Chapter 3

Management of Patients with Gastrointestinal Disorders

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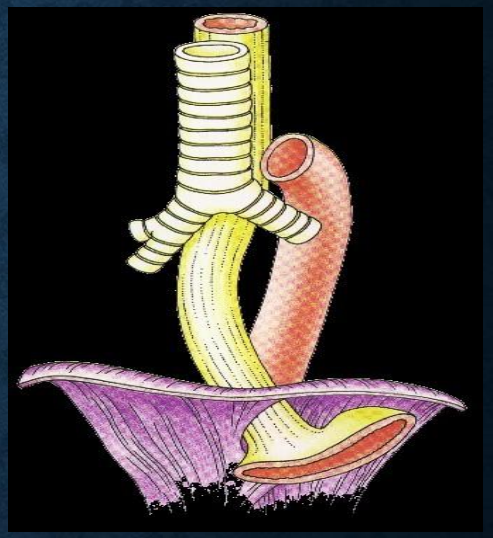
3.2. Management of Patients With Esophageal Disorders

ESOPHAGEAL DISORDERS

- Anatomic & Structural
- Motility disorder
- Reflux
- Pill induced and eosinophilic esophagitis

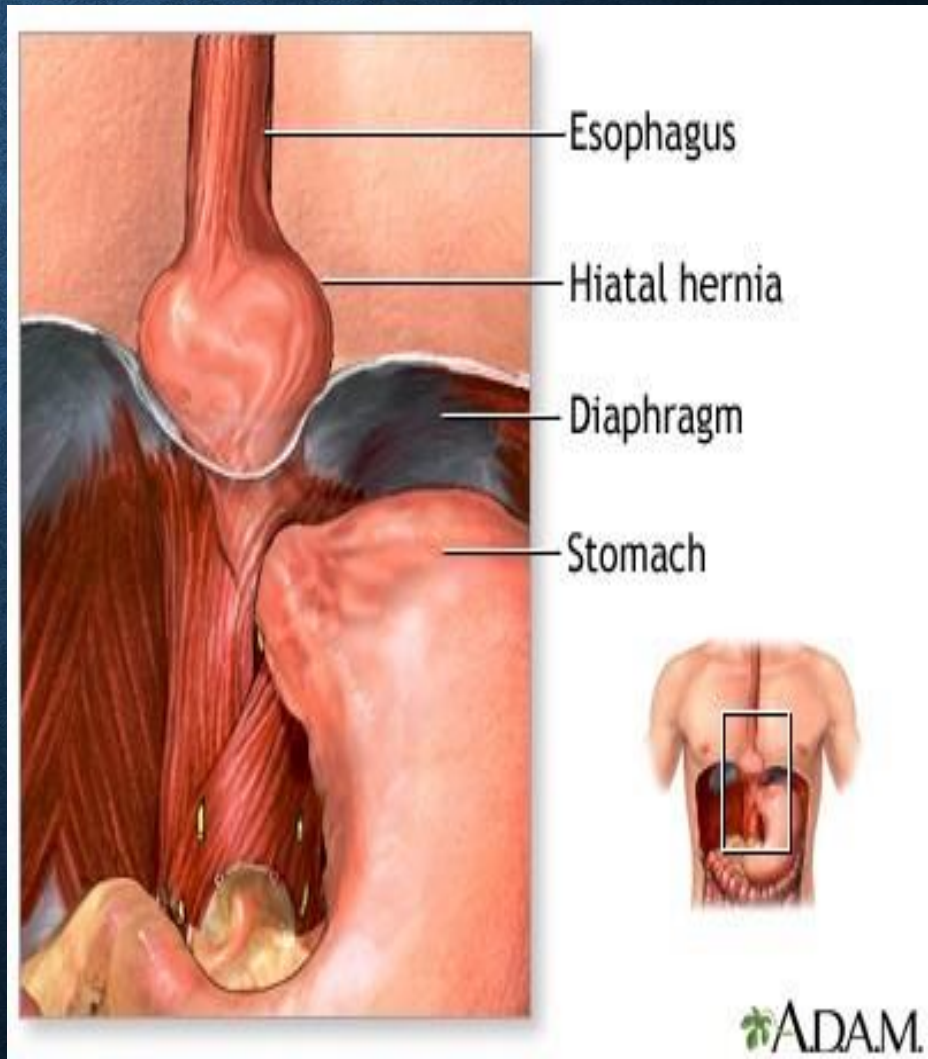


STRUCTURAL DISORDER

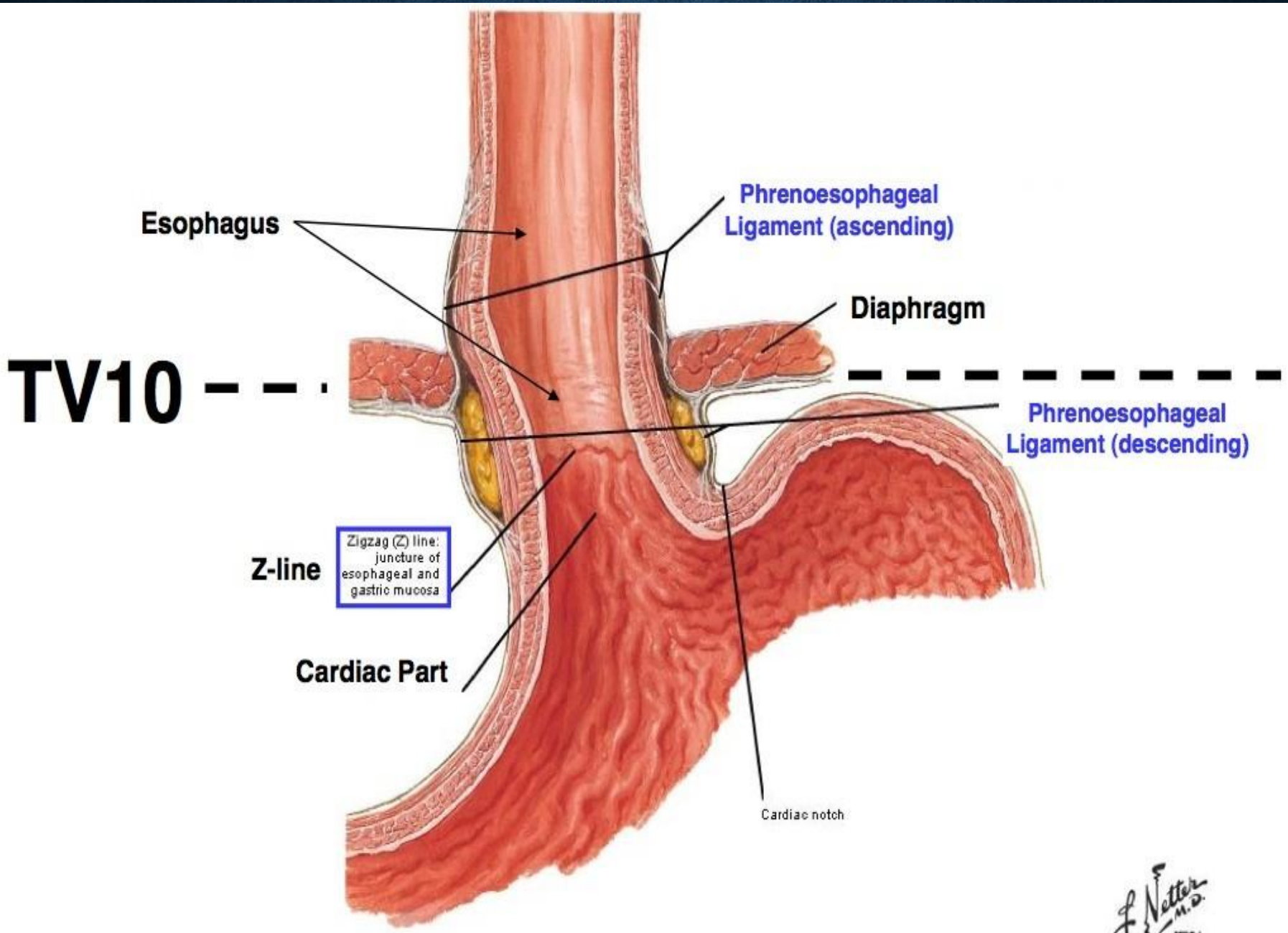
- Hiatal hernia
- Rings and Webs
- Diverticula
- Tumors

HIATAL HERNIA

Herniation of viscera, most commonly stomach into mediastinum through the esophageal hiatus of the diaphragm.



