

Reflux Esophagitis

Mechanisms • Morphology • Clinicopathologic interpretation

Gastroesophageal reflux disease and esophageal injury

Dr Abu Suraih Sakhri

MBBS, MD (Pathology)

www.abusuraihsakhri.com

Learning outcomes

- Explain the lower esophageal sphincter mechanisms underlying reflux.
- Identify the gross and microscopic features of reflux esophagitis.
- Relate symptoms and investigation choices to mucosal pathology.
- Differentiate reflux injury from eosinophilic and infectious esophagitis.

GERD is not synonymous with erosive esophagitis

- GERD is a clinical condition linked to reflux of gastric contents.
- Reflux esophagitis denotes mucosal injury in the esophagus.
- Symptoms may occur with little or no visible histologic abnormality.
- When injury occurs, the distal squamous-lined esophagus is exposed.

Clinical symptoms and structural injury need not track together.

Three linked—but distinct—observations

Reflux event

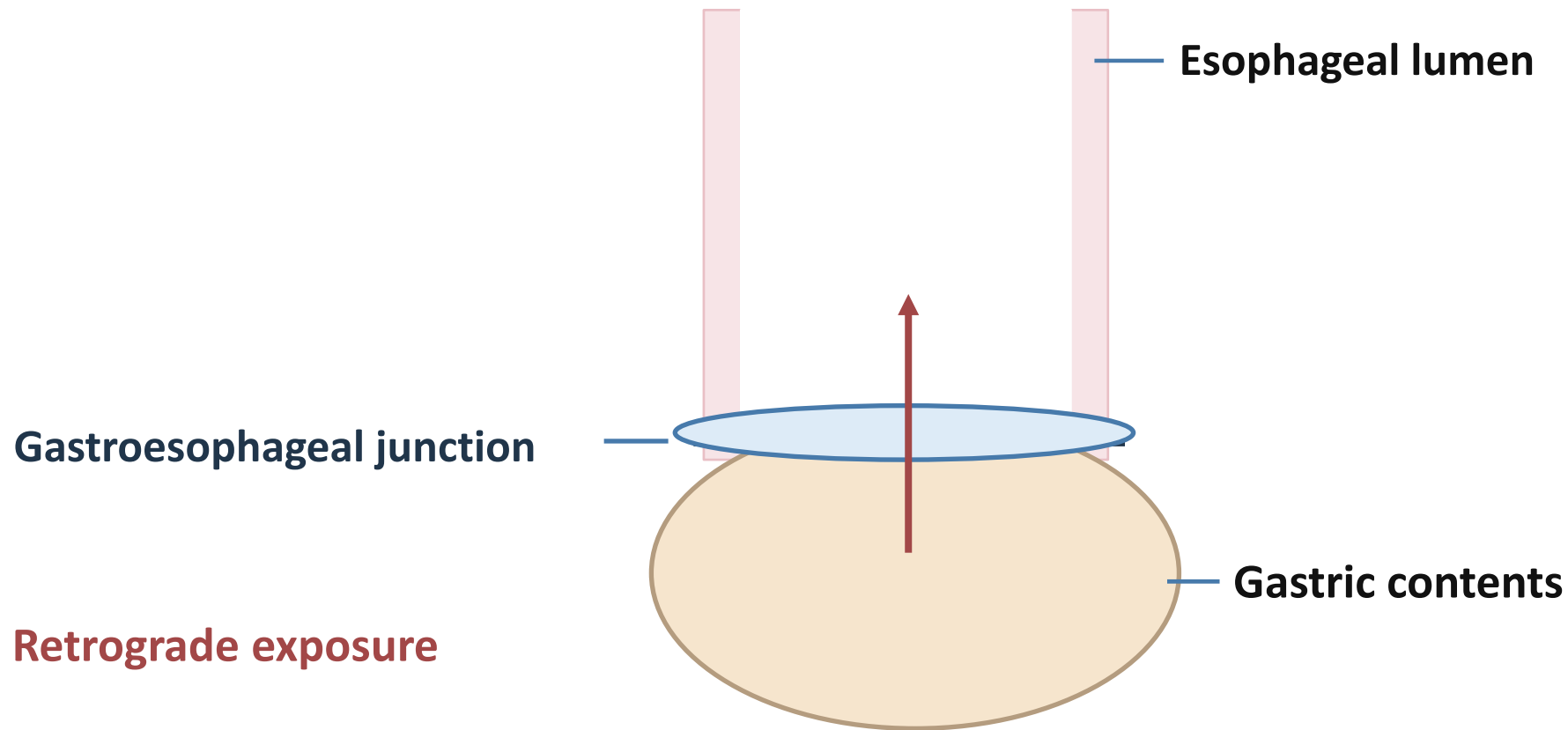
- Gastric contents pass proximally.
- Often facilitated by transient sphincter relaxation.

Mucosal response

- Esophageal squamous epithelium contacts gastric acid.
- Hyperemia, erosion, or microscopic reactive changes may follow.

Anatomic orientation: The distal esophagus

Schematic of the distal esophagus and stomach



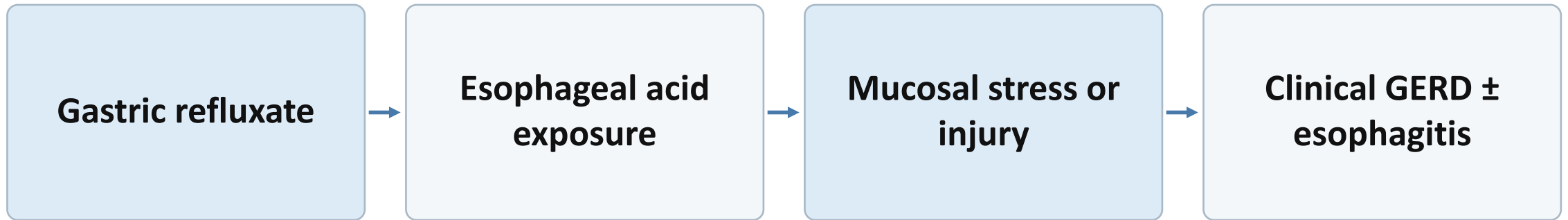
Simplified anatomy / histology schematic — not an observed specimen

The squamous lining resists abrasion, not acid

- The esophageal epithelium is stratified squamous.
- Its mechanical resilience does not ensure resistance to gastric acid.
- The dominant disease mechanism is exposure of this lining to gastric refluxate.

