

Esophageal Obstruction

Structural and functional barriers to swallowing

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Learning objectives

- Distinguish physical from functional esophageal obstruction.
- Explain characteristic dysmotility patterns and related diverticula.
- Describe strictures, webs, rings, and achalasia through morphology and clinical consequences.
- Use dysphagia patterns to frame a focused differential.

The esophagus is a transit organ

- Carries swallowed solid food and fluids to the stomach.
- Functions as a distensible muscular conduit rather than a storage organ.
- Effective transit requires a patent lumen and coordinated muscle activity.

Two mechanisms prevent bolus transit

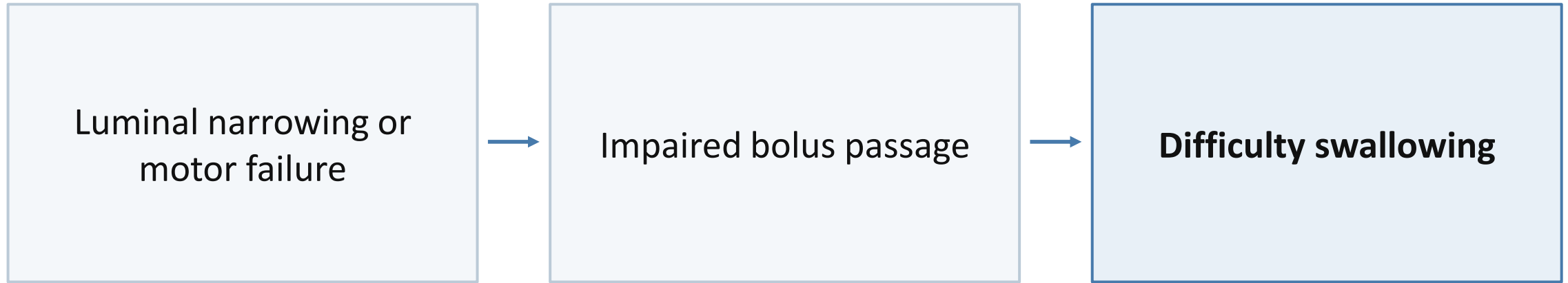
Physical obstruction

- A structural lesion reduces usable lumen.
- The wall may thicken or the lumen may be constricted.
- Examples: Stricture, mucosal web, esophageal ring.

Functional obstruction

- The lumen may be anatomically patent.
- Uncoordinated contraction or poor sphincter relaxation impedes transit.
- Examples: Esophageal spasm and achalasia.

From obstruction to dysphagia



The mechanism—not the symptom alone—determines whether the barrier is structural or functional.

Dysphagia must be interpreted mechanistically

- Dysphagia denotes impaired passage of swallowed material.
- Progressive difficulty with solids suggests a narrowing lesion.
- Motor dysfunction may cause obstructive symptoms without fixed stenosis.
- History guides mechanism; Structural and functional causes must remain distinct.

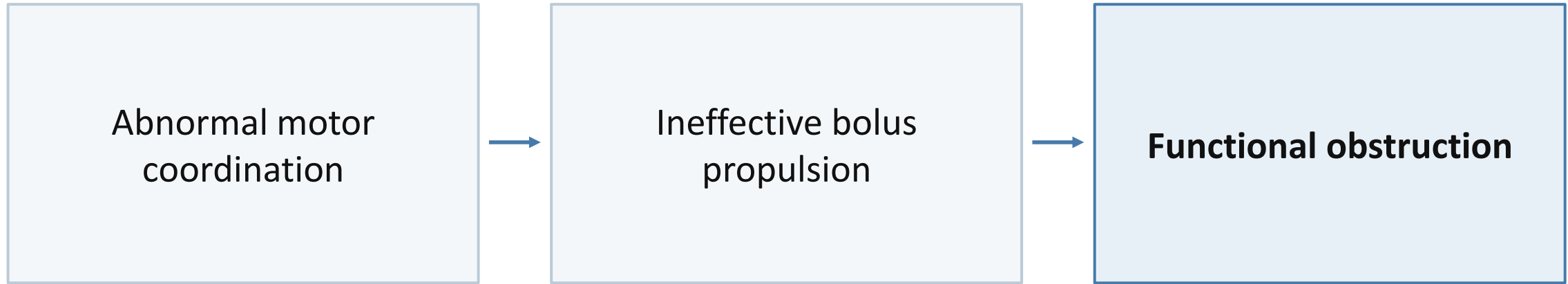
At the bedside: Key distinctions

Clue	Structural narrowing	Motility-related dysfunction
Anatomic lumen	Constricted by a lesion	May remain patent
Principal defect	Bolus cannot fit through	Bolus is not properly propelled
Illustrative lesions	Stricture, ring, web	Spasm, LES dysfunction
Diagnostic emphasis	Define narrowed segment	Assess contraction and relaxation

Normal motility coordinates transit

- Propulsive peristalsis follows swallowing.
- Circular and longitudinal smooth-muscle actions must be coordinated.
- Relaxation of the lower esophageal sphincter allows entry into the stomach.
- Breakdown of coordination can obstruct transit without an intraluminal mass.

Functional obstruction: The causal chain



Disordered timing, force, or sphincter relaxation can each prevent normal transit.

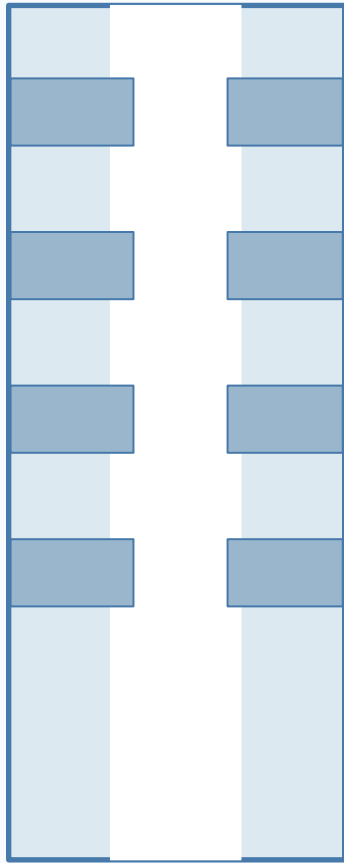
Three patterns of esophageal dysmotility

Pattern	Primary abnormality
Nutcracker esophagus	Intense high-amplitude, uncoordinated contractions
Diffuse esophageal spasm	Repetitive simultaneous distal contractions
Lower esophageal sphincter dysfunction	High resting pressure and/or incomplete relaxation

Nutcracker esophagus: Excessive contractile force

- Produces a functional obstruction from intense contractions.
- Contractions involve inner circular and outer longitudinal smooth muscle.
- The motor activity is high amplitude and uncoordinated.
- Strength of contraction does not ensure effective forward transit.

