



Chapter 89 – Esophagus, Stomach & Duodenum

Episode Overview:

1. List the the types of dysphagia. What is an ED approach to this condition?
2. What are 4 areas of narrowing in the esophagus that FBs get stuck?
3. List 8 causes of esophageal obstruction & List 3 therapies for a food bolus.
4. What are the indications for removal of an esophageal foreign body?
5. What are the indications for removal of a gastric foreign body?
6. List 6 causes of esophageal perforation – where does the perforation usually happen?
7. List 4 CXR findings of esophageal perforation and 3 other studies that can be performed.
8. What is the ED management of a patient with esophageal perforation?
9. List causes of esophagitis.
10. List 10 agents or conditions associated with GERD and list 3 complications of GERD
11. List 6 lifestyle modifications for someone with GERD and 3 medical therapies
12. List 6 causes of gastritis and 6 ddx
13. List the 2 main causes of PUD and describe the management of each.
14. How are prostaglandins used the setting of GI disorders?
15. What are the types of gastric volvulus? List risk factors for each. Describe the ED management.

Wisecracks

1. Differentiate between chest pain from ACS and that of an esophageal origin.
2. What is the mechanism of NSAID toxicity in PUD? List 3 at risk populations and 2 methods of preventing PUD in these people.
3. List 4 features of chest pain in PUD and 4 complications of PUD
4. What is Borchardt's triad?
5. Describe the mechanism of action of H2 blockers and PPIs
6. What are the potential complications of antacid use?

Rosen's in Perspective

Join us for a journey through the upper GI tract....we'll cover the tidbits of RIP as we move from section to section: throat-esophagus and then stomach.

1) List the the types of dysphagia. What is an ED approach to this condition?

Swallowing is divided into oral, pharyngeal, and esophageal phases. Failure at any one of these levels results in dysphagia, which literally means “difficulty Swallowing.”

When it comes to causes, just think M&M. The problem is either a mechanical block or a motility problem. The upper swallowing action is very complex neuro-muscular action that involves the lips, tongue, pharynx, etc the most common causes of OPD are motility-related.



a. List 4 causes of oropharyngeal dysphagia: “immediate or transfer dysphagia”

- i. Neuromuscular - swallowing muscles don't work
 1. **Due to a cerebrovascular accident** - #1 cause!
 2. **Degenerative aging:**
 - a. Alzheimer's, ALS, Diabetic neuropathy, muscular dystrophy, Parkinson's, Brain tumour
 3. **Immunologic** - poly/dermatomyositis / MS / Myasthenia Gravis / scleroderma
 4. **Infectious** - botulism, diphtheria, polioMyelitis, rabies, Sydenham's chorea, tetanus
 5. **Metabolic** - thyrotoxicosis, lead poisoning, Mg deficiency
- ii. Obstructive
 1. Dysphagia lusoria

b. List 6 causes of esophageal dysphagia. “Delayed / gets stuck”

- i. Again think M&M
 1. Mechanical
 - a. Intrinsic issue: strictures, webs, rings, tumours,
 - i. Esophagitis, ***foreign bodies***
 - b. Extrinsic compression: osteophytes, ***mediastinal masses***, aortic aneurysm, thyroid goitre
 2. Motility
 - a. Intrinsic: Achalasia, diffuse esophageal spasm, hypertensive LES, scleroderma, CREST syndrome, nutcracker esophagus
 - b. Extrinsic:
 - i. Gastric volvulus
 - ii. Alcoholism, diabetes, GERD,

Key History points to ask:

- Immediate vs. Delayed swallowing difficulty?
- Solids, liquids, or both?
- Intermittent or progressive symptoms?
- Associated symptoms, GI history, family history?

Key physical exam:

- HEENT
- Observed swallowing attempt
 - People with neuromuscular disease can't swallow while prone (they depend on gravity to do the work!)
- ii. Oropharyngeal - OPD
 1. Hx:
 - a. “Can't begin / initiate a swallow; sputter and gag; choke; drool; regurgitate through the nose”



- b. ****repeated swallowing attempts****
- iii. Esophageal - EsD
 1. Hx:
 - a. *“Difficulty 2-4 seconds after initiation of swallowing” - may have delayed regurgitation*
 - b. Achalasia: insidious onset, solids AND liquids, better with raising arms above head/standing straight; “birds/corkscrew beak” sign on barium swallow
 - c. Chest / retrosternal pain
 - c. Management:
 - i. Supportive! Based on the etiology (e.g. Thick fluids or solids for the elderly with transfer dysphagia)
 - ii. Achalasia - is one of the few disorders that can get better with:
 1. Nitrates
 2. CCB's
 3. Botox injection
 4. Surgical dilation
- d. **List 5 diagnostics tests that may be utilized for dysphagia.**

