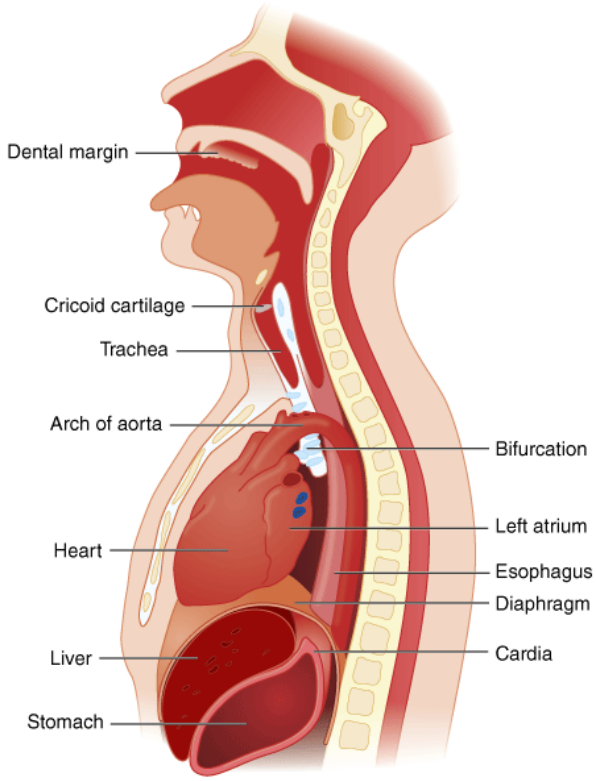
OESOPHAGEAL EMERGENCIES

COMPLAINTS OF DYSPHAGIA, ODYNOPHAGIA OR INGESTED FOREIGN BODY IMMEDIATELY IMPLICATE THE OESOPHAGUS AND THEY MAY PRESENT WITH CHEST PAIN, UGI BLEEDING

PATHOPHYSIOLOGY:

- Majority of the oesophagus is located in the posterior mediastinum, posterior to and slightly lateral to the trachea



- Upper third is made up of striated muscle, and the lower half down is all smooth muscle
- Two sphincters regulate passage of material → upper sphincter prevents air form entering oesophagus and food refluxing out
    - Lower sphincter regulates passage of food into the stomach and prevents retrograde flow of stomach contents
- THREE ANATOMIC CONSTRICTIONS IN ADULT:
    - CRICOPHARYNGEUS (C6)
    - AORTIC ARCH (T4)
    - GASTROOESOPHAGEAL JUNCTION (T10-11)
        - In kids, there is also the thoracic inlet and tracheal bifurcation
        - These areas are important as they represent where FB can get lodged

- Innervation of the heart mirrors that of the oesophagus and a convergence of visceral and somatic stimuli occur within the sympathetic system → makes cardiac and oesophageal pain NOTORIOUSLY SIMILAR

DYSPHAGIA:

- DIFFICULTY WITH SWALLOWING
- Most have an identifiable, organic cause
- TWO BROAD GROUPS → transfer dysphagia → as food moves from oropharynx through the upper sphincter, i.e. very early in swallowing. TRANSPORT DYSPHAGIA → impaired movement of the bolus down the oesophagus and through the lower sphincter, perceived later, "getting stuck"
    - Provides valuable framework for identifying underlying pathology
    - Obstructive disease is USUALLY PROGRESSIVE → solids first then liquids

Table 80-1 Dysphagia

Transfer Dysphagia (Oropharyngeal)	Transport Dysphagia (Esophageal)
Discoordination in transferring bolus from pharynx to esophagus	Improper transfer of the bolus from the upper esophagus into the stomach
Swallowing symptoms—gagging, coughing, nasal regurgitation, inability to initiate swallow, need for repeated swallows	Swallowing symptoms—food "sticking," retrosternal fullness with solids (and eventually liquids), possibly odynophagia
Risk of aspiration present	Risk of aspiration present, generally less pronounced than in transfer dysphagia
Long term—weight loss, malnutrition, chronic bronchitis, asthma, multiple episodes of pneumonia	Long term—malnutrition, dehydration, weight loss, systemic effects of cancer
Neuromuscular disease (80%)—cerebrovascular accident, polymyositis and dermatomyositis, scleroderma, myasthenia gravis, tetanus, Parkinson's disease, botulism, lead poisoning, thyroid disease	Obstructive disease (85%)—foreign body, carcinoma, webs, strictures, thyroid enlargement, diverticulum, congenital or acquired large-vessel abnormalities
Localized disease—pharyngitis; aphthous ulcers; candidal infection; peritonsillar and retropharyngeal abscesses; carcinoma of tongue, pharynx, larynx; Zenker diverticulum; cricopharyngeal bar; cervical osteophytes	Motor disorder—achalasia, peristaltic dysfunction (nutcracker esophagus), diffuse esophageal spasm, scleroderma
Inadequate lubrication—scleroderma	Inflammatory disease