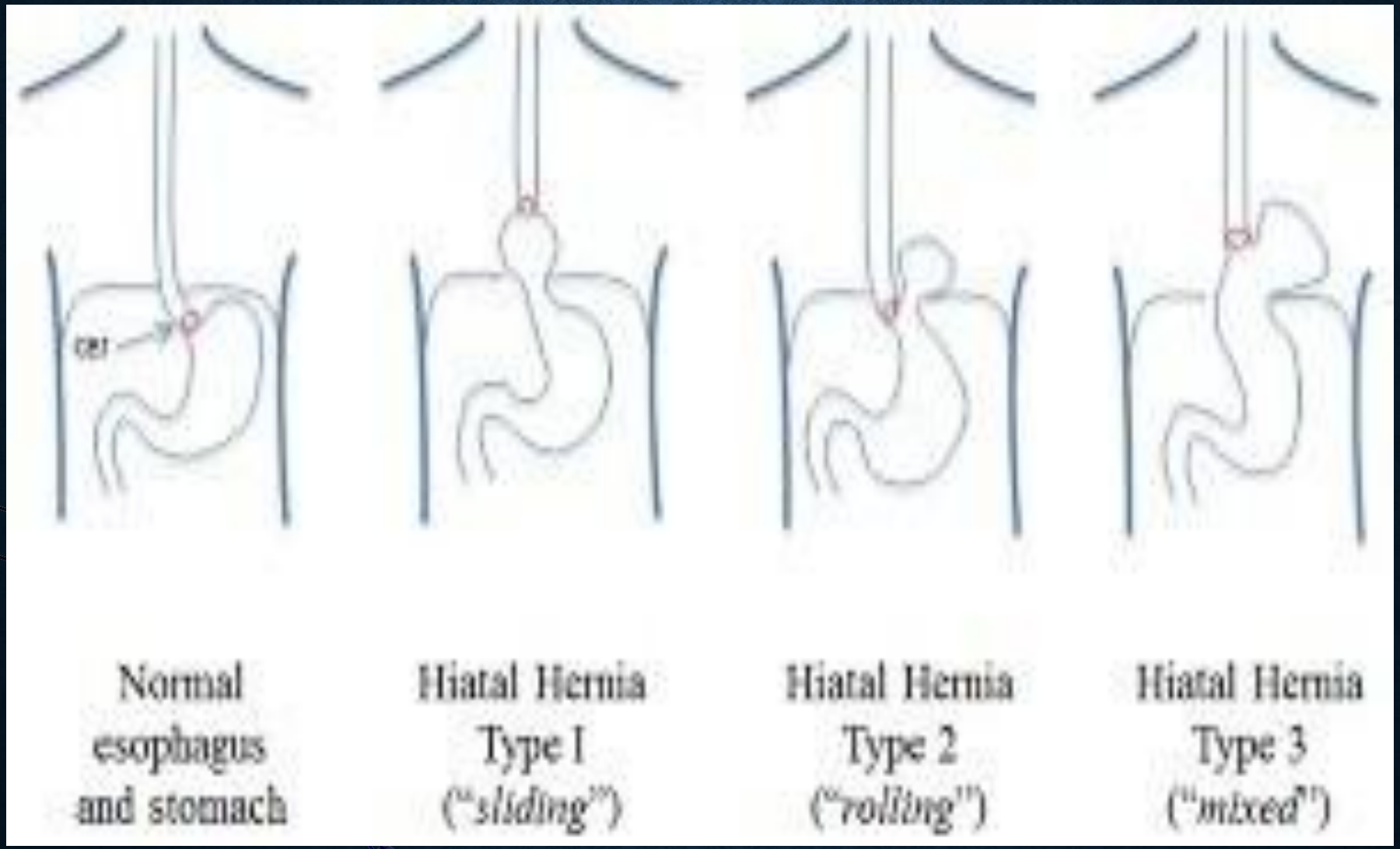
- Type 1 or sliding hiatal hernia:
- 95% of total
- Gastroesophageal junction and gastric cardia slide upward
- As a result of weakening of the phrenoesophageal ligament


Esophagus
Phrenoesophageal Ligament (ascending)
Diaphragm
TV10
Phrenoesophageal Ligament (descending)
Z-line
Zigzag (Z) line: juncture of esophageal and gastric mucosa
Cardiac Part
Cardiac notch
F. Netter M.D.
©IBN

N 236a *4th Ed.*

- Type II, III, and IV are all subtypes of para esophageal hernia
- Type II and type III the gastric fundus also herniates
- Difference: in type II GE junction remains fixed at the hiatus
- Type III: mixed sliding /paraesophageal hernia

λTYPE IV: VISCERA OTHER THAN THE STOMACH HERNIATES, MC THE COLON


GEJ
Normal esophagus and stomach
Hiatal Hernia Type I ("sliding")
Hiatal Hernia Type 2 ("rolling")
Hiatal Hernia Type 3 ("mixed")

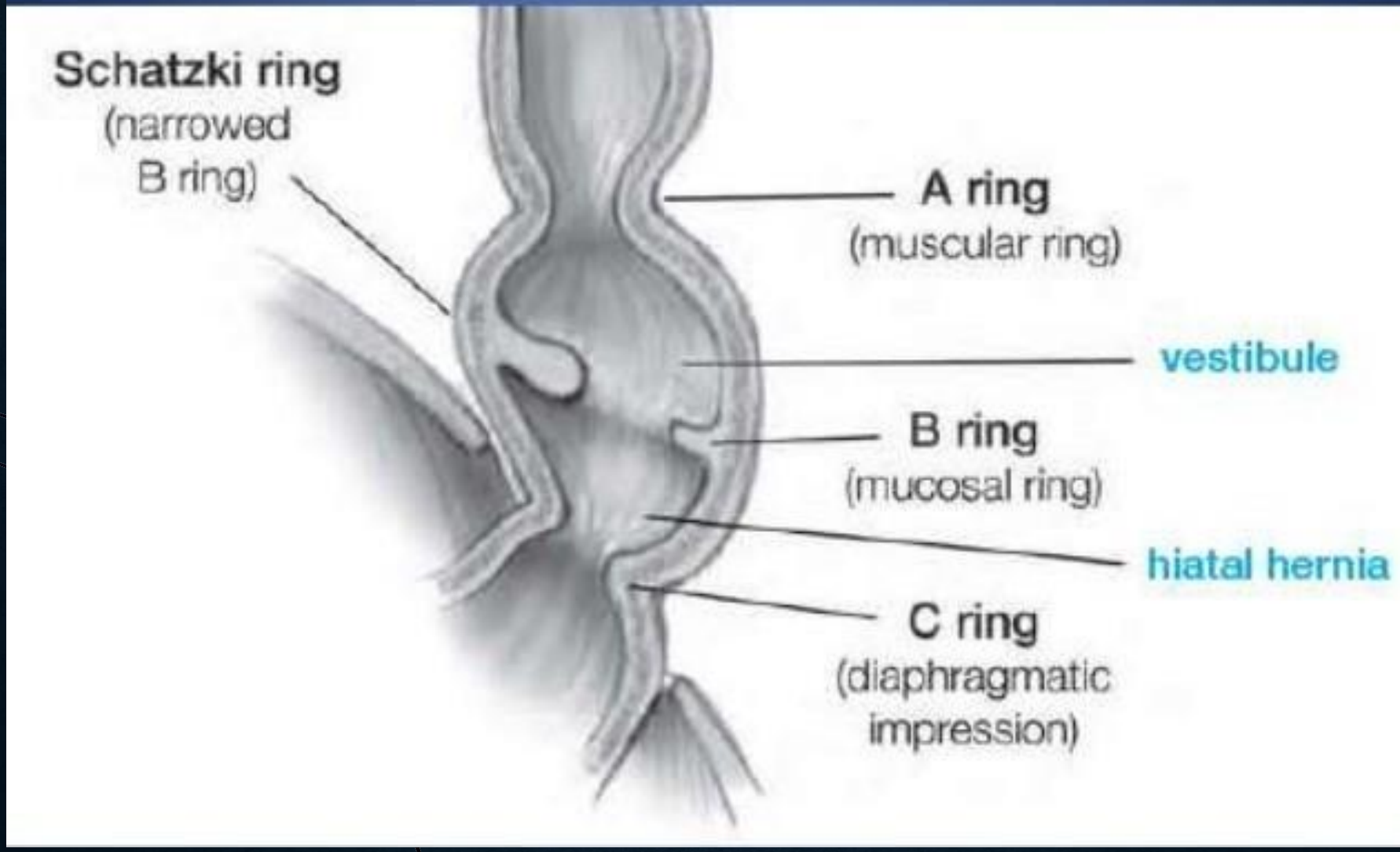
RINGS AND WEBS

- A lower esophageal mucosal ring (B ring) thin membranous narrowing at the squamocolumnar mucosal junction
- Its origin is unknown
- Usually asymptomatic
- When lumen diameter is less than 13 mm can cause episodic solid food dysphagia and is called Schatzki ring.