Nutcracker pattern: Contraction versus passage



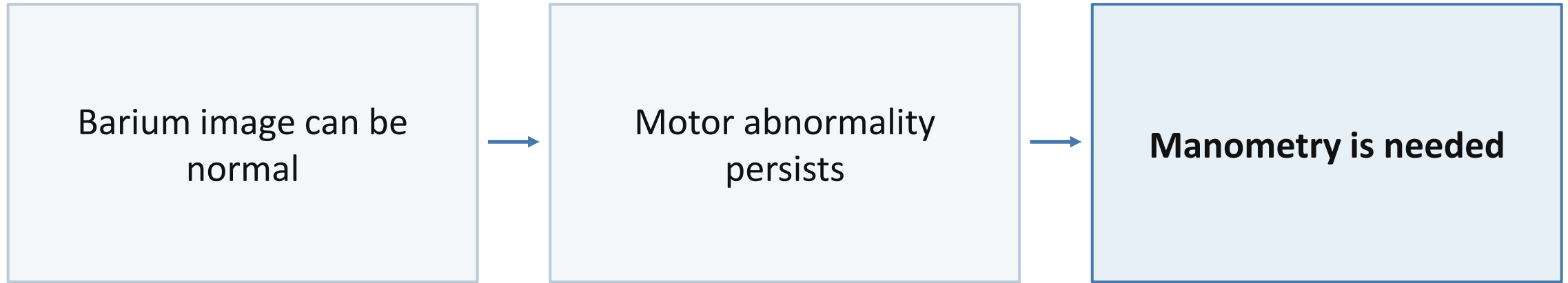
Repeated high-force contractions

- Marked contractions at more than one level
- Segments may narrow while adjacent regions distend
- Appearance is schematic, not a patient barium study

Nutcracker esophagus on barium swallow

- A contrast study may appear normal.
- Alternatively it may show a nutcracker-like pattern of narrow and distended regions.
- A normal barium examination does not exclude the contractile disorder.
- The text requires manometry for diagnosis.

Why manometry matters in nutcracker esophagus

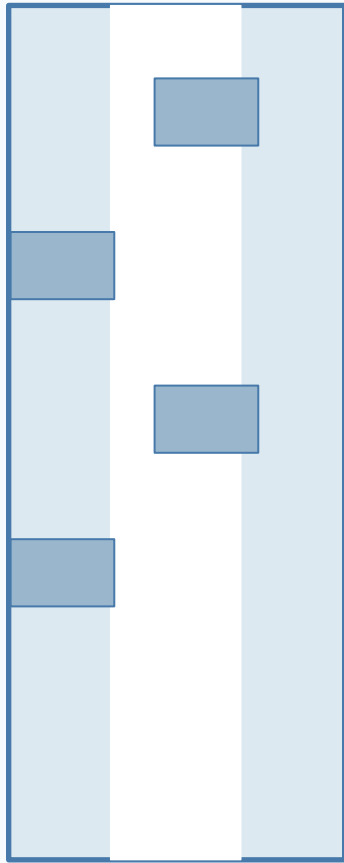


Manometry assesses motor behavior rather than relying on the outline of the contrast column.

Diffuse esophageal spasm: Disordered timing

- Repetitive contractions occur simultaneously rather than as an orderly propulsive wave.
- The distal esophageal smooth muscle is involved.
- description, amplitudes are normal.
- The key abnormality is coordination, not excessive force.

Diffuse spasm: The corkscrew appearance



Alternating simultaneous constrictions

- Simultaneous distal contractions distort the contour
- Repetitive narrowing produces the corkscrew descriptor
- Schematic rendering of the barium-swallow concept

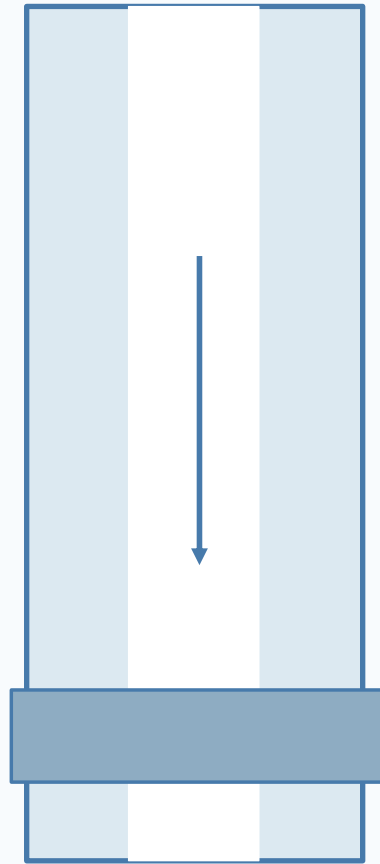
Nutcracker esophagus versus diffuse spasm

Property	Nutcracker esophagus	Diffuse esophageal spasm
Contraction amplitude	High	Normal in Robbins description
Coordination	Uncoordinated	Simultaneous/repetitive
Barium descriptor	Nutcracker-like or normal	Corkscrew
Diagnostic emphasis	Manometry required	Distal motor pattern

The lower esophageal sphincter is a gate

- Relaxation permits bolus entry into the stomach.
- Persistently high resting pressure may resist passage.
- Incomplete relaxation can create a functional bottleneck.
- Abnormal LES behavior may be isolated or part of a broader disorder.