Surface injury depends on exposure, not only on a microscopic defect.

From reflux episode to tissue injury



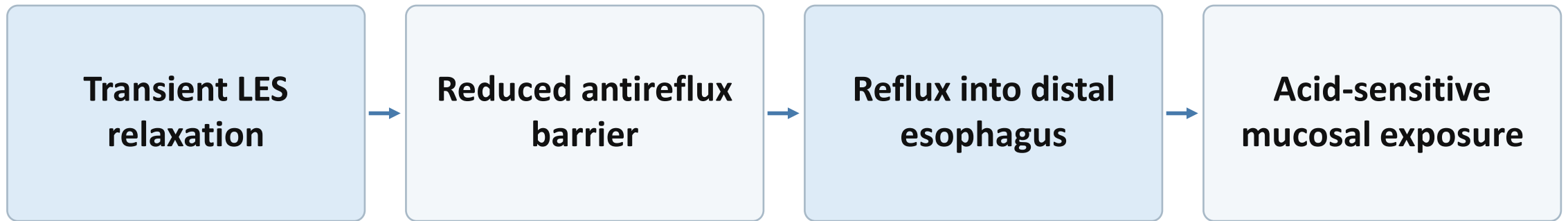
This is a causal teaching schematic, not a fixed sequence occurring in every patient.

GERD is common but geographically variable

- Substantial regional differences in GERD prevalence.
- The cited prevalence approaches 30% in several Western and Middle Eastern regions.
- East Asia and Australia are described as having prevalence at or below 10%.
- These are textbook estimates—not a current population survey.

Do not generalize one region's prevalence to another.

Main initiating mechanism: Transient LES relaxation



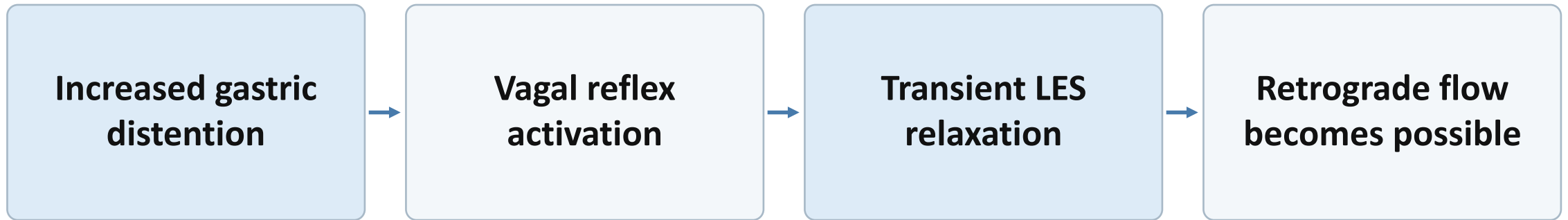
Transient lower esophageal sphincter relaxation is considered a major mechanism.

Vagal control of transient LES relaxation

- Transient relaxation of the lower esophageal sphincter involves vagal pathways.
- Gastric distention can trigger these relaxation events.
- This allows gastric contents to enter the esophagus without requiring a fixed obstruction.

A functional barrier failure can be intermittent.

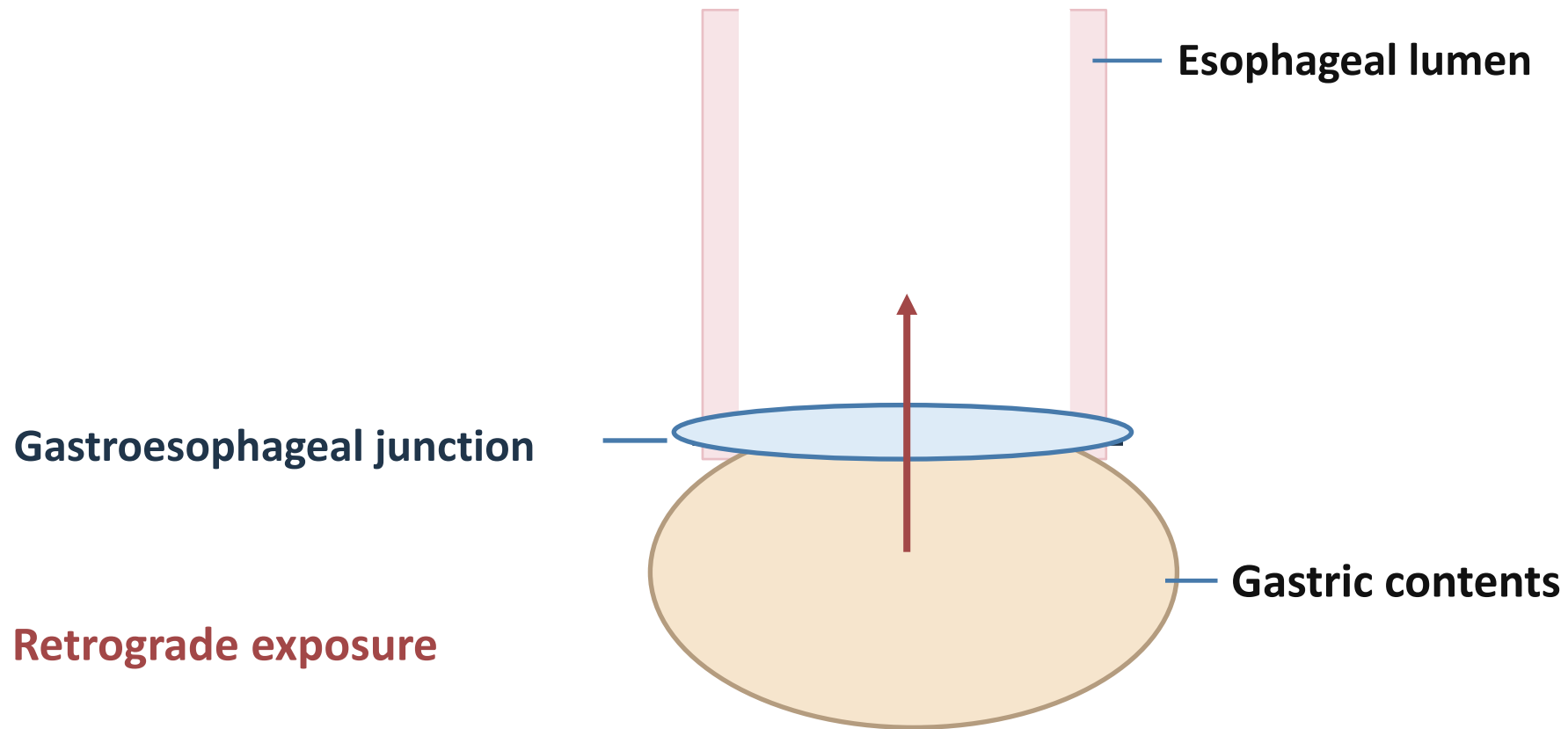
Gastric distention: A plausible reflux trigger



