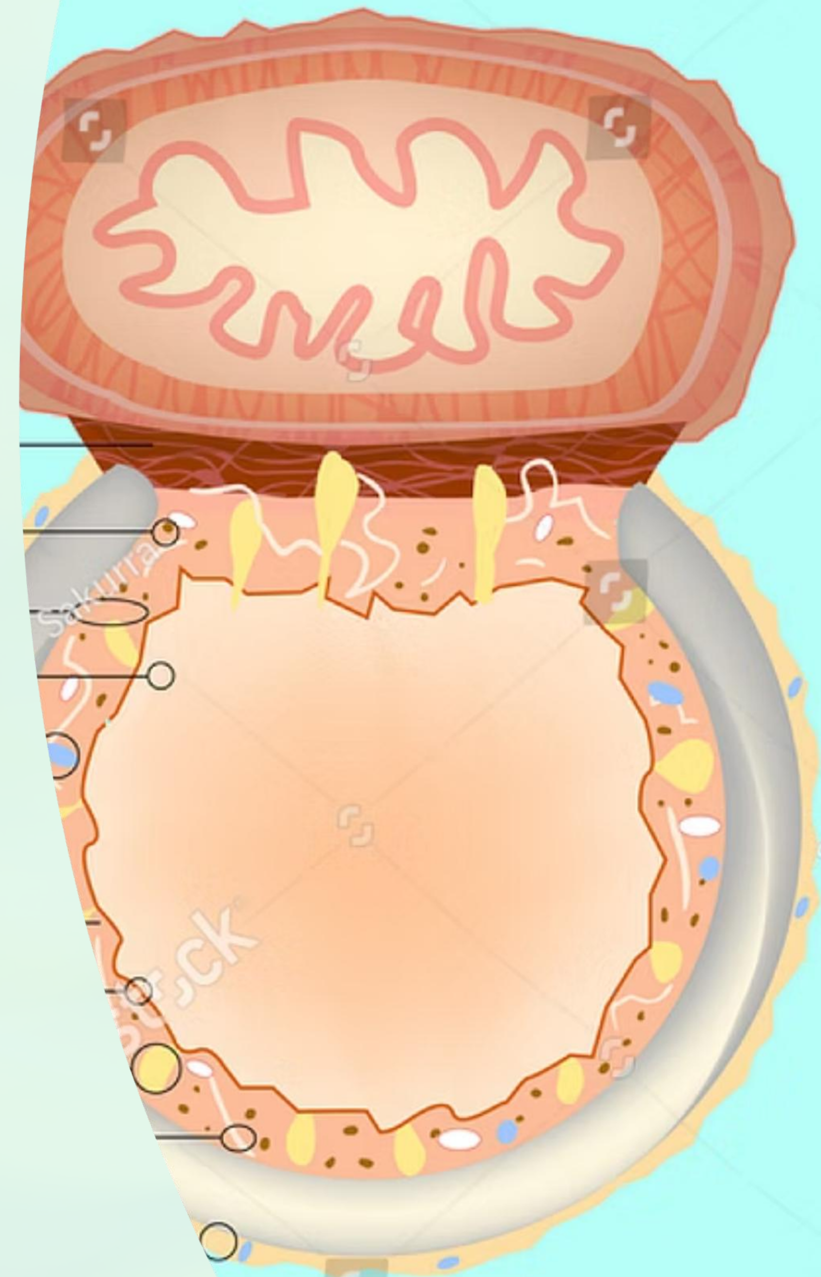
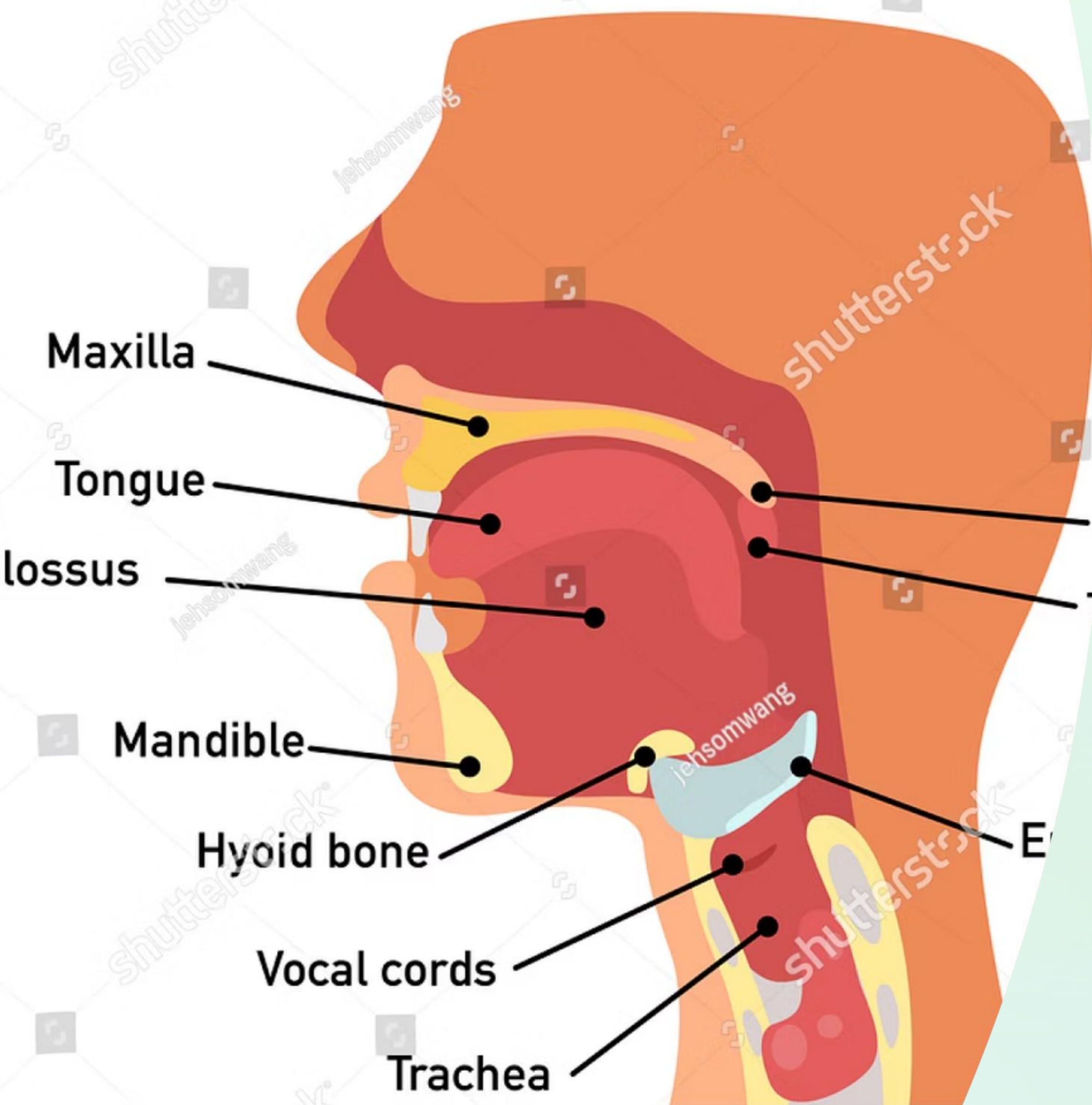
Esophageal Disorders

A comprehensive overview of esophageal pathophysiology — covering dysphagia, achalasia, pyrosis, GERD, and Barrett's esophagus. Designed for pathophysiology education with emphasis on etiology, pathomechanisms, and symptomatology.

