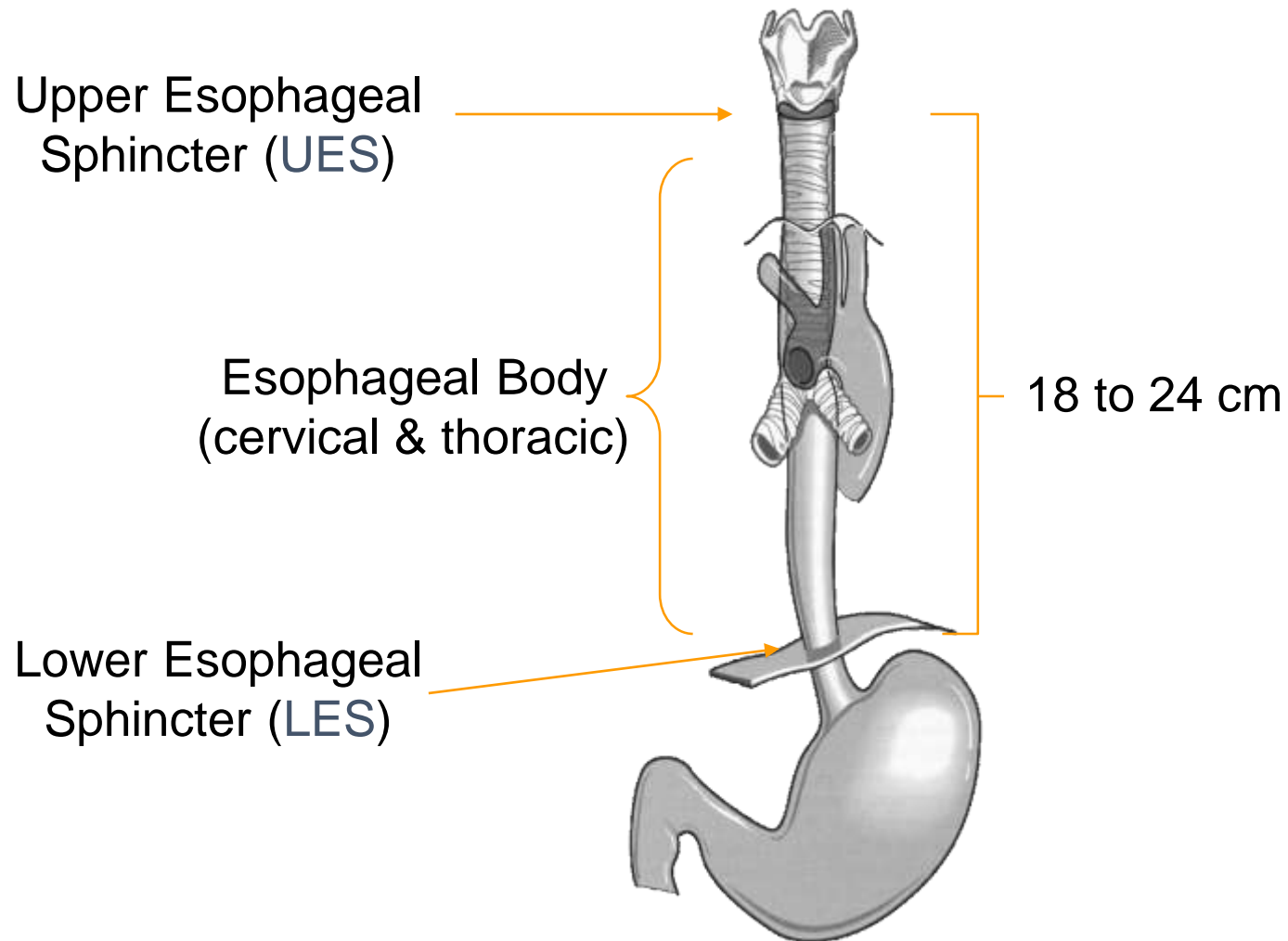


Esophageal Disorders

Esophageal Disorders

- Motility
- Anatomic & Structural
- Reflux
- Infectious
- Neoplastic
- Miscellaneous