- Is the MCC of intermittent food impaction
- Also known as Steakhouse syndrome
- Symptomatic rings treated by dilatation

- Web-like constrictions higher in the esophagus can be of congenital or inflammatory in origin
- When circumferential cause intermittent dysphagia to solids similar to schatzki rings
- Symptomatic proximal esophageal webs+IDA in middle aged women is called Plummer-Vinson synd.

Esophageal rings

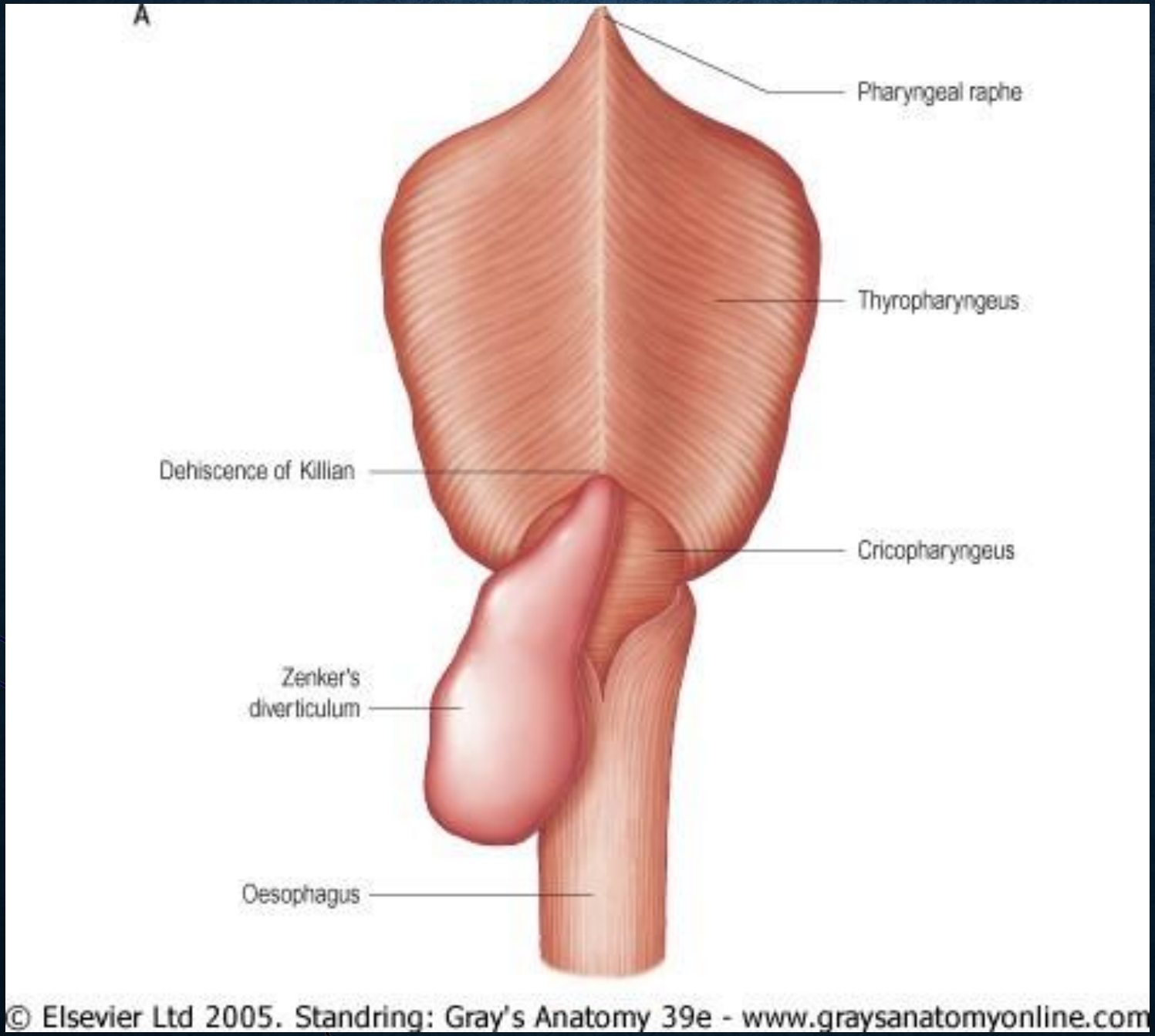


DIVERTICULA

- Categorized by location:
- Epiphrenic
- Hypopharyngeal (zenker)
- Mid esophageal

- Epiphrenic and zenker are both false diverticula(herniation of mucosa and sub mucosa through muscular layer of the esophageal layer)
- Result from increased intraluminal pressure A/W distal obstruction

- IN ZENKER HERNIATION OCCURS AT THE NATURAL WEAKNESS WHICH IS CALLED **KILLIAN TRIANGLE**


A
Pharyngeal raphe
Thyropharyngeus
Dehiscence of Killian
Cricopharyngeus
Zenker's diverticulum
Oesophagus
© Elsevier Ltd 2005. Standring: Gray's Anatomy 39e - www.graysanatomyonline.com

- SMALL ZENKER DIVERTICULA IS USUALLY ASYMPTOMATIC IF IT ENLARGE ENOUGH TO RETAIN SALIVA AND FOOD IT CAN CAUSE DYSPHAGIA, HALITOSIS, ASPIRATION.

- Treatment: surgery

- **EPIPHRENIC DIVERTICULA** USUALLY A/W ACHALASIA OR DISTAL ESOPHAGEAL STRICTURE
- **MID ESOPHAGEAL DIVERTICULA** :MAY BE CAUSED BY TRACTION FROM ADJACENT INFLAMMATION(CLASSICALLY TB)
- Both are asymptomatic until they enlarge and cause dysphagia and regurgitation

TUMORS

- Esophageal cancer occurs in about 4.5:100,000 in the U.S
- Mortality is 4.4:100,000
- Two types: adenocarcinoma and SCC
- RFs: adenocarcinoma strongly related to the GERD and Barrett metaplasia
- SCC : smoking, alcohol , caustic injury, HPV infection
-

Harrison principle of internal medicine 18th edition

- Typical presentation: progressive solid food dysphagia and weight loss
- Associated symptoms: odynophagia, iron deficiency anemia, and with mid esophageal tumors: hoarseness from left recurrent laryngeal nerve injury

GERD

- It has been estimated that 15% of adults in the United States are affected by GERD.

Harrison principle of internal medicine 18th edition

SYMPTOMS

- Typical symptoms: heart burn and regurgitation
- Less common : chest pain and dysphagia
- Extra esophageal symptoms **(asthma, globus, laryngitis, cough, throat clearing)**
- Atypical symptoms :dyspepsia, epigastric pain, nausea, bloating

COMPLICATIONS

- Esophagitis
- Bleeding
- Stricture
- Barret esophagus
- Esophageal adenocarcinoma

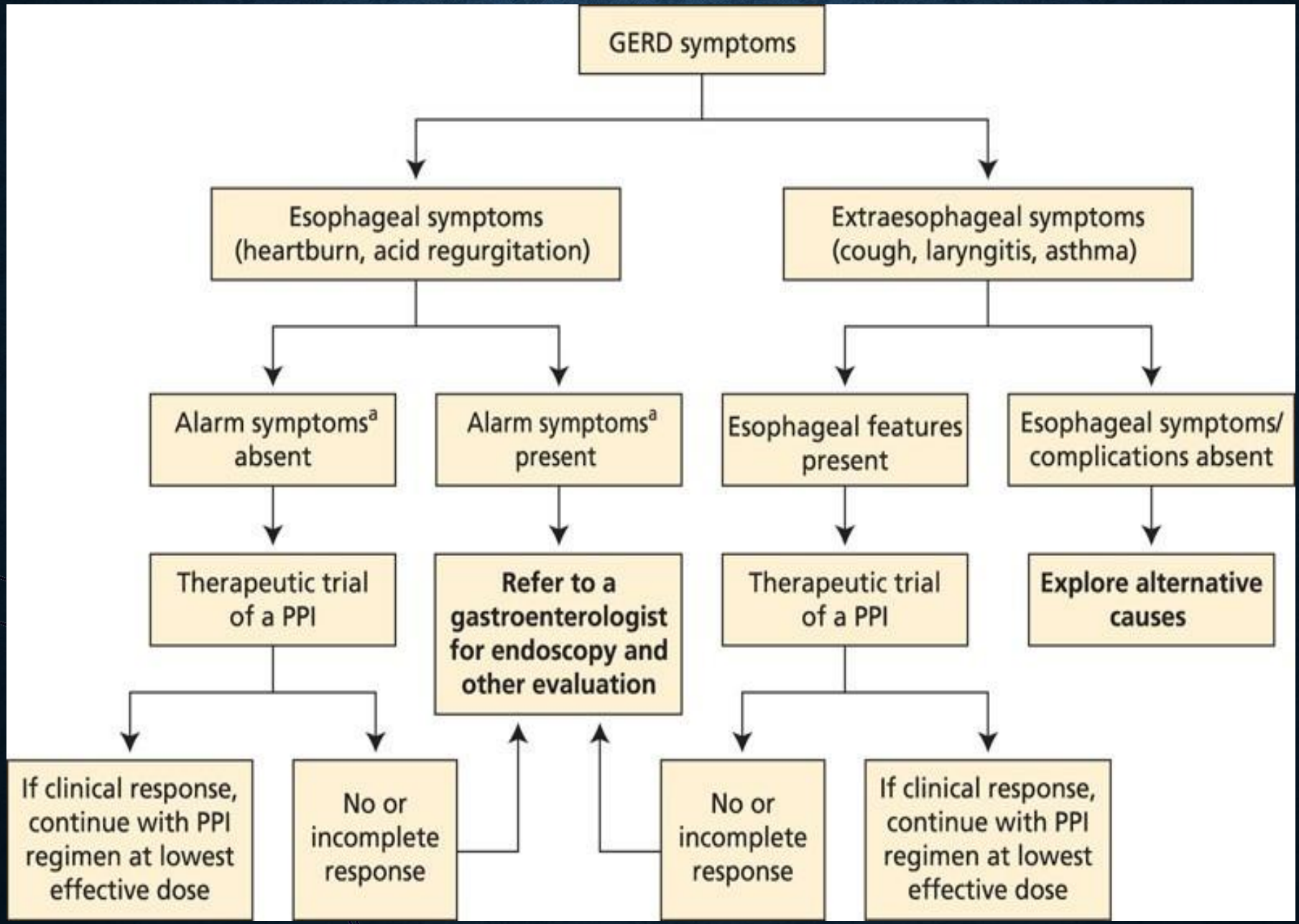
Secondary causes:	Ascites Eosinophilic esophagitis Esophagitis Obesity Pregnancy Scleroderma Surgical destruction of the LES Tobacco use
Medications:	Anti-cholinergic agents Benzodiazepines β-blockers Calcium-channel blockers Nitrates Prostaglandins Sildenafil Tricyclic antidepressants
Foods:	Alcohol Chocolate Coffee High-fat foods Oranges or orange juice Peppermint or spearmint Tomato products