Not every distention episode causes symptomatic or erosive disease.

Gastric reflux crosses the junction

Schematic of the distal esophagus and stomach



Simplified anatomy / histology schematic — not an observed specimen

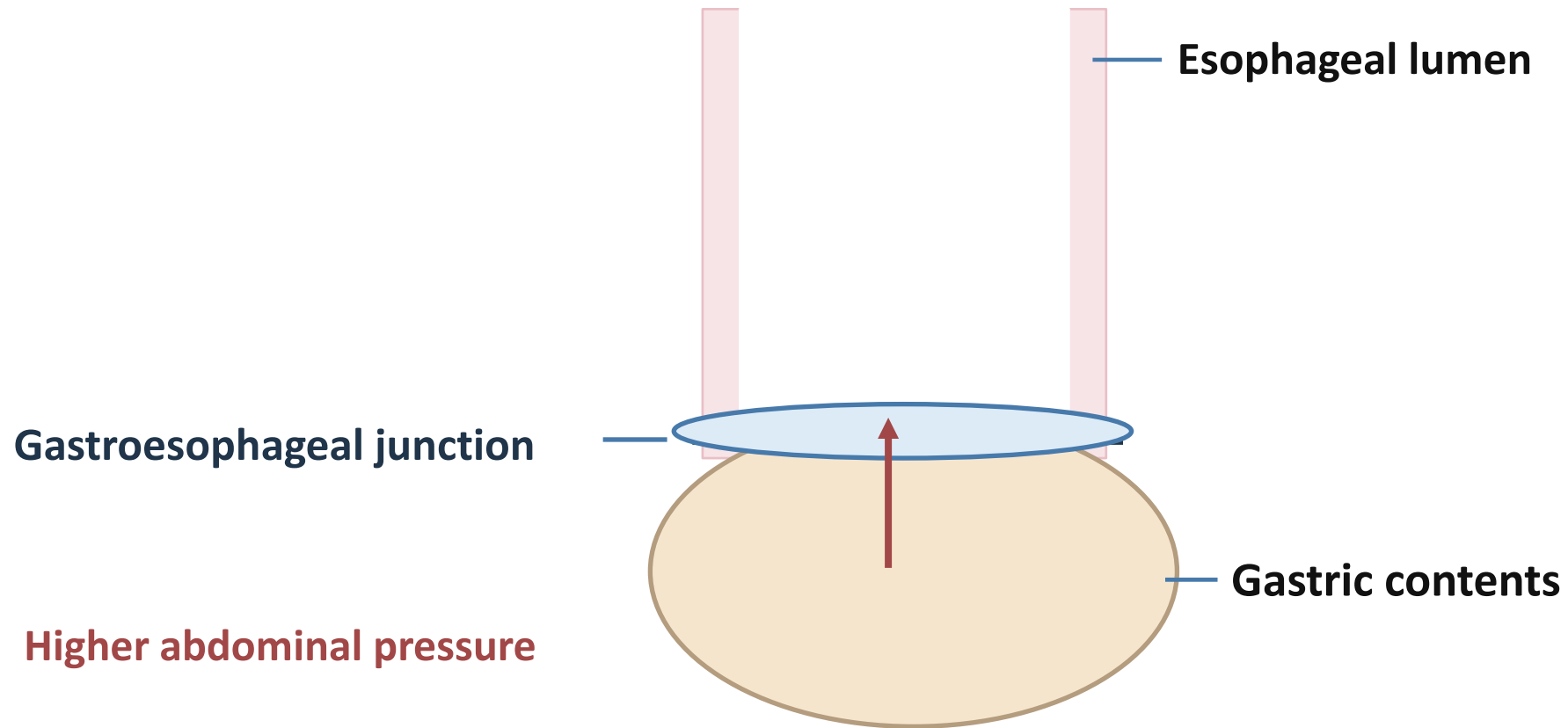
Abrupt intra-abdominal pressure increases

- Coughing, straining, and bending may abruptly increase abdominal pressure.
- These pressure changes can provoke gastroesophageal reflux.
- This mechanism differs from vagally mediated transient LES relaxation.

Different initiating mechanisms converge on the same reflux event.