PATHOPHYSIOLOGY MEDICAL EDUCATION





Dysphagia — Definition & Oropharyngeal Causes

What Is Dysphagia?

Dysphagia is the subjective sensation of difficulty swallowing. Patients may experience a feeling that food or fluid is "stopping" or failing to pass normally from the mouth to the stomach. Many patients are unaware they suffer from dysphagia. A psychogenic variant — phagophobia (fear of swallowing) — also exists.

Oropharyngeal Dysphagia — Neurological Causes

- Cerebrovascular stroke
- Multiple sclerosis (Sclerosis multiplex)
- Myasthenia gravis
- Parkinson's disease and parkinsonism
- Amyotrophic lateral sclerosis (ALS)

These conditions impair the neuromuscular coordination required to initiate the swallowing reflex, leading to oropharyngeal dysfunction.

Dysphagia — Esophageal Causes

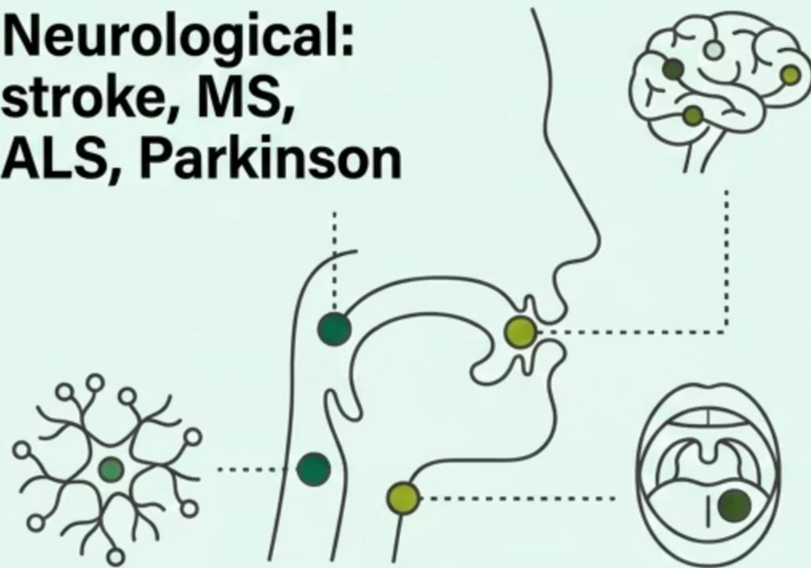
Oropharyngeal — Additional Causes

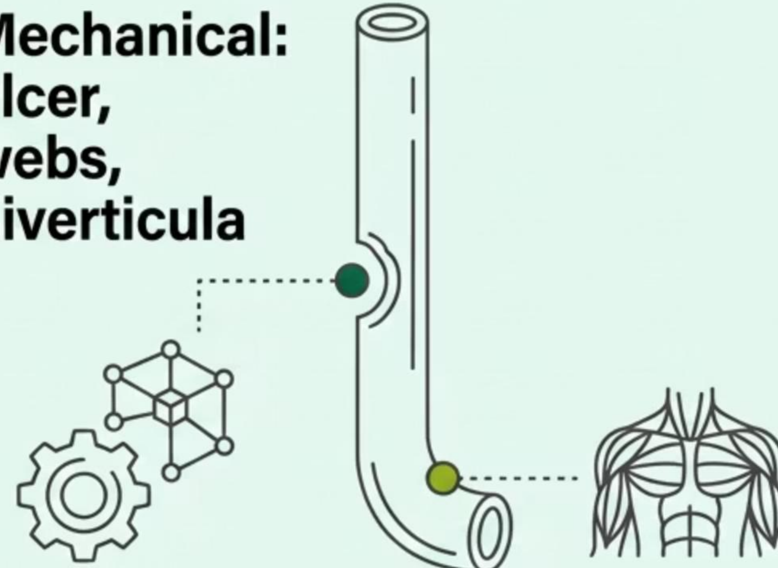
- Xerostomia (dry mouth — reduced lubrication)
- Radiation therapy to the head/neck
- Neck malignancies causing external compression
- Neurotoxins (e.g., snake venom)
- Eosinophilic esophagitis
- Pharyngitis

Esophageal Dysphagia

Mechanical causes: Peptic ulcer, esophagitis, esophageal or gastric cardia carcinoma, external compression, esophageal diverticula, esophageal webs, leiomyoma, systemic sclerosis.

Functional causes: Achalasia, myasthenia gravis, and other motility disorders that impair coordinated peristalsis without structural obstruction.





Odynophagia & Achalasia — Introduction

Odynophagia

Odynophagia is painful swallowing — distinct from dysphagia, which is difficulty without necessarily pain. It arises from inflammation, mucosal damage, or spasm anywhere along the esophageal lumen.

Causes of Odynophagia

- Motility disorders (spasm)
- Mechanical obstruction
- Infection (Candida, HSV, CMV)
- Gastroesophageal reflux
- Esophagitis (chemical, radiation, eosinophilic)

Achalasia — Overview

Also known as **cardial achalasia**, cardiospasm, or esophageal aperistalsis. Achalasia is a primary esophageal motility disorder affecting the smooth muscle layer and the **lower esophageal sphincter (LES)**.

It is characterized by **incomplete relaxation of the LES**, increased LES resting tone, and loss of coordinated peristalsis — in the absence of secondary causes such as malignancy or fibrosis.

Food, fluids, and saliva accumulate in the esophagus, creating a risk of **aspiration pneumonia**.

Achalasia — Etiology, Pathomechanism & Symptoms

Etiology

- **Idiopathic neuronal loss**

  Destruction of parasympathetic (inhibitory) ganglia in the myenteric plexus — loss of nitric oxide–releasing neurons that mediate LES relaxation.

- **Secondary causes**

  Esophageal carcinoma (pseudoachalasia), Chagas disease (*T. cruzi* destroys ganglia), Triple-A syndrome (rare autosomal recessive).

