after injury.          |

## Protection is distributed

- Failure in one layer stresses the others.
- Prostaglandins support several layers.
- Luminal acid alone does not explain injury.

## Four linked defenses preserve the mucosa



- Protection fails when these functions are insufficient relative to insult.

## Mucus provides a physical separation layer

- Foveolar cells secrete a thin mucus layer into the lumen.
- Phospholipids contribute to the protective luminal surface film.
- The film limits direct contact of large food particles with epithelium.
- Mucus also supports a stable, bicarbonate-enriched fluid microenvironment.

# Luminal acidity and epithelial surface pH differ

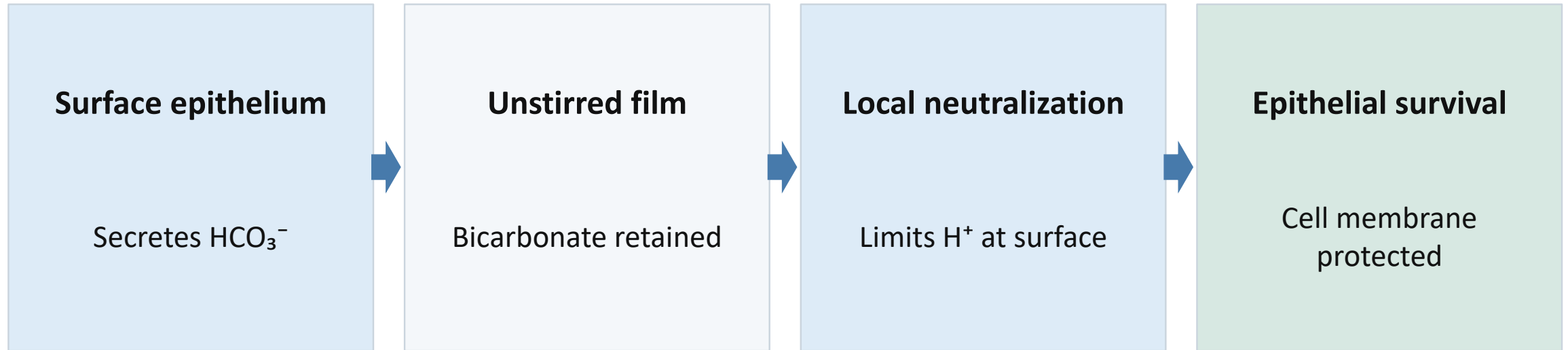
## Gastric lumen

- Highly acidic milieu supports digestion.
- Acid and peptic enzymes remain potentially damaging.
- The lumen is not a safe environment for unprotected cells.

## Epithelial surface

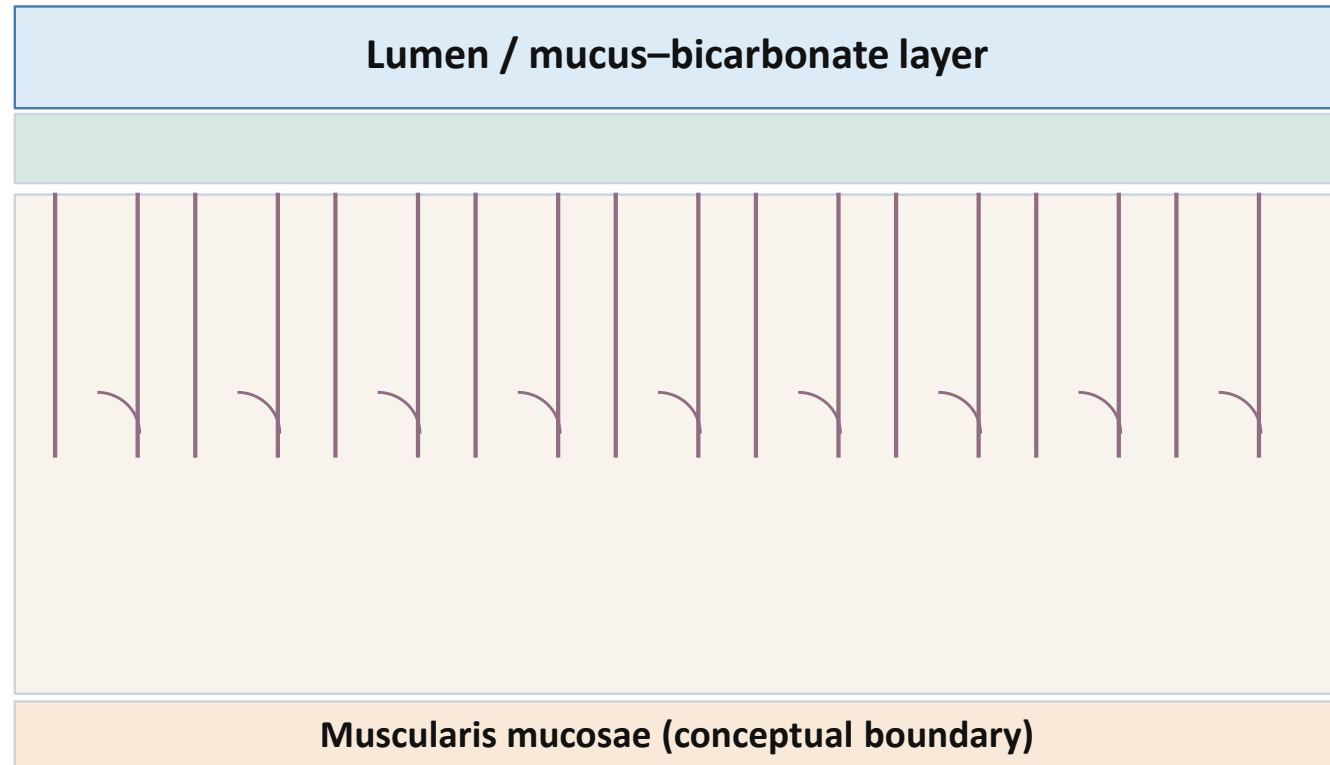
- An unstirred mucus-associated layer traps bicarbonate.
- Local pH can remain near neutral.
- A steep gradient reduces acid exposure to epithelial cells.

# Bicarbonate protects the epithelial interface



- Loss of bicarbonate secretion or transport reduces this protective gradient.

# Simplified surface defense architecture



## Interpretation

- Mucus lies above an intact epithelial surface.
- Gastric pits descend into mucosa.
- Lamina propria and muscularis mucosae lie beneath.

Original editable schematic — simplified architecture; Not a histologic specimen.

