QUIZ/TEST REVIEW NOTES
SECTION 2 GASTRIC PHASE OF DIGESTION
DIGESTIVE SYSTEM CHAPTER 21

I. ENTERIC NERVOUS SYSTEM

a. Defined and Correlation with Short Reflexes
   - Found in walls of LUMEN
   - Involved in short reflexes
     - Reflexes that originate within the enteric nervous system (ENS) and are integrated without outside input
     - It can also coordinate functions with autonomic neurons bringing signals from CNS
     - Control Motility, Secretion, Growth

b. Responses/Reflexes
   - Food bulk stretches the fundus of stomach, activating mechanoreceptors
   - GI peptides activate chemoreceptor’s
     - GI Peptides excite or inhibit motility and secretion
     - GI Peptides secreted may act as hormones or Paracrine signals
     - GI Peptides are secreted into the blood and may act on GI tract, accessory organs (pancreas), distant organs (brain)

At this stage it is no longer a Feedforward/cephalic reflex mechanism because food is present

Learning Objectives:
- Differentiate long and short reflexes of digestion
- Identify source and function of secretary products of stomach mucosal glands
- Explain molecular basis of gastric acid production, regulation of acid production, and treatment options for acid reduction, including proton-pump inhibitors
- Describe process of digestion, absorption, motility and secretion in stomach
- Describe neuroendocrine controls of gastric hase
- Describe digestive processes of carbohydrates, fats, and proteins in stomach
II. GASTRIC PHASE: GASTRIC SECRETIONS

a. Beginning

- Digestive activity in stomach begins with long vagal reflex of cephalic phase even before food arrives.
- When food arrives in stomach, stimuli in gastric lumen initiate series of short reflexes that constitute gastric phase of digestion.
- Distension/Expansion of stomach and peptide/amino acid presence in lumen activates: endocrine cells and enteric neurons.
  - Hormones, Neurocrine secretions, and Paracrine molecules influence motility and secretion.

b. Mucus Secretions

- Mucus surface cells secrete mucus and Bicarbonate ($\text{HCO}_3^-$).
- Mucus forms a physical barrier and bicarbonate creates chemical buffer.
- Bicarbonate is a chemical barrier that neutralizes acid on the mucus layer.
  - Drops the pH level down by $1 \times 10^5$; from a pH of 2 to a pH of 7 (acidic $\rightarrow$ neutral).

![Diagram of GastricJuice](image)

Gastric secretions influenced by both Long and Short Reflexes


c. Gastric Acid Secretion

1. G Cells
   - Secrete Gastrin
   - Stimulated by ACh, peptides, amino acids
   - Function: Stimulate gastric acid secretion

2. Enterochromaffin-like Cells (ECL)
   - Secrete Histamine
   - Stimulated by ACh, gastrin (from G Cells)
   - Function: Stimulates gastric acid secretion

3. Parietal Cells
   - Secrete Gastric Acid (HCL) and Intrinsic Factor
   - Stimulated by ACh, gastrin, histamine
   - Function:
     - HCL = Activates pepsin and kills bacteria
     - Intrinsic Factor = Complexes with Vitamin $\text{B}_{12}$ to permit absorption
   - Acid secretion is linked to bicarbonate absorption
d. Gastric Enzyme Secretion
   1. Chief Cells
      - Secrete pepsin (from pepsinogen) and gastric lipase
      - Stimulated by ACh, Acid, Secretin
      - Functions:
        • Pepsin: Digest proteins
        • Gastric Lipase: Digest fats

e. Hormone Secretion
   1. D Cells
      - Secreted somatostatin
      - Stimulated by Acid in the stomach
      - Function: Inhibits gastric acid secretion
OVERVIEW GASTRIC SECRETION