Incomplete sphincter relaxation creates a bottleneck



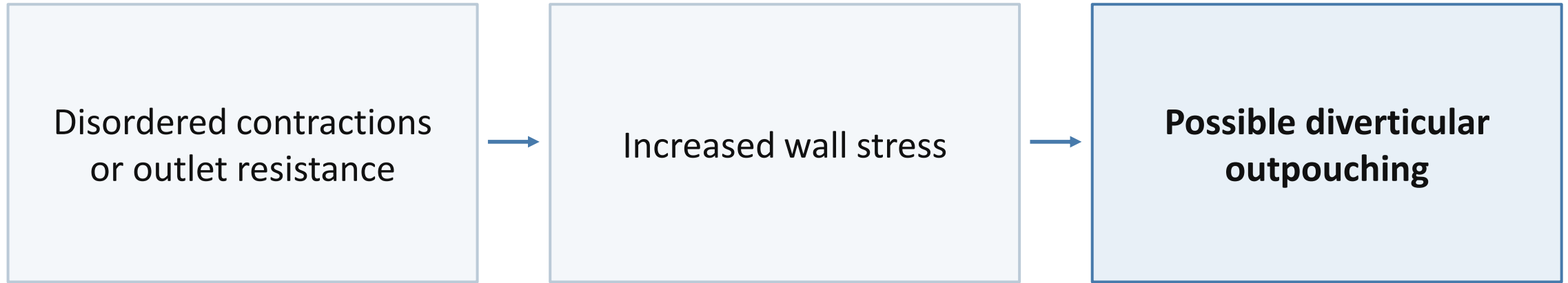
Distal high-tone sphincter

- Approaching bolus meets a poorly relaxing distal outlet
- Obstruction is physiologic rather than an intrinsic luminal mass
- Schematic illustrating the functional gate

Sphincter dysfunction need not occur alone

- High resting LES pressure may be an isolated anomaly.
- Incomplete LES relaxation may be isolated.
- Either may accompany nutcracker esophagus or diffuse esophageal spasm.
- These patterns may overlap clinically, so terminology requires care.

Dysmotility increases esophageal wall stress

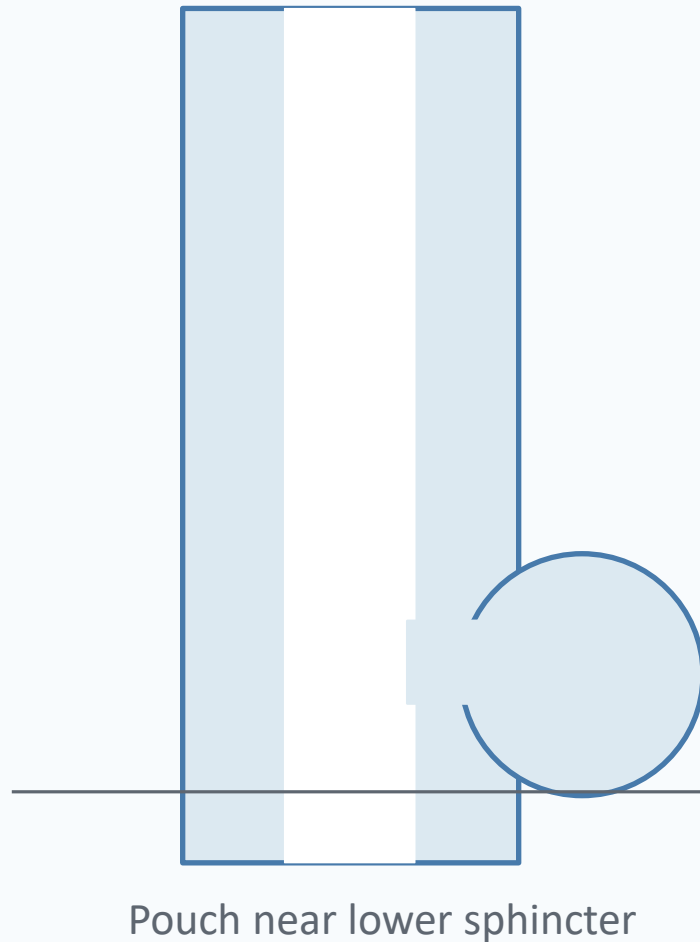


Two sites highlighted by Robbins are the epiphrenic esophagus and the pharyngoesophageal junction.

Epiphrenic diverticula arise near the LES

- Small diverticula can form with esophageal dysmotility.
- They typically occur immediately above the lower esophageal sphincter.
- Increased wall stress provides the mechanistic link.
- The diverticulum is therefore a possible consequence of the motor abnormality.

Epiphrenic diverticulum: Site and mechanism

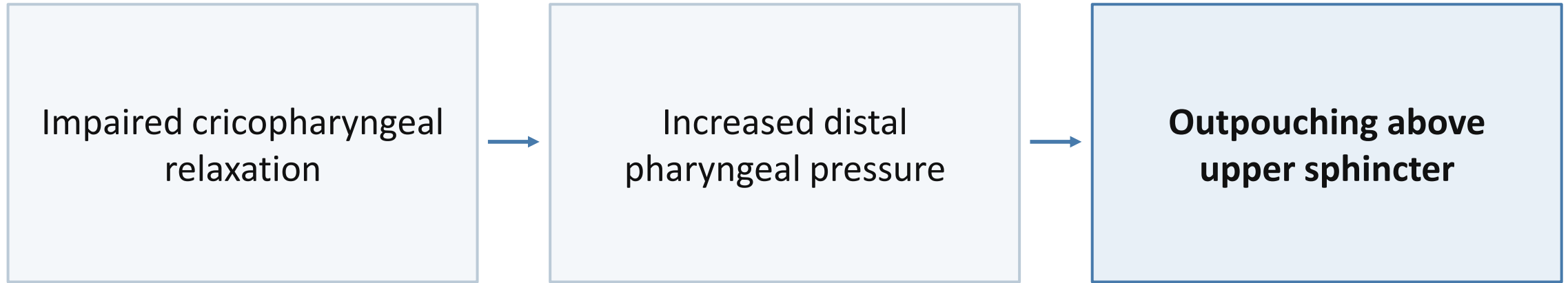


- Diverticular outpouching is immediately proximal to LES
- Raised wall stress follows dysmotility
- Site is different from the pharyngoesophageal (Zenker) region