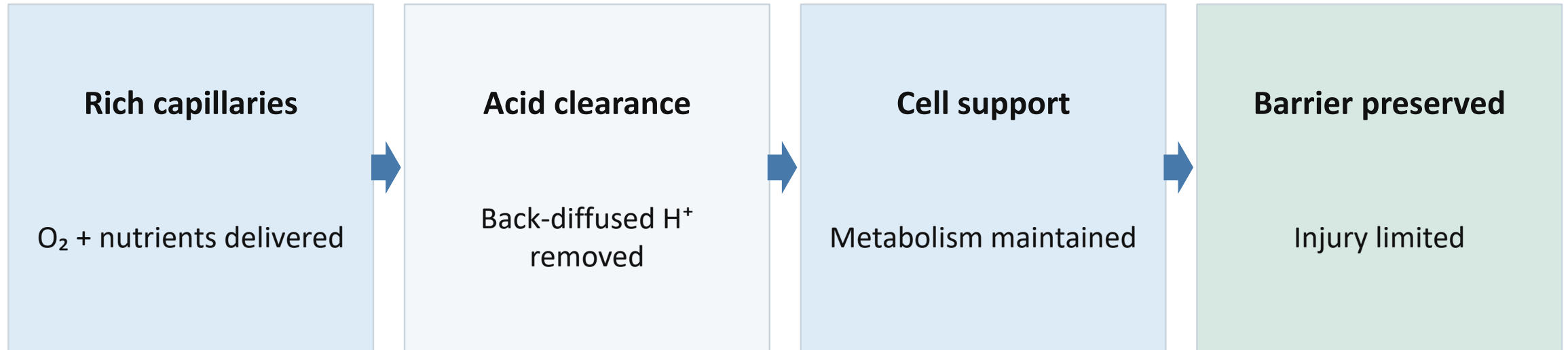
## Epithelial continuity limits acid back-diffusion

- Surface epithelial cells form a physical barrier to luminal contents.
- The barrier restricts acid diffusion into the lamina propria.
- It also limits leakage of digestive enzymes and other materials.
- Loss of epithelial integrity exposes deeper tissue to corrosive injury.

## Rapid replacement supports epithelial restitution

- Surface foveolar cells are renewed approximately every 3–7 days.
- Parietal and chief cells are comparatively long-lived.
- Continual renewal replaces damaged superficial epithelium.
- Agents that suppress cell division may impair barrier restoration.

# Mucosal blood flow is an active defense



- Ischemia weakens nutrient delivery and acid washout simultaneously.

# Two consequences of impaired perfusion

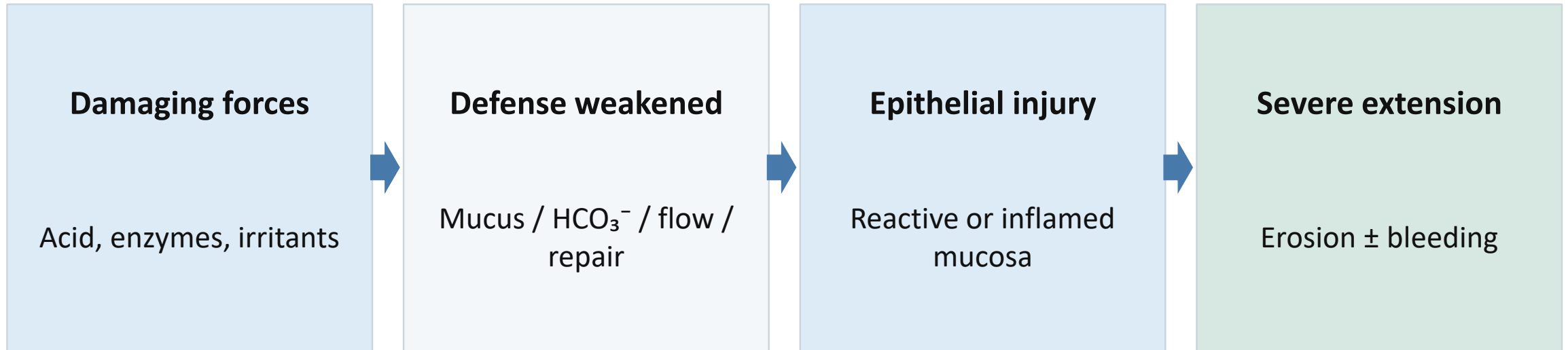
## Oxygen deficit

- Surface epithelium cannot maintain normal cell function.
- Repair becomes less effective.
- Barrier injury may become more likely.

## Reduced acid washout

- Back-diffused luminal acid persists in tissue.
- A protective pH gradient becomes harder to sustain.
- Damage may intensify despite unchanged luminal acid.

# Disease follows an imbalance, not a single insult



- Different exposures can converge on the same pattern of mucosal injury.

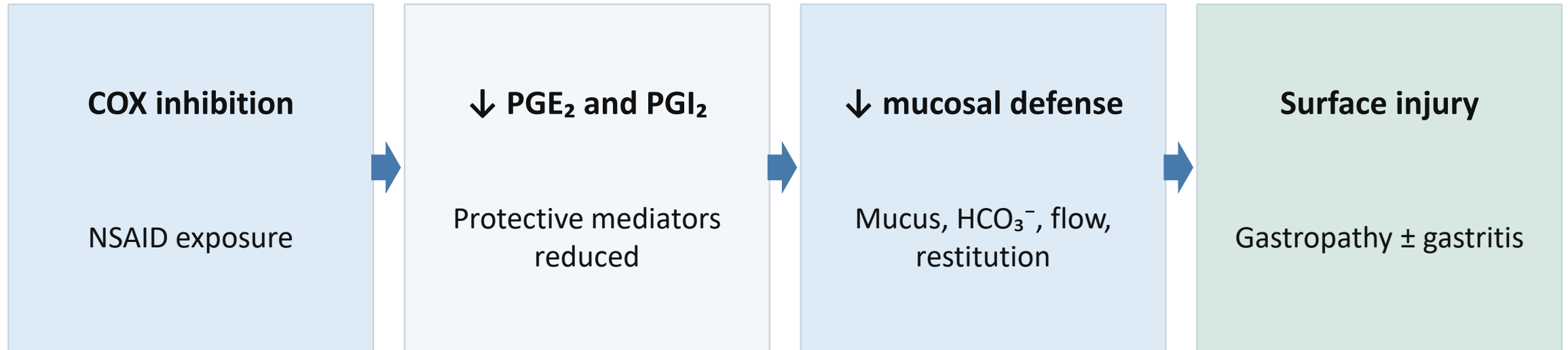
## Common exposures and their principal effects

| Exposure                 | Mechanism highlighted in Robbins           | Potential mucosal result              |
|--------------------------|--------------------------------------------|---------------------------------------|
| NSAIDs                   | Cyclooxygenase / prostaglandin suppression | Loss of multiple protective functions |
| Alcohol / bile           | Luminal irritant exposure                  | Reactive epithelial injury            |
| Caustics                 | Severe direct chemical injury              | Pronounced mucosal damage             |
| Radiation / chemotherapy | Direct cytotoxicity and impaired renewal   | Failure of epithelial protection      |

## NSAIDs are a major cause of gastric injury

- Aspirin, ibuprofen and naproxen inhibit cyclooxygenase.
- NSAID injury results largely from impaired prostaglandin-mediated protection.
- Both gastropathy and gastritis may occur after exposure.
- Exposure history is essential when interpreting reactive gastric injury.