Pathomechanism

LES Failure

Food Retention

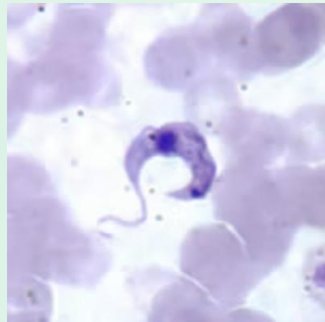
Symptoms

- Progressive dysphagia (solids and liquids equally)
- Regurgitation of undigested food
- Chest pain (especially early disease)
- Weight loss due to reduced oral intake
- Nocturnal cough / aspiration risk

Achalasia — Imaging & Histology

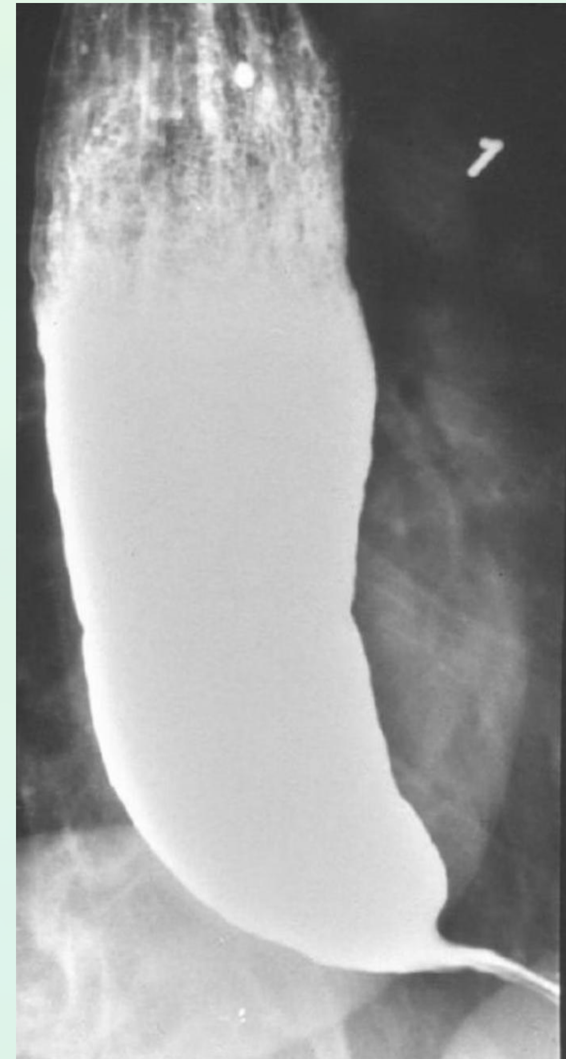
Radiological and histological findings are essential for confirming the diagnosis of achalasia. The classic barium swallow demonstrates a dilated esophageal body with a smooth, tapered narrowing at the gastroesophageal junction — the characteristic "bird beak" sign. Chest radiograph may reveal a widened mediastinum or absent gastric air bubble.

Chagas Disease — Histology



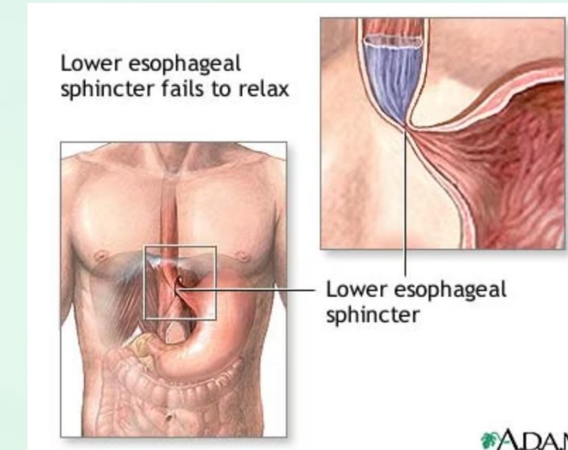
Trypanosoma cruzi amastigotes in tissue — responsible for ganglionic destruction in Chagas-related achalasia.

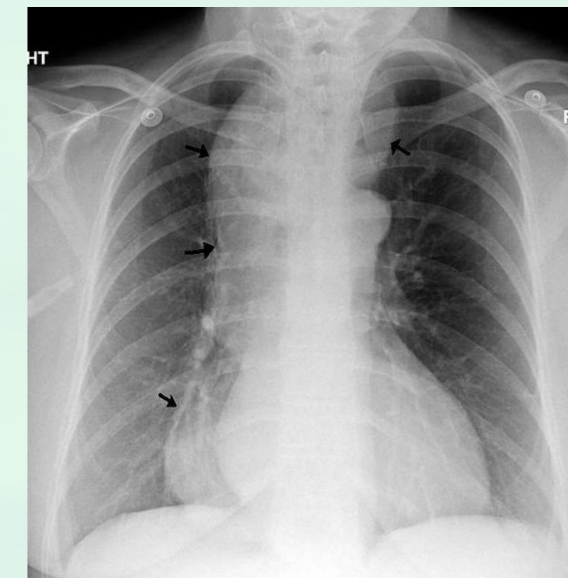
Barium Swallow — "Bird Beak" Sign



Barium esophagram showing massive esophageal dilation with tapered distal narrowing — classic for advanced achalasia.

LES Anatomy & Chest X-Ray





Top: LES anatomy. Bottom: Chest radiograph showing mediastinal widening and esophageal food/fluid retention (arrows).

Pyrosis — Definition, Causes & Pathomechanism

Definition

Pyrosis (heartburn) is a retrosternal burning sensation that can radiate to the neck, throat, or mandible. It is one of the most common symptoms in gastroenterology. A distinction is made between GERD-related pyrosis and functional burning, the latter diagnosed by Rome III criteria when no structural or motility cause is identified.

Rome III Criteria for Functional Burning

1. Burning sensation behind the sternum
2. Absence of another cause of gastroesophageal reflux
3. No evidence of esophageal motility disorders

Causes

GERD

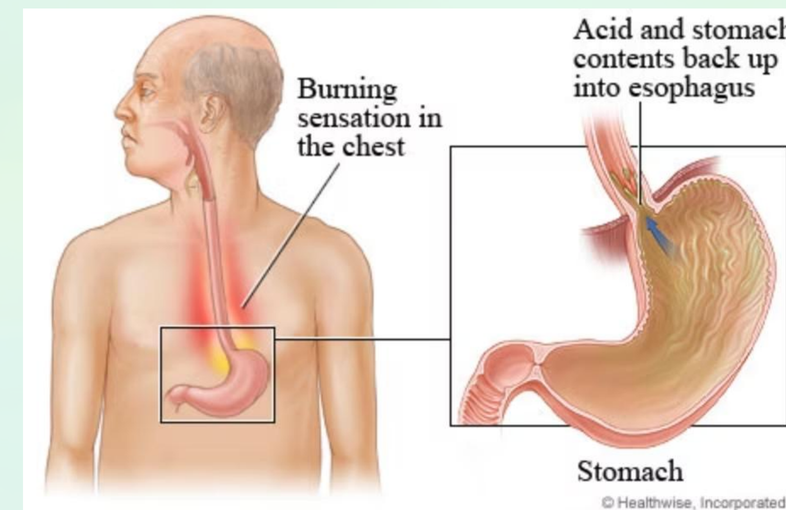
Most common cause — retrograde flow of acidic gastric content into the esophagus, causing mucosal irritation.

Functional Burning

Associated with functional GI disorders (e.g., irritable bowel syndrome) with visceral hypersensitivity — no acid exposure identifiable.

Esophageal Hypersensitivity

Lowered pain threshold in the esophageal mucosa, even with physiological acid exposure levels.



Acid and stomach contents backing up into the esophagus — the primary mechanism of heartburn.

Pyrosis — Symptomatology & Triggers

Clinical Presentation

- Burning sensation behind the sternum ("heartburn")
- Occurs after meals or during the night (supine position)
- Worsens when lying down or bending forward
- Frequently reported in pregnancy (due to LES pressure changes and progesterone effect)
- May radiate to neck, throat, or jaw

Dietary & Lifestyle Triggers

Large Meals

Increased gastric distension raises intragastric pressure, promoting LES relaxation and reflux.

Spicy Foods

Capsaicin and other compounds directly irritate esophageal mucosa and stimulate acid secretion.