Cricopharyngeal dysfunction raises pharyngeal pressure

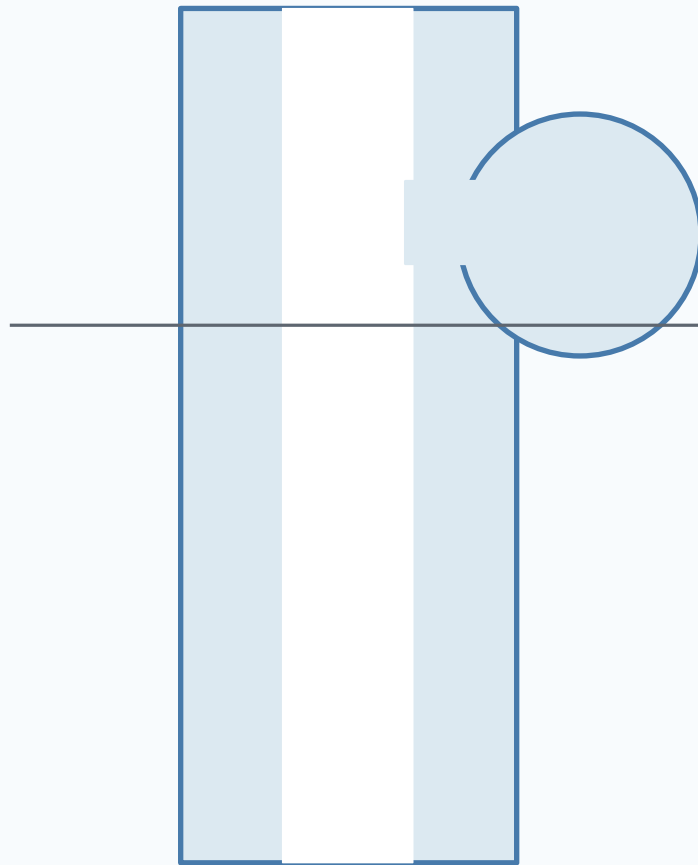
- Impaired relaxation may follow swallowing.
- Cricopharyngeal spasm also resists passage.
- Pressure rises within the distal pharynx.
- The consequence can be a Zenker (pharyngoesophageal) diverticulum.

Zenker diverticulum: Mechanistic sequence



This is a pressure-related pharyngoesophageal diverticulum, anatomically distinct from the epiphrenic type.

Zenker diverticulum lies above the upper sphincter



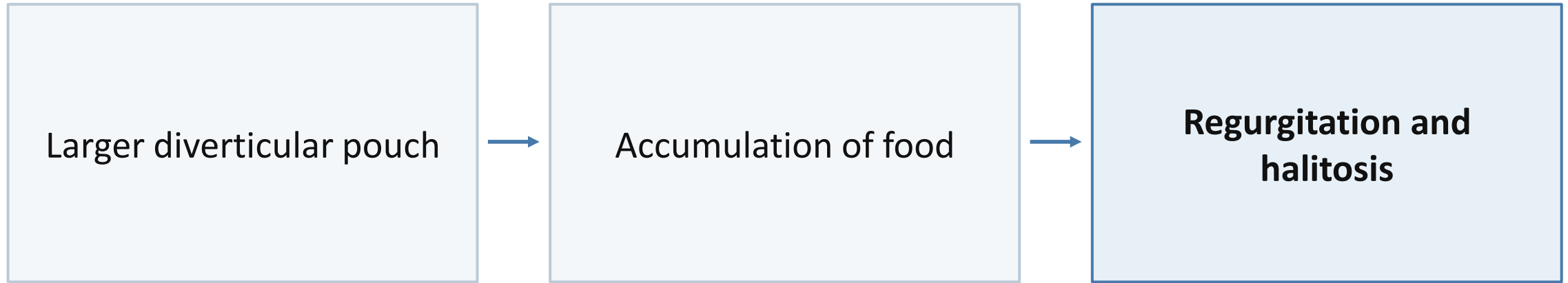
Pouch above upper sphincter

- Located immediately superior to upper esophageal sphincter
- Arises from distal pharyngeal pressure increase
- Original schematic; Not an endoscopic image

Zenker diverticula: Who is affected?

- Uncommon lesions.
- Typically develop after age 50.
- May enlarge to several centimeters.
- Small lesions may produce no symptoms.

Larger Zenker diverticula retain food



Symptoms follow food retention; The pouch may also act as a mass.

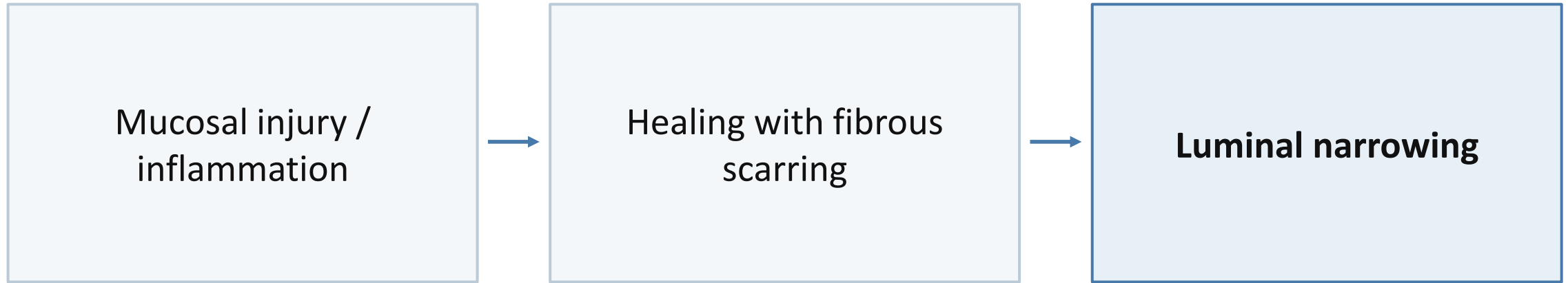
Two diverticula, two sphincter regions

Feature	Zenker	Epiphrenic
Anatomic site	Above upper esophageal sphincter	Immediately above lower sphincter
Precipitating physiology	Cricopharyngeal relaxation failure/spasm	Esophageal dysmotility with raised wall stress
Characteristic Robbins symptom	Regurgitation, halitosis from retention	Specific symptoms not assigned in excerpt

Mechanical stricture is persistent narrowing

- Benign and malignant strictures obstruct the lumen physically.
- Dysphagia is commonly progressive.
- Initial difficulty often involves solid food.
- Cause matters because benign scar and malignant narrowing have different implications.