Esophageal Anatomy



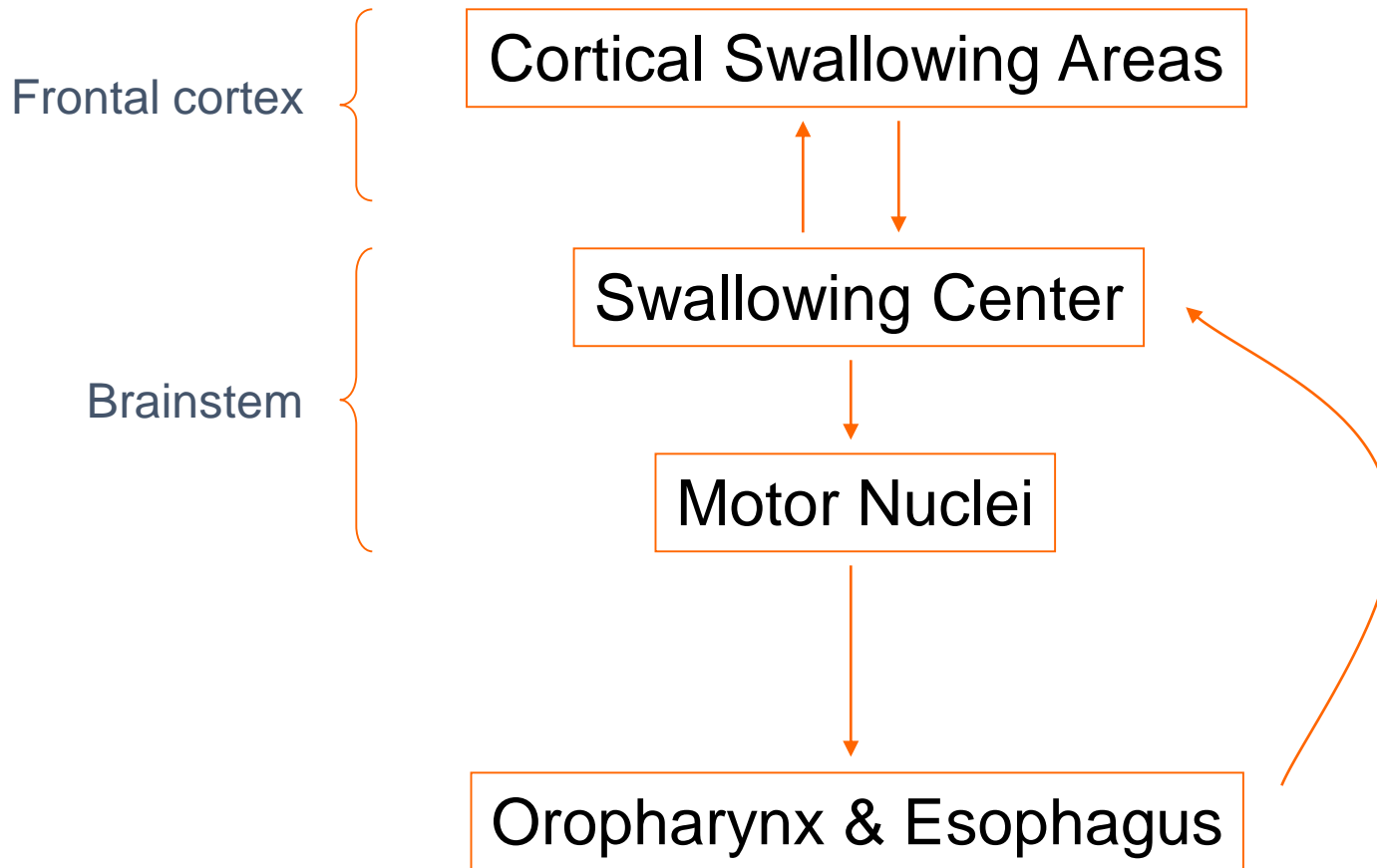
Normal Phases of Swallowing

- Voluntary
 - oropharyngeal phase – bolus is voluntarily moved into the pharynx
- Involuntary
 - UES relaxation
 - peristalsis
 - LES relaxation

Normal Phases of Swallowing

- Between swallows
 - UES prevents air entering the esophagus during inspiration and prevents esophagopharyngeal reflux
 - LES prevents gastroesophageal reflux
 - peristaltic and non-peristaltic contractions in response to stimuli
 - capacity for retrograde movement (belch, vomiting) and decompression