# NSAIDs impair the prostaglandin defense axis



- Cyclooxygenase inhibition links drug exposure to several defense failures.

# Prostaglandins protect through multiple effects

## Luminal protection

- Promote mucus output.
- Promote bicarbonate output.
- Support phospholipid secretion.

## Tissue support

- Preserve mucosal blood flow.
- Facilitate epithelial restitution.

## Reduced aggression

- Decrease gastric acid secretion.
- Act through COX-dependent synthesis.

# COX-1 and COX-2 both contribute to protection

## COX-1

- Has the larger role in mucosal protection.
- Inhibition strongly compromises protective prostaglandins.
- Nonselective inhibitors suppress this pathway.

## COX-2

- Also contributes to gastric mucosal protection.
- Selective inhibition does not abolish gastric injury risk.
- Selective COX-2 inhibitors can still cause gastropathy.

## NSAID selectivity does not confer zero gastric risk

| Drug group                  | Examples from textbook       | Teaching implication                            |
|-----------------------------|------------------------------|-------------------------------------------------|
| Nonselective COX inhibitors | Aspirin, ibuprofen, naproxen | Greatest gastric injury risk in this comparison |
| Selective COX-2 inhibitors  | Celecoxib, etoricoxib        | Can still cause gastropathy or gastritis        |

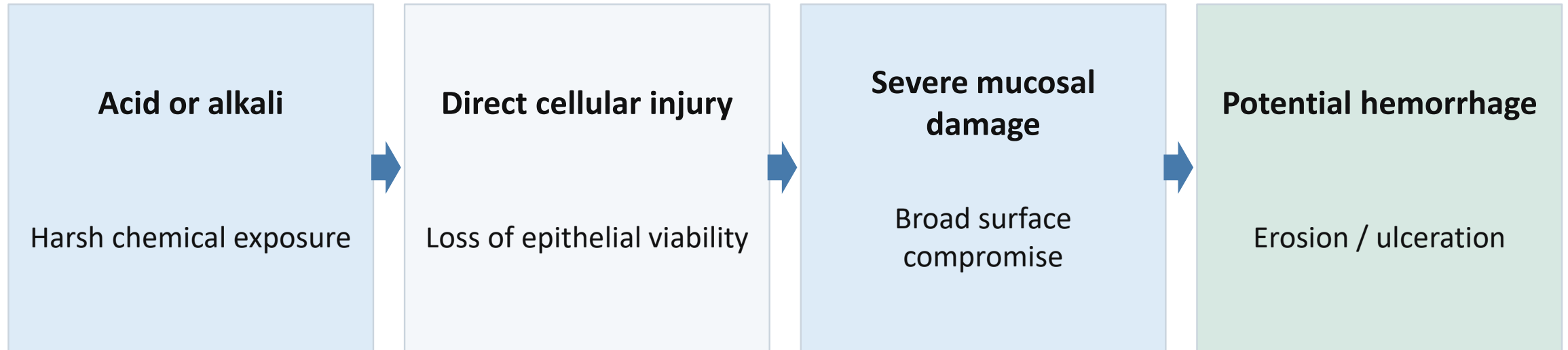
## Bile is a luminal irritant

- Bile is listed among common causes of gastropathy.
- Exposure of gastric mucosa to bile can disturb surface protection.
- Histology may show a reactive injury pattern with limited inflammation.
- An exposure pattern alone does not establish a specific histologic diagnosis.

## Alcohol can produce direct epithelial injury

- Alcohol is a common irritant associated with gastropathy.
- Direct cellular damage contributes to alcohol-related gastritis.
- Injury may occur without a prominent inflammatory infiltrate.
- Assess the epithelial reaction and the presence of neutrophils separately.

# Caustic ingestion produces a different scale of injury

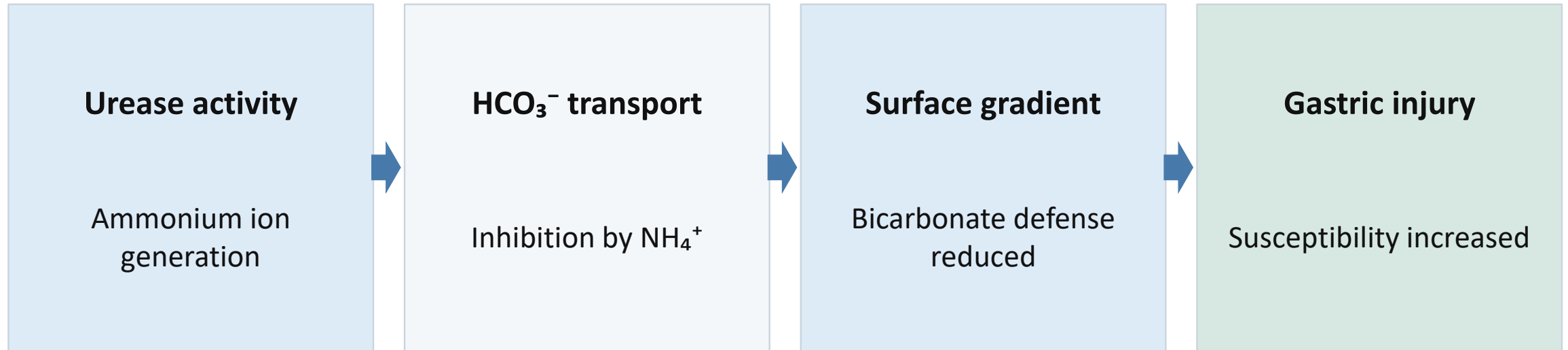


- Caustics as causing severe injury; Extent is exposure-dependent.

## Uremia can compromise bicarbonate-related protection

- Ammonium ions may inhibit gastric bicarbonate transporters.
- This mechanism can contribute to gastric injury in uremic patients.
- The key lesion is failure of an epithelial protective function.
- The morphologic outcome overlaps with other chemical or reactive insults.