How inflammation produces a benign stricture

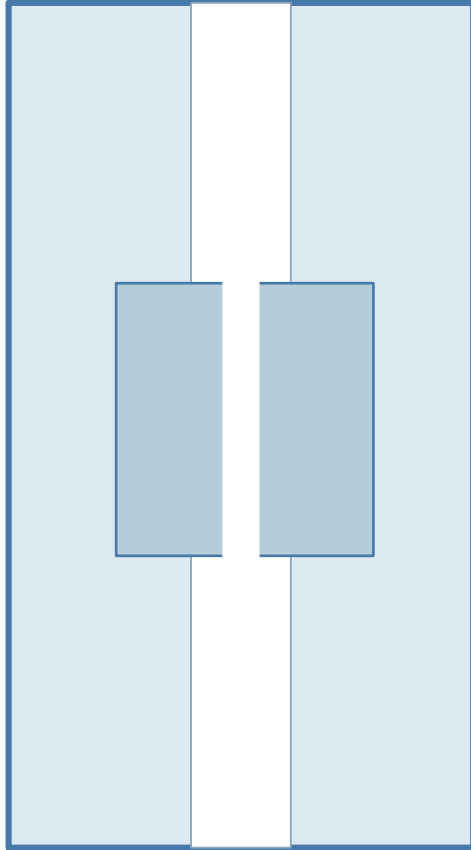


Thickened submucosa with secondary muscular and epithelial changes.

Benign stricture: Submucosal fibrosis

- Fibrous thickening of the submucosa is the principal structural change.
- Increased connective tissue reduces compliance and luminal caliber.
- The lesion reflects a chronic healing response rather than a primary contraction disorder.

Stricture morphology: Thickened wall, smaller lumen



Narrowed lumen in a thickened wall

- Prominent submucosal thickening narrows the passage
- Atrophic muscularis propria may accompany fibrosis
- Schematic cross-section, not histopathology

Muscularis propria atrophy accompanies stenosis

- Benign stricture is associated with atrophy of the muscularis propria.
- The muscular change occurs alongside submucosal fibrosis.
- A structural lesion may therefore coexist with impairment of the wall musculature.

Secondary epithelial damage in benign strictures

- The overlying epithelium may show secondary damage.
- Epithelial injury occurs in the context of chronic underlying wall disease.
- The major lumen-narrowing substrate remains fibrous submucosal thickening.

Acquired stricture: Three scarring settings

Setting	Basis for narrowing
Chronic gastroesophageal reflux	Inflammation followed by scarring
Irradiation	Treatment-related tissue injury and fibrosis
Caustic injury	Corrosive mucosal damage followed by scar formation

Reflux-associated stricture is a scar complication

- Chronic gastroesophageal reflux is a cause of acquired stenosis.
- Repeated inflammation can culminate in fibrous remodeling.
- Progressive solid-food dysphagia signals the mechanical consequence.
- The narrowed lumen represents a scarring complication of chronic injury.

Radiation-associated stenosis

- Irradiation is listed as a cause of inflammatory scarring.
- Late fibrous remodeling can convert tissue injury into fixed narrowing.
- The result is a mechanical obstruction rather than an isolated motility pattern.

Caustic injury may culminate in stricture

- Corrosive agents injure the esophageal wall.
- Subsequent healing can produce a fibrotic luminal narrowing.
- The obstructive effect may persist long after the initial exposure.

Congenital versus acquired stenosis

Congenital

- Stenosis can be present as a developmental lesion.
- A thickened wall causes partial or complete obstruction.
- Do not assume every stricture is reflux-related.

Acquired

- Inflammation and scarring are frequent causes.
- Reflux, radiation and caustic injury are named examples.
- Fibrous submucosal thickening is characteristic.

A malignant stricture is an essential alternative

- Malignant narrowing also produces mechanical dysphagia.
- Weight loss is often associated with malignant strictures.
- The pattern is a clinical warning, not proof of cancer.
- Dysphagia history alone cannot establish malignant histology.

Benign versus malignant stricture: Clinical clues

Feature	Benign	Malignant
Mechanical dysphagia	Common	Common
Course	May be progressive	Often progressive
Appetite / weight	Generally maintained	Weight loss often associated
Interpretive caveat	Clinical tendency, not absolute	Clinical tendency, not diagnostic

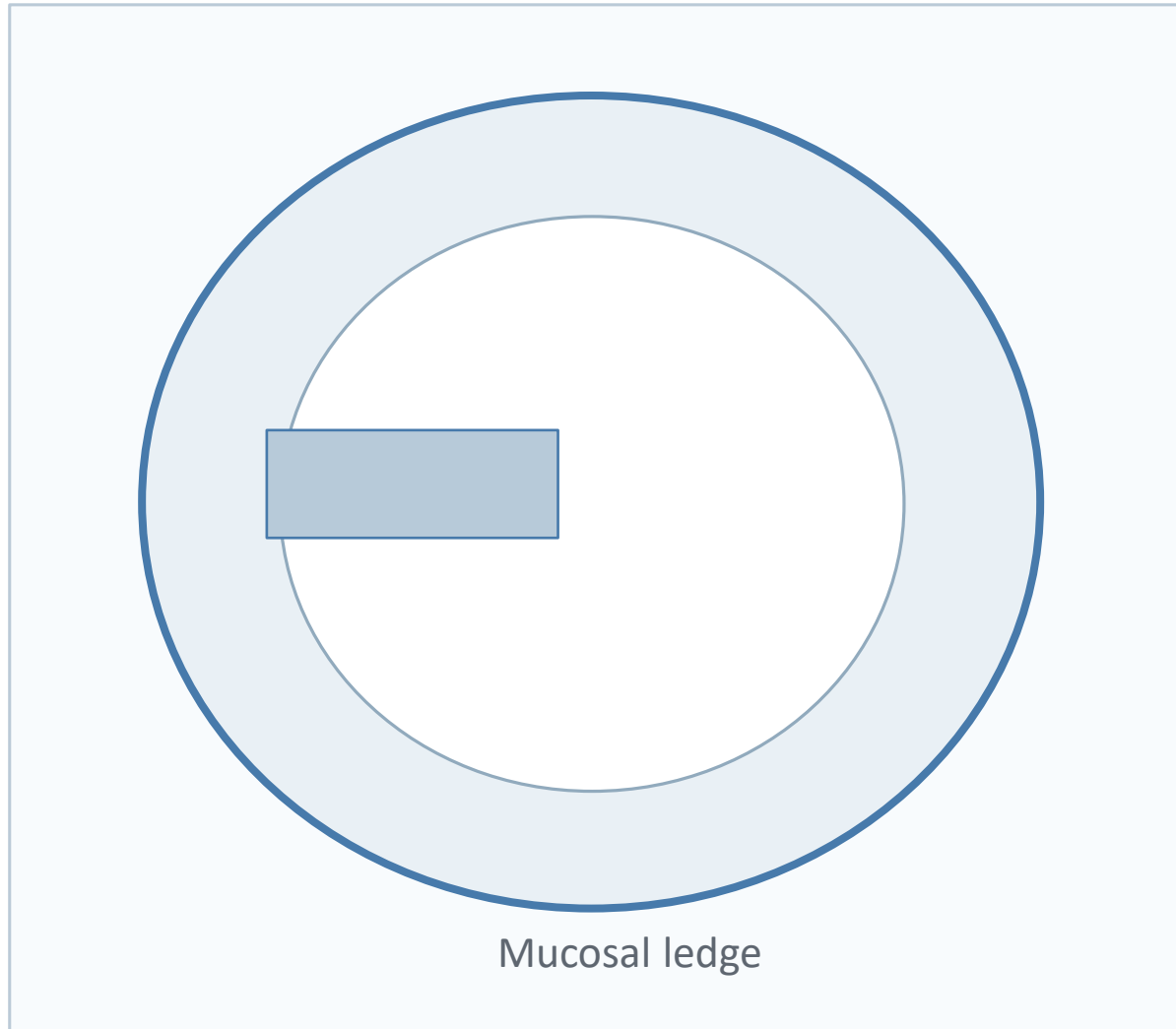
Progressive solid-food dysphagia indicates narrowing