CLINICAL FEATURES:

- Can be associated with ODYNOPHAGIA (painful swallowing) or with chest pain (GORD)
- Ask about speed of onset? Dysphagia to solids, liquids or both? Intermittent or progressive? Sensation of food getting stuck? Where? History of oesophageal disease?
- An impacted food bolus can be the presenting complaint for a variety of underlying oesophageal pathologies?
- Oesophageal filling proximal to the impacted bolus can cause inability to swallow secretions and can present an airway/aspiration risk
- Physical exam → signs of CVA, Cachexia, cervical nodes (cancer) May be normal

DIAGNOSIS:

- Diagnosis of underlying cause is often made on OUTPATIENT BASIS with endoscopy
- OBSTRUCTIVE/STRUCTURAL CAUSES:
    - NEOPLASM → oesophagus or surrounding structure can be site. 95% of oesophageal malignancies are SCC (RF include alcohol, smokingm achalsia). Adenocarcinoma in 5%, with Barretts oesophagus being pre-malignant condition in this case. Malignancy characterised by relatively rapid progression from dysphagia to solids then liquids. Diagnosis made at endoscopy (suspect in new onset dysphagia >40yo), survival is dismal, median 1 year.
    - STRICTURE → result of scarring from GORD or other chronic inflammation. Generally occur in distal oesophagus. Treatment is dilation and must first exclude malignancy as cause
    - SCHATZKI RING → most common cause of intermittent dysphagia of solids → consists of a diaphragm-like stricture near gastro-oesophageal junction, present in up to 15%. Treatment is dilation.
    - OESOPHAGEAL WEBS → treatment is dilation
    - DIVERTICULA → found throughout the oesophagus, usually after age 50. Suspect with halitosis and transfer dysfunction.
- SELECTED MOTOR LESIONS CAUSING DYSPHAGIA:
    - NEUROMUSCULAR DISORDERS → result in misdirection of the food bolus, liquids more difficult to handle. CVA is most common cause.
    - ACHALASIA → dysmotility disorder of unknown cause→impaired swallowing-induced relaxation of lower sphincter along with absence of peristalsis. Can be associated with oesophageal spasm and chest pain. Dilation of the oesophagus can be large enough to impinge on the trachea and cause airway symptoms. Treatment options → myotomy, oral medications, dilations, endoscopic botox injection.
    - DIFFUSE OESOPHAGEAL SPASM → chest pain a common symptom, treat with PPI.

<u>CHEST PAIN OF OESOPHAGEAL ORIGIN:</u>

- Differentiating oesophageal pain from ischaemic chest pain can be difficult or impossible in the ED
- Common reports → onset of pain at night, spontaneous onset of pain, regurgitation, odynophagia, dysphagia, meal-induced heartburn → but these symptosm are also found in IHD and THERE IS NO HISTORICAL FEATURE THAT IS SENSITIVE ENOUGH TO DIFFERENTIATE THE TWO
    - Best ED default assumption is that the pain is cardiac in nature and not oesophageal →incidence of oesophageal diseaes in those with normal coronary arteries is 20-60%

GORD:

- Wide array of symptoms and long-term effects
- The primary mechanism causing reflux is TRANSIENT RELAXATION OF LOWER SPHINCTER

- Contributing factors outlined below:

Table 80-2 Causes of Gastroesophageal Reflux Disease		
Decreased Pressure of Lower Esophageal Sphincter	**Decreased Esophageal Motility**	**Prolonged Gastric Emptying**
High-fat food	Achalasia	Medicines (anticholinergics)
Nicotine	Scleroderma	Outlet obstruction
Ethanol	Presbyesophagus	Diabetic gastroparesis
Caffeine	Diabetes mellitus	High-fat food
Medicines (nitrates, calcium channel blockers, anticholinergics, progesterone, estrogen)		
Pregnancy		