## A urease-dependent route to epithelial injury

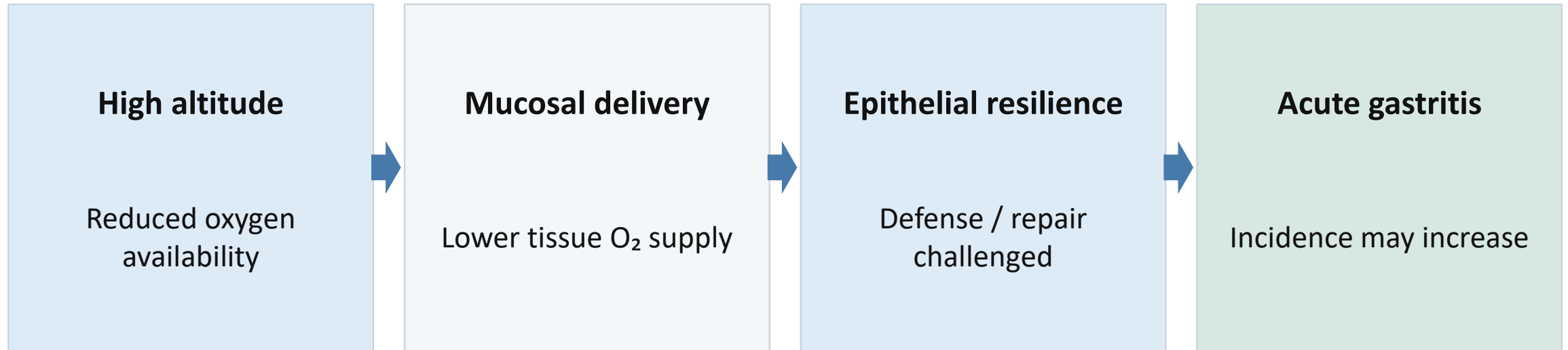


- Urease-secreting H. Pylori as an example. Detailed chronic H. Pylori gastritis is a separate topic.

## Aging may increase susceptibility to gastritis

- Reduced mucin secretion is a proposed contributing factor in older adults.
- Reduced bicarbonate secretion is another suggested mechanism.
- These are proposed explanations rather than a single proven pathway.
- Weakened luminal defenses increase vulnerability to ordinary irritants.

# High altitude highlights the oxygen requirement



- The source proposes decreased oxygen delivery as a possible mechanism.

# Radiation and chemotherapy damage through two routes

## Direct toxicity

- Radiation therapy can directly damage surface cells.
- Some chemotherapy agents also injure epithelial cells.
- Barrier integrity may decline rapidly.

## Impaired renewal

- DNA synthesis and mitotic inhibitors limit replacement of damaged cells.
- Restitution cannot keep pace with injury.
- Repeated damage can outstrip repair.

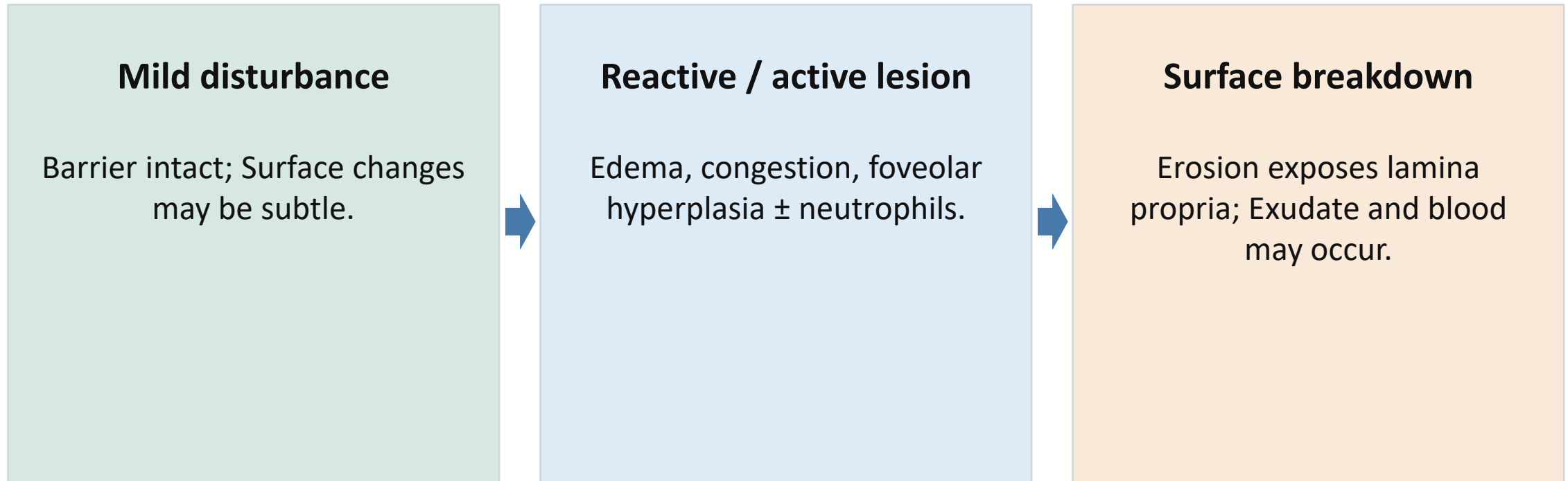
## Anti-proliferative therapy undermines epithelial renewal

- Mucosal replacement requires ongoing DNA synthesis and cell division.
- DNA synthesis inhibitors and mitotic inhibitors can limit renewal.
- A previously manageable insult may now produce more extensive injury.
- This mechanism is distinct from direct chemical damage.

## Classify mechanisms before interpreting morphology

| Mechanism                     | Representative setting         | Defense or injury axis                           |
|-------------------------------|--------------------------------|--------------------------------------------------|
| Prostaglandin loss            | NSAID use                      | Mucus, $\text{HCO}_3^-$ , perfusion, restitution |
| Chemical cytotoxicity         | Alcohol, acids / alkalis       | Direct cellular integrity                        |
| Impaired turnover             | Antimitotic chemotherapy       | Epithelial replacement                           |
| Oxygen / perfusion limitation | High altitude; ischemic stress | Metabolism and acid washout                      |