- A constricted passage first restricts larger solid boluses.
- Patients may initially accommodate liquids more readily.
- Progressive symptoms warrant consideration of a stricture.
- The symptom does not by itself distinguish benign from malignant disease.

Esophageal webs are mucosal ledges

- Uncommon protrusions of the esophageal mucosa.
- Ledge-like and only partly encircle the lumen.
- Can form a partial obstacle to swallowed food.
- Unlike fibrotic strictures, the typical lesion is a focal mucosal projection.

Webs: Semicircumferential ledge into the lumen



- Partial mucosal shelf projects toward the lumen
- Projection does not form a complete ring
- Schematic view, not an endoscopic specimen

Microscopic composition of an esophageal web

- Webs contain fibrovascular connective tissue.
- The core is covered by epithelium.
- The lesion is semicircumferential rather than a full-thickness fibrous stenosis.
- The fibrovascular ledge explains partial obstruction without a long stricture.

Webs: Patient pattern and associations

- Usually occur in women older than 40.
- Associated conditions include gastroesophageal reflux.
- Chronic graft-versus-host disease and blistering skin diseases are also associated.
- Association is not equivalent to a proved single cause.

Plummer–Vinson syndrome: Associated features

- An upper esophageal web is one component.
- Iron deficiency anemia is part of the associated clinical pattern.
- Glossitis and cheilosis are accompanying oral findings.
- These are associated findings, not a proposed sequential causal chain.

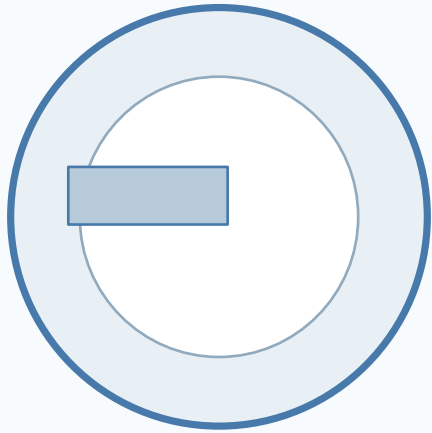
Why incompletely chewed food exposes a web

- A web leaves a partial luminal obstruction.
- Larger, poorly chewed food fragments are more difficult to pass.
- The reported symptom pattern is nonprogressive dysphagia.
- Compare this with the typical progressive course of a fixed stricture.

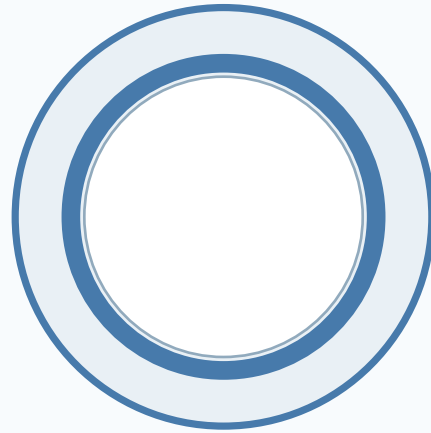
Esophageal rings encircle the lumen

- Schatzki rings are circumferential rather than semicircumferential.
- They are thicker than mucosal webs.
- They may include deeper wall layers than the mucosal web.
- Their location and surface lining distinguish A from B rings.

Rings versus webs: Geometry



WEB — partial ledge



RING — complete band

Cross-sectional schematics

- Web: A mucosal ledge occupies only part of the circumference
- Ring: A thick band extends around the entire circumference
- The structural distinction changes the geometry of luminal narrowing

Rings have more tissue layers than webs

- Mucosa and submucosa participate in esophageal rings.
- Hypertrophic muscularis propria may sometimes be present.
- Thus, rings are structurally thicker than simple fibrovascular mucosal webs.

A ring: Distal esophagus above the junction

- A rings occur in the distal esophagus.
- They lie above the gastroesophageal junction.
- Their covering is squamous mucosa.
- Anatomic level and lining are the identifiers supplied by Robbins.

B ring: The squamocolumnar junction

- B rings lie at the squamocolumnar junction of the lower esophagus.
- Their undersurface may carry gastric cardia–type mucosa.
- Distinguish B rings from A rings by their precise level and lining.