- Video esophagography
- Barium swallow
- Manometry
- Impedance monitoring
- CT - imaging with/without contrast for vascular / malignancy / external compression

These are RARELY indicated in the ED workup of a patient, and should be coordinated with the PCP or consultant

- **Admit those patients who:**
 - **1) Cannot swallow safely (risk for aspiration or risk for esophageal rupture)**
 - **2) Are unable to maintain adequate hydration**

2) What are 4 areas of narrowing in the esophagus that FBs get stuck?

75% of esophageal foreign body ingestions occur in Peds: 18-48 months.

4 natural areas of narrowing:

1. Cricopharyngeus (upper esophageal sphincter) → #1 spot things get stuck
 - In kids things get stuck at the cricopharyngeus; adults @ UES
2. Aortic arch
3. Left mainstem bronchus
4. Lower esophageal sphincter (LES) at the diaphragmatic hiatus



3) List 8 causes of esophageal obstruction & list 3 therapies for a food bolus.

Clinical presentation:

- Dysphagia, odynophagia, neck/chest pain / epigastric pain
- Complete obstruction = unable to swallow secretions (spit basin); choking; stridor; cough
 - Cafe' coronary: sudden cyanosis and collapse after a piece of swallowed food lodges in the airway leading to hypoxic arrest/syncope
 - Steakhouse syndrome: large piece of food (steak) causes complete obstruction of the distal esophagus. (fish bones RARELY obstruct the esophagus)
- Partial obstruction = usually the case in children, who present atypically:
 - Fever, wheezing, stridor, rhonchi, poor feeding

Causes of esophageal obstruction:

1. Mechanical obstruction:
 - a. Internal
 - i. Foreign body
 1. Adults - meat/bones; dentures; pen caps, etc.
 2. Kids - coins/batteries
 - ii. Strictures / rings (*Schatzki's ring*) / webs
 - iii. Eosinophilic esophagitis
 - iv. Malignancy
 - b. External factors
 - i. Large Left atrium / ventricle
 - ii. Goiter
 - iii. Mediastinal tumour
2. Motor factors (remember that the distal 2/3rds are innervated by involuntary smooth muscle)
 - a. Achalasia
 - b. Infectious (botulism, tetanus, etc.)

Food bolus therapies: (from Ep 60)

- Glucagon (0.5 to 2 mg) IV (Glucagon receptor agonist) – Theoretical action is lowering the smooth muscle tone at the lower esophageal sphincter without inhibiting normal esophageal peristalsis. Data is unsupporting of its use in modern treatment, and it is NO longer recommended.
- Benzodiazepine (GABA agonist): Anxiolytic +/- improves effect of Glucagon. Probably safe, but not shown to be better than placebo.
- Nitroglycerin (Nitric oxide which is a potent vasodilator) thought to work to relax lower esophageal sphincter tone
- Nifedipine (Calcium Channel Blocker) – thought to work to relax lower esophageal sphincter tone
- Enzymatic degradation w/ proteolytic enzyme papain or coca-cola – ineffective and has fallen out of favour



In summary; Do not use:

- Glucagon
- Meat tenderizer (papain)
- Effervescent agents

4) What are the indications for removal of an esophageal foreign body?

We covered some of this previously: <https://canadiem.org/crackcast-e060-foreign-bodies/>

- First you have to diagnose the foreign body:
 - AP and lateral radiographs of: neck soft tissues/chest/abdomen (as applicable)
 - Nasopharyngoscopy → laryngoscopy → endoscopy
 - Contrast swallow studies - are high risk for aspiration / perforation
 - CT - more sensitive for that elusive fish bone/chicken bone or to look for signs of inflammation/free air.

INDICATIONS FOR REMOVAL: (endoscopy, foley, Magill, etc).

- Button batteries (can cause severe tissue damage in 2 hrs!)
- Large objects (e.g. dentures, tooth brush)
- Sharp objects
- Coins lodged in the proximal esophagus
- Complete esophageal obstructions (preventing liquid past)
 - Incomplete obstructions must be removed in 24 hrs

Three mechanisms by which button batteries cause damage:

- Leakage of alkaline electrolyte
- Pressure necrosis
- Generation of electrical current - causing electrolysis of tissue fluids

5) What are the indications for removal of a gastric foreign body?