Normal Swallowing



Esophageal Motility Disorders

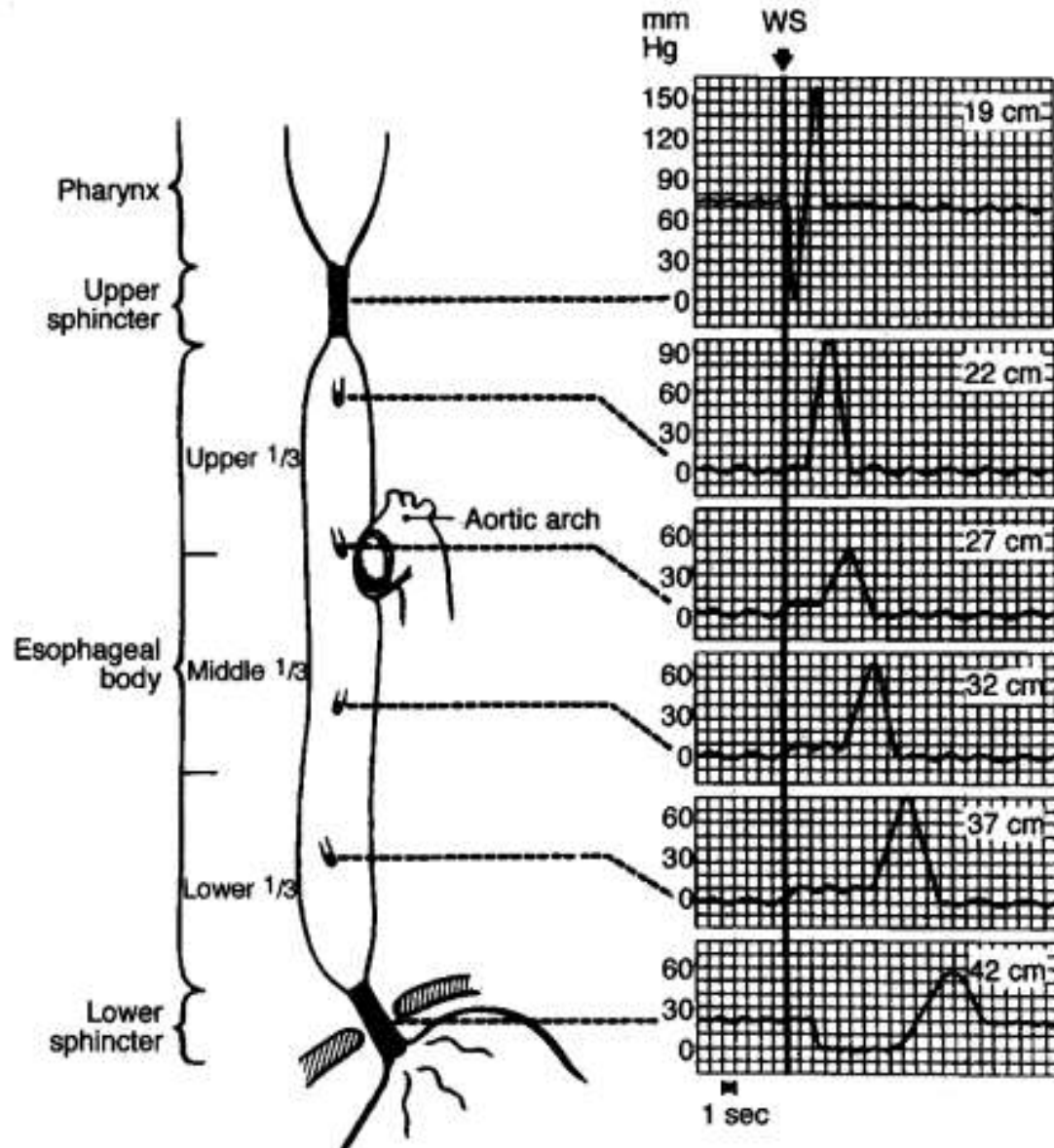
Motility Disorders

- upper esophageal
 - UES disorders
 - neuromuscular disorders
- esophageal body
 - achalasia
 - diffuse esophageal spasm
 - nutcracker esophagus
 - nonspecific esophageal dysmotility
- LES
 - achalasia
 - hypertensive LES
- primary disorders
 - achalasia
 - diffuse esophageal spasm
 - nutcracker esophagus
 - nonspecific esophageal dysmotility
- secondary disorders
 - severe esophagitis
 - scleroderma
 - diabetes
 - Parkinson's
 - stroke