A rings versus B rings

Property	A ring	B ring
Level	Above gastroesophageal junction	At squamocolumnar junction
Surface	Squamous mucosa	Undersurface may bear gastric cardia-type mucosa
Shared type	Circumferential esophageal ring	Circumferential esophageal ring

Web, ring, stricture: Three structural lesions

Property	Web	Ring	Benign stricture
Geometry	Semicircumferential ledge	Circumferential thick band	Longitudinally narrowed segment
Principal tissue	Fibrovascular mucosa	Mucosa/submucosa $\pm$ muscle	Fibrotic submucosa
Illustrative course	Nonprogressive dysphagia	Partial obstruction	Progressive solid dysphagia

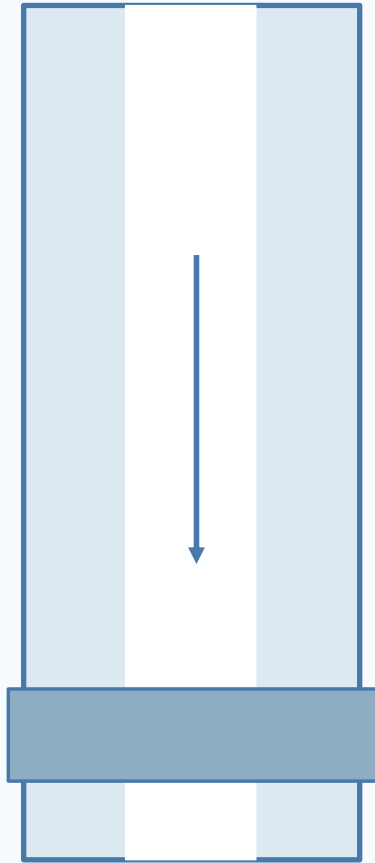
Achalasia: A specific sphincter–motor disorder

- The defining abnormality involves lower esophageal sphincter relaxation.
- It also features elevated sphincter tone and esophageal aperistalsis.
- Together these abnormalities cause functional outflow obstruction.
- The syndrome is distinct from a fixed anatomic ring or stricture.

Achalasia has a three-part physiologic signature

- Incomplete lower esophageal sphincter relaxation.
- Increased lower esophageal sphincter resting tone.
- Esophageal aperistalsis.
- These simultaneous physiological features define the syndrome.

Achalasia: Esophageal pump and distal gate fail



Distal high-tone sphincter

- Absent coordinated peristalsis limits bolus propulsion
- High-tone LES relaxes incompletely
- Schematic functional outflow obstruction

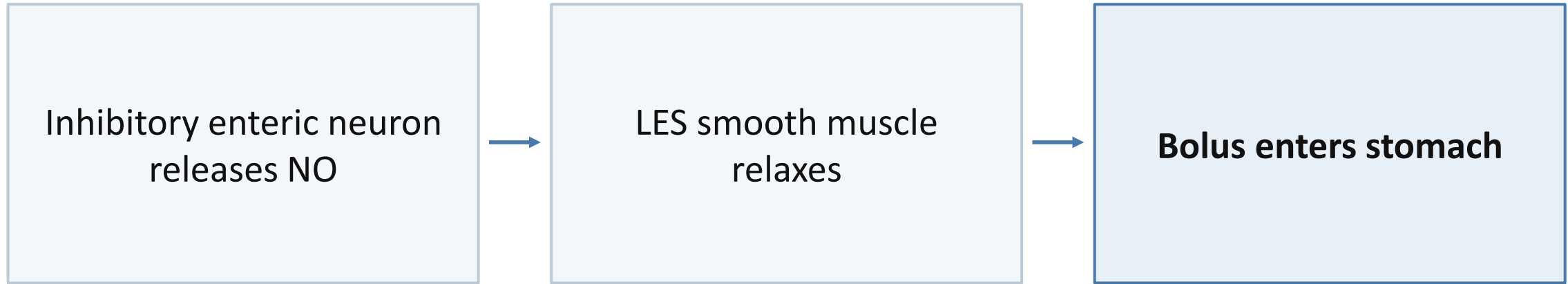
Symptoms reflect impaired distal outflow

- Dysphagia is a cardinal symptom.
- Difficulty belching is also described.
- Chest pain can accompany the motor disorder.
- Symptom combinations arise from failure of transit and sphincter function.

Primary achalasia affects inhibitory neurons

- Degeneration involves nitric oxide–producing neurons.
- These neurons ordinarily induce relaxation of the lower sphincter.
- Loss of inhibitory signaling explains incomplete relaxation.
- The key disease process is neurogenic, not simply fibrotic narrowing.

Nitric oxide neurons normally relax the LES



Primary achalasia disrupts this inhibitory pathway; The precise initiating cause remains unknown.

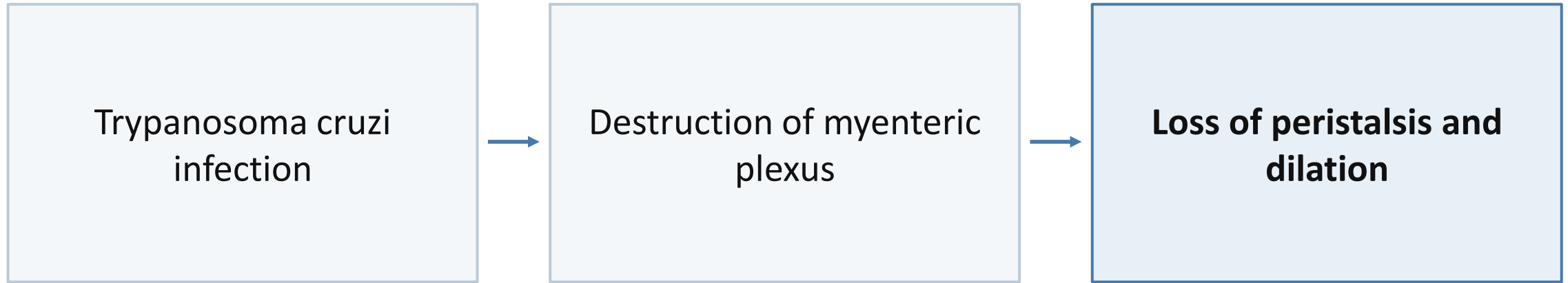
Primary achalasia may involve extrinsic pathways

- Degenerative changes may extend to the extraesophageal vagus nerve.
- The dorsal motor nucleus of the vagus may also be affected.
- The supplied source does not establish a single initiating lesion.
- Rare familial examples are described.

Immune mechanisms remain a hypothesis

- Some cases have an association with remote HSV-1 infection.
- Immunoregulatory gene polymorphisms have been linked to achalasia.
- Occasional coexistence with Sjögren syndrome or autoimmune thyroid disease is described.
- Together, these observations suggest—but do not prove—immune-mediated neuron loss.

