UPPER GI PATHOLOGIES
OESOPHAGEAL MOTILITY DISORDERS
PEPTIC ULCER DISEASE

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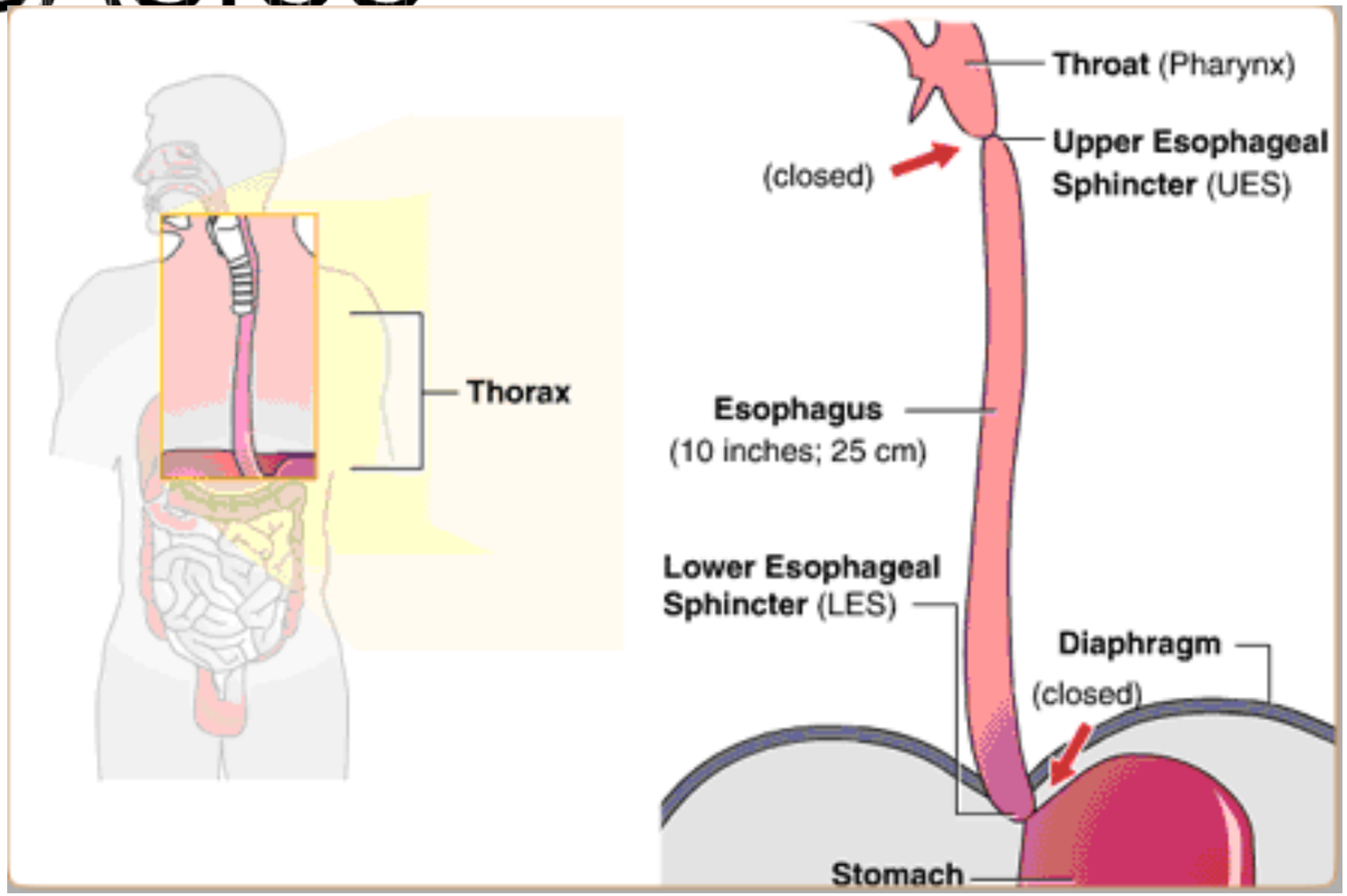
28 July 2016 MO teaching

THE OESOPHAGUS – BACK TO BASICS



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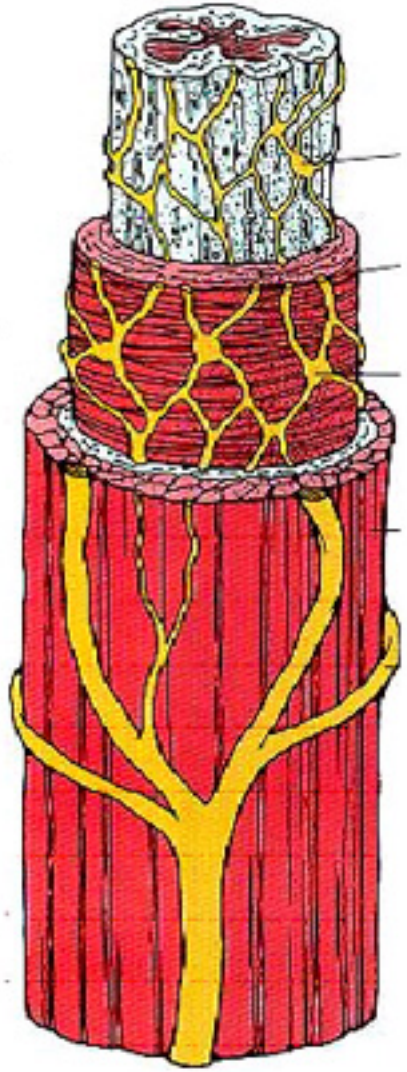


1 **MUCOSA and SUBMUCOSA**

2 **CIRCULAR MUSCLE: contraction causes an increase in luminal pressure**

3 **LONGITUDINAL MUSCLE: contraction causes shortening**

INNERVATION = (1) MEISSNER/SUBMUCOSAL PLEXUS; (2) AUERBACH/MYENTERIC PLEXUS; (3) VAGUS NERVE

OESOPHAGEAL MOTILITY DISORDERS

CONTENTS

- Classfication of oesophageal motility disorders
- Symptoms
- Investigations
- Special focus:
  - Achalasia
  - Diffuse oesophageal spasm

CLASSIFICATION

PRIMARY

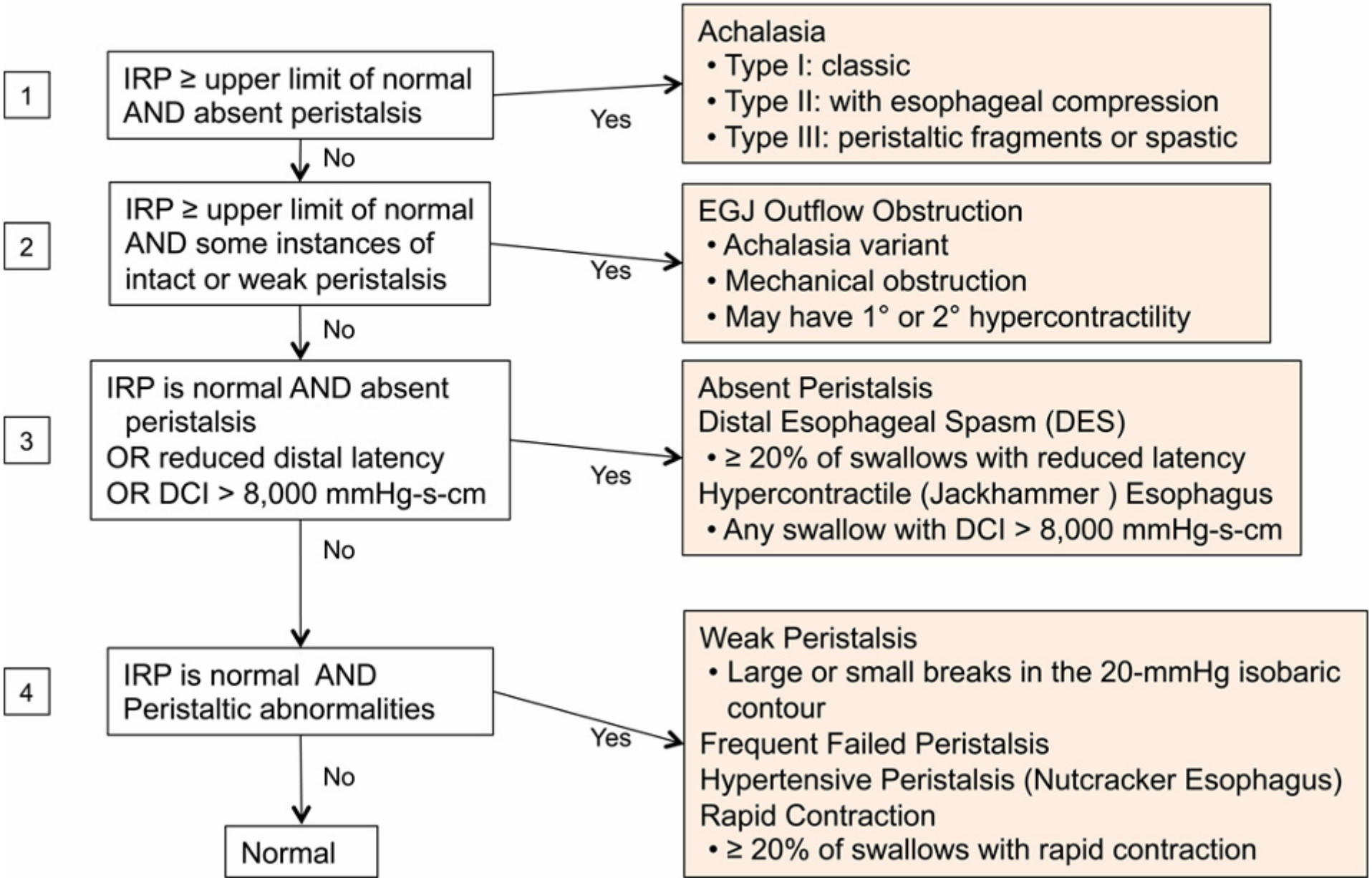
- Achalasia
- Discoordinated motility
  - Diffuse oesophageal spasm
- Hypercontracting oesophagus
  - Hypertensive peristalsis
  - Hypertensive LES
- Hypocontracting oesophagus
  - Ineffective oesophageal motility
  - Hypotensive LES