Diagnostic Tools

- videofluoroscopy
- barium esophagram
- esophageal manometry
- endoscopy

Normal Manometry



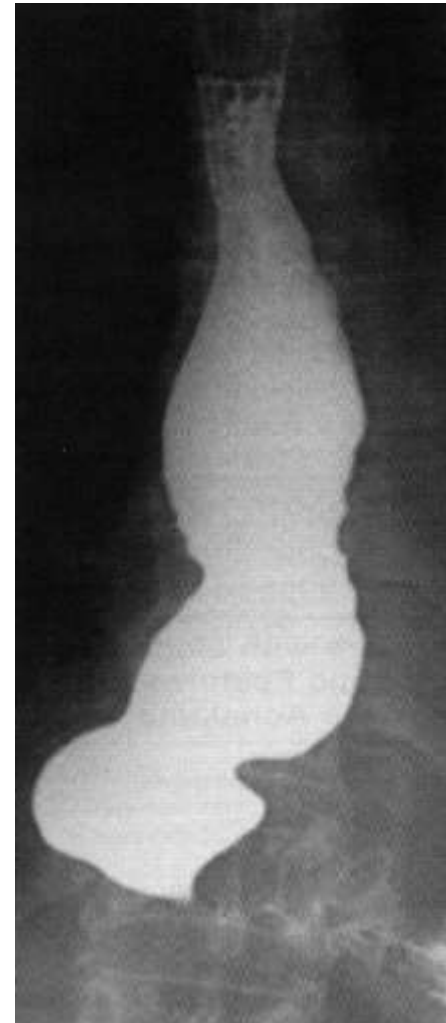
Motility Disorders Based on Manometry

- **Achalasia**
 - Inadequate LES relaxation
- **Diffuse Esophageal Spasm**
 - Uncoordinated contraction
- **Nutcracker Esophagus**
 - Hypercontraction
- **Ineffective Esophageal Motility**
 - Hypocontraction

Achalasia

Achalasia

- first clinically recognized esophageal motility disorder
- described in 1672, treated with whale bone bougie
- term coined in 1929
- epidemiology
 - 1-2 per 200,000 population
 - usually presents between ages 25 to 60
 - male=female
 - Caucasians > others
 - average symptom duration at diagnosis: 2-5 years



Pathophysiology

- Degeneration of NITIRIC OXIDE (NO) producing inhibitory neurons
 - loss of ganglionic cells in the myenteric plexus (distal to proximal)
 - vagal fiber degeneration
 - underlying cause: unknown
 - autoimmune? (antibodies to myenteric neurons in 50% of patients)
- that affect relaxation of LES
- Basal LES pressure rises

Mechanical End Result

- dual disorder
 - LES fails to appropriately relax
 - resistance to flow into stomach
 - *not spasm of LES* but an increased basal LES pressure often seen (55-90%)
 - loss of peristalsis in distal 2/3 esophagus

Clinical Presentation

- clinical presentation
 - solid dysphagia 90-100% (75% also with dysphagia to liquids)
 - post-prandial regurgitation 60-90%
 - chest pain 33-50%
 - pyrosis 25-45%
 - weight loss
 - nocturnal cough and recurrent aspiration

Diagnostic Work Up

- plain film (air-fluid level, wide mediastinum, absent gastric bubble, pulmonary infiltrates)
- barium esophagram (dilated esophagus with taper at LES) *Bird peak*
 - good screening test (95% accurate)
- endoscopy (rule out GE junction tumors, esp. age>60)
- esophageal manometry (absent peristalsis, ↓ LES relaxation, & resting LES >45 mmHg)



Manometric Features

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

Treatment of Achalasia

Goals

- *reduce LES pressure and*
- *increase emptying*

Nitrates and Calcium Channel Blockers

- Isosorbide dinitrate

 - Reduces LES Pressure 66% for 90 min

- Nifedipine

 - Reduces LES pressure 30-40% for > 60 minutes

 - 50-70% initial response; <50% at 1 year
 - limitations: tachyphylaxis and side-effects

Botulinum Toxin

- prevents ACH release at NM junction
- 90% initial response; 60% at 1 year
- Needs repetitive sessions