# Progression from intact epithelium to denudation

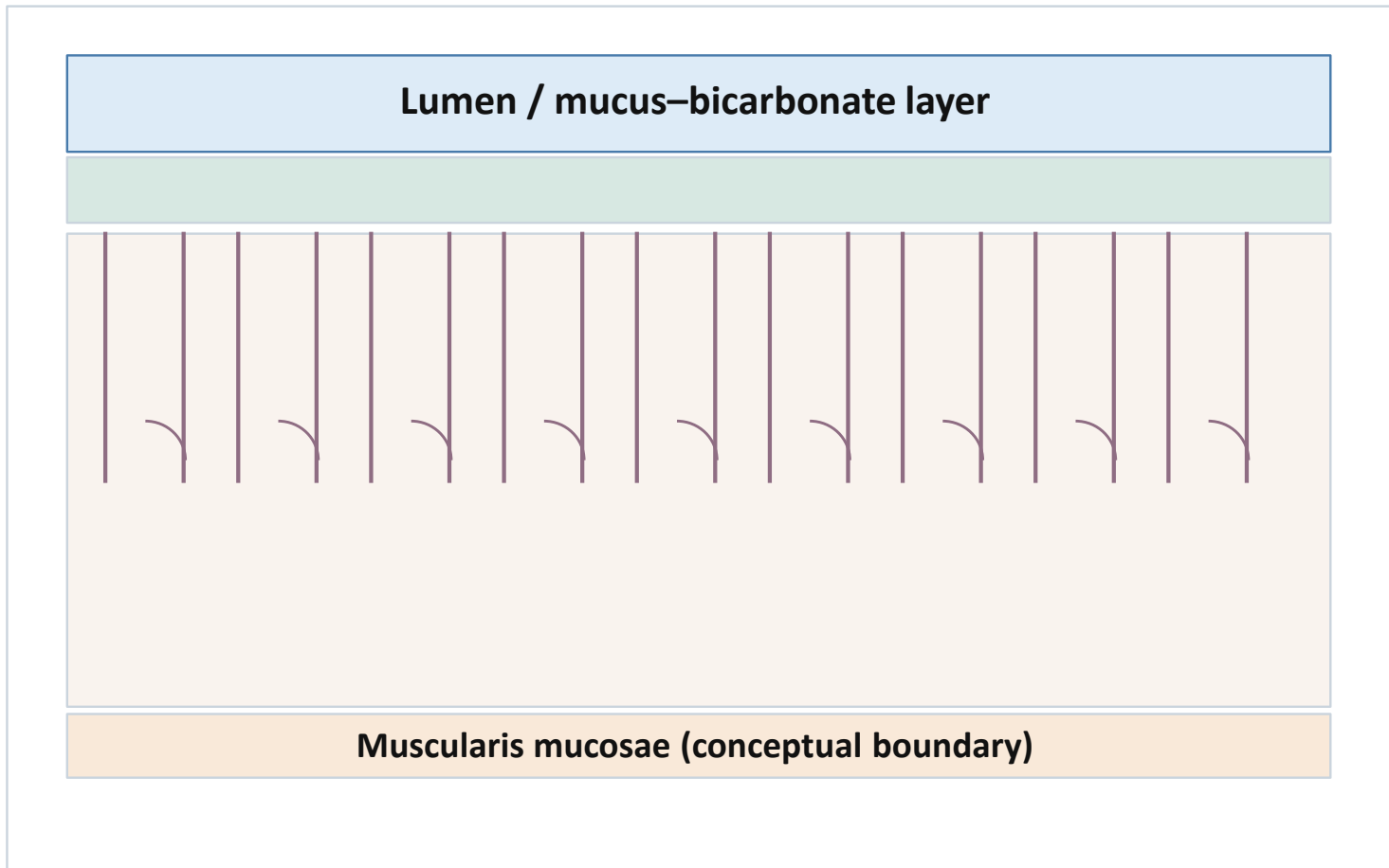


- Progression is not inevitable; The severity depends on injurious and defensive forces.

## Mild gastropathy may be histologically subtle

- Surface epithelium can remain intact.
- Lamina propria may show only modest edema and congestion.
- Reactive foveolar changes are often more distinctive than inflammation.
- The biopsy must be interpreted in clinical and exposure context.

# Lamina propria edema: A subtle tissue reaction



## Pathology context

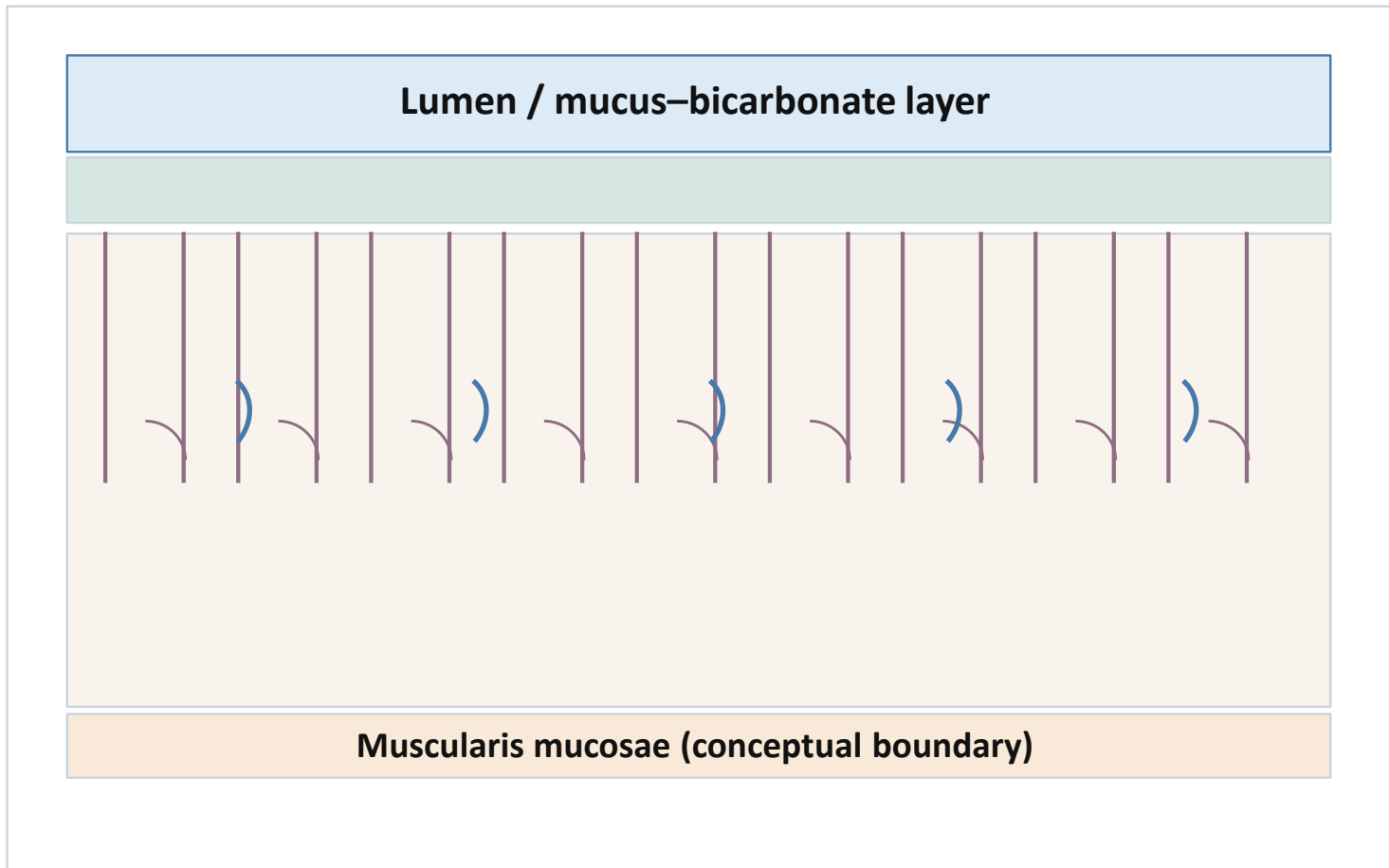
- A mild increase in stromal fluid may be apparent.
- This finding is nonspecific by itself.
- Assess it alongside epithelial changes and inflammation.