Reflux during increased abdominal pressure

Schematic of the distal esophagus and stomach



Simplified anatomy / histology schematic — not an observed specimen

Conditions associated with GERD

Physiologic	Structural / motility	Exposures
Pregnancy	Hiatal hernia	Alcohol
Obesity	Delayed gastric emptying	Tobacco
Increased gastric volume	LES incompetence (hernia-related)	CNS depressants

Obesity is an associated condition

- Obesity among conditions associated with GERD.
- It is a relevant clinical context when assessing reflux symptoms.
- The textbook excerpt does not quantify its individual attributable risk.

An association is not an individual-level diagnostic criterion.

Pregnancy may coexist with reflux symptoms

- Pregnancy appears in the source list of GERD-associated conditions.
- Clinical history should recognize this context.
- The excerpt does not assign a pregnancy-specific injury pattern on biopsy.

Potential modifiers of reflux

Patient exposures

- Alcohol use is associated with GERD.
- Tobacco use is associated with GERD.

Drug-related context

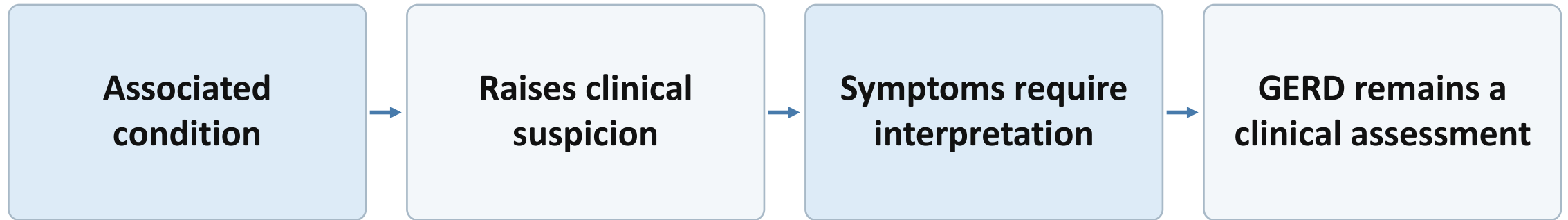
- Central nervous system depressants are associated with GERD.
- The source does not specify agent-by-agent risk estimates.

Delayed gastric emptying and gastric volume

- Delayed gastric emptying is among the associated conditions.
- Increased gastric volume is another listed factor.
- These are particularly relevant when considering a distended stomach as a reflux trigger.

Stomach-related factors can affect an esophageal disorder.

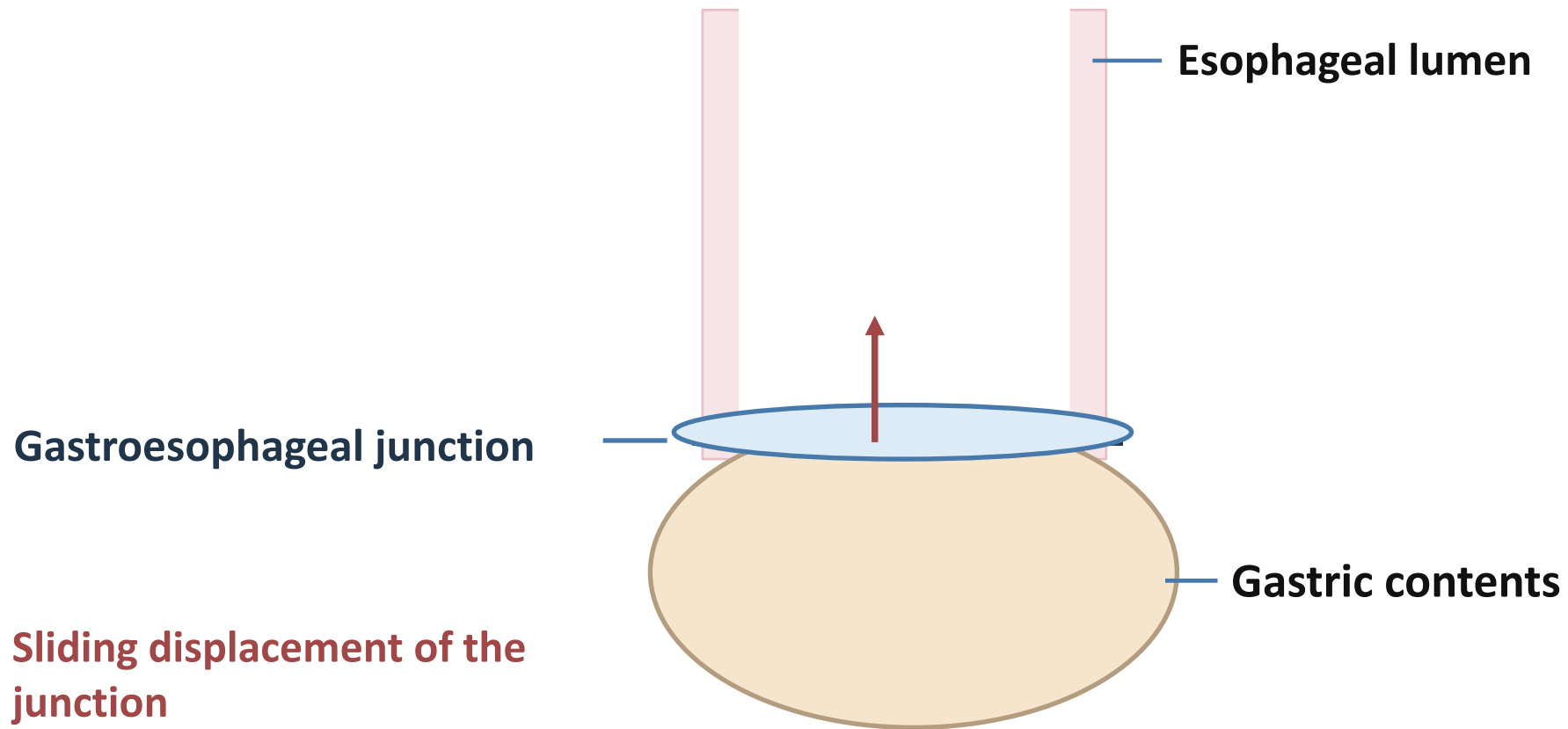
Why risk factors are not diagnostic tests



Presence of obesity, pregnancy, or hernia alone does not establish reflux esophagitis.