Pneumatic Dilatation

- Balloon dilatation to 300 psi
- disrupt circular muscle
- 60-95% initial success; 60% at 5 years
- recent series suggest 20-40% will require re-dilatation
- Success increases with repeat dilatations
- risk of perforation 1-13% (usually 3-5%); death 0.2-0.4%

Surgical Treatment

- surgical myotomy (open or minimally-invasive)
 - >90% initial response; 85% at 10 years; 70% at 20 years (85% at 5 years with min. inv. techniques)
 - <1% mortality; <10% major morbidity
 - 10-25% acutely develop reflux, up to 52% develop late reflux



Spastic Motility Disorders of the Esophagus

Spastic Motility Disorders of the Esophagus

- Diffuse Esophageal Spasm
- Nutcracker Esophagus
- Hypertensive LES
- Nonspecific Esophageal Dysmotility

Epidemiology

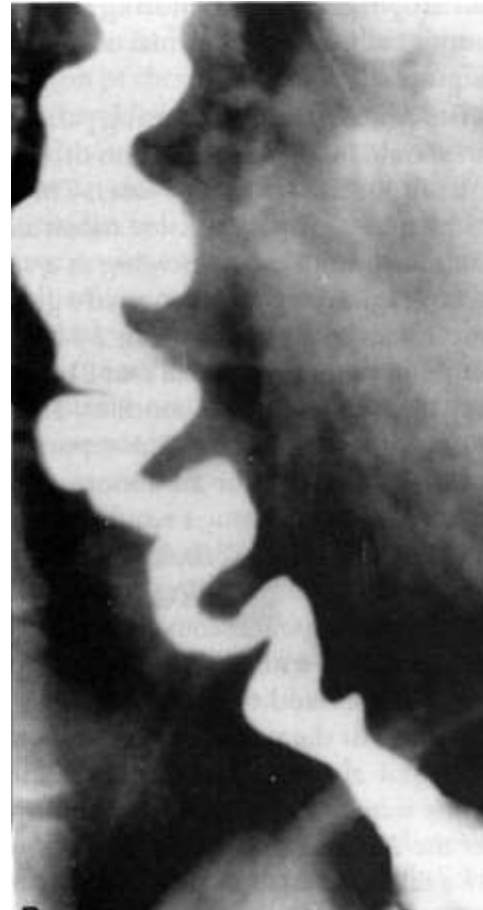
- Any age (mean 40 yrs)
- Female > Male

Clinical Presentation

- Dysphagia to solids and liquids
 - intermittent and non-progressive
 - present in 30-60%, more prevalent in DES (in most studies)
- Chest Pain
 - constant % across the different disorders (80-90%)
 - swallowing is not necessarily impaired
 - can mimic cardiac chest pain
- Pyrosis (20%) and IBS symptoms (>50%)
- Symptoms and Manometry correlate poorly

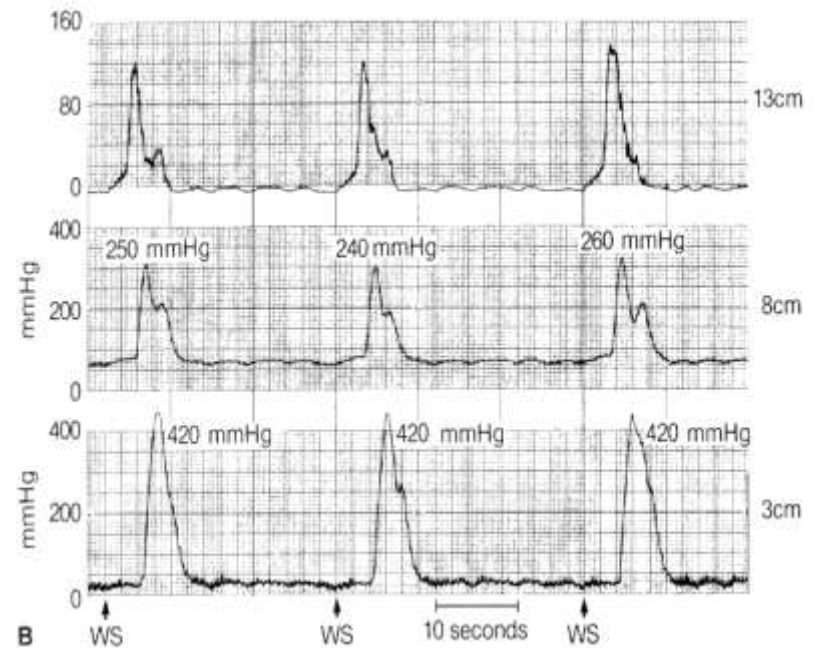
Diffuse Esophageal Spasm

- frequent non-peristaltic contractions
 - simultaneous onset (or too rapid propagation) of contractions in two or more recording leads
 - occur with >30% of wet swallows (up to 10% may be seen in “normals”)