- Cephalic reflex, parasympathetic neurons from Vagus nerve stimulate G cells to release gastrin into the blood (1). The presence of amino acids or peptides in the lumen triggers a short reflex for gastrin release.
- Gastrin in turn promotes acid release, both directly and indirectly by stimulating histamine release (2).
- Histamine is released from ECL cells in response to gastrin and ACh from the enteric nervous system (1). Histamine diffuses to its target, the parietal cells, and stimulates acid secretion by combining with $H_2$ receptors on parietal cells.
- Acid in the stomach lumen stimulates pepsinogen release from chief cells through a short reflex (3). In the lumen, acid converts pepsinogen into pepsin, and protein digestion begins.
- Acid also triggers somatostatin release from D cells (4). Somatostatin acts via negative feedback to inhibit secretion of gastric acid, gastrin, and pepsinogen.
III. GASTRIC MOTILITY

a. Stimulated by
   1. Distension
   2. Long And Short Reflexes
      - Influenced by hormones, Paracrine signals and autonomic nervous system
         - Parasympathetic: Rest and digest/Enhance GI functions
         - Sympathetic: Fight or flight/Inhibit GI functions
      - Short Reflexes of ENS effect myenteric neurons that influence motility

b. Mixing
   1. Autorhythmic slow waves of peristalsis
      - Spontaneous cycles of depolarization and repolarization
      - Does not reach threshold with each cycle
      - Graded according to the amount of $Ca^{2+}$ that enters the fiber
      - The longer the duration of the slow wave the more actions potentials that fire, and the greater the contraction force in the muscle
      - Can be modified by neurotransmitters, hormones, or Paracrine molecules

\[\text{Diagram of Membrane potential (mV) and Force of muscle contraction over time.}\]

\[\text{Diagram shows action potentials firing with slow wave potentials reaching threshold.}\]

\[\text{Diagram shows force and duration of muscle contraction are directly related to the amplitude and frequency of action potentials.}\]

c. Gastric Emptying
   1. Peristalsis propels chime through pyloric sphincter
      - Peristaltic contractions create forward movement
      - Progressive waves of contraction that occur behind the bolus

\[\text{Diagram of time zero and seconds later showing direction of movement and bolus moving forward.}\]

2. Rate of emptying slowed by intestinal phase factors
IV. GASTRITIS AND PEPTIC ULCER DISEASE

a. Compromise of the mucus-bicarbonate barrier

**Definition:** Is a inflamed area in the stomach or duodenum that extends from a break in the surface epithelium past the muscularis mucosae;

1. Peptic (Gastric) ulcers
   - 10% Stomach
   - Most caused by bacterium H. Pylori, rest caused by high levels of acid from enhanced enzyme production
2. Frequency of Duodenal ulcers
   - 90% Duodenal
   - Most caused by bacterium H. Pylori, rest caused by high levels of acid from enhanced enzyme production
   - Can be found in duodenum from gastric acid and stomach enzymes chime may carrier, or which is present in the E.C.F. when the stomach passes into the duodenum

b. Causes

1. Helicobacter Pylori
   - Bacterium that thrives in high pH environment of stomach and Duodenum
   - H. Pylori exploit the large amounts of urea released by the stomach in protein/amino acid digestion
   - H. Pylori used urea to protect themselves from the acidic environment
2. Hypersecretion of acid (Dyspepsia)
   - Excessive gastrin production by pancreatic tumors
   - Hyperactive Vagus stimulation
     - Increased amount of parasympathetic stimulation
3. Aggravating factors
   - Alcohol
   - NSAIDs
     - Most common causes of ulcers
     - Nonsteroidal Anti-Inflammatory Drugs (Aspirin/Tylenol)
   - Caffeine and spices

c. Treatment

1. Elimination of Helicobacter Pylori
2. Acid control
   - Vagotomy
   - Acid Suppression
     1) Antacids
     2) H₂-Receptor Blocker
       - Histamine receptor antagonists
       - Bind competitively to histamine H₂ receptors on parietal cells
       - Histamine is a Paracrine that stimulates acid secretion by the stomach; so by inhibiting it you inhibit acid section
     3) Proton Pump Inhibitors
       - By inhibiting the efflux of protons from the parietal cells Cl⁻ has nothing to bind with; can no longer bind with H⁺ to form HCL
       - Block H⁺-K⁺-ATPase Pump

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**G.E.R.D = Gastro Esophageal Reflux Disease**