SECONDARY

- Systemic sclerosis
- Diabetes mellitus
- Chagas disease
- Chronic GERD

Hierarchical Analysis of Esophageal Motility

The Chicago Classification

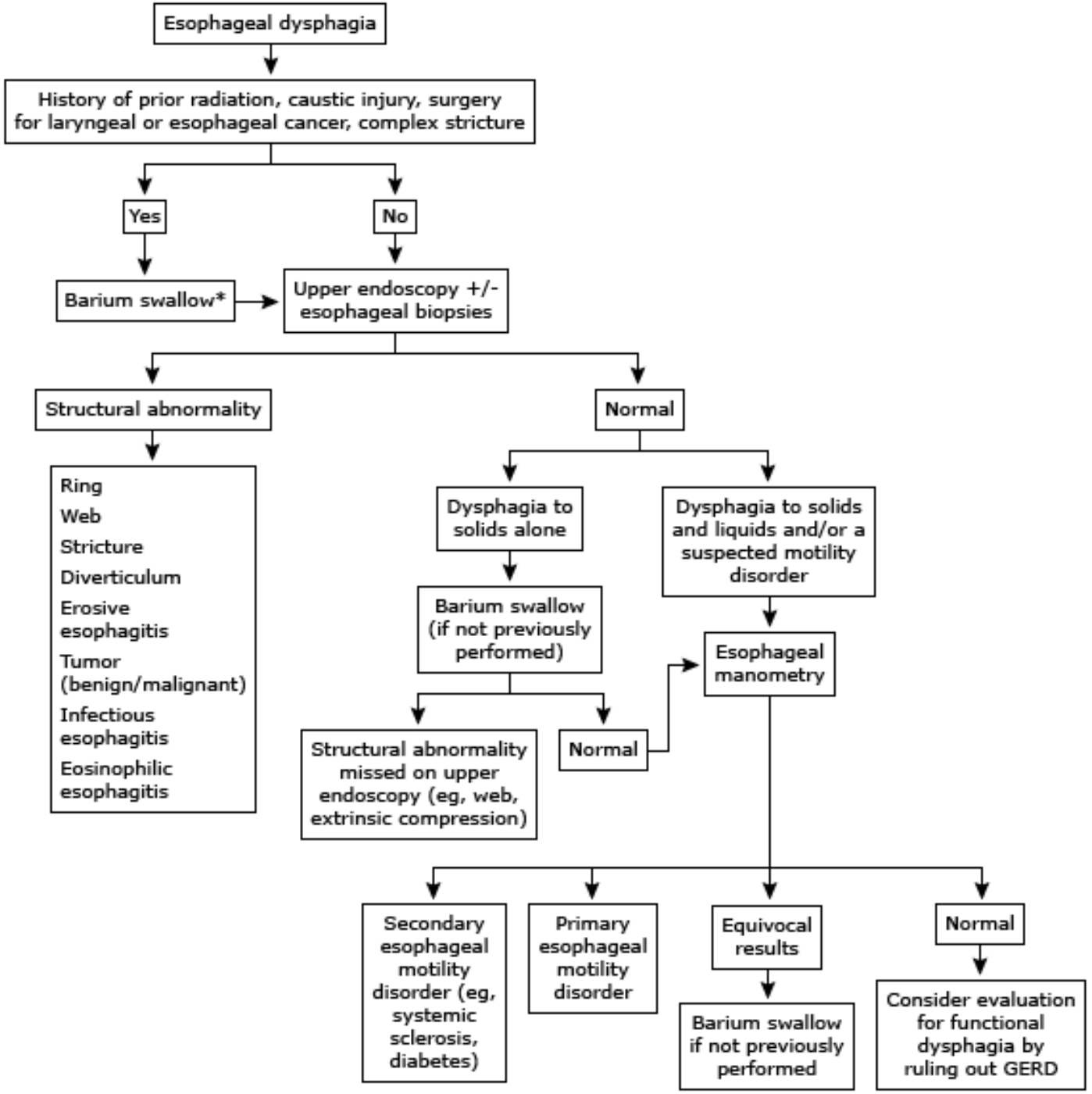


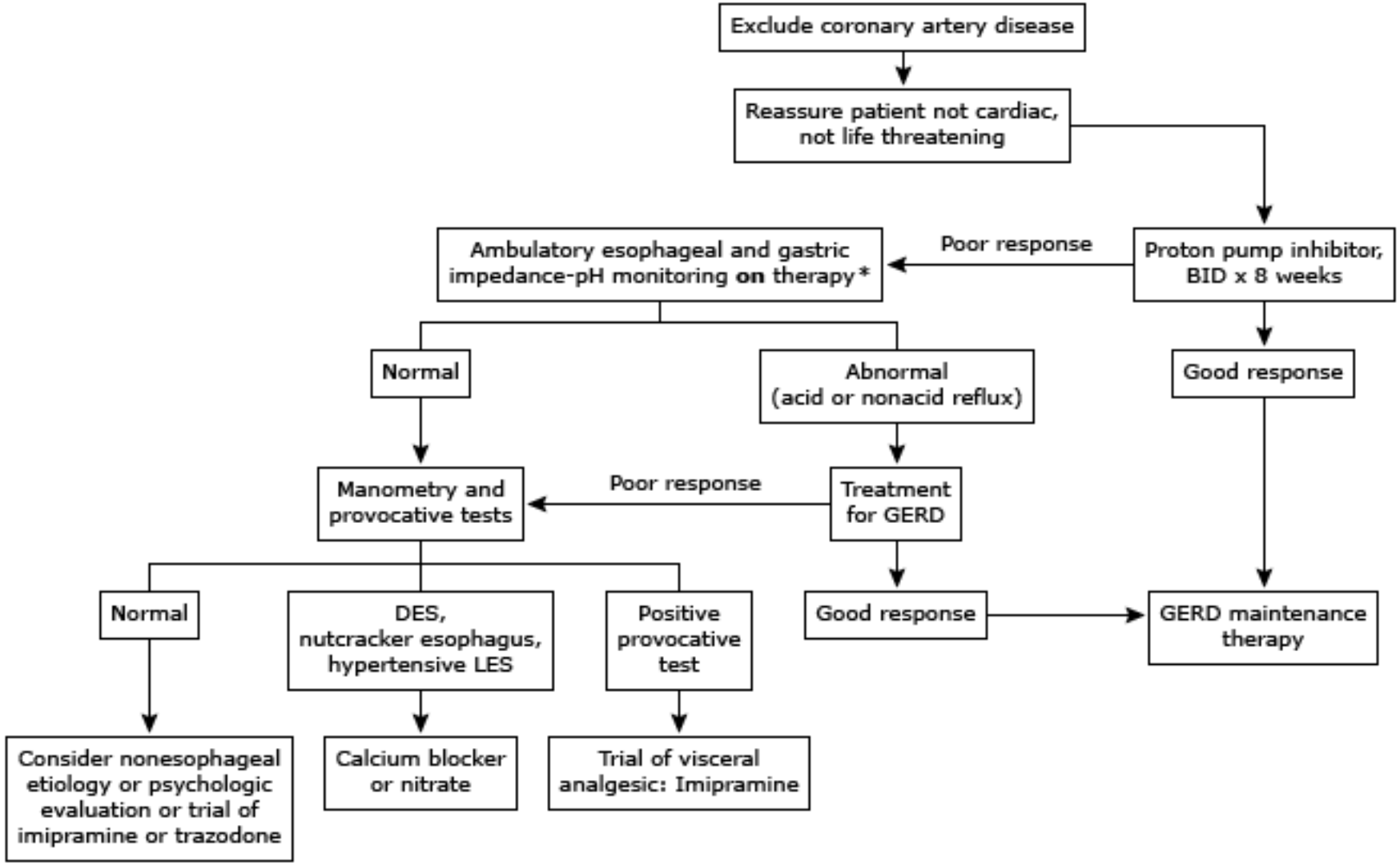
WHEN TO SUSPECT?

- Dysphagia
  - Both solids and liquids
- Chest pain
  - Retrosternal, non cardiac
- Refractory GERD symptoms
  - Regurgitation, heartburn

WHAT DO WE DO?