Nutcracker Esophagus

- high pressure peristaltic contractions
 - avg pressure in 10 wet swallows is >180 mm Hg
- 33% have long duration contractions (>6 sec)
- may inter-convert with DES



Hypertensive LES

- high LES pressure
 - >45 mm Hg
- normal peristalsis
- often overlaps with other motility disorders

Nonspecific Esophageal Dysmotility

- abnormal motility pattern
- fits in no other category
 - non-peristalsis in 20-30% of wet swallows
 - low pressure waves (<30 mm Hg)
 - prolonged contractions

Diagnosis of Spastic Motility Disorders of the Esophagus

- Manometry
- Barium Esophagram
- Endoscopy
- PH monitoring

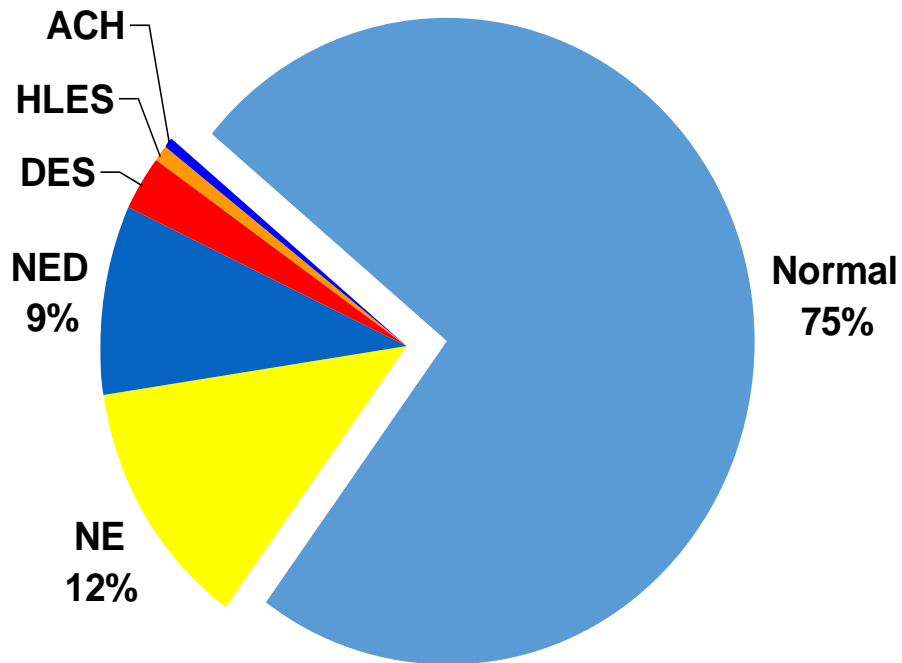
Spastic Motility Disorders of the Esophagus