Hiatal hernia and sphincter competence

Schematic of the distal esophagus and stomach



Simplified anatomy / histology schematic — not an observed specimen

Sliding and paraesophageal hiatal hernias

Sliding type

- More common type.
- Gastroesophageal junction rises into the thorax.
- Can impair lower sphincter competence.

Paraesophageal type

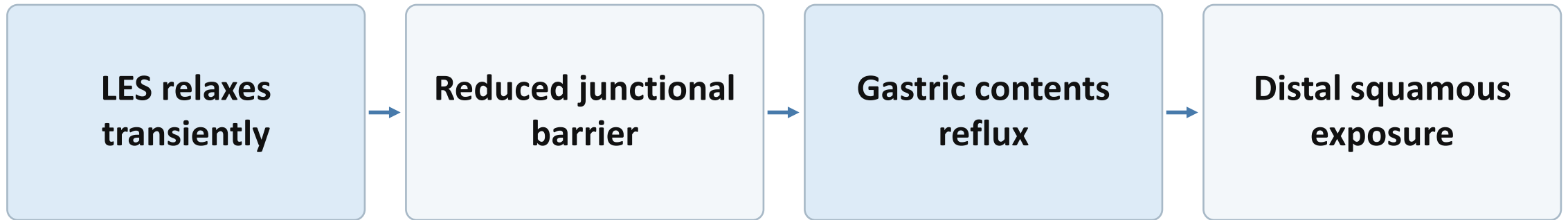
- Less common; Penetrates phrenoesophageal membrane defect.
- May involve stomach or other organs.
- Generally requires surgical repair.

A hiatal hernia is not equivalent to GERD

- Fewer than 10% of adults with hiatal hernia are symptomatic.
- Incompetence of the antireflux barrier can produce GERD-like symptoms.
- The structural finding alone does not explain every symptom.

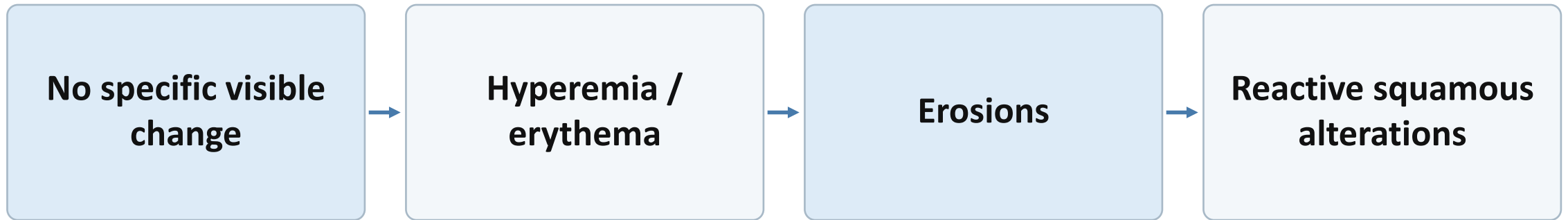
Interpret anatomy and symptoms together.

Two complementary routes to reflux



Pressure-provoked reflux is an additional route, not a step in this diagram.

Reflux injury forms a morphologic spectrum



Erosions and microscopic reactive changes are not mandatory in every clinical GERD case.

Mild reflux: The mucosa may look normal

- The source describes simple hyperemia as a possible sole endoscopic alteration.
- Histology is often unremarkable in mild GERD.
- A normal or nonspecific biopsy therefore cannot exclude clinical reflux symptoms.

Mild disease can evade morphology-based confirmation.

Erythema is a nonspecific endoscopic observation

- Hyperemia is seen endoscopically as redness.
- Redness alone offers limited etiologic specificity.
- Clinical context remains important in interpreting an erythematous esophagus.

Erosions indicate more substantial injury

- More significant reflux may produce esophageal erosions.
- An erosion reflects loss of superficial mucosal integrity.
- Endoscopic demonstration establishes visible mucosal injury, unlike uncomplicated symptomatic reflux.

Compare visible erosion with histologic reaction.

Microscopic pattern: The reactive triad

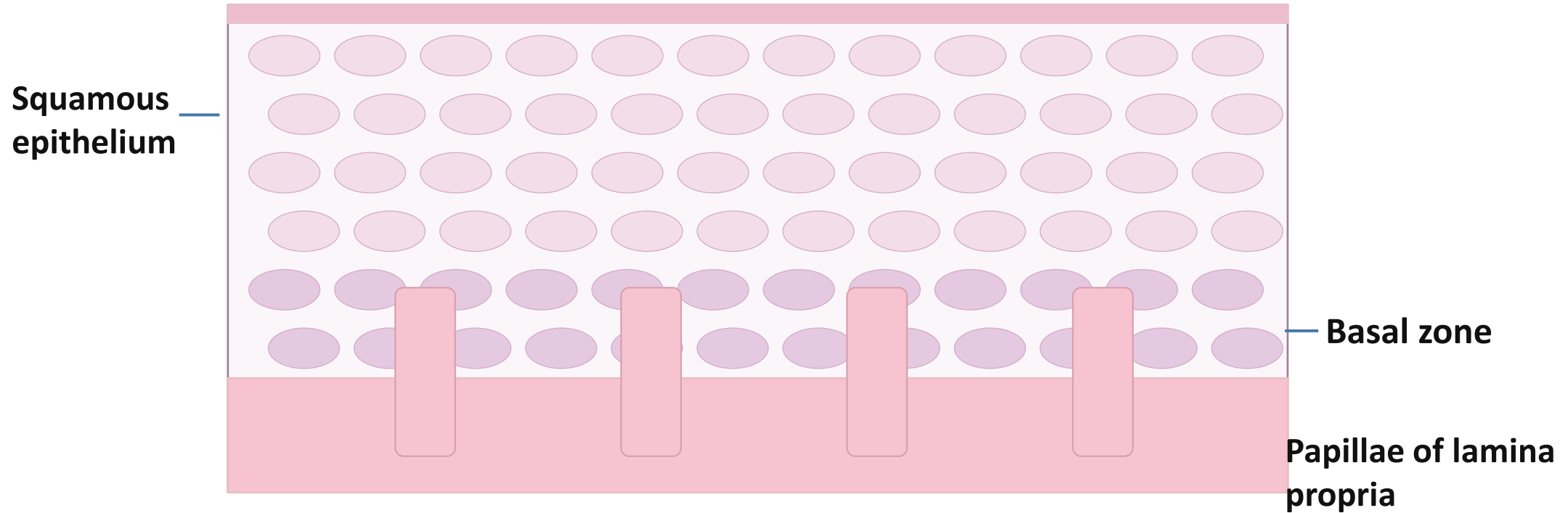
Three findings are emphasized in the Robbins description