Chagas disease causes secondary achalasia



The mechanism is acquired neuronal loss associated with the infectious disease.

Chagas disease can affect several organs

- Esophageal myenteric plexus destruction impairs peristalsis.
- Duodenal and colonic plexi may also be affected.
- Ureteric plexi are another reported site.
- The esophageal motor disorder is part of a broader distribution of neuronal injury.

Other causes of achalasia-like disease

Category	Examples in Robbins
Autonomic neuropathy	Diabetes
Infiltrative disease	Malignancy, amyloidosis, sarcoidosis
Systemic disease	Systemic sclerosis
Central neurologic lesion	Dorsal motor nuclei damage, including after polio

Down syndrome and LES dysfunction

- Lower esophageal sphincter dysfunction occurs in association with Down syndrome.
- This is a stated association, not a claim that all such patients have achalasia.
- Clinical findings still require identification of the underlying motor pattern.

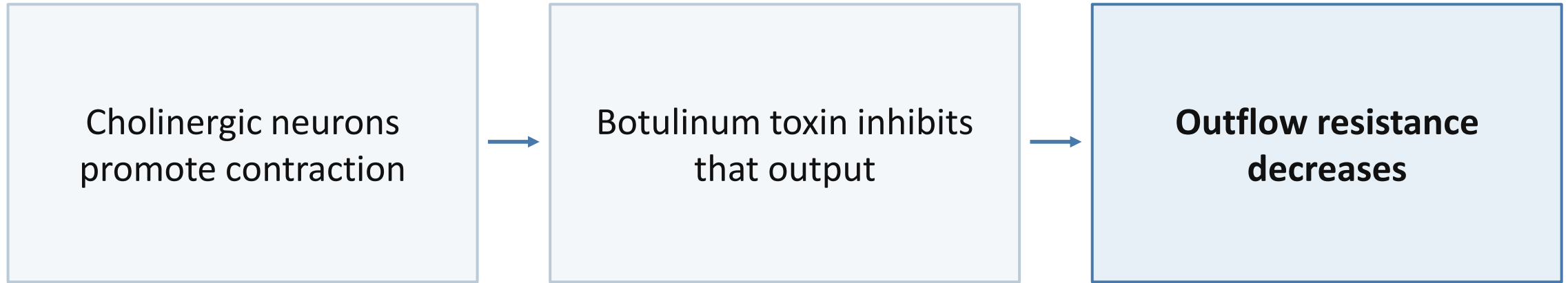
Primary versus secondary achalasia

Aspect	Primary	Secondary
Causal framework	Unknown etiology; neuronal degeneration	Identifiable associated cause
Named mechanism	Loss of nitric oxide–producing neurons	T. cruzi destruction of myenteric plexus
Related settings	Rare familial and possible immune links	Chagas and other achalasia-like disorders
Inference guardrail	Hypotheses are not proven causes	Exclude mimics before attributing mechanism

Management targets the functional barrier

- Treatment aims to overcome the obstructing distal sphincter.
- Approaches include pneumatic balloon dilation.
- Botulinum neurotoxin can reduce contraction.
- Surgical division of esophageal muscle is another approach.

Botulinum neurotoxin: Why relaxation improves



This is a mechanism of symptomatic treatment; It does not restore the missing inhibitory neurons.

Achalasia treatments and immediate targets

Intervention	Physical / neurophysiologic target
Pneumatic balloon dilation	Mechanically reduces outlet resistance
Botulinum neurotoxin	Inhibits contraction-promoting cholinergic signaling
Surgical muscle division	Reduces resistance by cutting muscle

Cancer risk in achalasia: Textbook framing

- The supplied text acknowledges some increased esophageal cancer risk.
- It states the risk is not high enough to warrant surveillance endoscopy.
- This is the textbook statement, not an independently verified current society guideline.
- Specific individualized surveillance decisions are outside this source-based lecture.

Case: Progressive solid-food dysphagia

A patient develops gradually worsening difficulty with solid meals and noticeable weight loss.

What broad mechanism should be prioritized?

Mechanical obstruction; Evaluate for malignant as well as benign narrowing.

Progressive solid dysphagia is typical of stricture; Weight loss is an accompanying warning for malignancy.

Case: Food regurgitation and halitosis

An older adult reports retained food coming back into the mouth, with persistent halitosis.

Which pressure-related lesion is suggested?

Zenker diverticulum.

Food can collect in a large pouch immediately above the upper esophageal sphincter.

Case: Nonprogressive trouble with rough food

A woman over 40 has longstanding nonprogressive dysphagia, especially for incompletely chewed food.

Which focal lesion best fits the pattern?

Esophageal mucosal web.

A semicircumferential fibrovascular ledge causes partial obstruction, unlike the usual progressive stricture.

Case: Normal-looking esophagogram

A patient has suspected high-amplitude esophageal contractions, but contrast imaging appears normal.

Does normal contrast imaging exclude the disorder?

No: Nutcracker esophagus still requires manometric assessment.

Barium swallow may be normal and manometry is required.

Integrated differential for esophageal obstruction

Clue	Think first	Supporting mechanism
Progressive solid dysphagia	Mechanical stricture	Reduced fixed lumen
Nonprogressive coarse-food dysphagia	Web	Semicircumferential ledge
Regurgitation + halitosis	Zenker diverticulum	Food retention proximal to UES
Corkscrew barium appearance	Diffuse spasm	Simultaneous distal contractions
Aperistalsis + impaired LES relaxation	Achalasia	Functional outlet failure

Five clinically useful conclusions

- Separate fixed luminal narrowing from defective propulsion or sphincter relaxation.
- Use the site and geometry to distinguish diverticula, webs, rings, and strictures.
- Understand how neuronal injury produces achalasia physiology.
- Interpret weight loss and dysphagia progression without overclaiming a diagnosis.
- Choose a test or therapy based on the mechanism it assesses or modifies.

Reference

Robbins, Cotran & Kumar. Pathologic Basis of Disease. 11th edition. Elsevier, 2026.

Lecture content is based on the “Esophageal Obstruction” section and its associated “Achalasia” subsection.

Original editable schematics are conceptual teaching illustrations, not clinical or histological specimens.