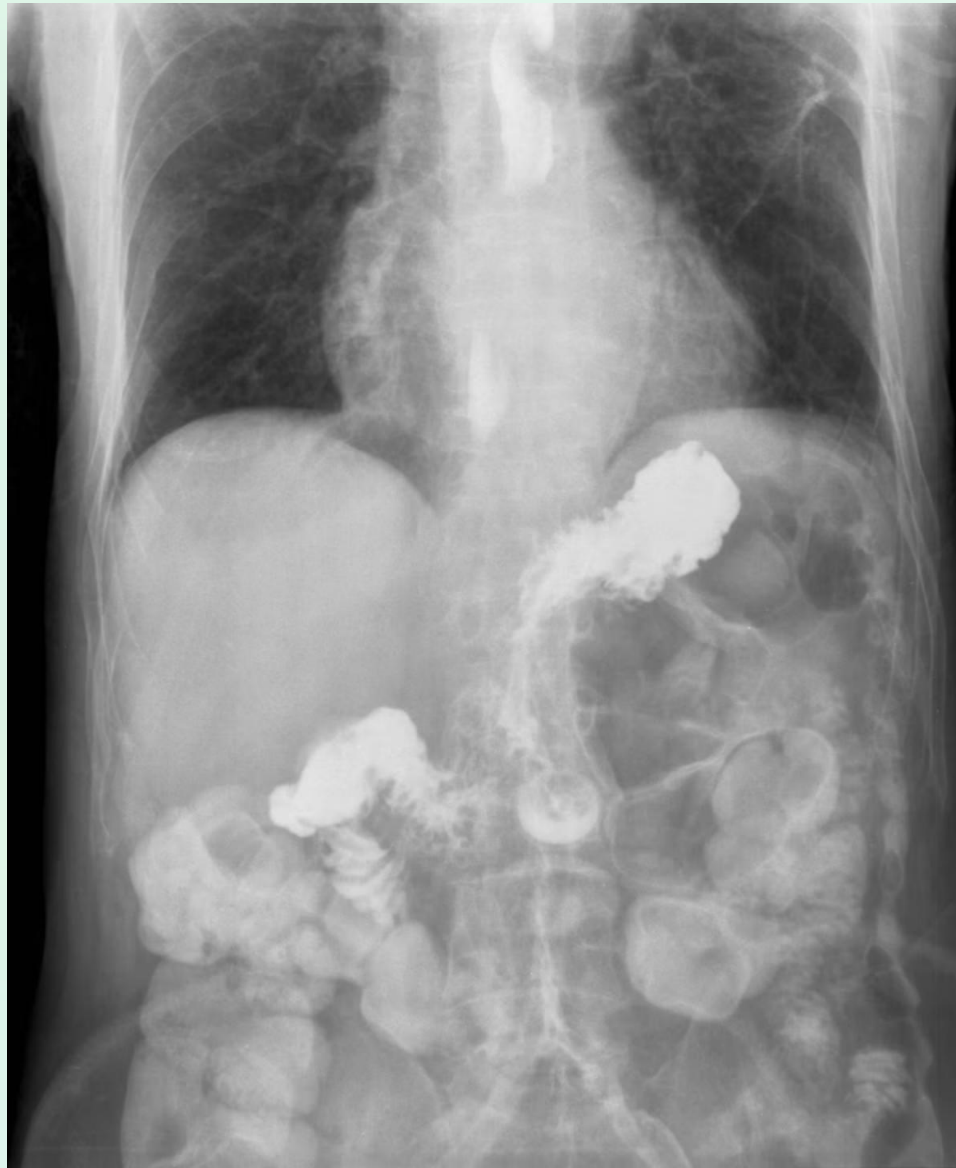
High-Fat Foods

Fat delays gastric emptying and decreases LES tone via cholecystokinin release.

Alcohol & Caffeine

Both decrease LES tone and increase gastric acid secretion, exacerbating reflux symptoms.

GERD — Definition, Etiology & Pathomechanism



Barium contrast study demonstrating gastroesophageal reflux — retrograde flow of barium from stomach into the esophagus.

Definitions

Gastroesophageal Reflux (GER): Non-voluntary movement of gastric (sometimes duodenal) content into the esophagus — a *normal physiological process* occurring 1–4×/hour in the 3 hours after a meal.

GERD: Chronic esophageal mucosal damage resulting from pathological GER — defined by frequency, duration, and associated complications.

Key Etiological Factors

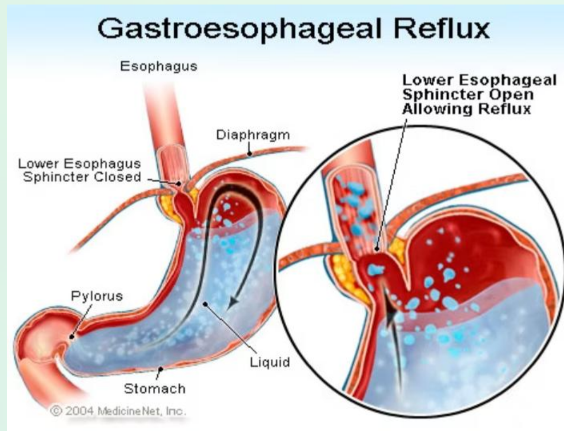
- Abnormal LES relaxation: Triggered by fat, chocolate, onion, garlic, alcohol, peppermint, smoking
- Hiatal hernia: Displacement of the gastric cardia into the thorax through the esophageal hiatus, altering LES position and tone
- Transient vs. permanent LES dysfunction

Protective Mechanisms (When Intact)

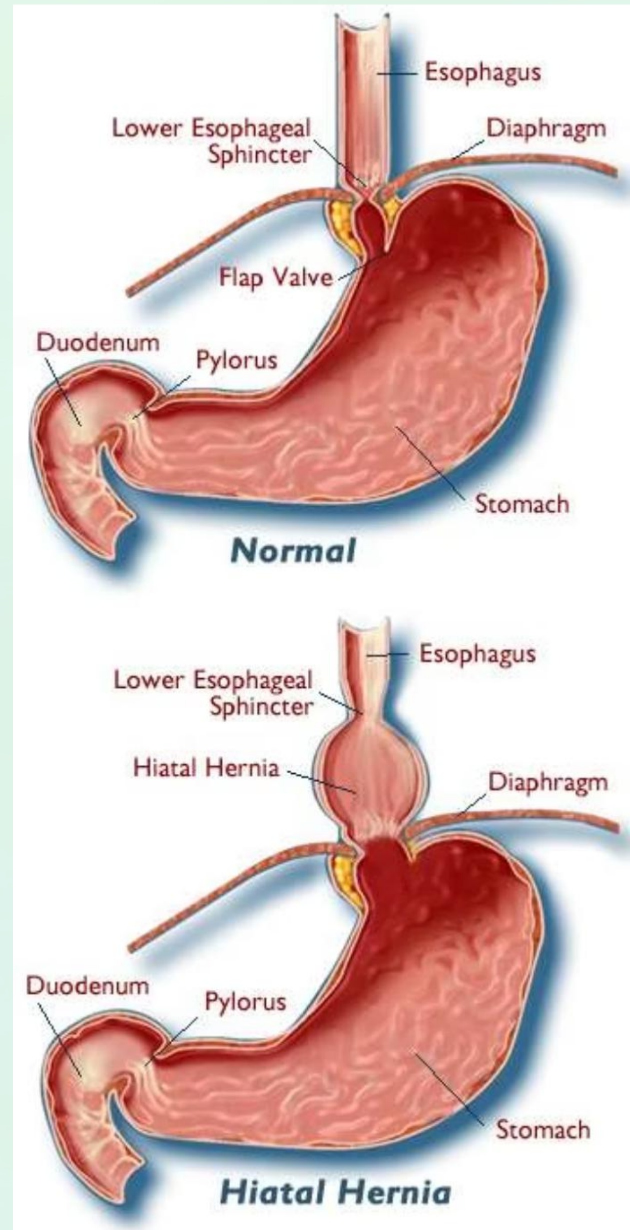
- Tonic LES contraction
- Esophageal peristalsis (acid clearance)
- Salivary bicarbonate neutralization