- Most can be observed conservatively, however if they have any of these features they may need to be removed:
 - Longer than 5 cm
 - Wider than 2.5 cm (rarely leave the duodenum)
 - Sharp and pointed objects (high risk of intestinal perforation)
 - Objects that remain in the stomach for more than 3-4 weeks
 - Objects that stay in the same intestinal location for 1 week



6) List 6 causes of esophageal perforation – where does the perforation usually happen?

- The esophagus has two layers: inner circular layer of muscle & the outer longitudinal layer of muscle
- Clinical presentation: odynophagia, fever, decreased PO intake, chest pain, dyspnea, hoarseness, subcutaneous air
- **Causes:**
 - Food stuffs:
 - Chicken / fish / animal bones
 - Large unchewed pieces (e.g. steak)
 - Button batteries
 - Coins
 - Forceful valsalva:
 - Vomiting
 - Childbirth
 - Coughing / heavy lifting
 - Iatrogenic:
 - Post EGD
 - Post NG placement
 - Post endotracheal intubation
 - Caustic substance ingestion
 - Severe esophagitis
 - Blunt / penetrating trauma
 - Carcinoma

90% of spontaneous perforations happen in the distal esophagus; other sites: pharyngoesophageal junction.

Iatrogenic injuries happen at the esophagogastric junction.

7) List 4 CXR findings of esophageal perforation and 3 other studies that can be performed.

CXR:

- Pneumothorax
- Subcutaneous air / emphysema
- Hydropneumothorax
- Pneumomediastinum
- Pleural effusions
- Pulmonary infiltrates
- Wide mediastinum



Other studies:

- Abdominal radiography
- Gastrograffin swallow (water soluble agent)
- CT chest
- Flexible EGD - 100% sensitivity (although has a risk of enlarging a minimal transmural opening due to insufflation of air)

8) What is the ED management of a patient with esophageal perforation?

Mackler's triad: subcutaneous emphysema, chest pain, vomiting = esophageal perforation

Misdiagnosis of people with esophageal perforation as ACS/PE/Aortic dissection/perforated ulcer/pancreatitis/pneumothorax/lung abscess - due to chest and abdominal pain!

Management:

- Broad spectrum abx
- Admission
- NPO

9) List causes of esophagitis.

Symptoms: dysphagia, odynophagia, chest pain, bleeding → hemorrhage → perforation!

- GERD (#1 cause)
- Eosinophilic esophagitis
 - Unknown etiology; typically in non-responders to PPI's and kids with atopy. Don't respond to high dose PPIs, may need steroids inhalers.
- Infection
 - Usually an immunocompromised (steroid use, ETOH, immunosuppression, or HIV seroconversion syndrome) person developing Candida (but can also be CMV/HSV). Usually have odynophagia not improved with antacids.
- Foreign body / toxic ingestion
 - Aka. Pill esophagitis - from large pills sticking to the esophageal wall. Can lead to erosions/ulcer/strictures.
- Post-radiation

10) List 10 agents or conditions associated with GERD and list 3 complications of GERD

Some physiologic GER occurs in everyone, when it becomes symptomatic then it is "GERD".

Conditions associated with GERD: (ie. anything that causes the LES to relax or become overwhelmed) - this comes from Box 79-2

- **Decreased LES pressure**
 - Meds:
 - CCB, nitrates, anticholinergics, albuterol, benzos,
 - Fatty meals / Chocolate / Caffeine / Ethanol / Peppermint / nicotine



- Pregnancy
- Estrogen / progesterone

- **Increased gastric emptying time / Increased intra-abd. Pressure**
 - Anticholinergic drugs
 - Pregnancy
 - Obesity
 - Coughing / bending /
 - Supine position
 - Gastroparesis / neuromuscular disease leading to gastric outlet obstruction

- **Decreased esophageal motility**
 - Achalasia
 - DM
 - Scleroderma

GEeRD = “gastric emptying; esophageal relaxation drugs” mnemonic

Complications of GERD

1. Esophagitis
 - Progressing to Barrett’s metaplasia, erosion, ulcerations, scarring
 - Stricture formation due to persistent scarring and inflammation
2. Reflux induced asthma
 - Microaspiration of
3. Adenocarcinoma of the esophagus
4. Esophageal perforation → mediastinitis

11) List 6 lifestyle modifications for someone with GERD and 3 medical therapies

Lifestyle mods:

- Avoidance of high risk foods (caffeine, chocolate, alcohol, fatty, spicy foods)
- Weight loss*
- Smoking cessation
- Elevation of the head of bed, and avoidance of lying down after meals*
- Exercise
- Alcohol cessation

Therapies:

- Acid neutralization
 - Milk of magnesia
 - TUMS
 - Pepto Bismol - bismuth salts
- Decrease acid production (most effective)
 - PPIs
 - Pantoprazole 20 - 40 mg PO - daily or BID before meals for 4-8 wks.
 - H2 blockers



- Ranitidine 150 mg PO
- Strengthening LES / motility amplification
 - Maxeran 10 mg
- Mucosal protection
 - Sucralfate 1 g - q 6 hrs (safe in pregnancy!)
 - Ideally given 30-60 mins before meals.

12) List 6 causes of gastritis and 6 DDX

True gastritis is a histology diagnosis.

Typically, we use the term clinically. Usually mild-moderate burning pain or bloating epigastrically. Few diagnostic tests are needed (but you need to exclude other things on the ddx!)

Causes:

1. Acute Helicobacter pylori infection
2. Acute phlegmonous gastritis (rare, but lifethreatening!)
3. Shock states
4. Lymphoma
5. Adenocarcinoma
6. NSAID use
7. ETOH abuse / use
8. Drugs:
 - a. 5FU, bisphosphonates, iron
9. Smoking
10. Bile / pancreatic secretions
11. Stress
12. Glucocorticoids

DDx:

- PUD
- Pancreatitis
- Mesenteric ischemia
- Biliary disease
- ACS - angina
- Pericarditis
- SBO



13) List the 2 main causes of PUD and describe the management of each.

1. H. Pylori infection - #1 cause

- 70% of duodenal/gastric ulcers thought to be caused by this!
- Oral-oral route/fecal-oral route/iatrogenic
- Up to 40% of the population is infected with this bacteria (but only 5-10% of people infected seem to get ulcers)
- Testing: serum antibody test, stool antigen testing, breath test, mucosal bx.
- Management:
 - i. Triple therapy:
 1. Clarithromycin 500 mg po BID for 10 days
 2. Amoxicillin 1 g bid PO for 10 days
 3. A PPI for 4-8 weeks.
 4. (USE metronidazole to replace #1,2 for penicillin allergy)
 - ii. Some ppl need quadruple therapy- bismuth/metronidazole/tetracycline/PPI
 - iii. Commercial combination packs exist (Prevpac)
 - iv. Use adjunct agents for GERD/gastritis

2. NSAID associated (chronic)

- Due to suppressed gastric prostaglandin synthesis
 - Varying risks for ulceration based on NSAID type and co-use of glucocorticosteroids, anticoagulants
 - Management:
 - i. Stop NSAID use
 - ii. PPI for 4-8 weeks
 - iii. Adjunct agents for GERD/gastritis
3. < 1% of PUD is caused by Zollinger-Ellison syndrome
4. Systemic illness (especially in infants and children)

Adjuncts:

- Prostaglandins (misoprostol)
- Sucralfate
- Bismuth salts
- Stop all GERD / gastritis causing foods/lifestyle choices/meds

Reasons to referral to GI:

- Age >55 yrs with:
 - New onset
 - Dyspepsia
 - Progressive weight loss
 - Persistent vomiting
 - Iron deficiency anemia
 - Epigastric mass



14) How are prostaglandins used the setting of GI disorders?

Prostaglandins protect the gastric mucosa! (Inhibit acid secretion, increase mucous/bicarbonate/stimulate blood flow).

Misoprostol (an analogue of prostaglandin) - used for high risk patients - to prevent progression of NSAID-induced ulcers.

15) What are the types of gastric volvulus? List risk factors for each. Describe the ED management.

This is a life-threatening closed-loop obstruction of the stomach! Because the stomach is only fixed at two points - it can easily twist on itself.

Defined by a few different ways:

1. Cause: **primary vs. secondary**
 - a. **Primary:**
 - i. subdiaphragmatic - stabilizing ligaments are too lax.
 - b. **Secondary:**
 - i. Supradiaphragmatic - due to diaphragmatic defects (hiatal hernia, gastric ulcer/cancer, diaphragm paralysis, and other).
2. Anatomy: **axis of rotation**
 - a. Twisting on its long axis (organoaxial)
 - b. Twisting on its short axis (mesenteroaxial)
3. Onset: **acute vs. chronic**

Look for the triad: severe epigastric pain and distension; vomiting with violent retching; inability to pass an NG tube.