DIAGNOSIS

- Typical symptoms such as heart burn, regurgitation or both is enough to diagnose GERD.
- A favorable response to PPI is also supportive evidence for GERD and is reasonable first step in patients without alarm symptoms.

- Endoscopy is first step in patient with alarms symptoms (dysphagia, anemia, vomiting or weight loss, age >50)

- If patients do not respond to PPI and have negative Upper endoscopy to make a definitive diagnosis ambulatory PH monitoring is usually performed.


GERD symptoms
Esophageal symptoms (heartburn, acid regurgitation)
Extraesophageal symptoms (cough, laryngitis, asthma)
Alarm symptoms[a] absent
Alarm symptoms[a] present
Esophageal features present
Esophageal symptoms/ complications absent
Therapeutic trial of a PPI
Refer to a gastroenterologist for endoscopy and other evaluation
Therapeutic trial of a PPI
Explore alternative causes
If clinical response, continue with PPI regimen at lowest effective dose
No or incomplete response
No or incomplete response
If clinical response, continue with PPI regimen at lowest effective dose

MEDICAL TREATMENT

- **Lifestyle modifications**

1. Weight loss is recommended for GERD patients who are overweight or have had recent weight gain.

2. Head of bed elevation and avoidance of meals 2–3 h before bedtime should be recommended for patients with nocturnal GERD.

- Routine global elimination of food that can trigger reflux (including chocolate, caffeine, alcohol, acidic and/or spicy foods) is not recommended in the treatment of GERD.

- Acid suppression via PPI's for 8 weeks,30-60 min before meal

FOR PATIENTS WITH PARTIAL RESPONSE TO ONCE DAILY THERAPY, TAILORED THERAPY WITH ADJUSTMENT OF DOSE TIMING AND/OR TWICE DAILY DOSING SHOULD BE CONSIDERED.

- λ NON-RESPONDERS TO PPI SHOULD BE REFERRED FOR EVALUATION

- λ Maintenance therapy with the lowest effective dose if needed.

- without erosive disease bedtime H_2 blocker therapy can be added to daytime PPI therapy in selected patients evidence of night-time reflux if needed.

- PPIs are safe in pregnancy.

INDICATIONS FOR SURGERY

- Failed optimal medical management
- Noncompliance with medical therapy
- High volume reflux
- Severe esophagitis by endoscopy
- Benign stricture
- Barrett's columnar-lined epithelium (without severe dysplasia or carcinoma)

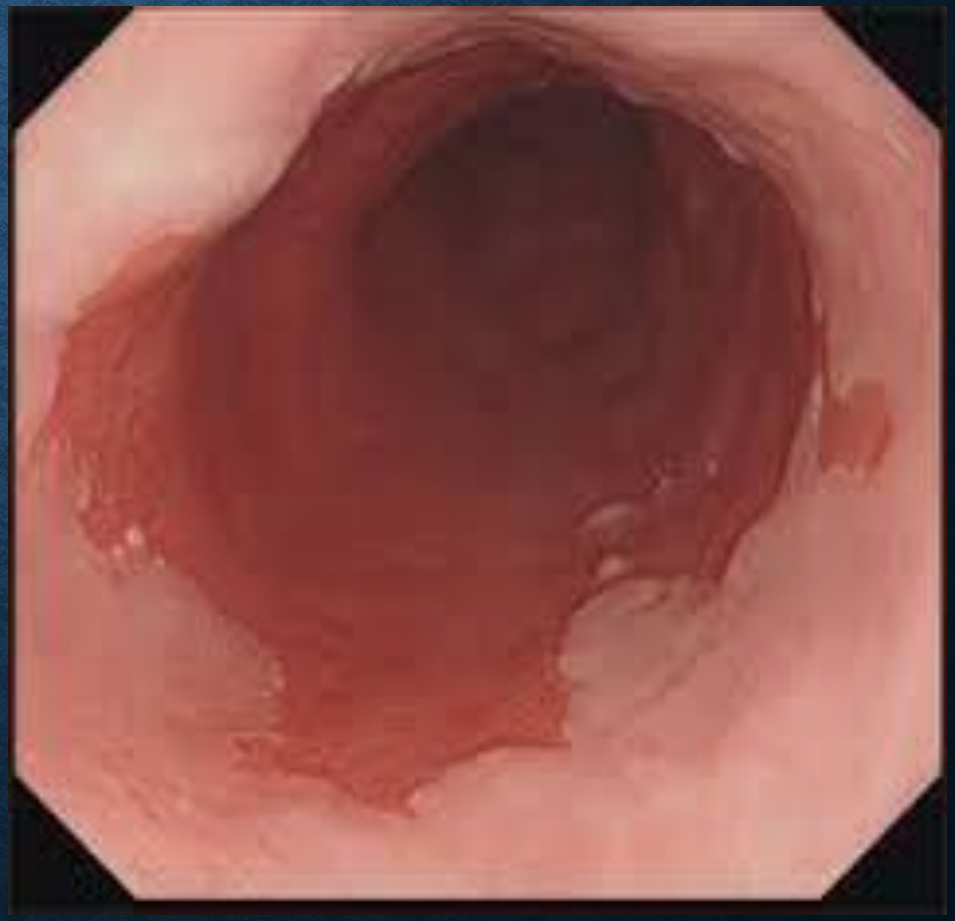
Up-to-date

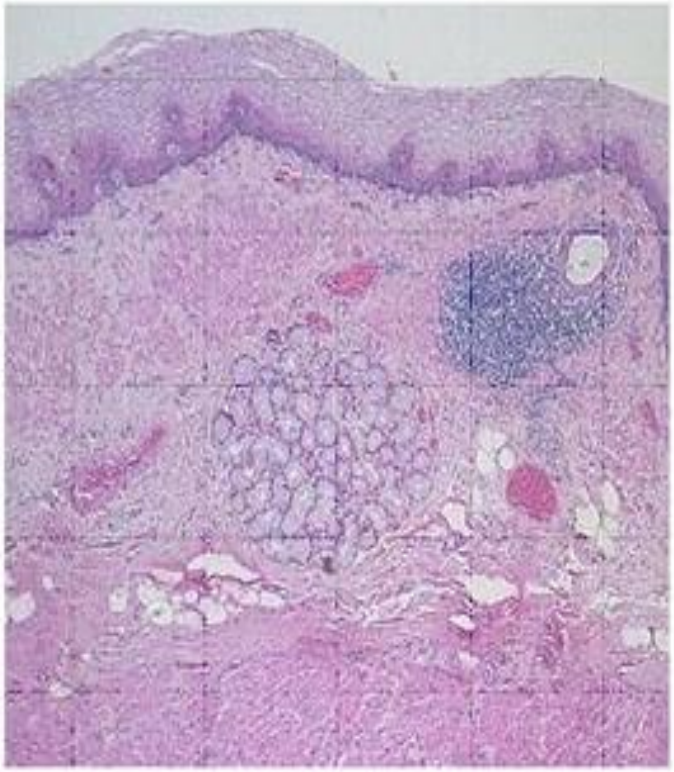
BARRETT ESOPHAGUS

- Is a complication of GERD
- Normal squamous epithelium of the distal esophagus is replaced by columnar epithelium