GERD — Anatomy & Hiatal Hernia Types

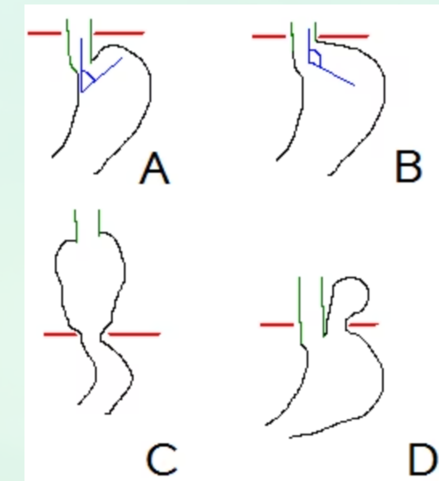
Understanding the anatomical basis of GERD is essential. The lower esophageal sphincter, diaphragmatic crura, and the His angle all contribute to the anti-reflux barrier. Disruption of any of these — particularly via hiatal hernia — predisposes to pathological reflux.



Mechanism of reflux: LES opens allowing gastric acid and liquid to back up into the esophagus.

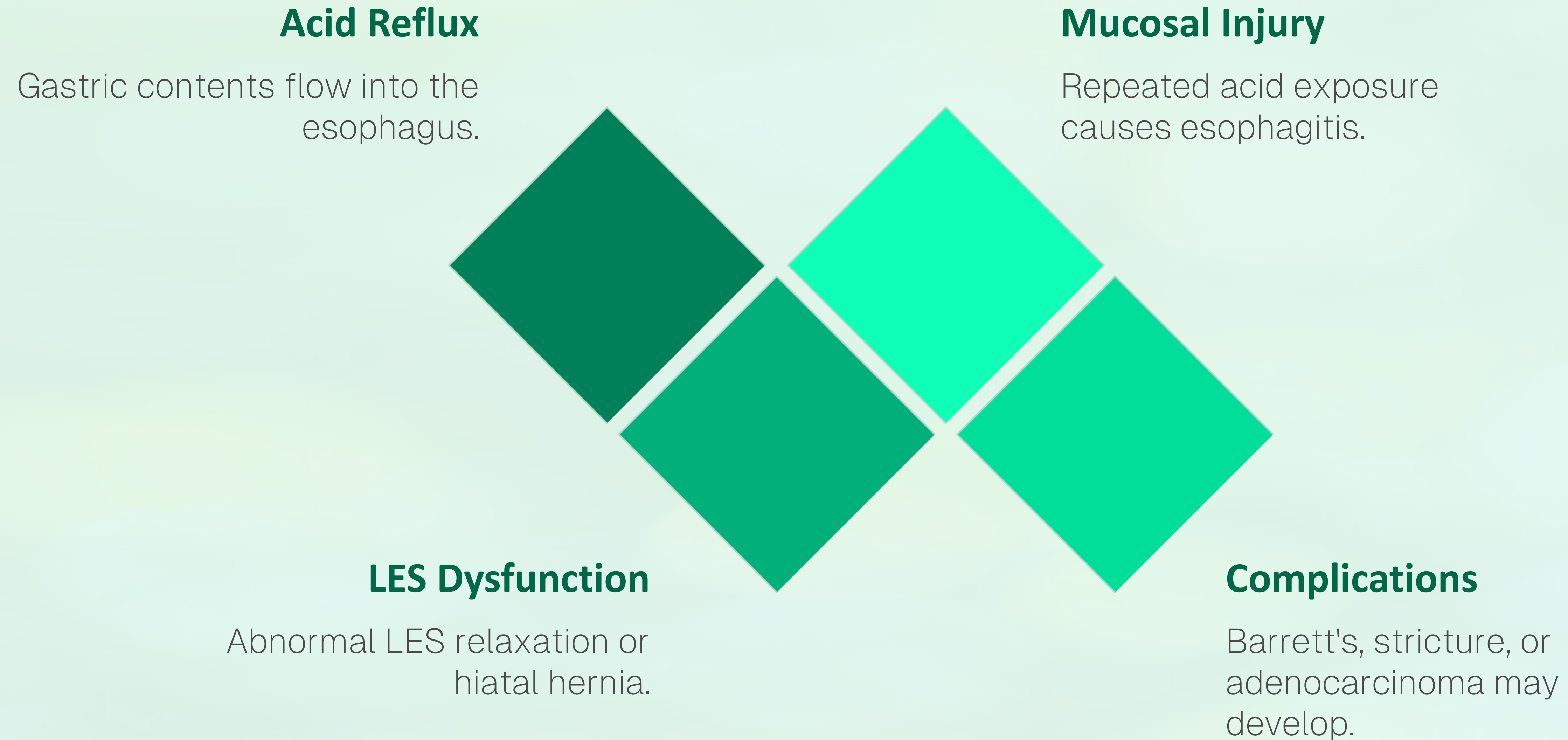


Normal anatomy vs. sliding hiatal hernia — note loss of LES position below the diaphragm.



A – Normal anatomy. B – Pre-hiatal hernia state. C – Sliding hiatal hernia (Type I, most common). D – Paraesophageal type (Type II).

GERD — Pathomechanism



GERD — Symptomatology

Cardinal Symptoms

- Pyrosis (heartburn) — retrosternal burning
- Regurgitation of acidic or bitter fluid
- Dysphagia and odynophagia
- Hypersalivation (water brash)
- Nausea, occasional vomiting

Extraesophageal Symptoms

- Chronic cough (microaspiration)
- Laryngitis, hoarseness, pharyngitis
- Bronchial asthma exacerbation