Basal zone hyperplasia

Papillary elongation

**Scattered intraepithelial
eosinophils**

Reactive squamous mucosa: Architectural clues



Simplified anatomy / histology schematic — not an observed specimen

Basal zone hyperplasia

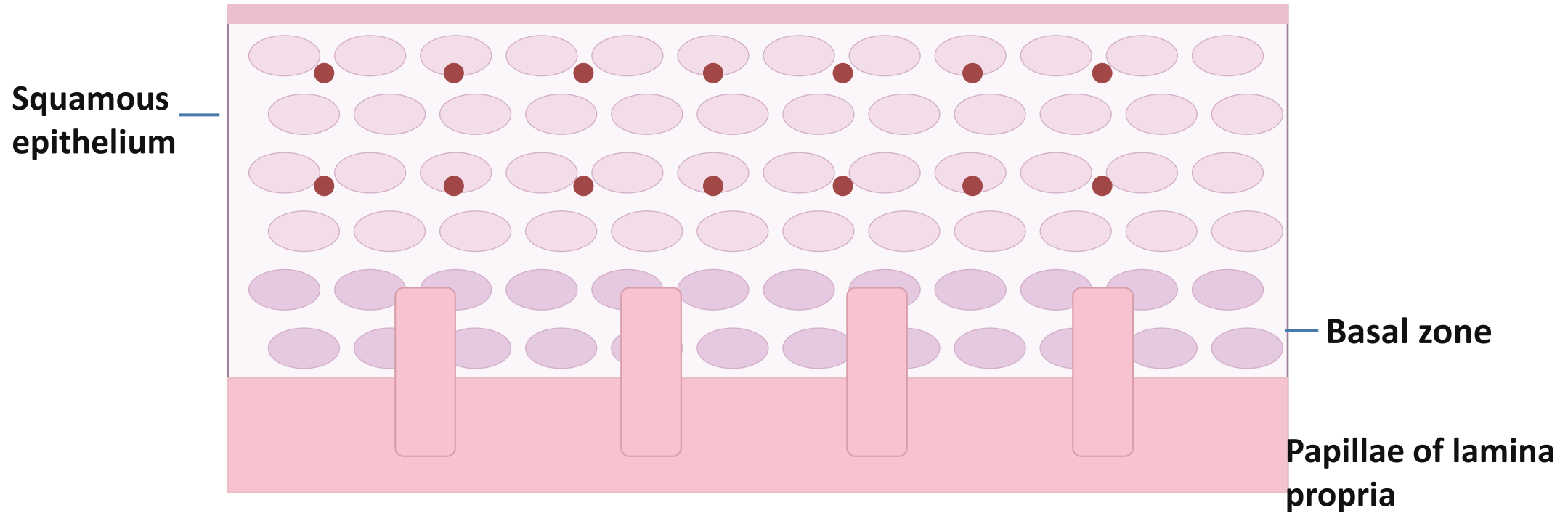
- The basal compartment of squamous epithelium becomes expanded.
- This is a tissue response associated with reflux-related injury.
- Interpret basal thickening with other changes rather than as a stand-alone GERD test.

Reactive architecture is a pattern, not a clinical diagnosis.

Elongation of lamina propria papillae

- Lamina propria papillae extend farther into the squamous mucosa.
- This architectural alteration commonly accompanies basal zone hyperplasia.
- It can be recognized in well-oriented mucosal sections.

Inflammatory cells in reflux-related mucosa



Simplified anatomy / histology schematic — not an observed specimen

Eosinophils may be intraepithelial

- Increased reflux can be associated with eosinophils in squamous epithelium.
- The reflux-associated pattern is described as scattered intraepithelial cells.
- Marked clusters or sheets shift concern toward eosinophilic esophagitis.

Cell distribution and density matter in the differential.

Neutrophils: Interpret cautiously

- Neutrophilic infiltration is less frequent in typical reflux esophagitis.
- Neutrophils more often with infection or chemical damage.
- Consider alternative or additional causes if prominent neutrophils are seen.

Inflammatory-cell type refines, but rarely settles, etiology.

The endoscope and biopsy answer different questions

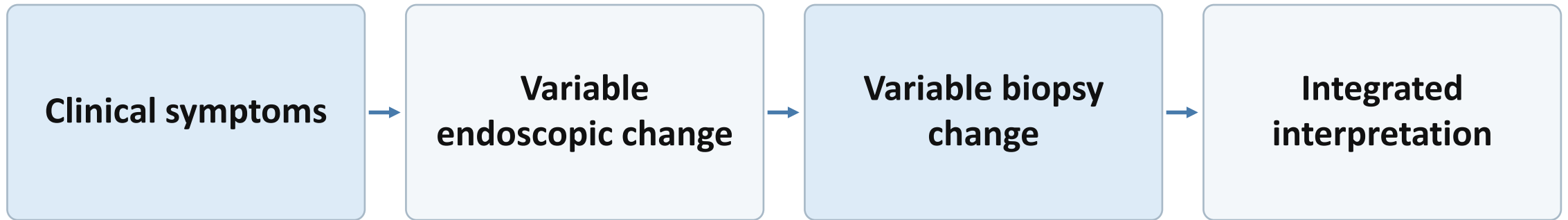
Endoscopy

- Shows redness, erosions, and other structural findings.
- May be normal in mild symptomatic disease.