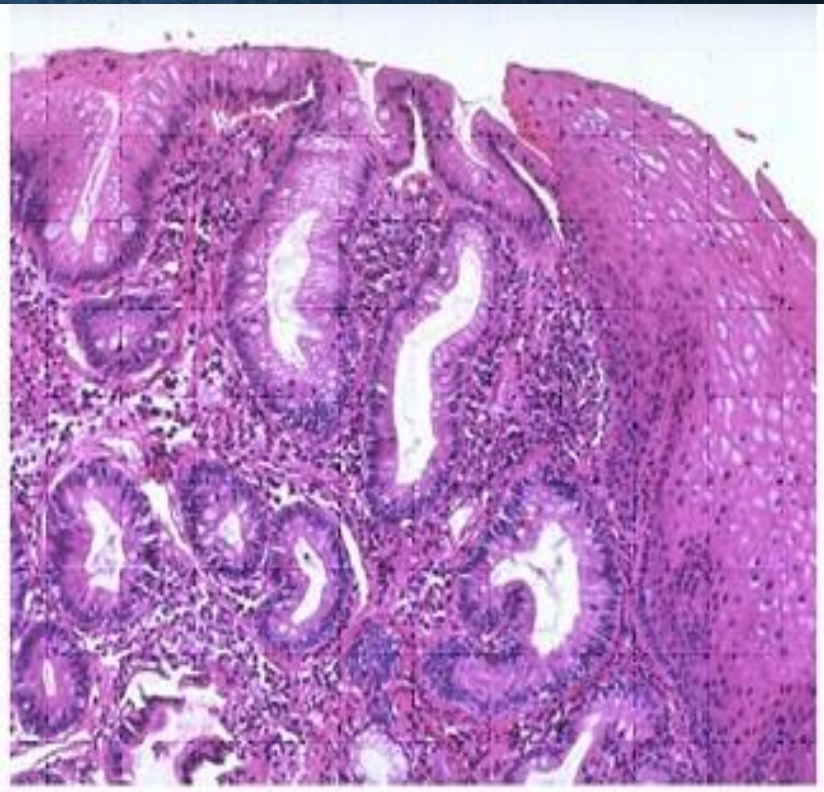
- Is premalignant
- Annual incidence of esophageal adenocarcinoma is 0.5%
- 10% of patients with chronic GERD symptoms have Barrett on endoscopy

- The diagnosis of BE is suggested by endoscopic findings and is confirmed histologically by the presence of specialized intestinal metaplasia with acid-mucin–containing goblet cells





This is normal esophageal squamous mucosa at the top, with underlying submucosa and muscularis propria

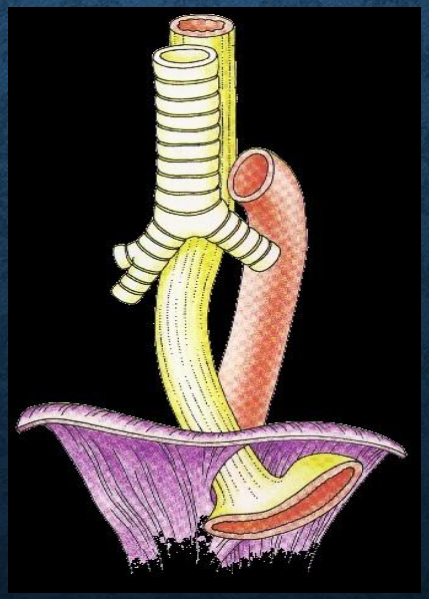


Barrett's esophagus in which there is gastric-type mucosa above the gastroesophageal junction. Note the columnar epithelium to the left and the squamous epithelium at the right.

Table 3. Practice Guidelines for Endoscopic Surveillance of Barrett Esophagus

Dysplasia Grade	Recommendation
None	Repeat endoscopy 1 year after diagnosis to detect prevalent dysplasia; if negative for dysplasia, endoscopic surveillance every 3 years
Low-grade	Confirmation by expert pathologist Repeat endoscopy 6 months after diagnosis to detect prevalent dysplasia, then endoscopic surveillance yearly
High-grade	Confirmation by expert pathologist Endoscopic evaluation for any focal lesion (may indicate more advanced neoplasia): if present, focal lesion(s) should be removed by endoscopic mucosal resection for diagnosis and staging Options for further management: esophagectomy, endoscopic ablation, surveillance every 3 months

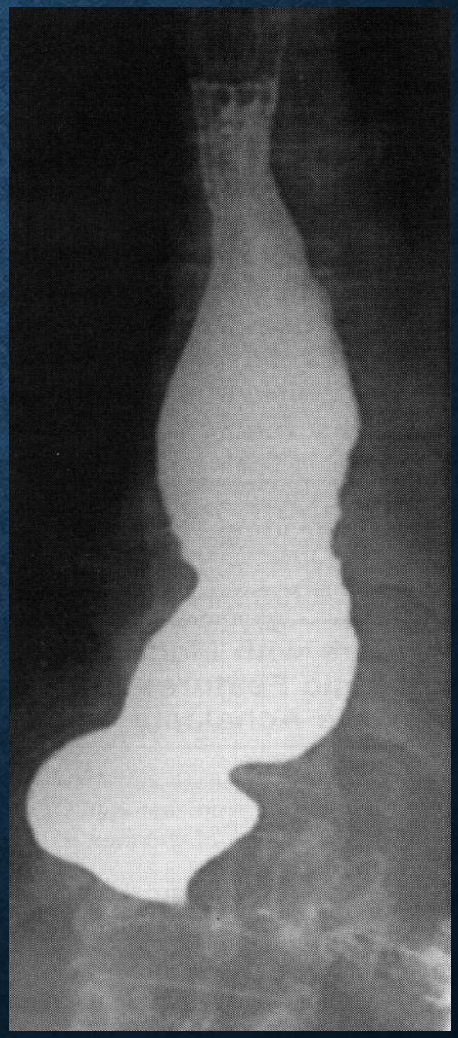
ESOPHAGEAL MOTILITY DISORDERS



ACHALASIA

ACHALASIA

- Incidence of 1:100,000 population annually
- usually presents between ages 30 to 60
- male=female
- No racial predilection



American college of gastroenterology guideline

PATHOPHYSIOLOGY

- Degeneration of NO producing inhibitory neurons
  - loss of ganglionic cells in the myenteric plexus (distal to proximal)
  - vagal fiber degeneration
- that affect relaxation of LES

- Basal LES pressure rises

underlying cause: unknown

- Autoimmune, viral immune, or neurodegenerative.

CLINICAL PRESENTATION

dysphagia (most patients report solid and liquid food dysphagia)

- regurgitation of food retained in the prox. Dilated esophagus
- chest pain (squeezing, retrosternal, radiates to jaw, neck, arms or back and worsen with food)
- weight loss
- nocturnal cough and recurrent aspiration
- Sensation of heartburn in 30% ,assumed to be related retained food fermentation and lactic acid formation

DDX

- DES

- Chagas(endemic in central brazil, Venezuela, Northern argentina)◊reduvid(kissing) bugs◊transmits Tryponosoma Cruzi◊destruction of autonomic ganglion cells in heart, urinary tract, gut and respiratory tract

- Pseudoachalasia: tumor infiltration ,MC seen with carcinoma of the gastric fundus or distal esophagus

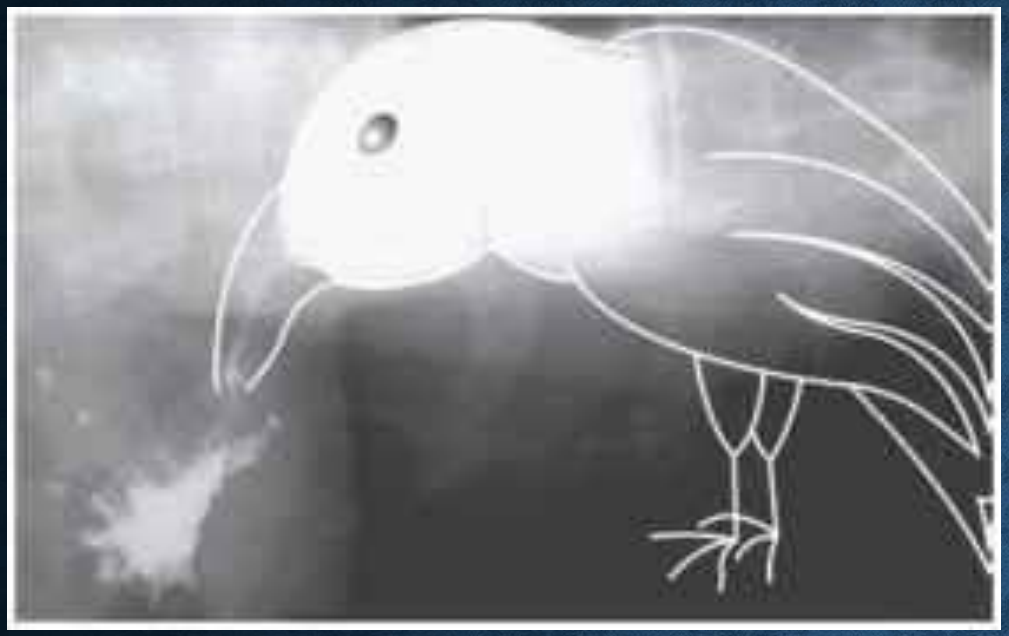
- More likely with advanced age, abrupt onset of symptoms(<1yr) and weight loss