Oral Manifestations

- Dental enamel erosion (acid contact)
- Tooth hypersensitivity
- Halitosis

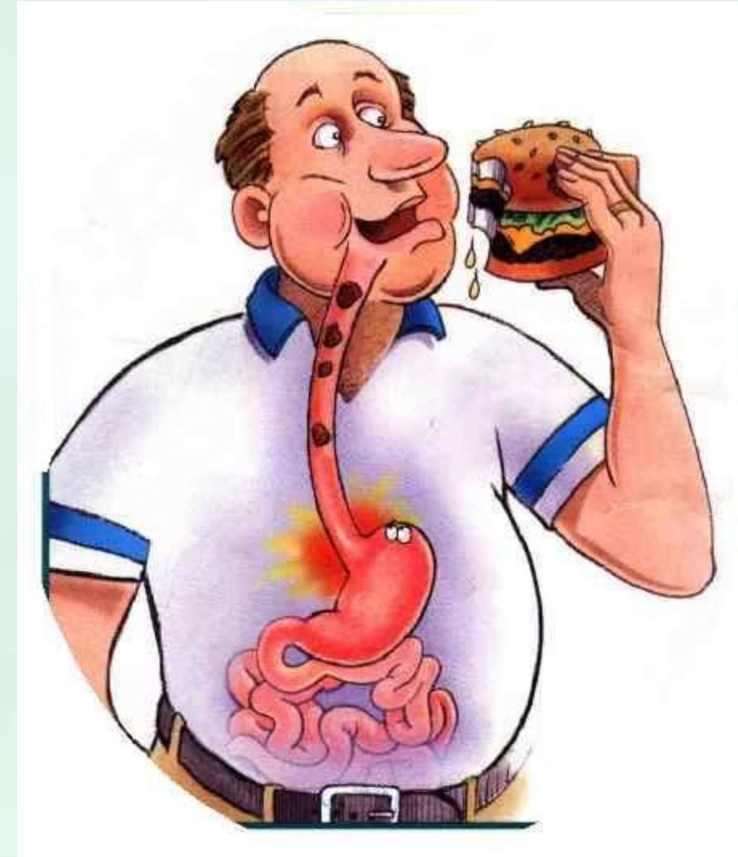
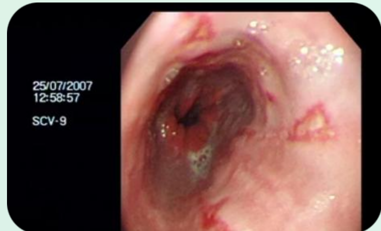


Illustration of acid reflux reaching the esophagus and oral cavity — explaining both GI and dental symptomatology.

⚠ Extraesophageal symptoms (cough, asthma, laryngitis) may be the **sole presenting complaint** in up to 30% of GERD patients — requiring a high index of suspicion.

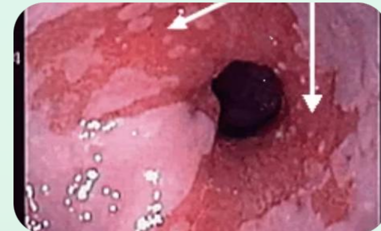
GERD — Complications

Untreated or undertreated GERD can progress through a well-defined sequence of complications, driven by repeated mucosal acid exposure and chronic inflammation.



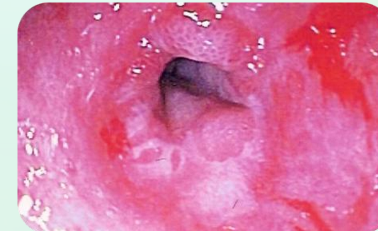
Reflux Esophagitis

Erosions and ulcerations of the distal esophageal mucosa. Graded by Los Angeles classification (A–D). Causes pain, bleeding, and dysphagia.



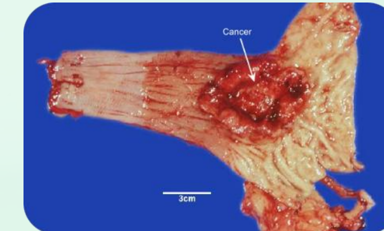
Barrett's Esophagus

Intestinal metaplasia — replacement of squamous epithelium by columnar (goblet cell) epithelium. A premalignant condition requiring endoscopic surveillance.



Esophageal Stricture

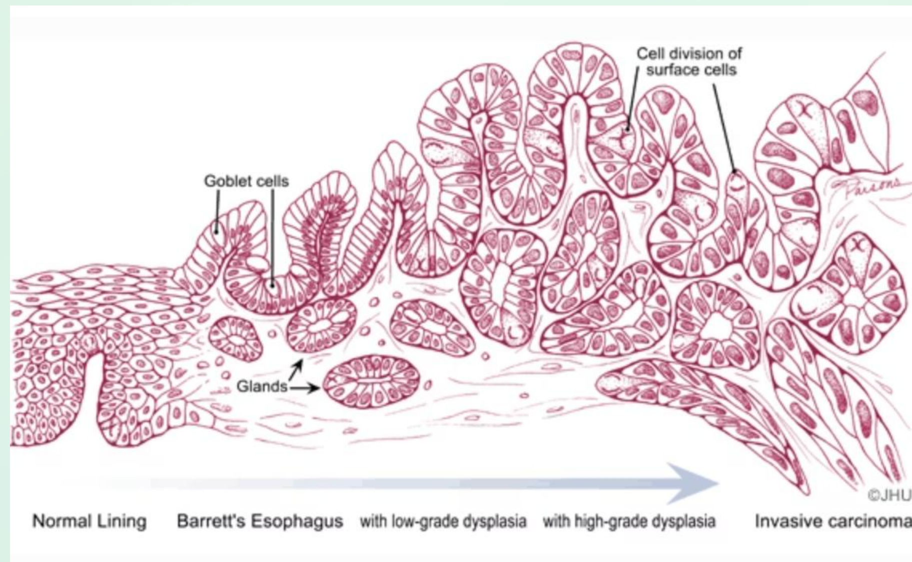
Fibrous narrowing from repeated mucosal injury and healing. Results in progressive solid-food dysphagia. Requires endoscopic dilation.



Esophageal Adenocarcinoma

The most feared complication — arises from Barrett's metaplasia via dysplasia. Incidence rising in Western countries. Poor prognosis if detected late.

Barrett's Esophagus — Pathomechanism & Endoscopy



Endoscopic view of Barrett's esophagus — characteristic salmon-colored columnar mucosa extending proximally from the gastroesophageal junction, replacing normal pale squamous epithelium.

Pathomechanism of Barrett's

Chronic acid (and bile) exposure triggers **epigenetic reprogramming** of basal esophageal stem cells. The squamous epithelium undergoes **intestinal metaplasia** — a defensive adaptation that, paradoxically, confers malignant potential. Goblet cells (hallmark of intestinal metaplasia) are required for diagnosis. Progression: **Metaplasia → Low-grade dysplasia → High-grade dysplasia → Adenocarcinoma.**

Risk Factors & Surveillance

- Long-standing GERD (>5 years)
- Male sex, age >50, obesity, smoking
- White ethnicity
- Hiatal hernia

Endoscopic surveillance with biopsy (Seattle protocol) every 3–5 years for non-dysplastic Barrett's; more frequent for dysplastic cases. Goal: early detection before invasive carcinoma develops.

GERD — Diagnosis & Investigations

Upper Endoscopy

24-hr pH-Impedance

Manometry

GERD — Treatment Principles

1

Lifestyle Modification

Weight loss, dietary changes (avoid triggers), elevate head of bed, small meals, avoid recumbency post-meal, smoking cessation.