Biopsy

- Samples reactive epithelium and inflammatory cells.
- May be histologically unremarkable in mild reflux.

Morphologic findings are suggestive, not universal



No single depicted finding is represented here as necessary for all GERD.

Typical clinical manifestations

- Heartburn is a characteristic symptom.
- Regurgitation of sour-tasting gastric contents is common.
- Dysphagia is another described presenting symptom.
- Symptoms often occur after meals.

The symptom pattern frames the clinical diagnosis.

Heartburn arises in the clinical reflux syndrome

- Heartburn is central to the GERD symptom complex.
- It is commonly linked temporally to meals.
- Its presence does not predict the extent of epithelial injury.

Regurgitation provides a mechanistic clue

- Patients may describe sour gastric contents returning to the esophagus or mouth.
- This history is compatible with retrograde gastric flow.
- Distinguish regurgitation from painful swallowing and mechanical obstruction.

Dysphagia requires careful interpretation

- Dysphagia may occur with GERD.
- Persistent or prominent dysphagia raises concern for structural complications or an alternative cause.
- Endoscopic evaluation becomes relevant when stricture or other disease is suspected.

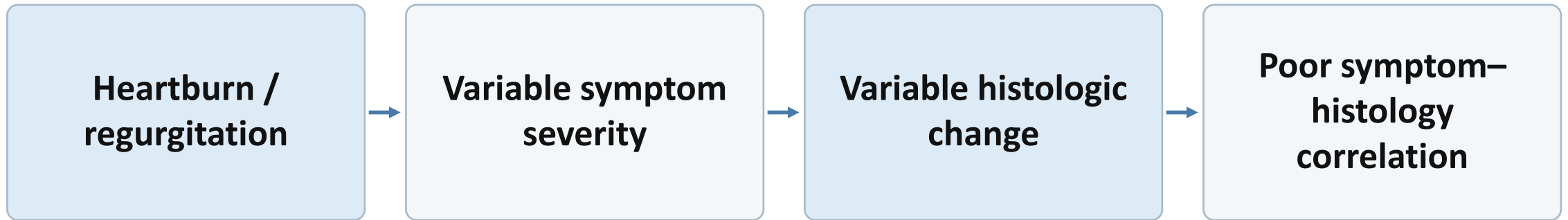
Not every dysphagia complaint is explained by acid reflux.

Chest discomfort can mimic cardiac pain

- Typical reflux symptoms can be mistaken for ischemic heart disease.
- Chest discomfort should not be attributed automatically to GERD.
- Pathology teaching must preserve the cardiovascular differential.

A presumed reflux explanation must not obscure cardiac evaluation.

Clinical symptoms and mucosal damage can diverge



The source explicitly says that the correlation is weak.

GERD diagnosis is usually clinical

- Endoscopy and biopsy are generally unnecessary for diagnosis.
- Symptom evaluation often establishes a working clinical diagnosis.
- Investigation becomes appropriate when the presentation is refractory or complications are suspected.

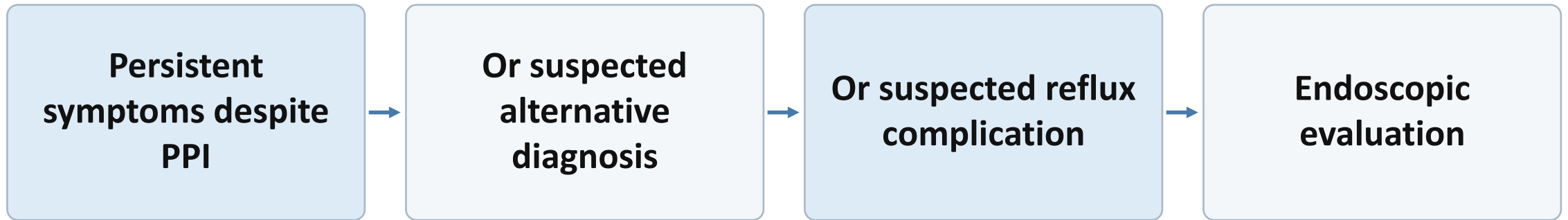
Routine biopsy is not a prerequisite for ordinary GERD.

Proton pump inhibitors address acidity

- Proton pump inhibitors suppress gastric acid production.
- Symptomatic relief in up to 90% of patients.
- Treatment response concerns symptoms; It does not provide direct histologic grading.

The textbook response estimate is not a guaranteed individual outcome.

When the source supports endoscopy

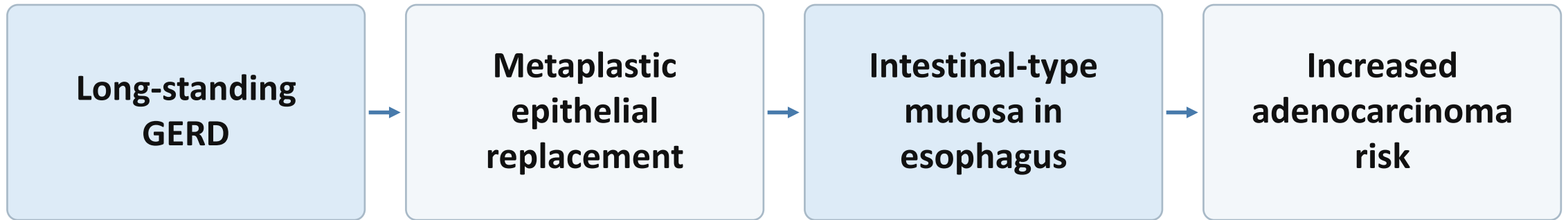


The diagram summarizes indications named by Robbins; It is not a comprehensive guideline.