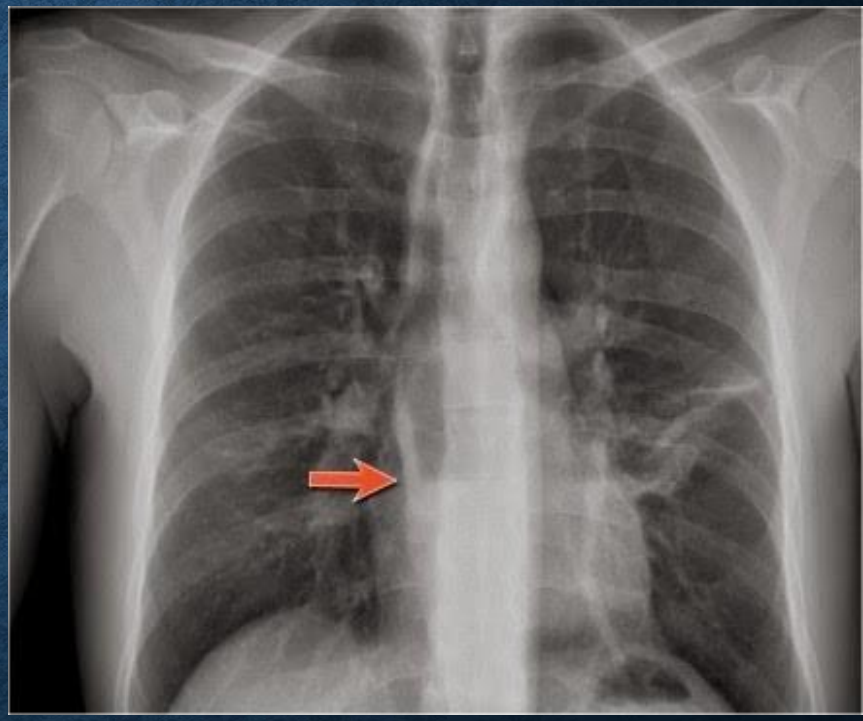
DIAGNOSTIC WORK UP

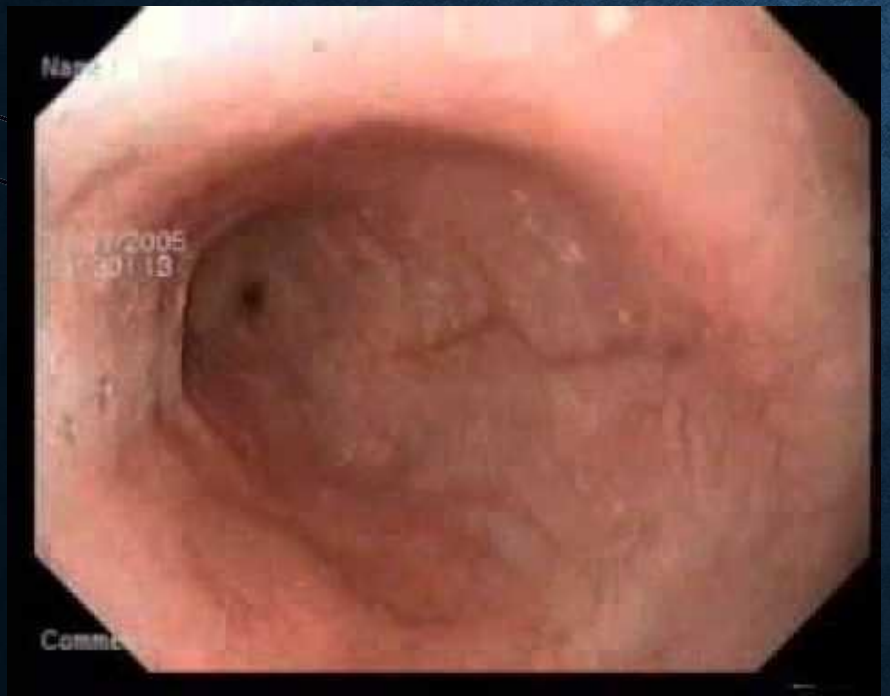
- λplain film (air-fluid level, wide mediastinum, absent gastric bubble)

- λBarium swallow (*Bird beak sign)*
  - λPrimary screening test (95% accurate)

- λendoscopy (rule out GE junction tumors)
- λesophageal manometry (absent peristalsis,
  - LES relaxation, & resting LES >45 mmHg)

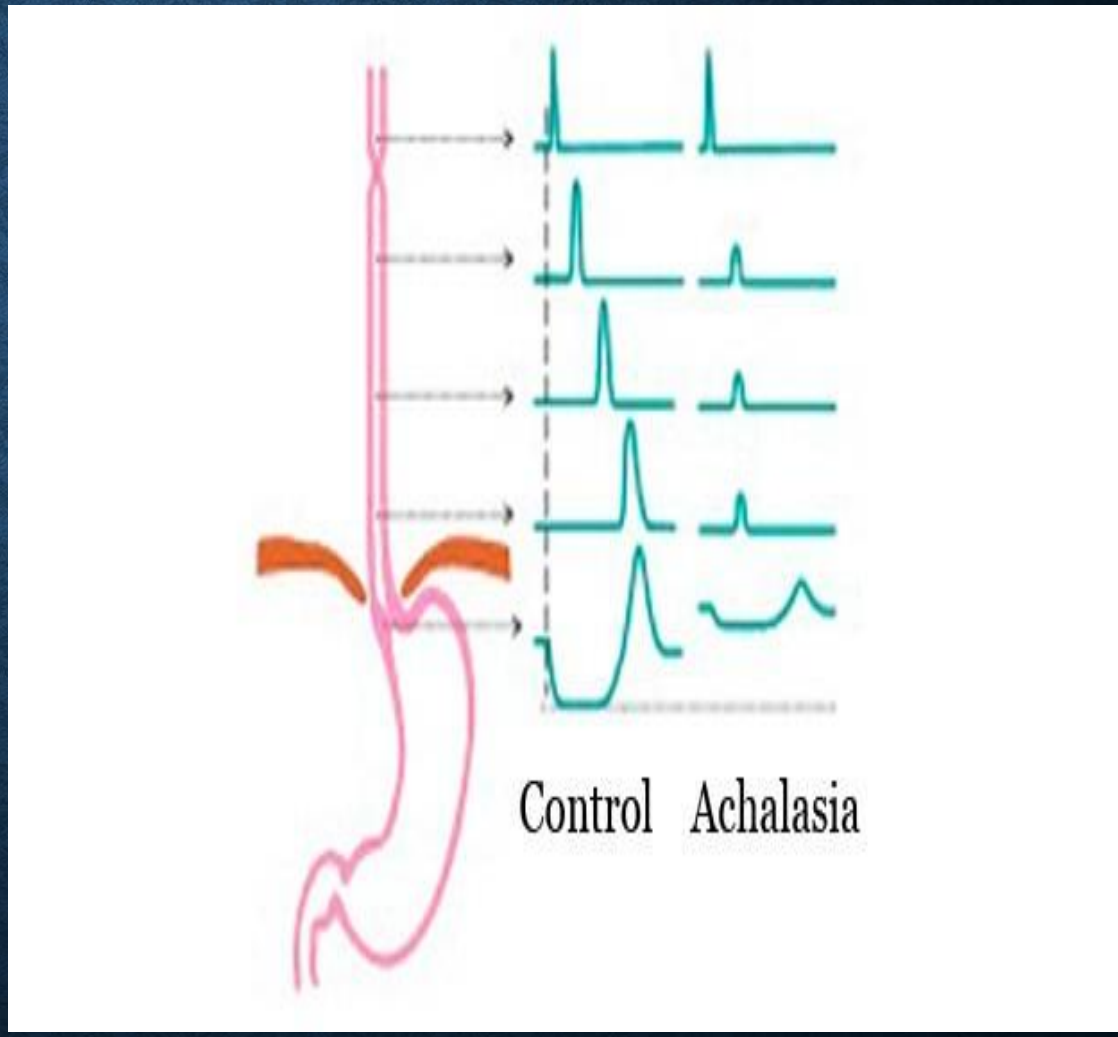






MANOMETRIC FEATURES

- Incomplete LES relaxation
- Elevated resting pressure (>45 mmHg)
- Aperistalsis of esophageal body



TREATMENT

- Achalasia is a chronic condition without cure
- *Goal of treatments:*
- *reduce LES pressure and*
- *increase stomach emptying*

PHARMACOLOGIC THERAPY

- Is the least effective therapy
- CCB and long acting nitrates are the MC medications to use
- Nifedipine, it should be used (10–30 mg) sublingually 30–45 min before meals for best response

- Sublingual isosorbide dinitrate
- Effective in 30–65%
- 5 mg only 10–15 min before meals.