2

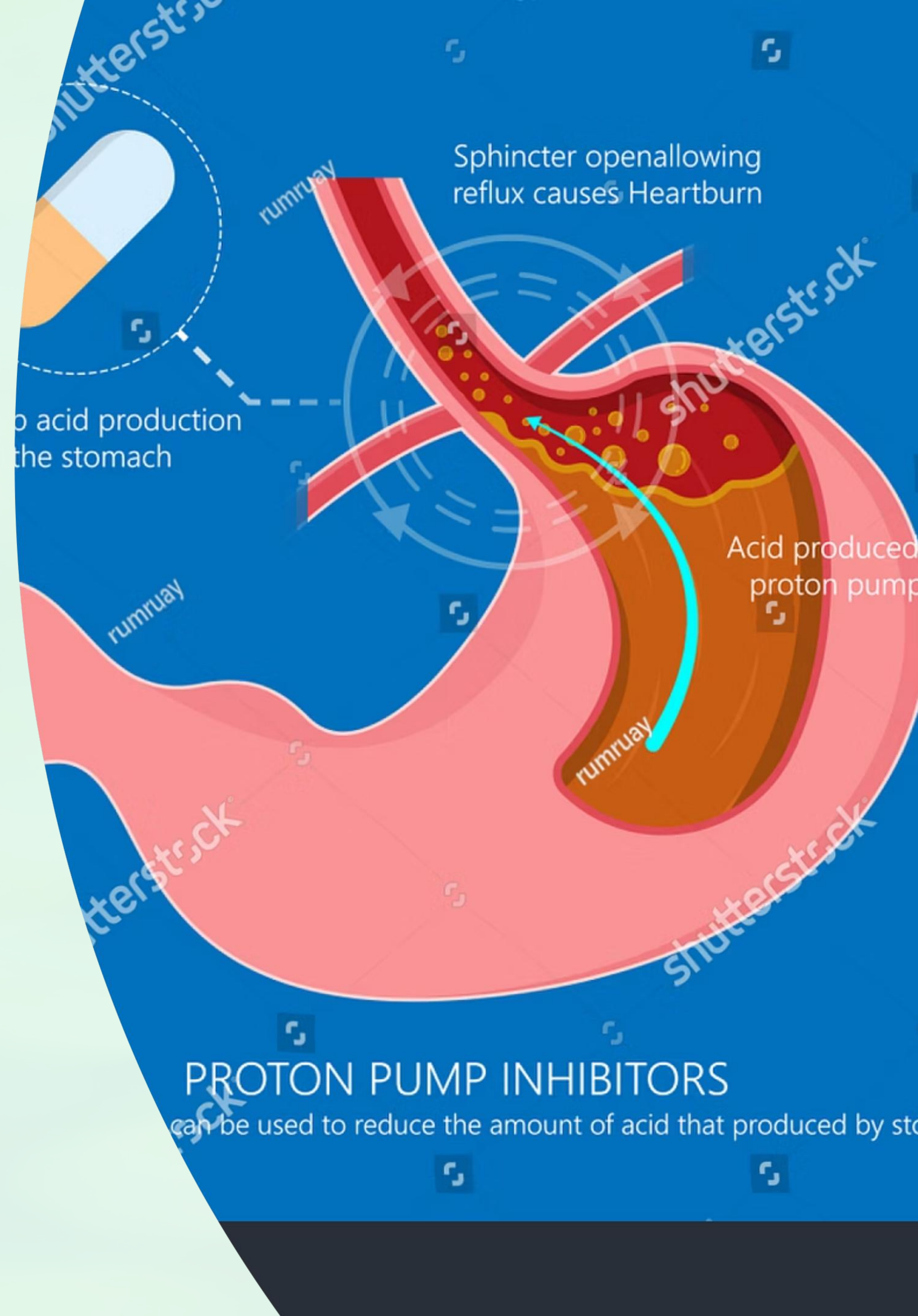
Pharmacological Therapy

PPIs (first-line), H2-receptor antagonists (adjunct), antacids (symptom relief), prokinetics (delayed gastric emptying). PPIs heal esophagitis in 80–90%.

3

Endoscopic & Surgical

Nissen fundoplication (laparoscopic) for refractory GERD or large hiatal hernia. Endoscopic ablation (radiofrequency/APC) for Barrett's dysplasia.

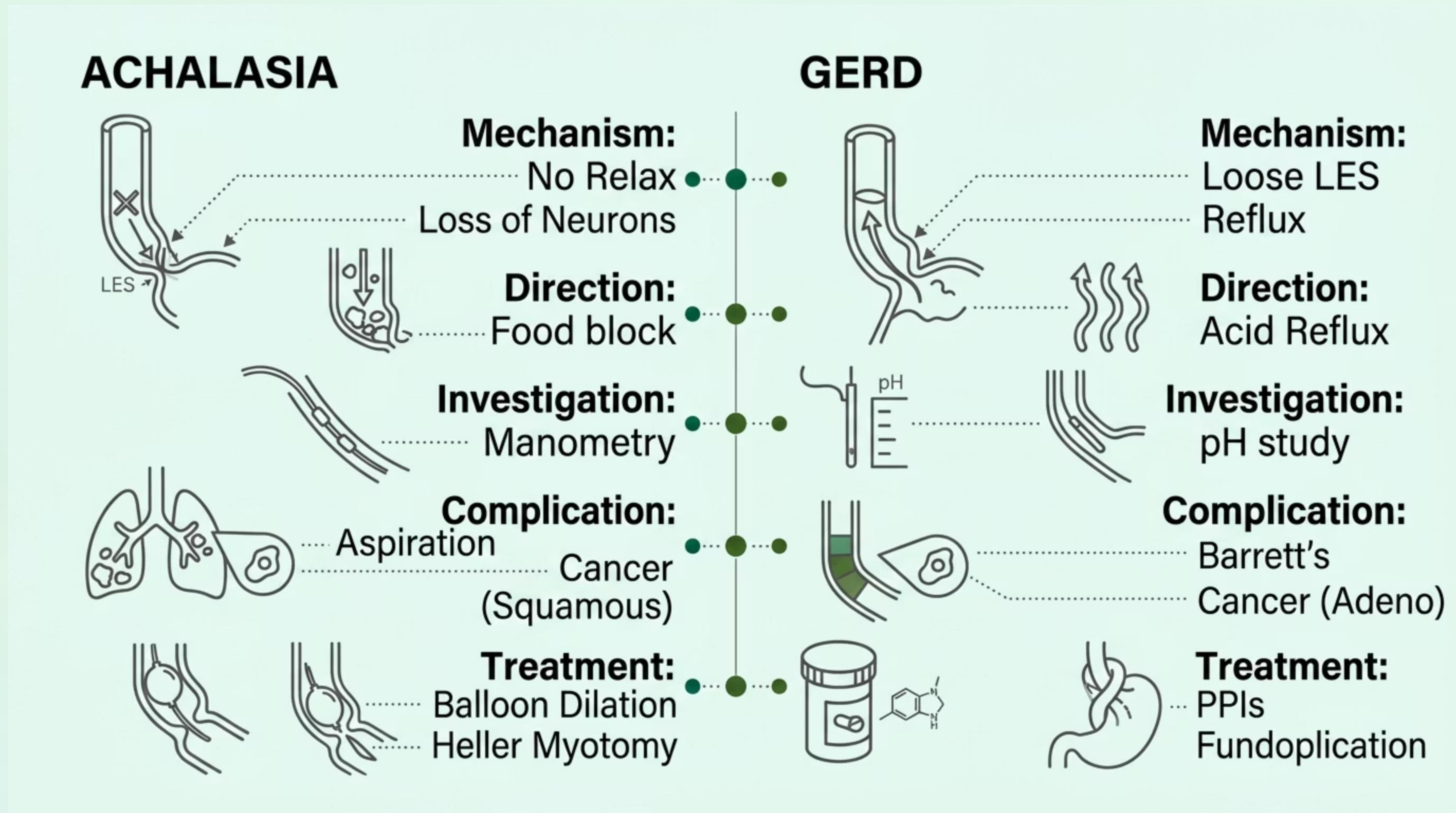


Esophageal Disorders — Comparative Overview

A side-by-side comparison of the major esophageal disorders covered in this presentation, highlighting key distinguishing features for clinical differentiation.