Suspected findings prompting endoscopy

Alternative diagnosis	Complication	Other concern
Eosinophilic esophagitis	Stricture	PPI-refractory symptoms
—	Ulceration	Persistent unexplained symptoms
—	Barrett esophagus	Assessment of mucosal injury

Chronic reflux and Barrett esophagus



Barrett is a complication of chronic GERD; Progression is not inevitable.

Barrett esophagus represents metaplasia

- Chronic GERD can be complicated by intestinal metaplasia of esophageal squamous mucosa.
- The process differs from acute superficial erosion.
- Barrett esophagus carries an increased risk of esophageal adenocarcinoma.

Not every patient with GERD develops Barrett esophagus.

Reflux esophagitis and Barrett esophagus

Reflux esophagitis

- Reactive squamous epithelium.
- Hyperemia or erosion.
- Basal hyperplasia and papillary elongation.

Barrett esophagus

- Columnar metaplastic epithelium above the junction.
- Goblet cells can establish intestinal metaplasia.
- Long-term neoplastic risk is clinically important.

Barrett esophagus: Endoscopic and biopsy clues

- Metaplastic red, velvety mucosa may extend proximally as tongues.
- Endoscopic location relative to the gastroesophageal junction is important.
- Goblet cells are a key histologic marker of intestinal metaplasia account.

Reflux may cause structural complications

- The Robbins discussion lists strictures and ulcerations among suspected GERD complications.
- Dysphagia may motivate evaluation for narrowing.
- The clinician should not infer a stricture from symptom severity alone.

Complication suspicion changes the investigation plan.

Eosinophilic esophagitis versus GERD

Reflux esophagitis

- Acid-exposure clinical context.
- Erosions may be visible.
- Scattered eosinophils with reactive squamous change.

Eosinophilic esophagitis

- Frequently associated with atopy.
- Food impaction and dysphagia can occur.
- Ringed/furrowed esophagus; Prominent superficial eosinophils.

Eosinophils alone do not establish EoE

- GERD can contain intraepithelial eosinophils.
- Eosinophilic esophagitis more typically has numerous superficial cells and clusters or sheets.
- Clinical and endoscopic context is needed because microscopic findings overlap.

Avoid diagnosing EoE from a nonspecific isolated cell finding.

Other esophagitis patterns to distinguish

Etiology	Characteristic context	Key clue
Reflux	Postprandial GERD symptoms	Reactive squamous pattern ± erosion
Pill / chemical	Corrosive or lodged medication exposure	Chemical mucosal injury
Candida	Susceptible host	Adherent gray-white pseudomembranes
HSV	Viral esophagitis	Punched-out ulcers; Epithelial inclusions
CMV	Immunosuppression	Ulcers; Stromal/endothelial inclusions

Case: Reflux symptoms with a normal biopsy

A patient reports recurrent postprandial heartburn and sour regurgitation. Endoscopy reveals no erosion, and the distal esophageal biopsy has no specific abnormalities.

Interpretive questions

- Does normal histology exclude GERD?
- Is biopsy necessary for all suspected GERD?

Case: Reactive distal squamous epithelium

A distal esophageal biopsy shows basal zone hyperplasia, elongated lamina propria papillae, and scattered intraepithelial eosinophils.

Interpretive questions

- What pattern does the biopsy support?
- What clinical information is needed?

Case: Dysphagia despite acid suppression

A patient has ongoing dysphagia despite treatment with a proton pump inhibitor. The physician suspects a stricture or eosinophilic esophagitis.

Interpretive questions

- Why is endoscopy relevant?
- Which findings could alter the diagnosis?

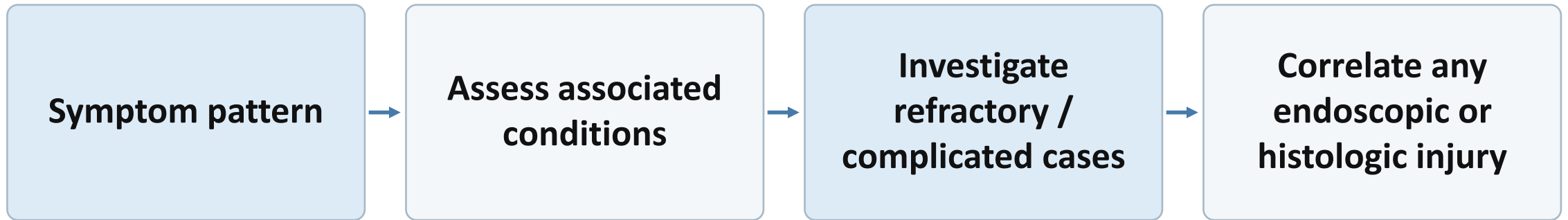
Case: A ringed esophagus

An adult with atopy and recurrent food impaction has an esophagus with ring-like folds and linear furrows. Biopsy shows numerous superficial intraepithelial eosinophils.

Interpretive questions

- Which diagnosis is favored over uncomplicated GERD?
- Why is merely finding eosinophils insufficient?

Integrated approach to suspected reflux



Do not expect severity of symptoms and injury to correlate closely.

Essential morphologic and clinical synthesis

- Reflux is commonly driven by transient LES relaxation and can be pressure-provoked.
- Early mucosal findings range from no change to hyperemia; Deeper injury may erode.
- Basal hyperplasia, papillary elongation, and scattered eosinophils are characteristic but nonspecific.
- GERD is often diagnosed clinically; Endoscopy targets refractory symptoms and suspected complications.

Separate the reflux mechanism, the observed tissue response, and the clinical syndrome.

Key source and further reading

Robbins, Cotran & Kumar

Pathologic Basis of Disease, 11th edition (2026)

The Gastrointestinal Tract — esophageal disorders

Lecture focus: reflux esophagitis, with selected adjacent material on eosinophilic esophagitis and Barrett esophagus.