- Oesophagogastroduodenoscopy (OGD)
- Barium swallow
- Oesophageal manometry


Esophageal dysphagia
History of prior radiation, caustic injury, surgery for laryngeal or esophageal cancer, complex stricture
Yes
No
Barium swallow*
Upper endoscopy +/- esophageal biopsies
Structural abnormality
Normal
Ring
Web
Stricture
Diverticulum
Erosive esophagitis
Tumor (benign/malignant)
Infectious esophagitis
Eosinophilic esophagitis
Dysphagia to solids alone
Dysphagia to solids and liquids and/or a suspected motility disorder
Barium swallow (if not previously performed)
Esophageal manometry
Structural abnormality missed on upper endoscopy (eg, web, extrinsic compression)
Normal
Secondary esophageal motility disorder (eg, systemic sclerosis, diabetes)
Primary esophageal motility disorder
Equivocal results
Normal
Barium swallow if not previously performed
Consider evaluation for functional dysphagia by ruling out GERD


Exclude coronary artery disease
Reassure patient not cardiac, not life threatening
Proton pump inhibitor, BID x 8 weeks
Poor response
Ambulatory esophageal and gastric impedance-pH monitoring on therapy*
Good response
Normal
Abnormal (acid or nonacid reflux)
Poor response
Manometry and provocative tests
Treatment for GERD
Normal
DES, nutcracker esophagus, hypertensive LES
Positive provocative test
Good response
GERD maintenance therapy
Consider nonesophageal etiology or psychologic evaluation or trial of imipramine or trazodone
Calcium blocker or nitrate
Trial of visceral analgesic: Imipramine

ACHALASIA

Pathophysiology → Symptoms → Diagnosis → Treatment

- Inflammatory degeneration of neurons in esophageal wall > ganglion cells in myenteric plexus
- Preferentially involves nitric oxide producing inhibitory neurons
- Spares the cholinergic neurons that result in LES contraction

- Largely idiopathic
- Secondary causes (pseudoachalasia)
  - Chagas disease: protozoan infxn with Trypanosoma cruzi
  - Malignancy: invasion of oesophageal neural plexus OR paraneoplastic syndrome with release of unknown humoral factors
  - Amyloidosis, sarcoidosis etc.

ACHALASIA

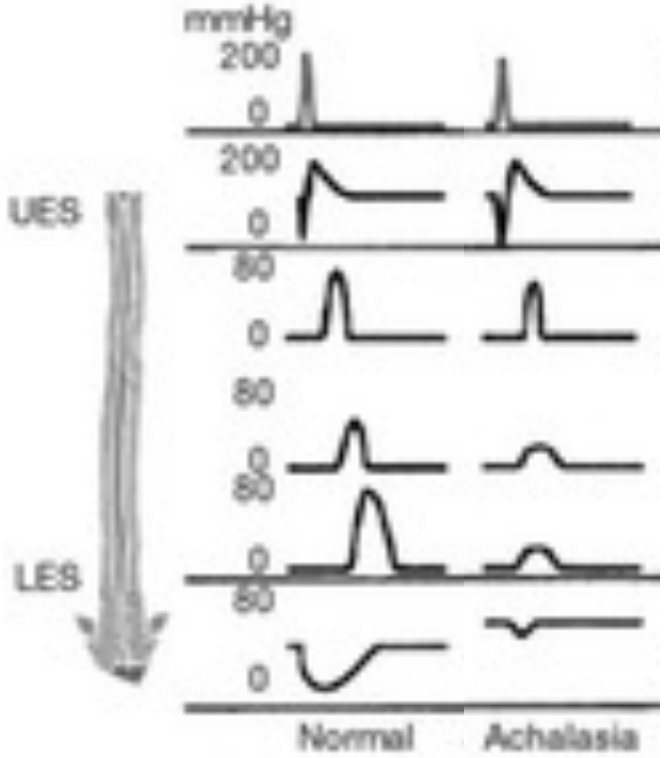
Pathophysiology → **Symptoms** → Diagnosis → Treatment

- Esophageal dysphagia for both solids and liquids
- Retrosternal chest pain
- Heartburn or regurgitation
- Aspiration
- Weight loss

ACHALASIA

Pathophysiology → Symptoms → **Diagnosis** → Treatment

- Barium swallow: "bird's beak" appearance
- Manometry:
  - Failure of LES relaxation (<75%)
  - High LES resting tone (>100mmHg)
  - Aperistalsis of esophagus body
- Biopsy: absence of myenteric nerve cells



ACHALASIA

Pathophysiology → Symptoms → Diagnosis → **Treatment**

Lifestyle changes

- Small frequent meals, avoid bedtime meals
- Raising head of bed to 35 degrees
- Avoid aggravating foods

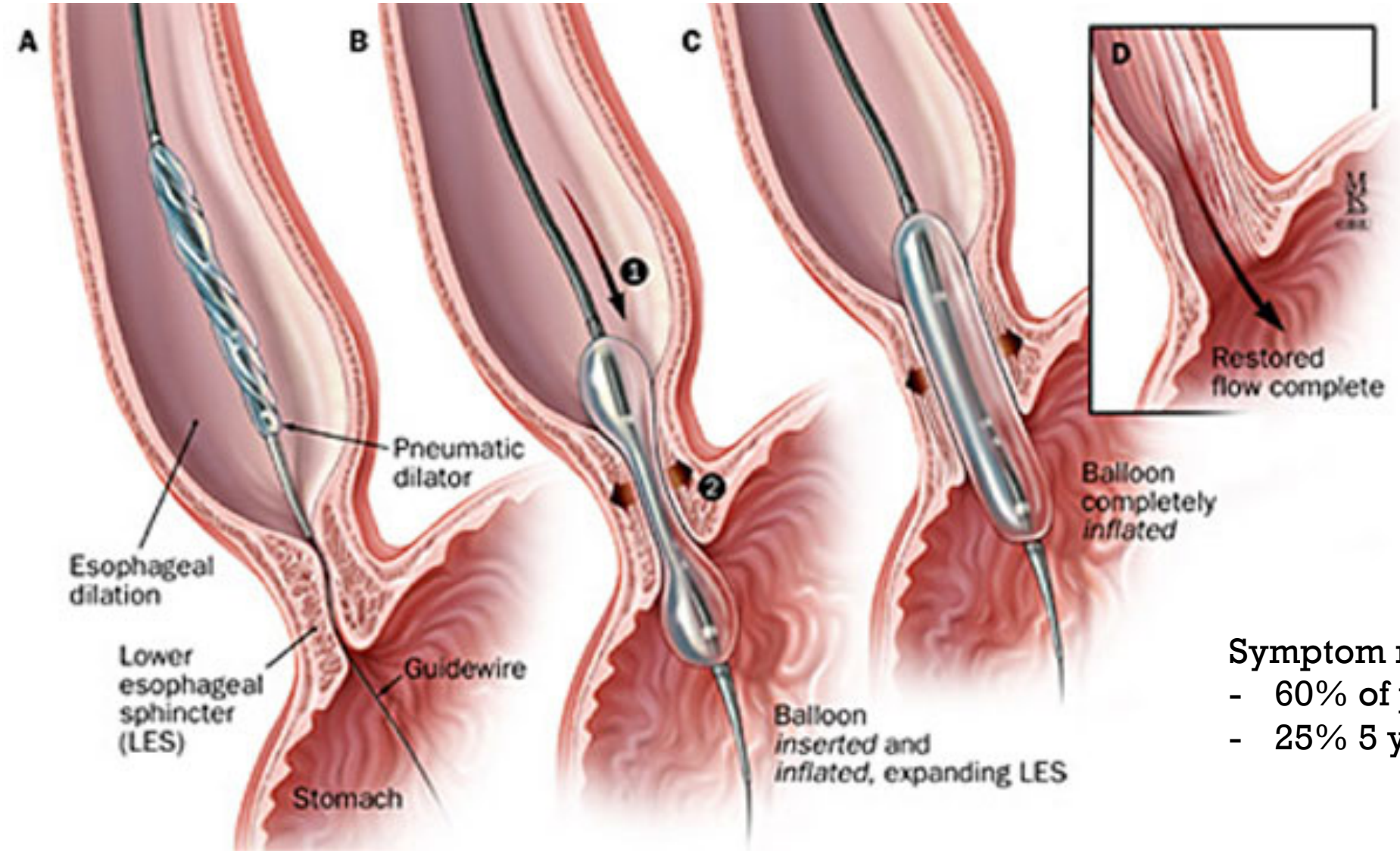
Medications

- Proton pump inhibitors
- Calcium channel blockers
- Nitrates
- Botulinum toxin injection

Procedures

- Pneumatic balloon dilatation
- Peroral endoscopic myotomy (POEM)
- Heller esophago-myotomy with partial fundoplication