- **treatment**

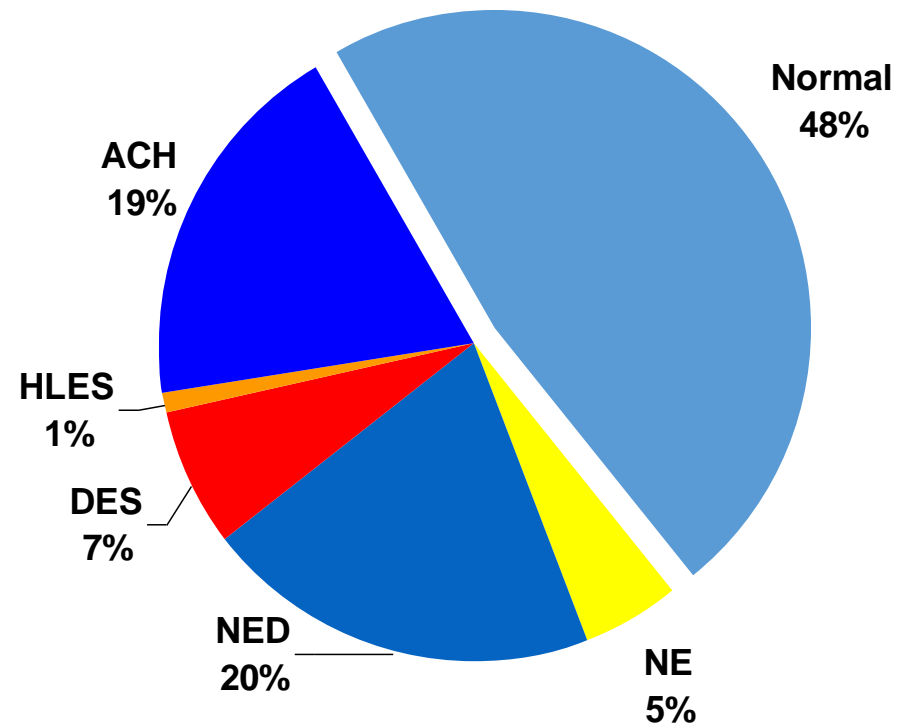
- reassurance
- nitrates, anticholinergics, hydralazine - *all unproven*
- calcium channel blockers - *too few data with negative controlled studies in chest pain*
- psychotropic drugs – *trazodone, imipramine and setraline effective in controlled studies*
- dilation - *anecdotal reports, probable placebo effect*

Manometry in Esophageal Symptoms

Non-Cardiac Chest Pain



Dysphagia



Hypomotility Disorders

- primary (idiopathic)
 - aging produces gradual decrease in contraction strength
 - reflux patients have varying degrees of hypomotility
 - more common in patients with atypical reflux symptoms
 - usually persists after reflux therapy
 - defined as
 - low contraction wave pressures (<30 mm Hg)
 - incomplete peristalsis in 30% or > of wet swallows

Hypomotility Disorders

- secondary
 - scleroderma
 - in >75% of patients
 - progressive, resulting in aperistalsis in smooth-muscle region
 - incompetent 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

Non ischemic Chest Pain

- remains poorly understood (*functional chest pain*)
- *enthusiastic* investigation finds numerous associations in studies
 - psychiatric disorders (depression, panic or anxiety disorder...)
 - esophageal disorders (GERD, motility disorders...)
 - musculoskeletal disorders
 - cardiac disease (microvascular, MVP, tachyarrhythmias...)

Non ischemic Chest Pain

- GERD is by far the most common, diagnosable, *esophageal* cause
 - 50-60% of patients have heartburn or acid regurgitation symptoms
 - 50% have abnormal esophageal pH studies (not always correlating to sxS)
 - *very* low incidence of endoscopic findings
 - “PPI Test” may be best and most cost-effective approach
- a small subset of patients with non-GERD NCCP display a variety of esophageal motility disorders
 - symptoms and motility findings correlate poorly
 - esophageal hypersensitivity/hyperalgesia may explain the symptoms

GERD

- **36-77% of all Americans experience GERD**
- **– 7% have daily GERD symptoms**
- **– 14-20% weekly symptoms**
- **– 15-50% monthly**
- **Symptoms include: heartburn, acid regurgitation, water brash, dysphagia,**
- **atypical symptoms (asthma, globus,**
- **laryngitis, cough, throat clearing)**

Pathophysiology

- Lower esophageal sphincter dysfunction
- Delayed gastric emptying
- Esophageal dysmotility
- +/- hiatal hernia
- Repetitive mucosal injury / esophagitis
- Barrett's Esophagus

Medical Treatment

- **Lifestyle modifications**
 - **avoid coffee, fatty foods, smoking; lose weight, raise head of bed, eliminate late night meals**
- **Acid suppressin via PPI's**

Indications for Surgery

- Failed medical management
- Need for lifelong medical therapy
- Hiatal hernia
- Atypical symptoms with (+) pH probe
- Complications
 - – Barrett's esophagus (5-15% develop BE)
 - – Erosive esophagitis

Surgical Treatment

- Pre-operative evaluation
 - – Esophagram
 - – EGD
 - – Manometry (resting LES >5, length >2cm)
 - – 24-hr esophageal pH monitoring