Original editable schematic — simplified architecture; Not a histologic specimen.

## Vascular congestion can accompany mild injury

- Slight congestion in gastropathy and mild acute gastritis.
- Congestion is not sufficient to determine the cause.
- A vascular reaction can coexist with intact surface epithelium.
- Neutrophil distribution remains essential for classifying gastritis.

# Foveolar hyperplasia indicates a reactive response



## Morphologic clues

- Elongated or tortuous gastric pits.
- Epithelial proliferation in response to injury.
- May appear without substantial inflammatory infiltrates.

Original editable schematic — simplified architecture; Not a histologic specimen.

## “Corkscrew” foveolae are characteristic

- Reactive foveolar hyperplasia may distort pit profiles.
- Elongated and tortuous pits create a corkscrew-like appearance.
- Pit architecture is interpreted together with lamina propria changes.
- Architectural reaction does not itself establish active gastritis.

# Reactive changes and inflammatory activity are separate

## Reactive epithelial component

- Foveolar cell hyperplasia and tortuous pits.
- Lamina propria edema and congestion.
- Epithelial surface may remain intact.

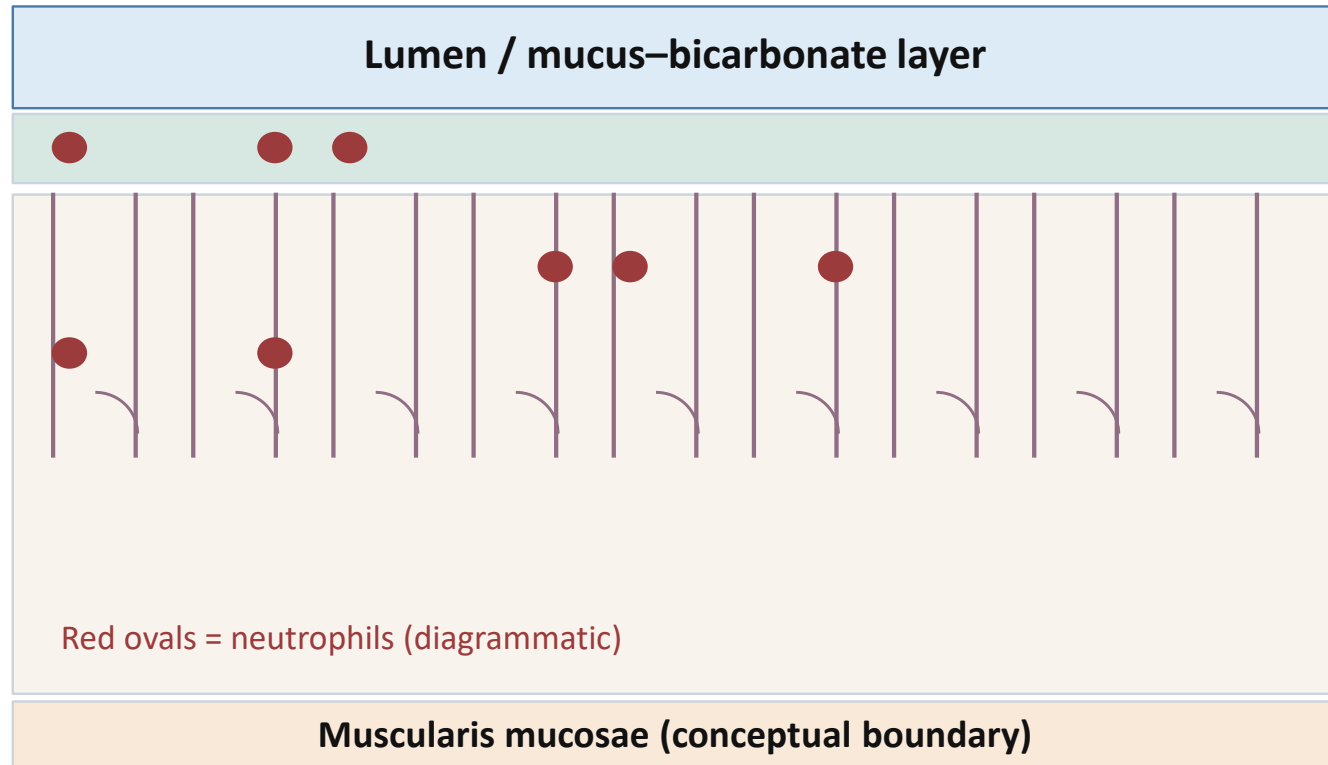
## Active inflammatory component

- Neutrophils in epithelium or glands.
- Evidence of epithelial contact by neutrophils.
- May accompany reactive changes and more severe damage.

## Mild acute gastritis has little chronic inflammation

- Only a few lymphocytes and plasma cells in the lamina propria.
- An extensive chronic infiltrate is not required for an acute process.
- Inflammatory activity is identified by neutrophils, not by edema alone.
- Evaluate location of neutrophils, not simply their overall number.

# Epithelial neutrophils establish inflammatory activity



## Decisive finding

- Neutrophils contact epithelial cells after crossing basement membrane.
- This is abnormal anywhere in the gastrointestinal tract.
- The pattern supports active gastritis rather than pure gastropathy.

Original editable schematic — simplified architecture; Not a histologic specimen.

## Neutrophils within gland lumina are a second clue

- In gastritis, neutrophils may be present within mucosal gland lumina.
- They can also lie between surface epithelial cells.
- Either distribution establishes an active inflammatory component.
- These findings may coexist with foveolar hyperplasia.