In children: look for sudden life-threatening events with apnea/cyanosis/respiratory distress!

Risk factors:

- Age 40-50 yrs
- Paraesophageal hernia
- **20% of cases occur in infants due to a congenital diaphragmatic defect (secondary)

Management:

- This disease can result in gastric ischemia, perforation and death.

Goal is REDUCTION!

- Attempt NG tube placement
- May go for attempted endoscopic reduction vs. immediate surgery! (call both!)



Wisecracks

1) Differentiate between chest pain from ACS and that of an esophageal origin.

Esophageal	--overlapping symptoms/signs--	ACS
<ul style="list-style-type: none"> ● prolonged, non exertional pain that: <ul style="list-style-type: none"> ○ Interrupts sleep / lying down ○ Is related to meals ○ Relief with antacids ○ Regurgitation ○ Dysphagia ○ Worse with swallowing 	<ul style="list-style-type: none"> ● substernal chest pain ● Crushing, squeezing pain ● Radiation to neck, shoulder, arm ● Response to nitroglycerin 	<ul style="list-style-type: none"> ● exertional symptoms ● Substernal discomfort ● Dyspnea ● (Atypical anginal symptoms may mislead us!)

These are clues, NOT slam-dunk rules! Think worst-first!

Once a workup is “negative” for the killers - then consider a GI source of the symptoms! (Gastritis, esophagitis, PUD, biliary tract disease).

2) What is the mechanism of NSAID toxicity in PUD? List 3 at risk populations and 2 methods of preventing PUD in these people.

See above!

- Prostaglandin inhibition due to COX inhibition!
- NSAID users, age > 60, hx of PUD, steroid/anticoag use
- Change to lower risk NSAID / stop use
- Concurrent PPI / acid suppressants.

3) List 4 features of chest pain in PUD and 4 complications of PUD

- Burning , gnawing epigastric pain
- Fullness, nausea, early satiety, bloating
- Onset of pain 2-5 hrs post meal, OR **worse around 2 am in the morning** (when acid production is at its highest!)
- Relief with eating (pain eating relief)



(Vomiting is atypical!!) - so think of other things: gastric volvulus, outlet obstruction, SBO, pancreatitis, biliary disease.

- Complications
 - Hemorrhage
 - Perforation (duodenal ulcers)
 - Penetration (e.g. posteriorly into the retroperitoneum)
 - Gastric outlet obstruction

4) What is Borchardt's triad?

"This condition may present as an acute abdominal emergency or a chronic cause of upper abdominal discomfort. **Borchardt's triad is believed to be diagnostic for acute gastric volvulus and consists of unproductive retching, epigastric pain and distention, and the inability to pass a nasogastric tube.** Carter et al suggest three additional findings that may be very suggestive of gastric volvulus: minimal abdominal findings when the stomach is in the thorax; a gas-filled viscus in the lower chest or upper abdomen on chest radiograph, especially when associated with a paraesophageal hernia; and obstruction at the site of the volvulus shown by upper gastrointestinal (GI) series. **Mortality from acute gastric volvulus has been reported to be as high as 30–50%.** As a result, quick recognition and prompt surgical correction remain key in therapy for acute gastric volvulus."

Copied from: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3072542/>

5) Describe the mechanism of action of H2 blockers and PPIs

Histamine - is the primary stimulus for gastric acid secretion. H2 blockers attach onto the H2 receptor of the parietal cell and prevent the release of hydrochloric acid.

The parietal cell's proton pump (H, K, ATP-ase) that produces HCl is irreversibly blocked by PPI's. Very effective - production of acid can be reduced up to 95%. Require regular use to achieve therapeutic benefit - (if a patient is taking drugs on a PRN basis a H2 blocker would be a better choice).

These therapies are novel - because ulcers can't develop in the absence of acid!

6) What are the potential complications of antacid use?

- Magnesium containing antacids:
 - Can cause diarrhea
- Calcium containing antacids: watch for **milk-alkali syndrome**
 - Acid rebound
 - Constipation
 - Hypercalcemia
 - Alkalosis
 - Renal insufficiency

CrackCast Show Notes – Abdominal Aortic Aneurysm – June 2017
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- Aluminum containing antacids:
 - Constipation
 - Electrolyte abnormalities
- Decrease Absorption of: (Reduce this by taking them 1-3 hrs post meals)
 - Warfarin
 - Digoxin
 - Antibiotics,
 - Anticonvulsants