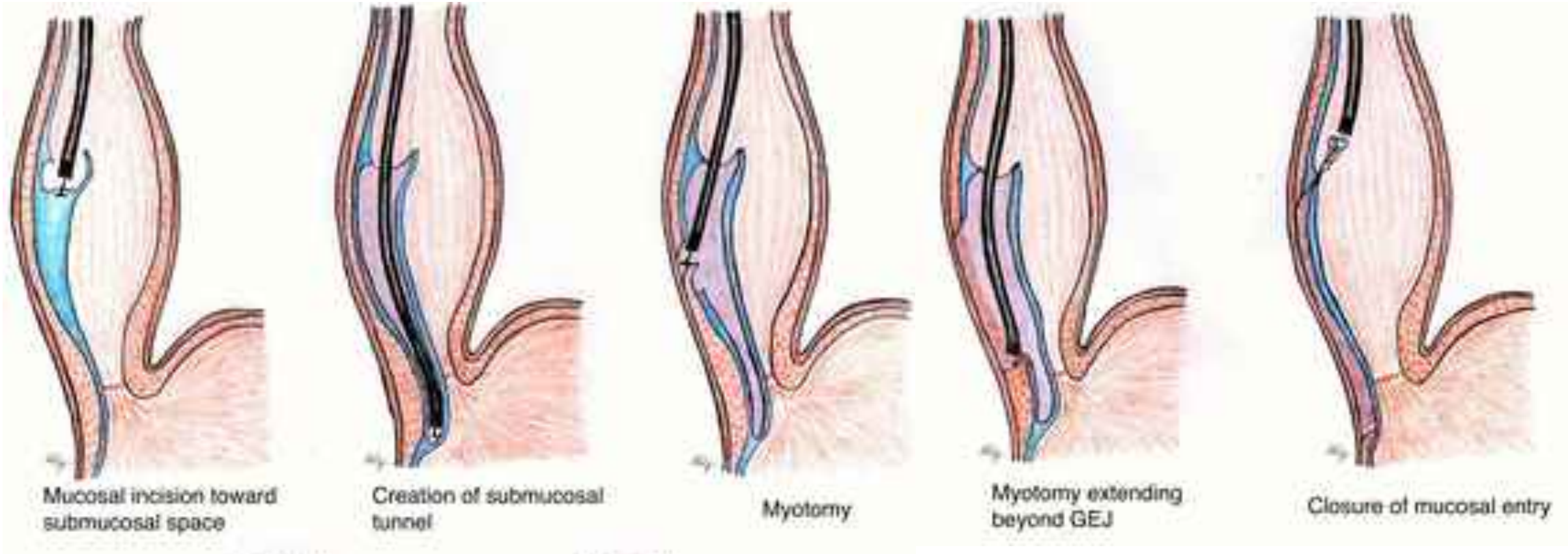
PNEUMATIC BALLOON DILATATION



Symptom relief

- 60% of patients in 1st year
- 25% 5 years after procedure

PERORAL ENDOSCOPIC MYOTOMY

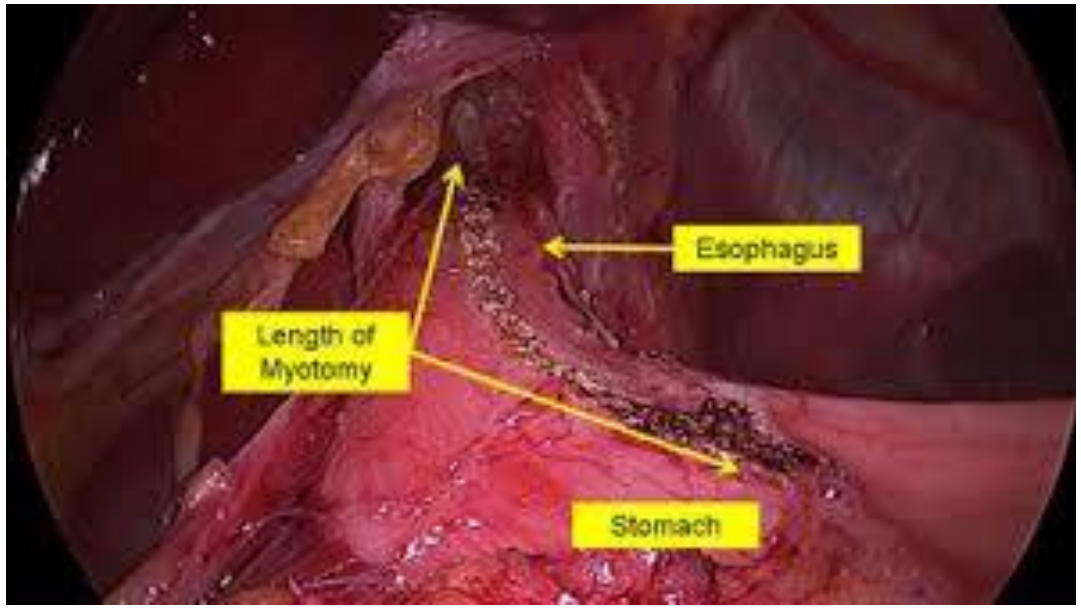


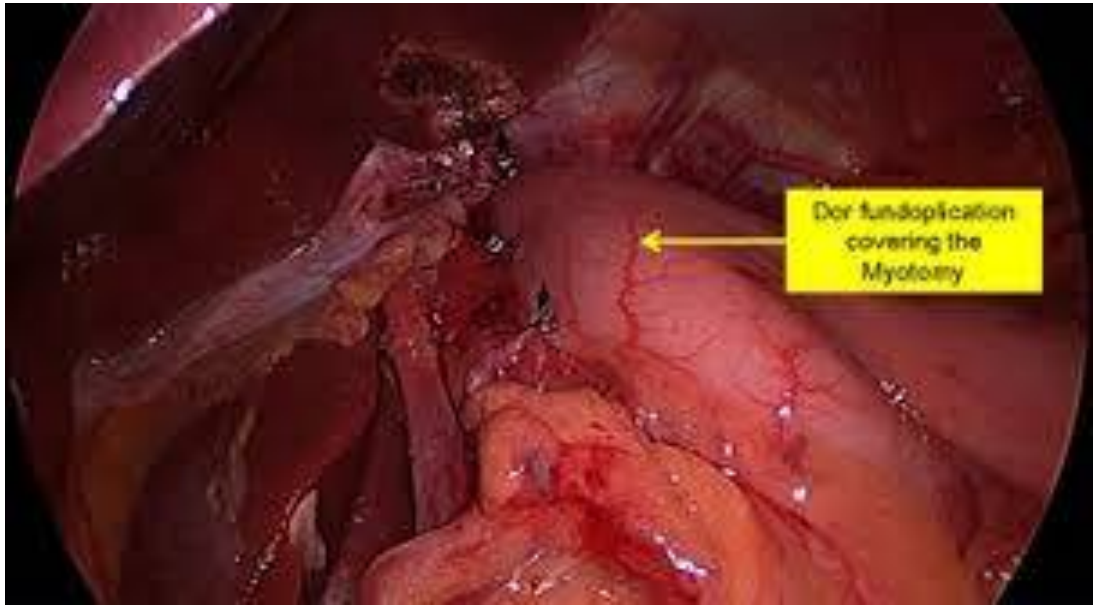
Symptom relief
- 80% of patients in 1st year

SE: 46% had mild GERD

LAPAROSCOPIC HELLER ESOPHAGOMYOTOMY

- Mobilisation of gastric fundus and mediastinal esophagus
- Myotomy (6cm on esophagus, 3cm on stomach)
  - Division of longitudinal then circular muscle fibers until bulging white mucosal plane
- Fundoplication (partial)
  - Dor: 180deg anterior wrap
  - Toupet: 270deg posterior wrap





DIFFUSE OESOPHAGEAL SPASM

Pathophysiology → Symptoms → Diagnosis → Treatment

- Impairment of inhibitory innervation -> premature and prolonged contractions of the oesophagus
- Malfunction of endogenous nitric oxide production/degradation

DIFFUSE OESOPHAGEAL SPASM

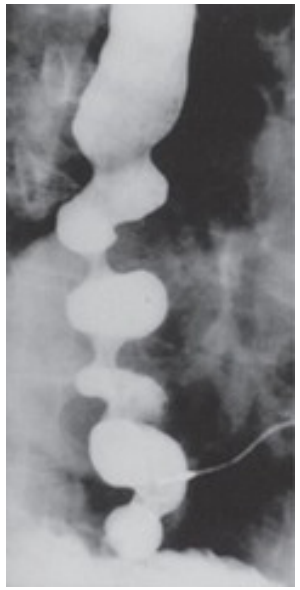
Pathophysiology → **Symptoms** → Diagnosis → Treatment

- Esophageal dysphagia for both solids and liquids
- Retrosternal chest pain
- Heartburn or regurgitation

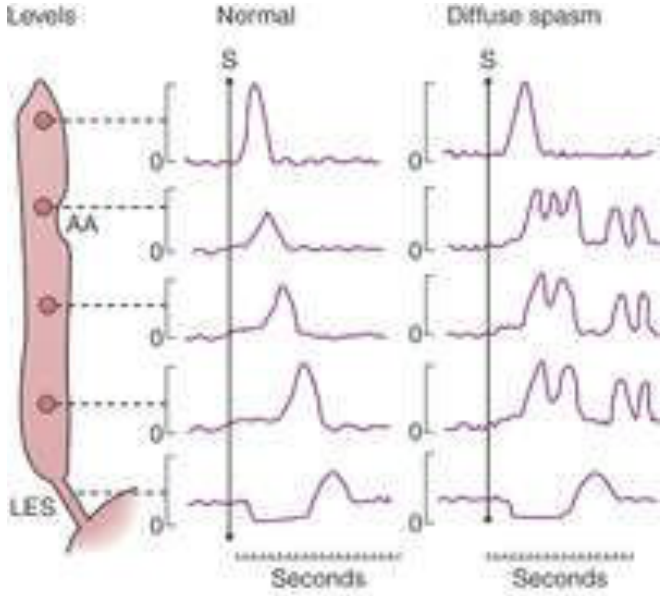
DIFFUSE OESOPHAGEAL SPASM

Pathophysiology → Symptoms → **Diagnosis** → Treatment

- Barium swallow: "corkscrew" appearance



- Manometry: increased simultaneous contractions (>20%, >30mmHg amplitude)