- **THE PHOSPHODIESTERASE-5-INHIBITOR, SILDENAFIL, HAS ALSO BEEN SHOWN TO LOWER THE LES IN ACHALASIA.**

- Headache, hypotension, and pedal edema are common limiting factors in their use, also they do not provide complete relief of symptoms

- Reserved for patients with achalasia who
- 1. Cannot or refuse to undergo more definitive therapies (PD or surgical myotomy)
- 2.Who have failed botulinum toxin injections.

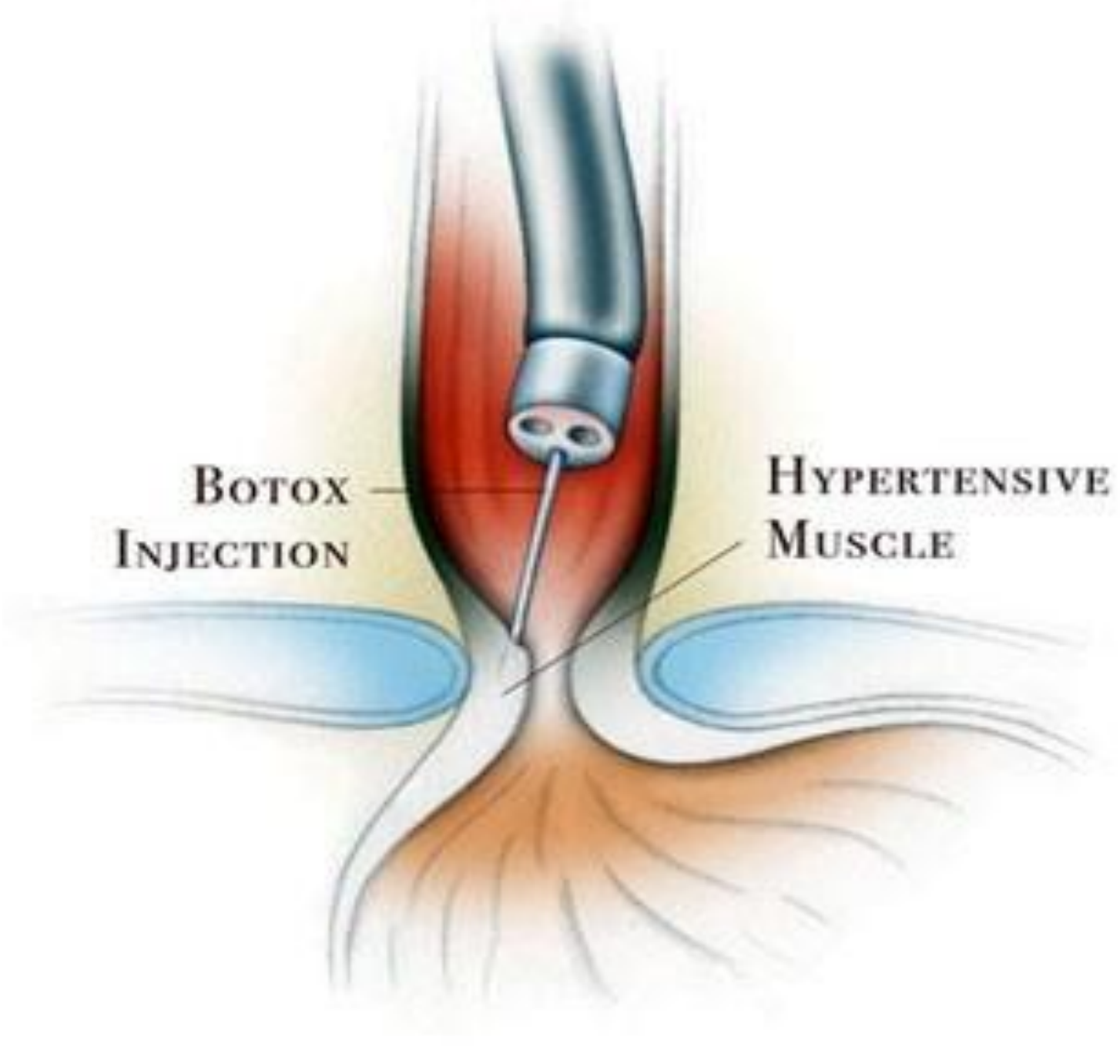
BOTULINUM TOXIN

- prevents ACH release at NM junction

- Approximately 50% of patients relapse and require repeat treatments at 6–24-month intervals

λSERIOUS SIDE EFFECTS ARE UNCOMMON

- λ 16–25% rate of developing chest pain and rare complications, such as mediastinitis and allergic reactions related to egg protein


BOTOX
INJECTION
HYPERTENSIVE
MUSCLE

PNEUMATIC DILATATION

- Is the most effective nonsurgical option

- All patients considered for PD must also be candidates for surgical intervention in the event of esophageal perforation needing repair.

- **After dilation radiographic testing by gastrograffin study should be done to exclude esophageal perforation.**

- Vomiting after procedure is possible.

- Developing severe chest pain with or without fever after discharge needs an immediate medical attention.

- Efficacy ranging from 32-98%
- Major complication: perforation with incidence of 1-5%
- Success increases with repeat dilatations

MYOTOMY

- Usually performed in conjunction with fundoplication
  - >90% initial response; 85% at 10 years; 70% at 20 years (85% at 5 years with min. inv. techniques)
    - <1% mortality; <10% major morbidity

ESOPHAGECTOMY

- Indication:

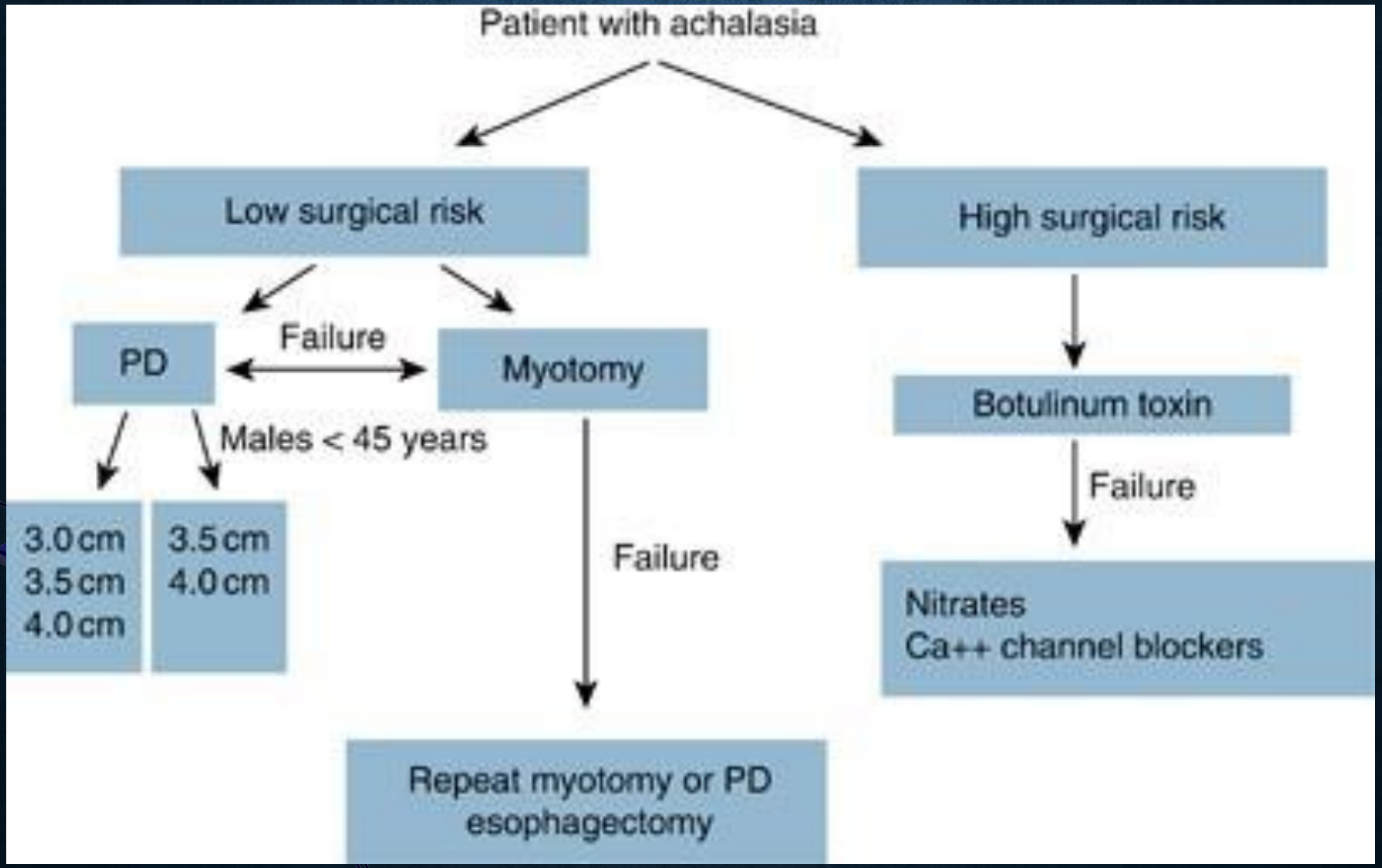
1. End-stage” achalasia or sigmoid esophagus