- **Heartburn is the classic symptom** → burning discomfort associated with other GI symptoms → odynophagia, dysphagia, acid regurgitation and hypersalivation. Classically pain and discomfort with meals points to GORD
    - **Relief with antacids classic but can occur in patients with IHD and angina**
    - GORD can be pressure-like and both cardiac and GORD pain associated with diaphoresis, pallor, N+V
    - Radiation pattern can be identical
    - CAUTION IS WARRANTED
- Over time, GORD can cause complications:
    - Strictures
    - Dysphagia
    - Inflammatory oesophagitis and odynophagia
    - Barrett Oesophagus in 10% → pre-malignant condition associated with adenocarcinoma
    - Less obvious presentations → pulmonary complications including asthma due to microaspiration inducing bronchospasm and inflammation
    - Dental erosion, vocal cord ulcers and granuloma, chronic sinusitis, chronic cough
- Mild disease often treated empirically with PPI or H2RA and avoidance of precipitants (alcohol, cigarettes, caffeine, chocolate, fatty foods)

OESOPHAGITIS:

- Can cause prolonged periods of chest pain and almost always causes odynophagia as well
- INFLAMMATORY OESOPHAGITIS:
    - GORD may induce inflammatory response in lower oesophagus → ulcerations, scarring and stricture formation
    - Warrants aggressive pharmacotherapy
    - Multiple medications implicated → NSAIDS, potassium, doxycycline, clindamicin
- INFECTIOUS OESOPHAGITIS:

- Patients with immunosuppression can develop infectious oesophagitis → diagnosis of oesophageal candidiasis in an otherwise healthy host suggestive of underlying immunosuppression

OESOPHAGEAL PERFORATION:

- Can occur secondary to any number of disparate processes but IATROGENIC PERFORATION MOST COMMON

Table 80-3 Causes of Esophageal Perforation

Cause of Perforation	Description
Iatrogenic	Intraluminal procedures
	Endoscopy
	Dilatation
	Variceal therapy
	Gastric intubation
	Intraoperative injury
Boerhaave syndrome	"Spontaneous," usually associated with transient increase in intraesophageal pressure
Trauma	Penetrating
	Blunt (rare)
	Caustic ingestion
Foreign body	Includes pill-related injury
Infection	Rare
Tumor	May be intrinsic or extrinsic cancer
Aortic pathology	Aneurysm
	Aberrant right subclavian artery
Miscellaneous	Barrett esophagus
	Zollinger-Ellison syndrome

- Rate of perforation during endoscopy in oesophagus is lower in the NON-DISEASED STATE → dilation of strictures increases risk significantly
- Perforation causes a dramatic presentation if oesophageal contents leak intot he mediastinal, pleural or peritoneal space
  - FULMINANT, NECROTISIN MEDIASTINITIS, PNEUMONITIS OR PERITONITIS MAY RESULT AND RAPIDLY LEADS TO SHOCK
  - Most spontaneous perforation occur through left posterolateral wall of distal oesophagus (proximal perforation is less severe with localised abscess the norm)
  - PAIN DESCRIBED AS ACUTE, SEVERE, UNRELENTING AND DIFFUSE AND IS REPORTED IN THE CHEST, NECK AND ABDOMEN → EXACERBATED BY SWALLOWING → dysphagia, dyspnoea, haematemesis and cyanosis are reported
  - Physical exam varies with severity of the rupture and elapsed time between rupture and presentation
    - Cervical cutaneous emphysema is common
    - MEDIASTINAL EMPHYSEMA TAKES TIME TO DEVELOP and its absence does not exclude perforation

        - HAMMAN CRUNCH → caused by air in the mediastinum overlying the heart
        - PLEURAL EFFUSIONS develop in half of those with intrathoracic perforation
    - CT CHEST OR EMERGENT ENDOSCOPY TO CONFIRM DIAGNOSIS → timely diagnosis is crucial and perforation is associated with a high mortality rate regardless of cause
- TREATMENT OF OESOPHAGEAL PERFORATION:
    - Rapid resuscitation
    - Broad spectrum antibiotics
    - Emergency surgical consultation
- BOERHAAVE SYNDROME → full-thickness perforation of the oesophagus after a sudden rise in intra-oesophageal pressure → mechanism is sudden, forcefull emesis in about ¾ cases → coughing, straining, seizures and childbirth have been implicated. Alcohol consumption is frequently an antecedent to this syndrome
- TRAUMA → implicated in 10% causes, PENETRATING INJURY TO THE NECK IN MOST CASES. Often masked by other more imminently life-threatening conditions → oesophagoscopy crucial to exclude these disorders

SWALLOWED FOREIGN BODIES:

PATHOPHYSIOLOGY:

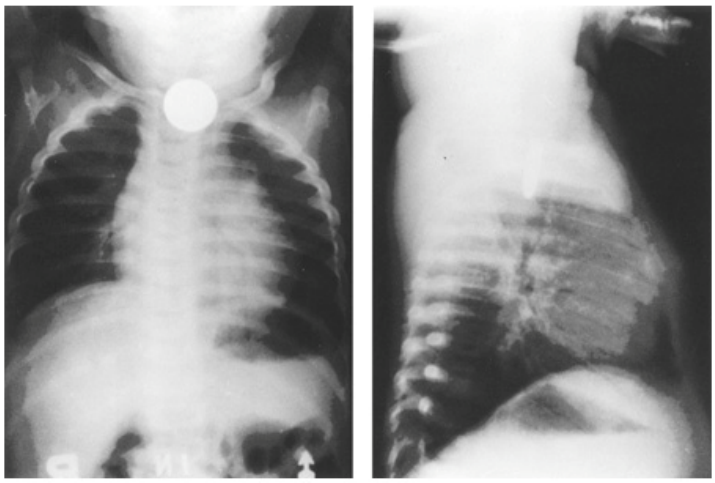
- Children 18-48 months account for 80% of all cases of ingested foreign bodies
- In children and adults, once an object has TRAVERSED THE PYLORUS, it usually continues through the GIT and is passed without issue
    - If, however, the object has an irregular or sharp edges or is particularly wide (>2.5CM) or long (>6cm) it may become lodged distal to the pylorus
- Oesophageal impaction can lead to airway obstruction, stricture or perforation

CLINICAL FEATURES:

- Adults provide unequivocal history
- Dysphagia, vomiting and choking are associated symptoms
- In children, the history can be unclear → refusal or inability to eat, vomiting, gagging/choking, stridor, neck or throat pain, drooling
    - High index of suspicion is necessary, especially in those <2

DIAGNOSIS:

- Plain films are used to screen for radio-opaque objects
    - Coins in the oesophagus present their circular face on AP films as opposed to coins in the trachea, which show that face on lateral films



- Ingested, impacted bones are only seen in 50%
- CT scan is very high yield and also delivers excellent information about perforation and subsequent infection

TREATMENT:

- Patients in extremis or with pending airway compromise are resuscitated in standard fashion and may require active airway management
- Other situations requiring urgent input are listed below:

Table 80-4 Circumstances Warranting Urgent Endoscopy for Esophageal Foreign Bodies
Ingestion of sharp or elongated objects (including toothpicks, aluminum soda can tabs)
Ingestion of multiple foreign bodies
Ingestion of button batteries
Evidence of perforation
Coin at the level of the cricopharyngeus muscle in a child
Airway compromise
Presence of a foreign body for >24 h

- In general terms, if endoscopy is clearly indicated, then advanced imaging plays little role
- If the object is distal to the pylorus and is not a shape or nature to cause concern and the patient is comfortable and tolerating oral intake then treatment is expectant and they can be discharged
    - For worrisome foreign bodies that are impacted in the distal GIT, surgery is required
- For distal oesophageal foreign bodies, glucagon has been used to relax the lower sphincter and allow passage → but success rates are poor and it may be no better than watchful waiting
- FOOD IMPACTION:
    - Meat is the most common offender → obstruction is complete and contains bony fragment, then emergency endoscopy is required
    - Food should not be allowed to remain impacted for >12-24 hours
    - Trial glucagon (1-2mg IV)

- COIN INGESTION:
  - Can attempt removal with foley catheter with fluoroscopy in Trendelenburg position
  - Complications → aspiration, airway compromise, mucosal laceration
- BUTTON BATTERY INGESTION:
  - Button battery lodged in the oesophagus is a TRUE EMERGENCY requiring prompt removal, because the battery may quickly induce mucosal injury and necrosis
  - Perforation can occur within 6 hours of ingestion
  - Lithium cells are associated disproportionately with adverse outcome
  - THREAT OF HEAVY METAL POISONING IS NON-EXISTENT
  - URGENT ENDOSCOPY IF PROXIMAL TO THE DISTAL OESOPHAGEAL SPHINCTER
    - If past oesophagus and asymptomatic, then no further input required
    - If symptomatic, then endoscopy indicated to delineate injury
- INGESTION OF SHARP OBJECTS:
  - Sharp objects in the oesophagus need immediate removal
  - Sharp objects may pass into the stomach, but intestinal perforation from ingested objects that pass distal to the stomach is up 35%, hence removal is mandatory
    - Endoscopic removal if they are in the stomach or duodenum
    - If intestinal perforation is to occur, it will often be at the ileocaecal valve
    - If the object is distal to the duodenum at presentation and the patient is asymptomatic, the objects passage should be documented and surgery indicated if intestinal injury develops
- NARCOTIC INGESTION (BODY-PACKERS):
  - Use condoms
  - Visible on plain films
  - Rupture of one packet may be fatal
  - Endoscopic removal is contraindicated