DIFFUSE OESOPHAGEAL SPASM

Pathophysiology → Symptoms → Diagnosis → **Treatment**

First line therapy:

- Calcium channel blockers: diltazem 180-240 mg daily
- Tricyclic antidepressants: imipramine 25-50mg ON

Second line therapy:

- Botulinum toxin
- Nitric oxide contributing drug: isosorbide 10mg or sildenafil 50mg ON

PEPTIC ULCER DISEASE

CONTENTS

- Introduction
- Pathophysiology
  - H.pylori and PUD
- Simple PUD
  - Presenting symptoms
  - Diagnosis
  - Management
- Complicated PUD
  - 5 'B's: **b**leed, **b**urst, **b**urrow, **b**lock, **b**ecome malignant
- Surgery in PUD

INTRODUCTION

- Break in the gastrointestinal mucosal that extends through the muscularis mucosae
- Annual incidence of 0.1 – 0.3 percent worldwide
  - 6 – 10 fold higher in patients with H. pylori infection

PATHOGENESIS

Aggravating factors

- Helicobacter pylori infection
- NSAIDs
- Gastric acid production
- Caustic agents

Protective factors

- Gastric mucosal barrier
- Mucosal blood flow
- Bicarbonate production
- Prostaglandin production

H. PYLORI AND PUD

- 80 – 95% of patients with duodenal ulcers have H.pylori infection
- 65 – 80% of patients with gastric ulcers have H.pylori infection
- Eradication of H.pylori reduces disease recurrence:
  - 6% in patients who underwent successful H.pylori treatment vs 67% in those without
- Pathogenesis of PUD:
  - Increased gastric acid production (via increased gastrin & decreased somatostatin)
  - Gastric metaplasia in D1 > provide foci of areas for H.pylori colonisation
  - Immune response > increased production of inflammatory cytokines
  - Downgrading of mucosal defense factors (e.g. bicarbonate production, growth factors)

SYMPTOMS

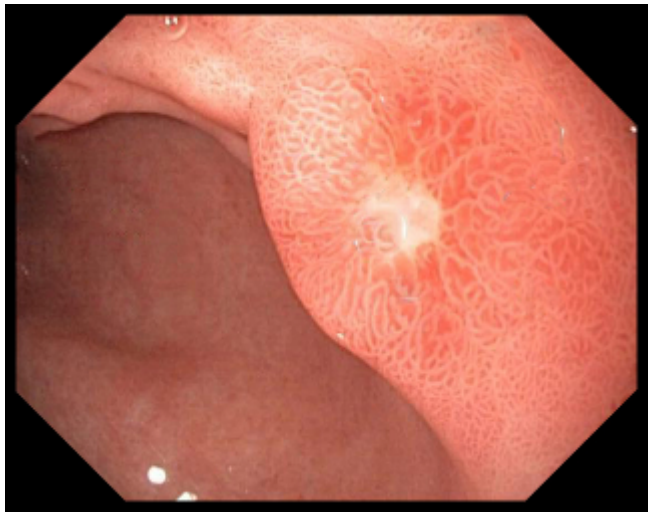
- Epigastric pain or discomfort
  - Gastric ulcers: worse with food
  - Duodenal ulcers: worse without food
- Early satiety, nausea

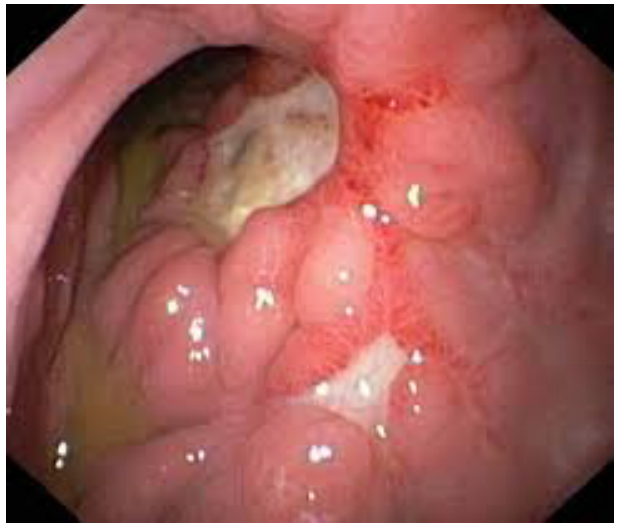
RED FLAGS

- GI bleeding > hemetemesis or malena, iron deficiency anemia
- Perforation > peritonism, shock
- Gastric outlet obstruction > vomiting, loss of weight, dysphagia/odynophagia
- Fistulisation > feculent vomiting, post prandial diarrhea
- Malignancy > lymphadenopathy, palpable mass, jaundice, weight loss

DIAGNOSIS

- Oesophagogastroduodenoscopy (OGD)
- Biopsy indications
  - All malignant looking ulcers
  - All gastric ulcers *controversial
  - Duodenal ulcers due to suspected Crohn's disease





Modified Johnson Classification of peptic ulcers

Type I: Ulcer along the lesser curve of stomach

Type II: Two ulcers present - one gastric, one duodenal/prepyloric

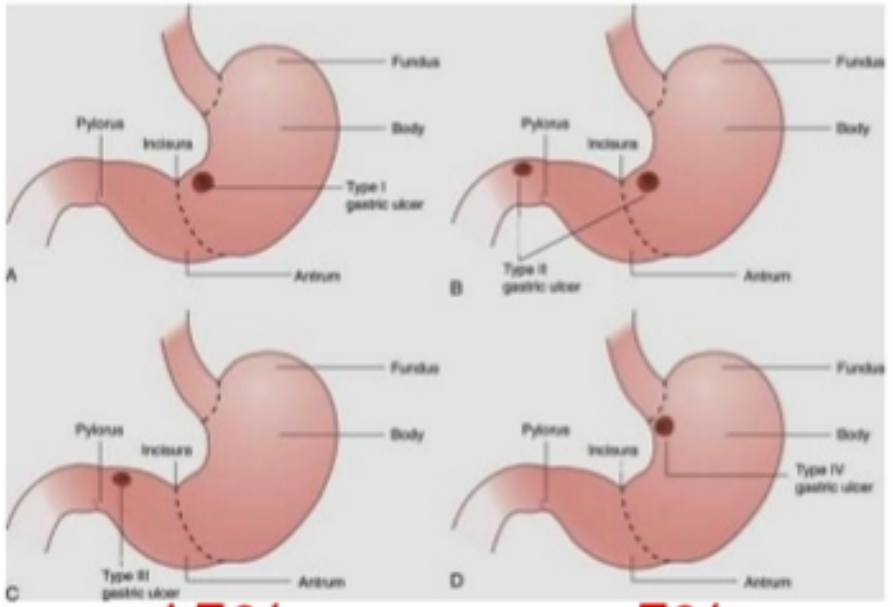
Type III: Prepyloric ulcer

Type IV: Proximal gastroesophageal ulcer

Type V: Anywhere (associated with chronic NSAID use)