- PD may be less effective

- A surgical myotomy may be an initial approach before consideration for esophagectomy.

2.WHO HAVE FAILED PD AND/OR MYOTOMY AND WHO ARE GOOD CANDIDATES FOR SURGERY

- λDysphagia requiring dilation may occur in up to 50% of patients after esophagectomy.


Patient with achalasia
Low surgical risk
High surgical risk
PD
Failure
Myotomy
Botulinum toxin
Males < 45 years
Failure
3.0 cm
3.5 cm
4.0 cm
3.5 cm
4.0 cm
Failure
Nitrates
Ca++ channel blockers
Repeat myotomy or PD
esophagectomy

SPASTIC MOTILITY DISORDERS OF THE ESOPHAGUS

SPASTIC MOTILITY DISORDERS OF THE ESOPHAGUS

- Diffuse Esophageal Spasm
- Nutcracker Esophagus

EPIDEMIOLOGY

- Any age (mean 40 yrs)
- Female > Male

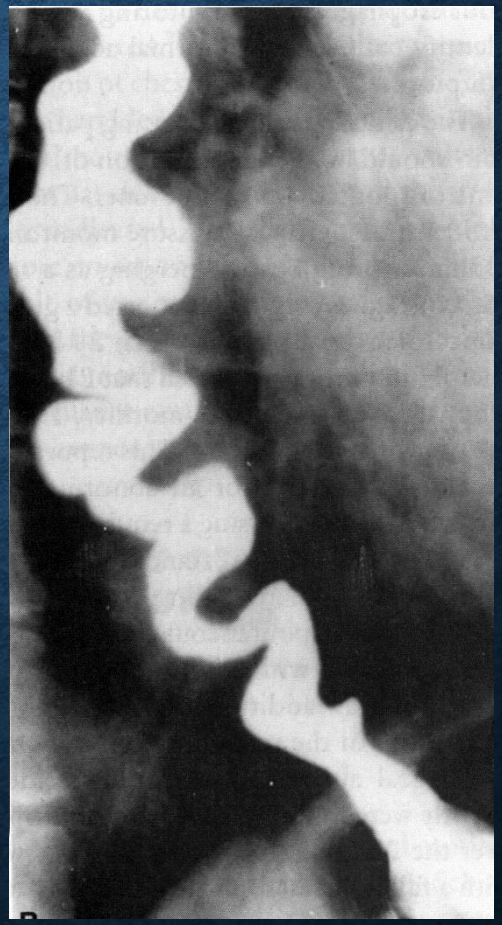
CLINICAL PRESENTATION

- Dysphagia to solids and liquids
  - intermittent and non-progressive
  - present in 30-60%, more prevalent in DES (in most studies)

- Chest Pain
  - swallowing is not necessarily impaired
  - can mimic cardiac chest pain

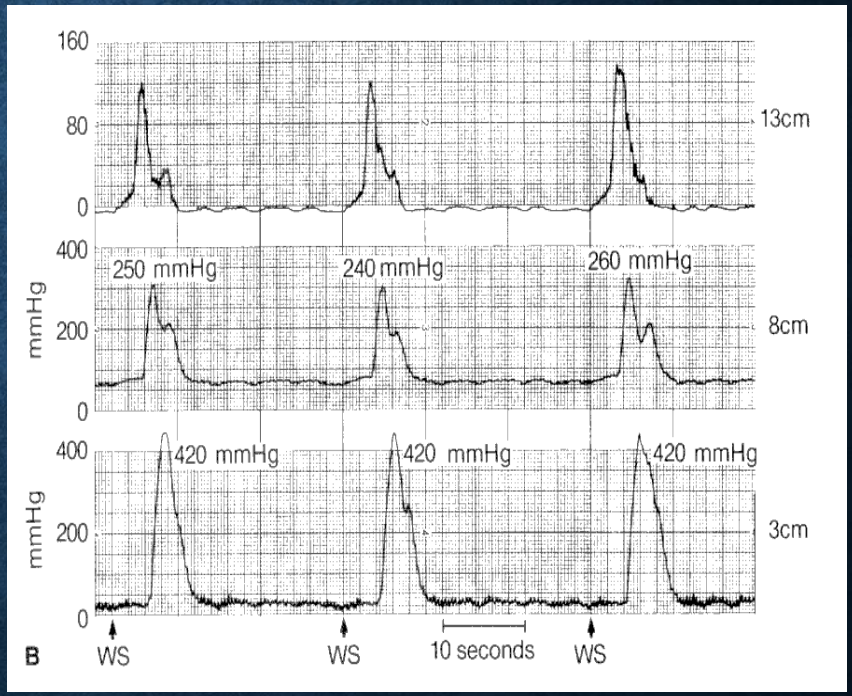
DIFFUSE ESOPHAGEAL SPASM

- Manometry shows intermittent , high amplitude (>30mmHg) , simultaneous and non-peristaltic contractions in response to swallowing.
- Diagnosis is made by clinical presentation, and typical findings on barium swallow and/or manometry following exclusion of other disorders.



NUTCRACKER ESOPHAGUS

- high pressure peristaltic contractions
  - avg pressure in wet swallows is >220 mm Hg
- 33% have long duration contractions (>6 sec)



TREATMENT

- Symptomatic relief
- CCBs are first-line treatment
- Trazodone and imipramine could be helpful
- Botox injection maybe considered when these therapies are unsuccessful

HYPOMOTILTY DISORDERS

- primary (idiopathic)
  - defined as
    - low contraction wave pressures (<30 mm Hg)
    - aging produces gradual decrease in contraction strength

HYPOMOTILTY DISORDERS

- secondary
  - scleroderma
    - in >75% of patients
    - aperistalsis in manometry
    - Smooth muscle atrophy and fibrosis◊loss of peristalsis and weakening of LES with reflux
  - other “connective tissue diseases”
    - CREST
    - polymyositis & dermatomyositis
  - diabetes
    - 60% with neuropathy have abnormal motility on testing (most asx)
  - other
    - hypothyroidism, alcoholism, amyloidosis, narcotics

EOSINOPHILIC ESOPHAGITIS

- Prevalence is 1:1000
- More common in white male
- Consider in cases of Dysphagia and food impaction regardless of the presence or absence of heartburn

Atypical chest pain and heartburn that is refractory to PPI.

- History of food allergy, asthma, eczema or allergic rhinitis

- Endoscopic finings: multiple esophageal rings, linear furrows and punctate exudates

- Histologic confirmation: >15 eos per HPF

GERD MUST BE EXCLUDED BY AMBULATORY PH MONITORING WITH LACK OF RESPONSE TO A THERAPEUTIC TRIAL OF A PPI TWICE A DAY FOR 6 WEEKS.

TREATMENT

-Swallowed aerosolized corticosteroid

-In refractory cases may need a combination of esophageal dilation, systemic corticosteroids, or a food elimination diet

PILL INDUCED ESOPHAGITIS

- Symptoms: odynophagia, dysphagia, and sometimes retrosternal chest pain.
- Tetracycline, iron sulfate, bisphosphonates, potassium, NSAIDs, and quinidine

- Diagnosis is suspected by medication review and is confirmed by endoscopy.

- Treatment typically includes temporary cessation of the culprit medication or taking the medication with a large bolus of water and avoiding a recumbent posture for 30 to 60 min.

Thank You

REFERENCES

- Harrison principle of internal medicine
- MKSAP
- Up-to-date
- Medscape
- American gastroenterology guidelines
- John Hopkin modules