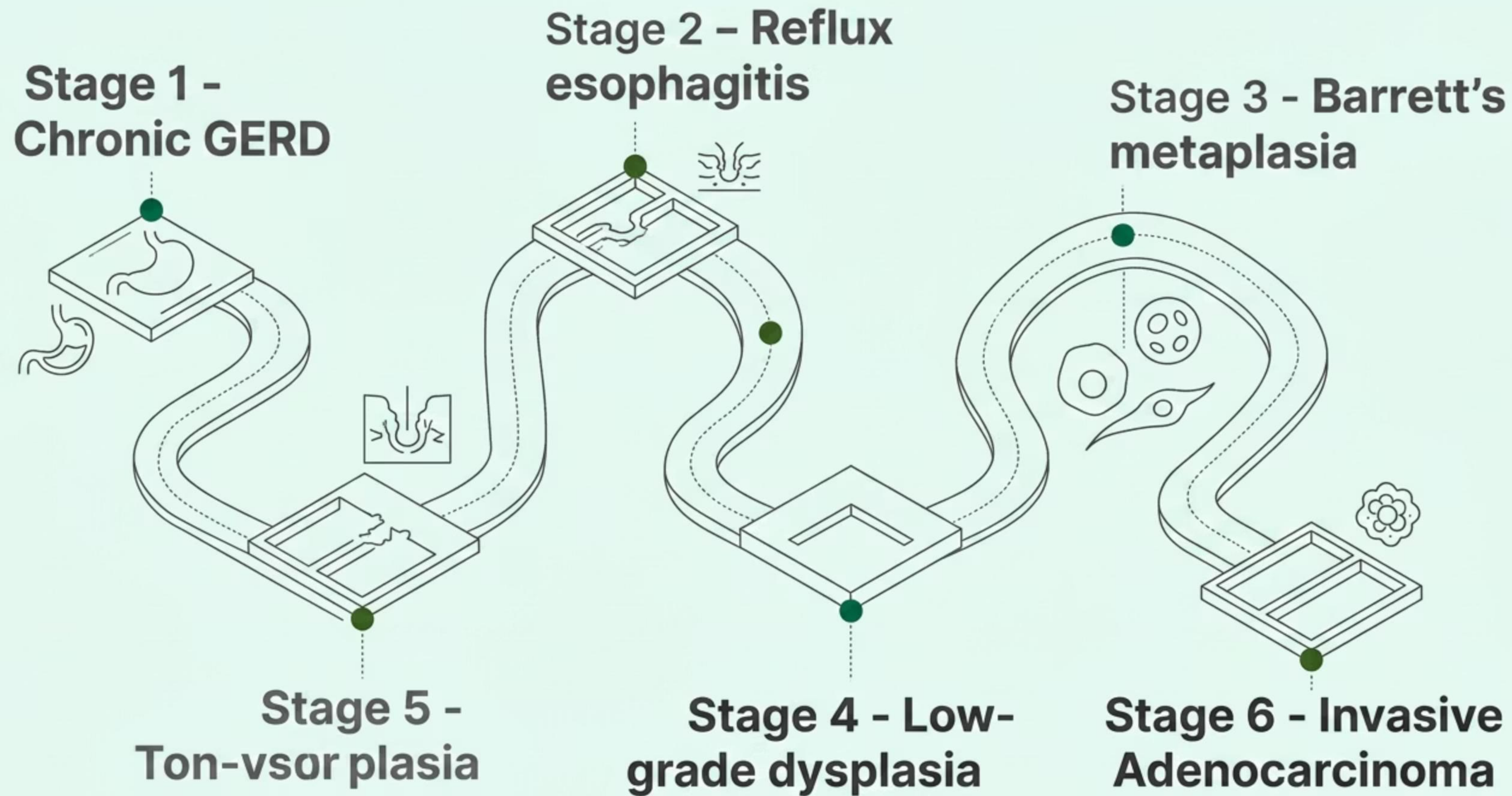
Condition	Core Mechanism	Key Symptom	Diagnosis	Main Complication
Oropharyngeal Dysphagia	Neuromuscular failure of swallowing initiation	Difficulty initiating swallow, coughing/choking	Videofluoroscopy	Aspiration pneumonia
Esophageal Dysphagia	Mechanical obstruction or motility disorder	"Food sticking" in chest	Endoscopy, manometry	Malnutrition, aspiration
Achalasia	Loss of inhibitory myenteric neurons → LES fails to relax	Dysphagia to solids AND liquids	Manometry (type I–III), barium swallow	Aspiration, esophageal carcinoma
Pyrosis / GERD	LES dysfunction → acid reflux → mucosal injury	Heartburn, regurgitation	Clinical ± endoscopy ± pH monitoring	Barrett's esophagus, adenocarcinoma
Barrett's Esophagus	Intestinal metaplasia from chronic acid exposure	Often asymptomatic (GERD symptoms)	Endoscopy + biopsy	Esophageal adenocarcinoma

Achalasia vs. GERD — Pathophysiology Comparison



Though both conditions affect the lower esophageal sphincter, their pathophysiology is diametrically opposed: achalasia results from **failure of LES relaxation** (hypertonia), while GERD results from **excessive or inappropriate LES relaxation** (hypotonia). This fundamental distinction drives all diagnostic and therapeutic decisions.

Esophageal Malignancy — Risk Pathway



Key Risk Amplifiers

- Duration and severity of GERD symptoms
- Obesity (increased intra-abdominal pressure)
- Male sex and age >50
- Smoking (impairs mucosal defense)
- Absence of *H. pylori* (paradoxically protective)

Interception Points

Each stage of the GERD-to-cancer progression represents an opportunity for clinical intervention: aggressive PPI therapy at Stage 1–2, endoscopic surveillance at Stage 3, endoscopic mucosal resection or ablation at Stage 4–5, and surgical resection at Stage 6. Early detection dramatically improves prognosis.

Key Takeaways

Dysphagia

Classify first: oropharyngeal (neurological) vs. esophageal (mechanical/functional). Etiology guides workup and treatment.

Achalasia

Loss of inhibitory myenteric neurons → LES fails to relax. Dysphagia to solids AND liquids. "Bird beak" on barium swallow. Confirmed by manometry.

Pyrosis & GERD

LES dysfunction allows acid reflux. Symptoms: heartburn, regurgitation, cough. PPIs are first-line. Alarm symptoms require urgent endoscopy.

Barrett's & Cancer

Chronic GERD → intestinal metaplasia → dysplasia → adenocarcinoma. A preventable progression with surveillance and timely intervention.

Understanding the **pathomechanism** of each condition — not just its symptoms — is the foundation of rational diagnosis and treatment in esophageal pathophysiology.