55%

25%

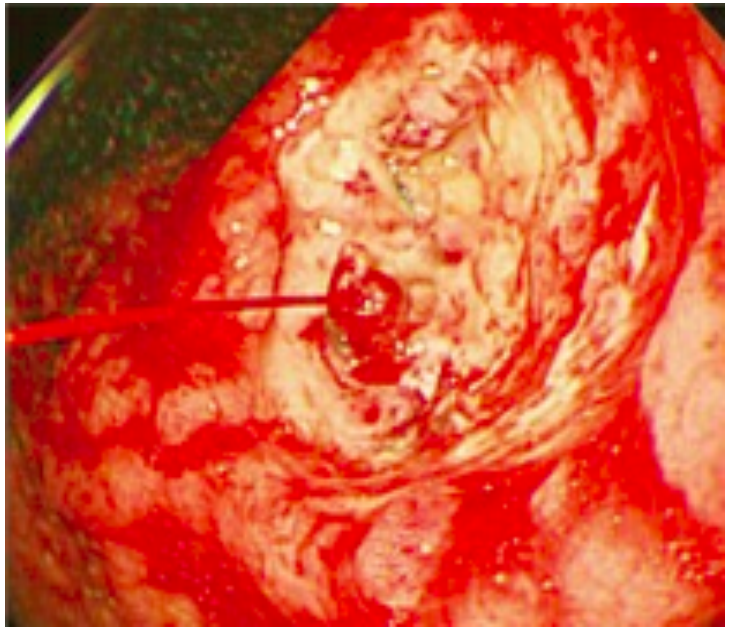
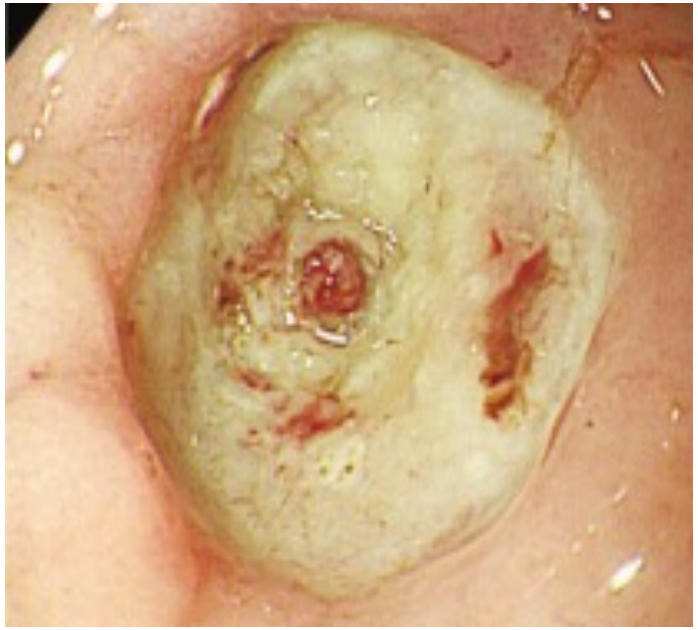
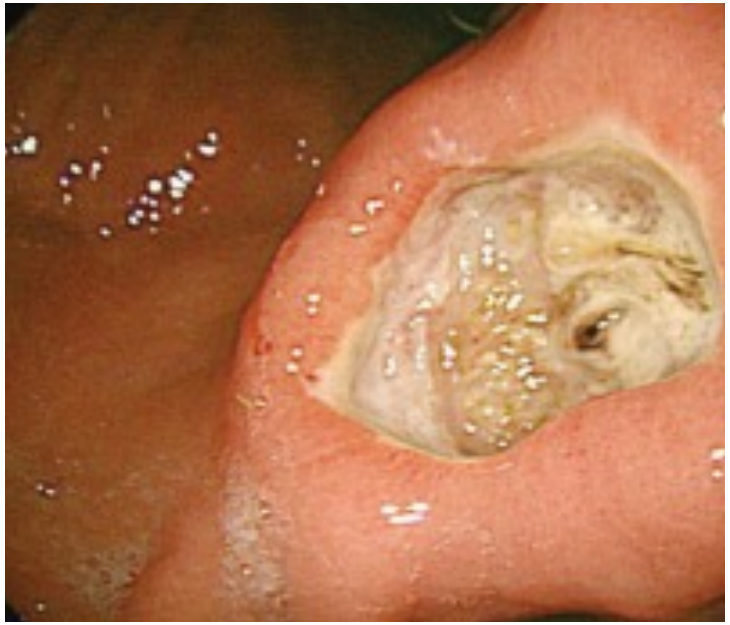
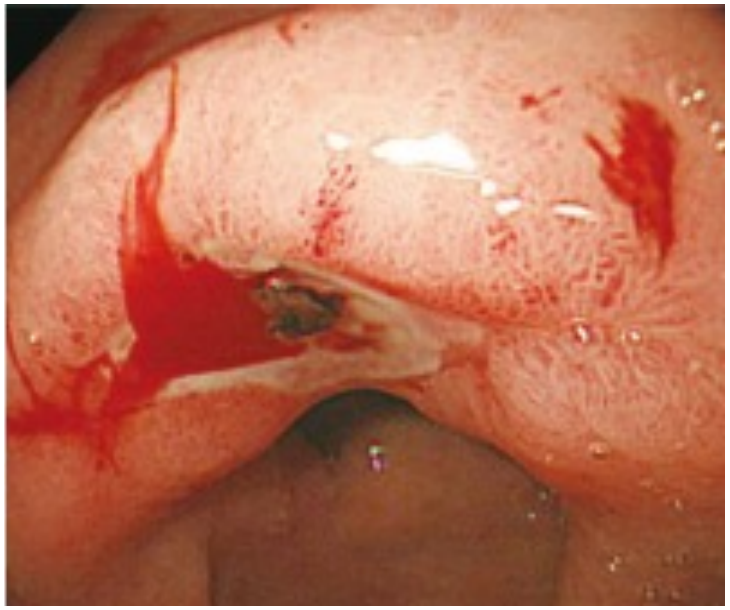
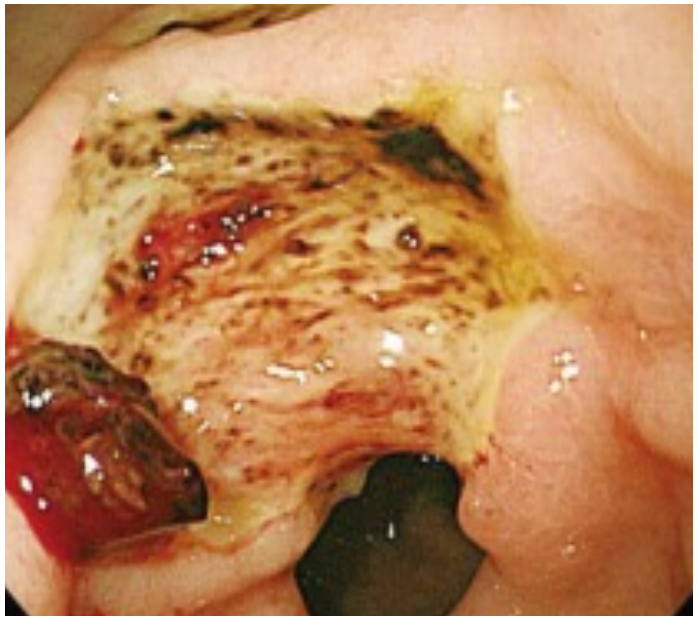
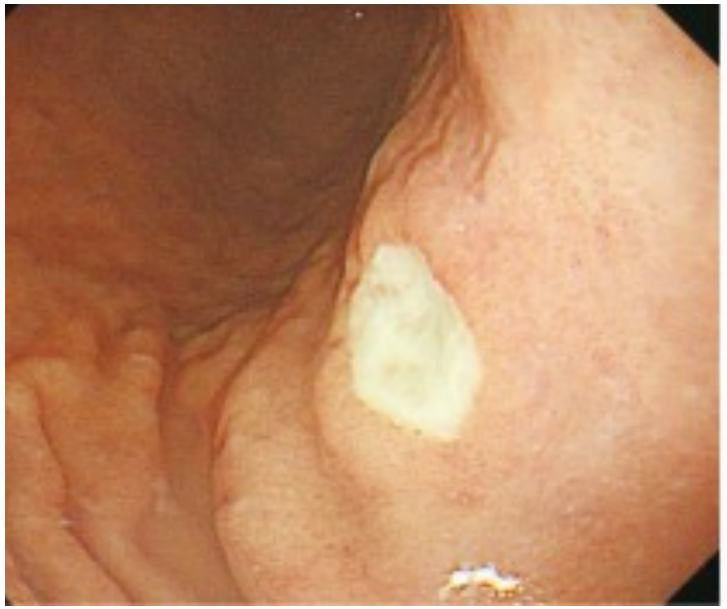


15%

5%

FORREST CLASSIFICATION

Forrest classification	Stigmata	Further bleeding (*N* =2,994)	Surgery for bleeding (*N* =1,499)	Mortality (*N*=1,387)
IA	Active spurting bleeding	55% (17–100%)	35% (20–69%)	11% (0–23%)
IB	Active oozing bleeding			
IIA	Non-bleeding visible vessel	43% (0–81%)	34% (0–56%)	11% (0–21%)
IIB	Adherent clot	22% (14–36%)	10% (5–12%)	7% (0–10%)
IIC	Flat pigmented spot	10% (0–13%)	6% (0–10%)	3% (0–10%)
III	Clean ulcer base	5% (0–10%)	0.5% (0–3%)	2% (0–3%)

TESTING FOR H. PYLORI

Endoscopic tests

- Biopsy urease test (CLO test)
  - From antrum or fundus
  - Beware false negatives
- Histology
- Bacterial culture

Non-invasive tests

- Urea breath test
- Serum serology test for IgG
- Stool antigen test

MANAGEMENT

- Stop aggravating drugs if possible e.g. NSAIDs, steroids
- Triple therapy for H.pylori eradication
  - Amoxicillin 1g BD, clarithromycin 500mg BD, omeprazole 20mg BD x14 days
- PPI therapy
  - Omeprazole (dose and duration dependent on ulcer type and location)
- Indications for maintenance PPI therapy
  - Persistent symptoms or recurrent ulcers
  - NSAIDs or H.pylori negative ulcers
  - Long term NSAID requirements
  - Failure of H.pylori eradication
  - Large >2cm ulcer or age >50yo

MANAGEMENT

- Follow up for H.pylori positive ulcers:
  - Confirm eradiation of H.pylori >4 weeks after triple therapy
  - Urea breath test or repeat OGD (if indicated for other reasons)
- Follow up endoscopy
  - Gastric ulcers: repeat OGD in 12 weeks to document ulcer healing
    - Persistent symptoms
    - Unclear etiology
    - Large gastric ulcer >2cm
    - Malignant features of ulcer
    - Biopsy not performed / inadequate sample during index OGD
    - Index OGD performed for bleeding PUD
    - Risk factors for gastric cancer
  - Duodenal ulcers: no need for repeat OGD unless symptoms persist or recur