# Inflammatory activity differs from disease duration

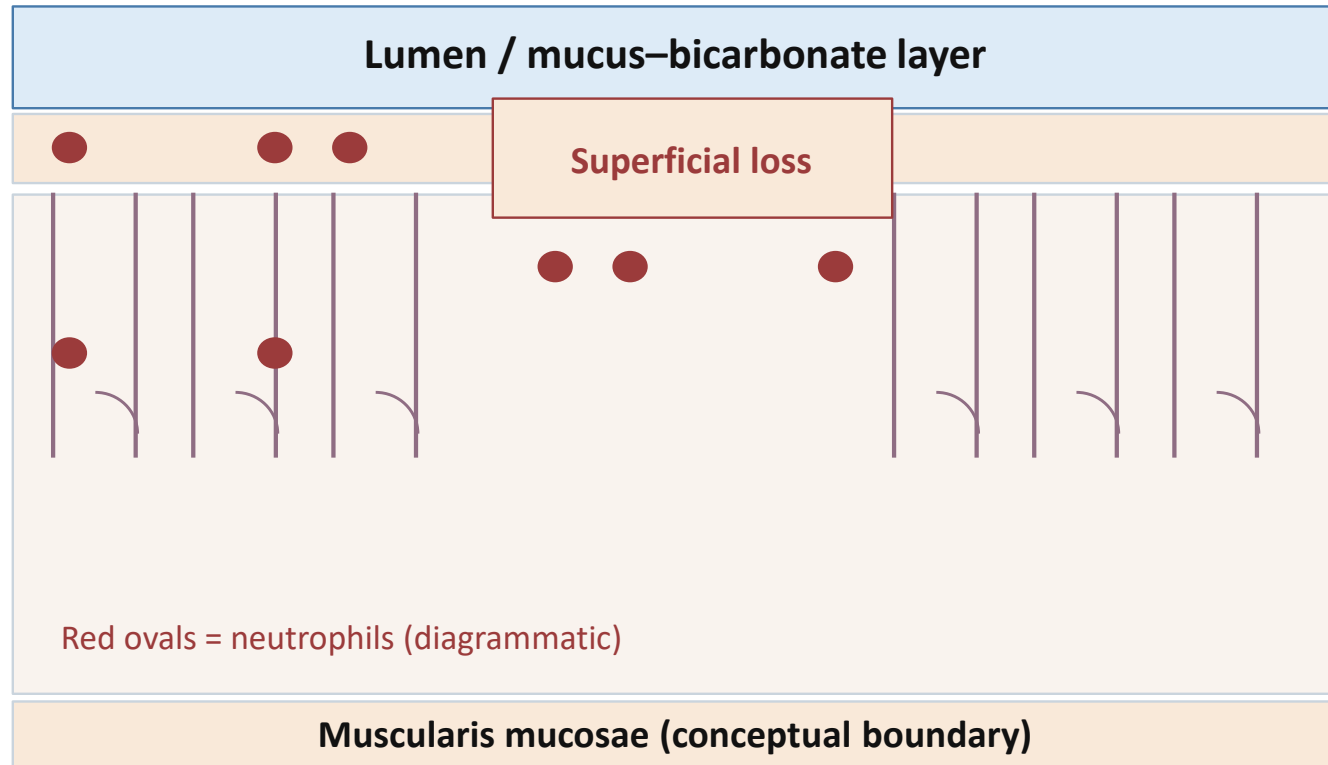
## Active inflammation

- A histopathologic pattern defined by neutrophils.
- May occur in short- or long-duration gastric disease.
- Prefer this label for inflammatory pattern.

## Acute gastritis

- Refers to an acute-duration clinical / pathologic process.
- May show neutrophils within surface or gland epithelium.
- Duration cannot be determined by neutrophils alone.

# An erosion is a superficial mucosal defect



## Depth of injury

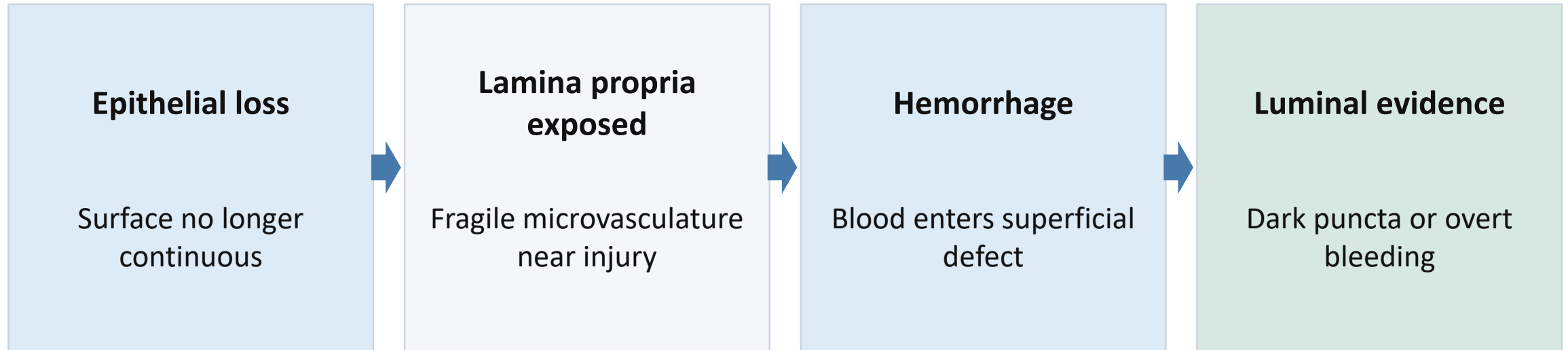
- Surface epithelium is lost in a limited area.
- Defects remain superficial in typical acute injury.
- Deep mucosal penetration is not implied by “erosion.”

Original editable schematic — simplified architecture; Not a histologic specimen.

## Fibrin-containing exudate accompanies erosions

- As injury advances, the mucosal surface can become denuded.
- Neutrophils may accumulate at the site of epithelial loss.
- A fibrin-containing exudate may cover erosions.
- Exudate indicates surface tissue injury rather than a purely reactive pattern.

# Why denudation increases bleeding risk



- The source describes dark puncta within hyperemic mucosa when bleeding occurs.

## Endoscopic appearance may include dark puncta

- Hemorrhage can create dark puncta in hyperemic gastric mucosa.
- Visible puncta indicate a bleeding component rather than only inflammation.
- The extent of injury is not reliably inferred from color alone.
- Histologic depth and epithelial loss remain separate considerations.

# Erosion and hemorrhage define a combined pattern

## Erosion

- Superficial mucosal defect.
- Often neutrophils and fibrin-containing exudate.
- May be extensive across the surface.

## Hemorrhage

- Blood associated with mucosal injury.
- Can produce dark puncta within hyperemic mucosa.
- Can coexist with erosions in acute erosive hemorrhagic gastritis.

## Acute erosive hemorrhagic gastritis: Core definition

- Concurrent mucosal erosion and hemorrhage constitute this pattern.
- Despite wide denudation, injury is typically superficial.
- A neutrophilic and fibrinous surface reaction may be present.
- This is a descriptive lesion pattern, not a unique etiologic diagnosis.

## A morphology-based comparison

| Feature             | Gastropathy                    | Active gastritis                     |
|---------------------|--------------------------------|--------------------------------------|
| Dominant lesion     | Reactive epithelial injury     | Neutrophilic inflammation            |
| Foveolae            | Hyperplasia; tortuous profiles | May be reactive as well              |
| Neutrophils         | Rare or absent                 | Within epithelium / gland lumina     |
| Additional findings | Edema, congestion              | Edema, congestion; possible erosions |

# Erosion is not identical to a chronic peptic ulcer

## Erosion

- Defect of the superficial mucosal surface.
- Can accompany acute gastric injury.
- Often heals without the deep injury of an ulcer.

