Esophageal Motility

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Alimentary Tract Motility

• Propulsion
  – Movement of food and endogenous secretions
• Mixing
  – Allows for greater contact of food with digestive enzymes and absorptive surface
• Reservoir

Determinants of GI Tract Motility

• Myogenic control
• Neurogenic control
• Endocrine factors

Myogenic Control

• Basic Electrical Rhythm:
  – intrinsic rhythmic fluctuation of smooth muscle membrane potential
• Pacemaker Cells:
  – set BER for the entire organ
• Slow waves:
  – spread from cell to cell via gap junctions

Enteric Nervous System

• Afferent and efferent arms
• Numerous interneurons in the ENS are highly integrated and receive input from:
  – CNS
  – efferent arm of ENS
• Afferent neurons receive input from ENS interneurons and these affect:
  – smooth muscle
  – blood vessels
  – secretory cells

Swallowing

• Oropharyngeal Phase
  – Involuntary & Voluntary Phases
  – Extremely rapid
  – Dx test: Video esophagram
• Esophageal Phase
  – Slow
  – Stereotyped
  – Dx test: Esophageal Manometry
Oropharyngeal Phase of Swallowing

- Moves ingested food and fluid into upper esophagus
- Prevents aspiration or regurgitation of the bolus
- Voluntary movement by the tongue of the bolus into the pharynx triggers the involuntary phase of swallowing

Esophageal Disease Symptoms

- Dysphagia
  - Oropharyngeal Dysphagia
  - Esophageal Dysphagia
- Pain
  - Odynophagia
  - Atypical Chest pain
- GE Reflux disease (GERD)
**Dysphagia**

- Oropharyngeal
  - Difficulty transferring bolus out of mouth
  - Associated with coughing & aspiration
- Esophageal
  - Sense of bolus "sticking in chest"
  - Mechanical causes
  - Motility disorders

**Esophageal Dysphagia**

**Mechanical Causes**

- Typically occurs with solid foods
- Frequently progressive, especially with malignancy
- Food impaction (with forced regurgitation) common
- Prominent weight loss only with malignancy

**Schatzki Ring**

**Esophageal Strictures**
Barrett's esophagus – definition

- “A change in the esophageal epithelium of any length that can be recognized at endoscopy and is confirmed to have intestinal metaplasia by biopsy”

Barrett's esophagus is a premalignant lesion for esophageal adenocarcinoma

Normal

- Endoscopy-negative reflux disease
- Erosive esophagitis
- ?
- Barrett’s esophagus
- ?
- Dysplasia

Esophageal adenocarcinoma
Esophageal Dysphagia

Motor Disorders

Achalasia

Esophageal Manometry
Normal Study

Achalasia

Achalasia

Achalasia
GERD
The sequellae of prolonged exposure of esophageal mucosa to caustic gastric refluxate

GERD: Pathogenesis

Defective Esophageal Clearance
- Ineffective peristalsis
- Reduced salivary secretion
- Reduced secretion from esophageal submucosal glands

LES ‘dysfunction’
- Inappropriate and prolonged transient relaxations
- Reduction in basal LES pressure/tone

Hiatal hernia
- May trap a reservoir of gastric contents above the diaphragm, increasing reflux
- May compromise LES function
Delayed gastric emptying

- May result in an increase in the volume of gastric contents available for reflux into the esophagus

- Exact role in GERD remains to be clarified

Bravo Capsule

Ambulatory pH Monitoring

- Strengths
  - Quantify reflux
  - Monitor during activities of daily living
  - Allows symptom correlation

- Limitations
  - Availability
  - Technique dependent
  - Cumbersome
  - Cost
  - Variability of response

Adapted from American College of Gastroenterology. An Update on GERD. 1995

Cumulative Remission On Omeprazole


Bravo Capsule

Nissen Fundoplication

Principles of Anti-Reflux Surgery

- Restore intra-abdominal esophagus
- Approximate diaphragmatic crura
- Reduce hiatus hernia
- Perform fundoplication
Mechanism of action of refluxate in GERD

Acid-peptic attack weakens cell junctions, leading to a widening of cell gaps and thus allowing acid penetration.

- Acid
- Pepsin
- Bicarbonate

Nerve ending
Tight cell junction
Widened cell junction

Orlando. Am J Gastroenterol 1996

Mechanism of action of refluxate in GERD

Penetration of acid and pepsin allows contact of acid with nerve endings and disrupts intracellular mechanisms leading to cell rupture and damage.

- Acid
- Pepsin
- Bicarbonate

Nerve ending

Orlando. Am J Gastroenterol 1996