PROGNOSIS

- 60 percent of PUDs heal spontaneously
- >90% of PUDs heal with H.pylori treatment
- Recurrence rate of 6-30% within 1st year despite H.pylori eradication
- 5-10% are refractory to PPI treatment
- Complication risks of 2-3% per year in patients with chronic PUD

REFRACTORY PUD

Exclude:

- H.pylori infections
- Chronic NSAID use
- Malignancy or other inflammatory conditions e.g. Crohns
- Zollinger-Ellinson syndrome and hyperparathyroidism
  - Fasting serum gastrin levels, serum calcium levels

Treat:

- Additional PPI therapy > omeprazole 40mg OM –BD x12 weeks
- Surgical management (last resort)

COMPLICATED PUD

- BLEEDING
- PERFORATION
- FISTULISATION
- OBSTRUCTION

COMPLICATED PUD

BLEEDING

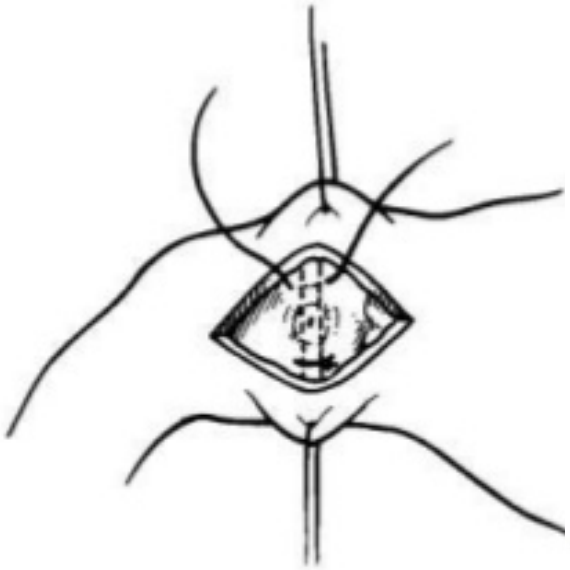
PERFORATION

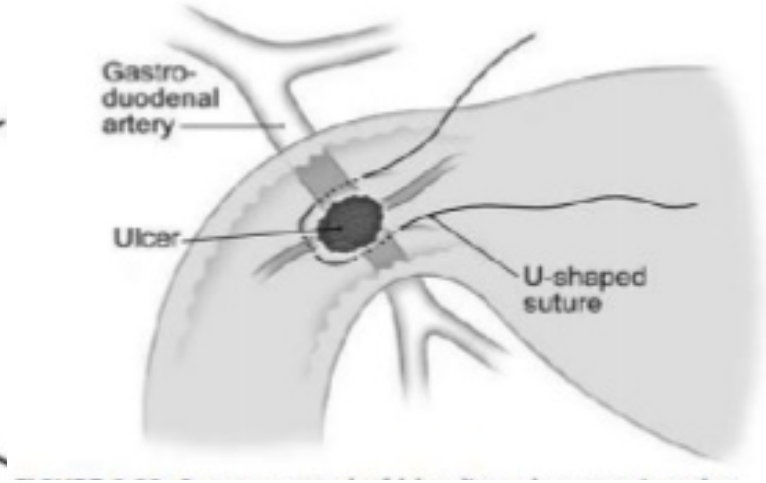
FISTULISATION

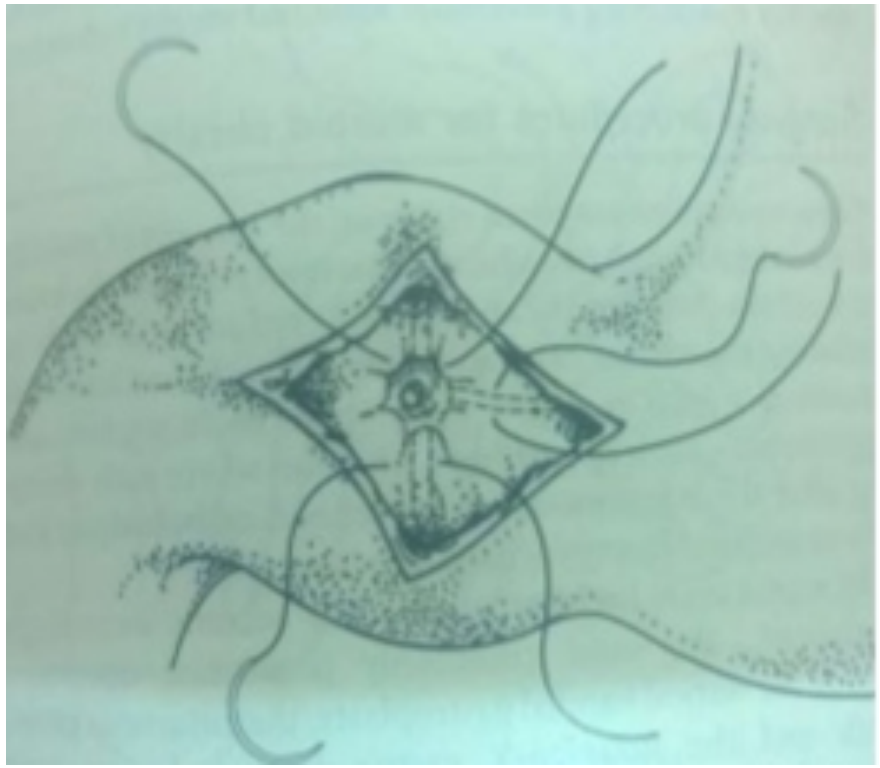
OBSTRUCTION

- Fluid resuscitation and transfusion
- Early high dose PPI – IV omeprazole 80mg bolus > 8mg/hr infusion
- OGD and endoscopic hemostasis – IV adrenaline + clipping + coagulation
- Interventional radiology – angioembolisation
- Surgery
  - Duodenal ulcer – duodenotomy, suture ligation of bleeding vessel (oversewing), KIV definitive surgery
  - Gastric ulcer – ulcerectomy / gastric resection, definitive acid reduction surgery

- Kocherise the duodenum
- Longitudinal duodenotomy
- Bimanual compression of gastroduodenal artery
- Three point ligation of GDA and transverse pancreatic branch







COMPLICATED PUD

BLEEDING

PERFORATION

FISTULISATION

OBSTRUCTION

- Resuscitate, IV fluids, NGT insertion
- IV PPI – omeprazole
- IV broad spectrum antibiotics – rocephin/flagyl

CONSERVATIVE

- Poor surgical candidates
- Sealed small perforation in stable patient
- Delayed presentation

*Consider percutaneous drainage

SURGICAL

-Majority of patients

-Duodenal:

- Omental patch repair
- Pyloroplasty incorporating perforation

-Gastric:

- Gastrectomy – partial versus total

COMPLICATED PUD

BLEEDING

PERFORATION

FISTULISATION

OBSTRUCTION

- Order of frequency: pancreas, biliary tract, liver, colon, vascular structures
- Dependent on ulcer location, type of fistula, patient demographics

COMPLICATED PUD

BLEEDING

PERFORATION

FISTULISATION

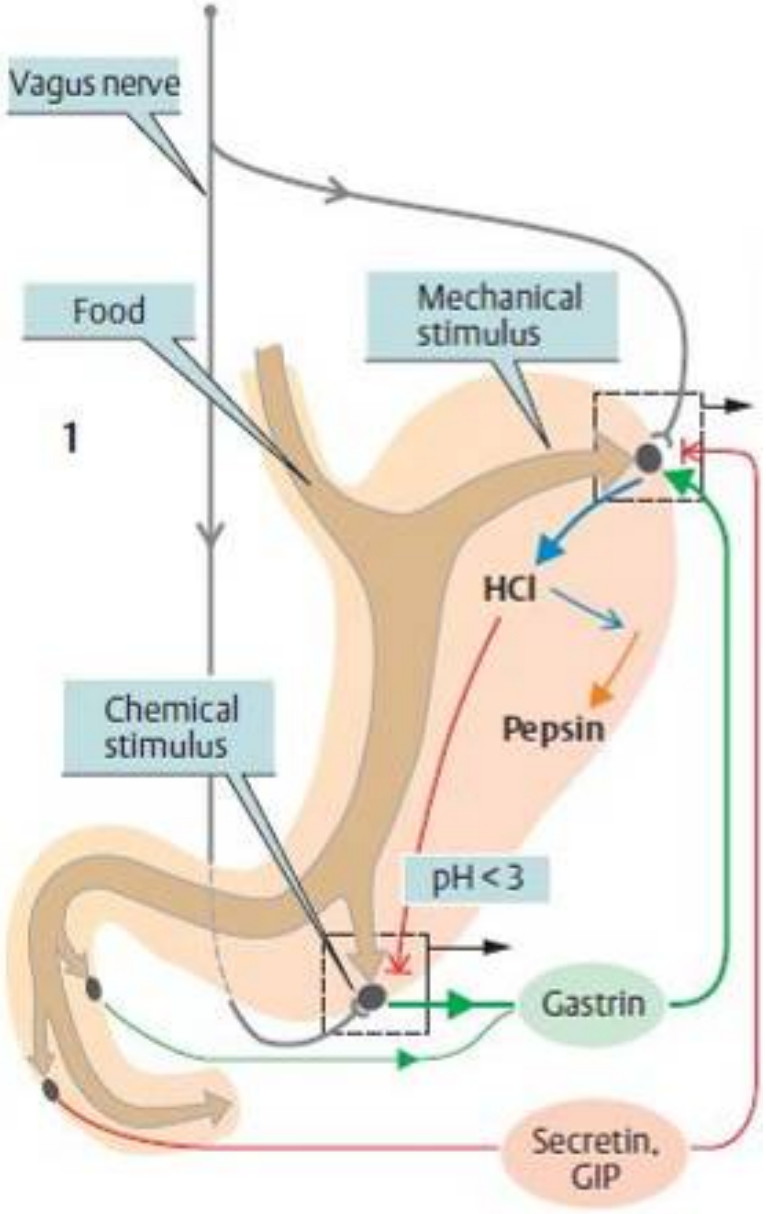
OBSTRUCTION

- NGT insertion for gastric decompression
- IV hydration, electrolyte replacement
- Biopsy TRO malignancy
- Surgery
  - Antrectomy/distal gastrectomy
  - Bypass surgery – gastrojejunostomy
  - Vagotomy and drainage (pyloroplasty)