## Ulceration

- Extends more deeply into gastric tissue than a superficial erosion.
- A broader outcome in severe gastric disease.
- Chronic peptic ulcer disease is addressed separately.

## Symptoms do not identify the microscopic category

- Epigastric pain can occur in gastropathy and gastritis.
- Nausea or vomiting may accompany either lesion.
- Both patterns may be asymptomatic.
- Histology and history provide the distinction that symptoms cannot.

# Bleeding manifestations reflect severity

## Occult / limited injury

- The mucosa may show erosion or superficial hemorrhage.
- Symptoms may be absent or nonspecific.
- A small injury can still have a distinctive biopsy pattern.

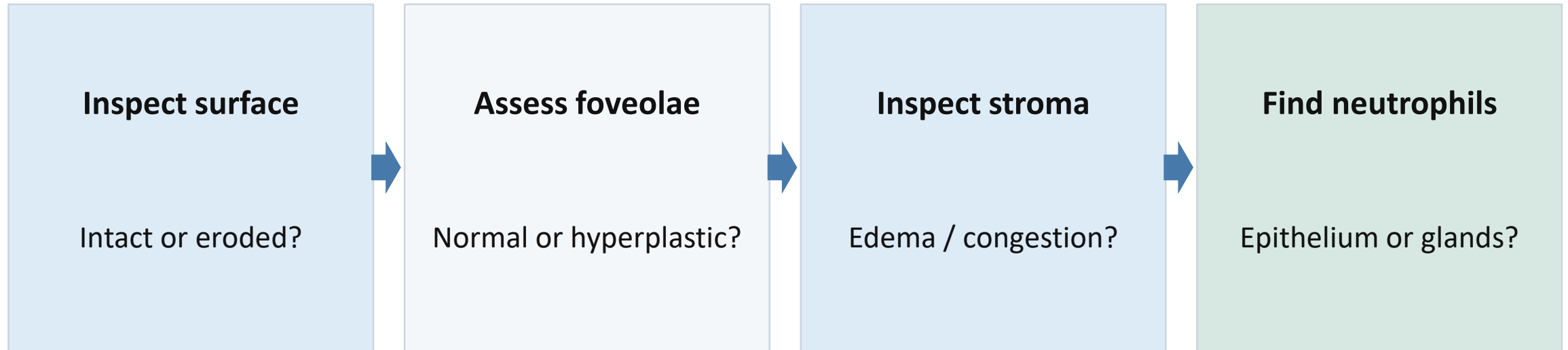
## Clinically visible bleeding

- Hematemesis indicates blood vomited from the upper GI tract.
- Melena may follow upper GI bleeding.
- Rarely, blood loss may be massive.

## Bleeding is a consequence, not a separate mechanism

- Multiple insults can converge on erosion and hemorrhage.
- The same lesion may result from chemical, drug-related or vascular injury.
- Interpret bleeding in conjunction with mucosal depth and cause.
- Do not equate all gastric bleeding with primary peptic ulcer disease.

# A structured biopsy-reading approach



- Report a reactive pattern separately from neutrophil-defined activity.

## Histologic language: What each observation supports

| Finding                     | Supported interpretation   | What it does not prove       |
|-----------------------------|----------------------------|------------------------------|
| Corkscrew foveolae          | Reactive epithelial change | Acute inflammation by itself |
| Lamina propria edema        | Mild tissue injury         | Specific exposure or cause   |
| Intraepithelial neutrophils | Active gastritis           | Duration of the process      |
| Erosion plus blood          | Erosive hemorrhagic injury | Single unique etiology       |

# Case: Epigastric discomfort after frequent analgesics

## Clinical scenario

An adult taking frequent ibuprofen develops epigastric discomfort. A gastric biopsy shows elongated, tortuous foveolae and congestion; Neutrophils are scarce.

**Is this pattern better termed gastropathy or active gastritis?**

Identify the feature that would be needed to call the lesion active.

## Case interpretation: NSAID-associated gastropathy

- Scant neutrophils favor gastropathy rather than active gastritis.
- Reactive foveolar hyperplasia supports epithelial injury.
- NSAID exposure can suppress protective prostaglandin pathways.
- The drug association is clinically plausible, but biopsy alone does not prove causation.

## Case: Acute bleeding and superficial defects

### Clinical scenario

A patient presents with hematemesis. The gastric mucosa has hyperemic areas with dark puncta. Biopsy reveals superficial epithelial defects, neutrophils and fibrinous exudate.

**What morphologic diagnosis best describes the combined findings?**

Distinguish the observed lesion pattern from an etiologic attribution.

## Case interpretation: Erosive hemorrhagic gastritis

- Superficial epithelial loss establishes erosion.
- Dark puncta and extravasated blood indicate hemorrhage.
- Neutrophils and surface exudate support active injury.
- Cause requires medication, exposure and clinical-context assessment.

## Relationship to neighboring gastric pathology topics

| This lecture                    | Adjacent focused lecture       | Boundary of coverage                           |
|---------------------------------|--------------------------------|------------------------------------------------|
| Reactive / acute mucosal injury | Stress-related mucosal disease | Severe systemic stress and stress ulcers       |
| Activity rather than chronicity | H. pylori gastritis            | Chronic infection pattern and its consequences |
| Superficial erosion             | Peptic ulcer disease           | Deeper persistent peptic ulceration            |

## Integrative summary: Defense → reaction → loss

### Defense maintained

Mucus,  $\text{HCO}_3^-$ , epithelium, perfusion and renewal offset luminal damage.



### Injury outweighs defense

Reactive epithelial changes develop; Neutrophils distinguish active gastritis.



### More severe injury

Erosion, fibrinous exudate and hemorrhage may occur.

- Etiology and morphologic pattern must be analyzed separately.

## Application question: Choose the decisive finding

### Clinical scenario

Two biopsies show foveolar hyperplasia and lamina propria edema. Only one has neutrophils in the surface epithelium.

**Which biopsy is active gastritis, and why?**

Answer using the definition of inflammatory activity, not clinical duration.

## Answer: Neutrophil–epithelium contact defines activity

- The biopsy with intraepithelial neutrophils has active gastritis.
- Both specimens may show reactive epithelial injury.
- Edema and foveolar hyperplasia alone do not establish activity.
- Neutrophils do not establish how long the disease has been present.

## References and related reading

- Robbins, Cotran & Kumar: Pathologic Basis of Disease, 11th ed. (2026). Chapter 17, “The Gastrointestinal Tract,” pp. 696–697.
- Primary section: Gastropathy and Acute Gastritis, pp. 696–697.
- Textbook Figure 17.13 reviews mechanisms of gastric mucosal injury and protection.
- Related sections: stress-related mucosal disease, H. pylori gastritis and peptic ulcer disease.