Surgical Treatment

- Laparoscopic Nissen Fundoplication
- Goals of antireflux surgery:
 - – Recreate Angle of His
 - – Reconstitute LES with wrap
- Predictors of good surgical outcome:
 - – typical symptoms (heartburn, regurg)
 - – abnormal pH score, but NML motility
 - – clinical response to acid suppression
- therapy

Other New Treatments

- Stretta...radiofrequency ablation of LES
- Enteryx, Gatekeeper...implanted biopolymer into LES
- Endocinch, Plicator...endoscopic suturing to recreate LES

GERD Controversies

- Are meds better than antireflux surgery?
- Does antireflux surgery allow regression of Barrett's esophageal better than meds?
- Which is more cost effective?
- Does symptom relief correlate with esophageal acid exposure?
- Where do the newer endoscopic therapies stand?

GERD Medical Vs Surgical Therapy

A multicenter Nordic study evaluated treatment failures of Omeprazole to Nissen fundoplication – failure defined as: mod/severe heartburn, dysphagia or regurg; grade 2 esophagitis; > 8 wks post-op requiring PPI At 12 months surgery was favored But at five year follow-up, open surgery appeared superior, but when allowing for escalating doses of PPI, each strategy was similar for symptom control
Lundell et al. Gastroenterology 114:A207, 1998.
Lundell et al. JACS 192:172-179, 2001

GERD Medical Vs Surgical Therapy

UK study evaluated *laparoscopic* Nissen to PPI therapy in 217 randomized patients with chronic GERD

At three months, LNF group had improved LES pressure, DeMeester acid exposure score, GI symptom and general well-being score as compared to PPI group, and lasted to twelve months

Mahon et al. Brit Journ Surg 92:695-699, 2005.

Regression Of Barrett's

PPI compared to LNF in 35 non-randomized pts with low-grade dysplasia detected on surveillance EGD

12 of 19 (63%) in PPI group had regression of LGD to Barrett's compared to 15 of 16 (93%) of LNF pts at 12 and 18 months

Is biliopacreatic reflux to blame for BE?

Rossi et al. Annals of Surgery 243:58-63, 2006.

DO Symptoms Correlate with Treatment (*Success/Failure*)

24 hr pH and DeMeester acid scores compared in 70 pts on no meds, on PPIs, or after antireflux surgery

LES pH decreased most by LNF

18 of 30 PPI pts asymptomatic but had pathologic pH probe testing

19 LNF pts complained of heartburn/regurg, only two had positive pH probe

Jenkinson et al. Brit Jour Surg 91:1460-1465, 2004.

Hiatal Hernia

Pathophysiology & Classification

- **Type I - sliding**
- **Type II - paraesophageal**
- **Type III - para and sliding component**
- **Type IV - other viscera involved**



Clinical Presentation

- **postprandial fullness (63%),**
- **Reflux (31%),**
- **Dysphagia (34%),**
- **Bleeding (24%)**
- **Regurgitation/vomiting (36%)**
- **Dyspnea (11%)**

Work Up



Surgical Treatment

- **Effective repair includes:**
 - **Excision of hernia sac**
 - **Reduction of hernia contents**
 - **Repair of crural defect**
- **– Fundoplication, gastropexy, PEG, esophageal lengthening (Collis gastroplasty)**

Upper Esophageal Motility Disorders

Overview

- cause oropharyngeal dysphagia (transfer dysphagia)
 - patients complain of *difficulty swallowing*
 - tracheal aspiration may cause symptoms
- pharyngoesophageal neuromuscular disorders
 - stroke
 - Parkinson's
 - poliomyelitis
 - ALS
 - multiple sclerosis
 - diabetes
 - myasthenia gravis
 - dermatomyositis and polymyositis
- upper esophageal sphincter (cricopharyngeal) dysfunction

Overview

- cricopharyngeal hypertension
 - elevated UES resting tone
 - poorly understood (reflex due to acid reflux or distension)
- cricopharyngeal achalasia
 - incomplete UES relaxation during swallow
 - may be related to Zenker's diverticula in some patients
- clinical manifestations
 - localizes as upper (cervical) dysphagia
 - within seconds of swallowing
 - coughing, choking, immediate regurgitation, or nasal regurgitation
- diagnosis: swallow evaluation & modified barium swallow