SURGERY FOR PUD

- Management of ulcer related complications versus Definitive ulcer surgery

- Definitive ulcer surgery
  >> AIM – reduction of gastric acid secretion
  - Vagus nerves > vagotomy
  - Antrum hormonal stimulation > antrectomy
  - Gastric parietal cells > subtotal gastrectomy



SURGERY FOR PUD

Vagotomy ± gastric drainage procedures

Gastrectomy and reconstruction

SURGERY FOR PUD

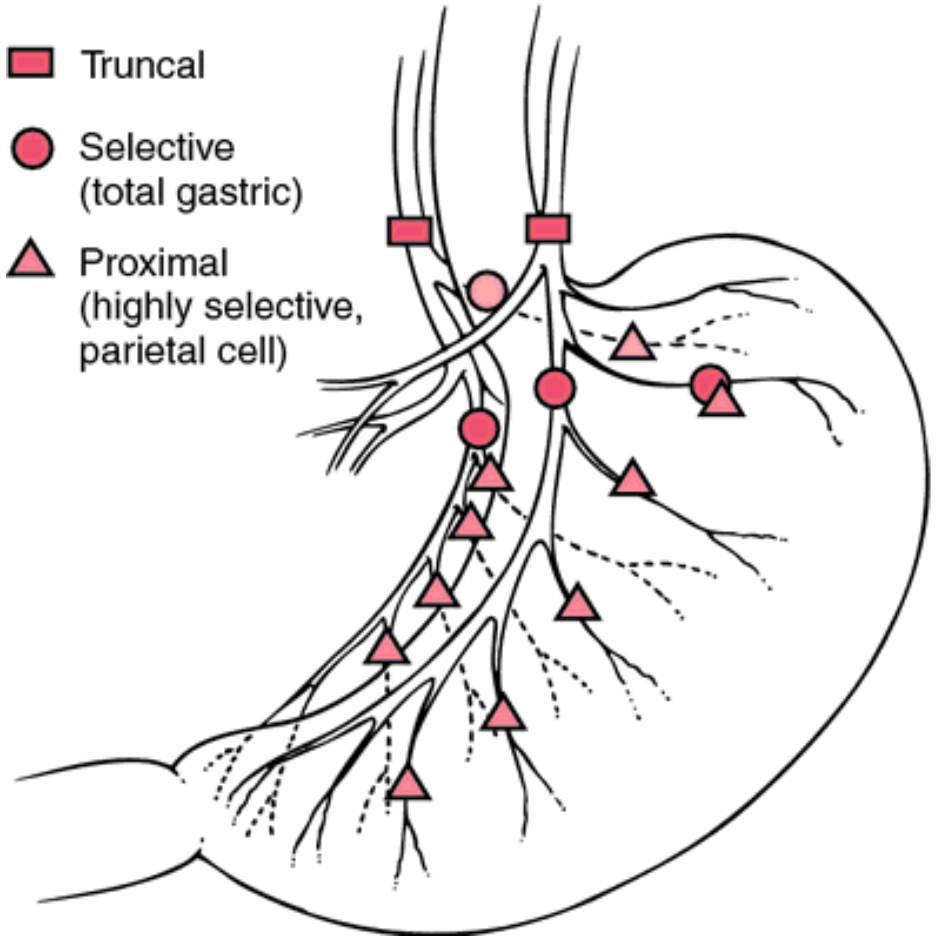
Vagotomy ± gastric drainage procedures

- Truncal vagotomy
- Selective vagotomy
- Highly selective vagotomy (HSV)

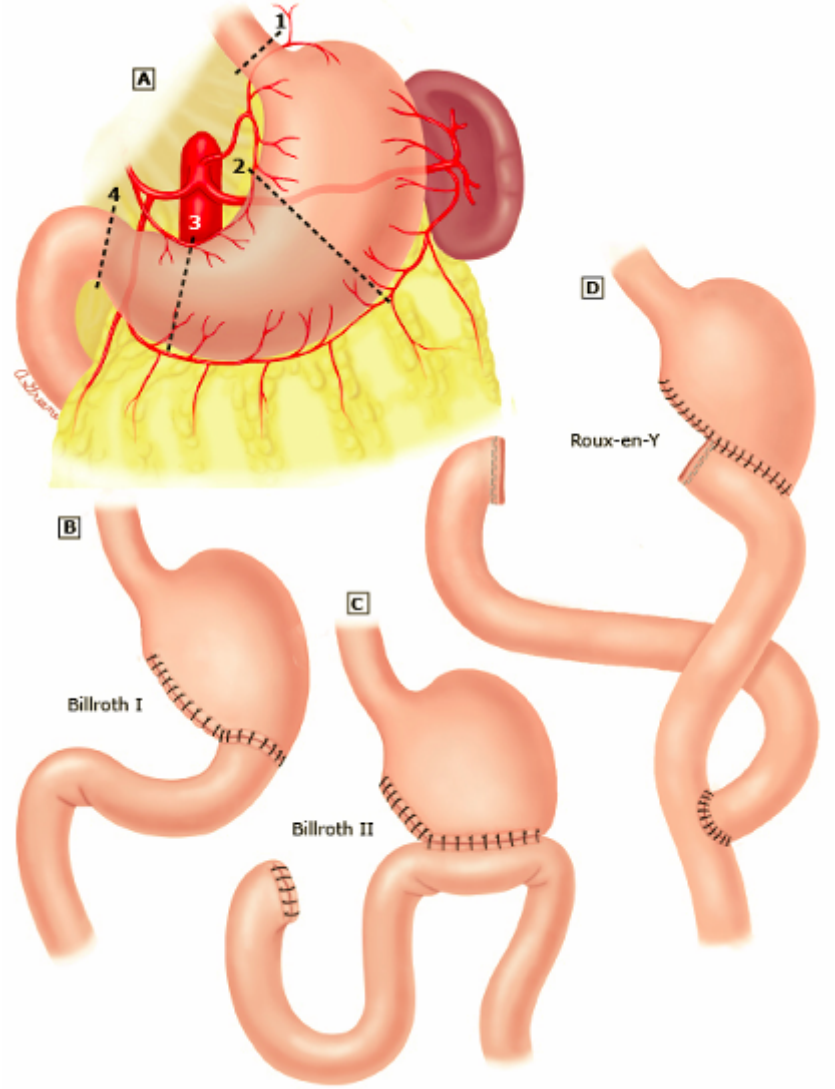
Gastric drainage procedures

- Pyloroplasty
- Gastrojejunostomy

*Not required for HSV



SURGERY FOR PUD



Gastrectomy and reconstruction

- Partial gastrectomy (to remove all parietal cells & gastrin producing antral tissue)
- Reconstruction e.g. Billroth II, Roux-en-Y

Q&A

Which of the following is false?

Achalasia of the cardia:

1. Is associated with hyperplasia of the lower oesophageal sphincter
2. Has an absence of ganglion cells in Auerbach's plexus
3. Presents in children with dysphagia
4. Diagnosis is confirmed by oesophageal function tests
5. Cardiomyotomy may not totally relieve symptoms

Q&A

During a routine examination, a 65yo woman complains of poor sleep for 3 months, associated with a burning and pressure sensation in the middle of her chest. She describes this as intermittent during the day, not relieved by antacids and not associated with exercise. She is afebrile, vitals are stable, examination is unremarkable. What is the most appropriate first step in her management?

1) Refer her to a psychologist

2) Prescribe cisapride

3) Order an ECG

4) Prescribe triple therapy antibiotics for H.pylori eradication

5) Prescribe a PPI – e.g. omeprazole

Q&A

The *preferred* drug treatment regimen for a penicillin-allergic *H. pylori*-positive 35-year-old man with epigastric pain and a duodenal ulcer documented by radiography is:

1. Lansoprazole + metronidazole
2. Ranitidine + bismuth citrate + clarithromycin
3. Omeprazole + metronidazole + clarithromycin
4. Bismuth subsalicylate + metronidazole + tetracycline

Q&A

The most common site of gastric ulcer is:

1. Incisura
2. Greater curvature
3. Fundus
4. Pylorus