Mammalian Physiology

Gastrointestinal System: Motility and Secretion
Objectives

• Describe the structure of the digestive system
• Describe the functions of the stomach and intestines
• Discuss the functions of the other digestive organs – liver, gall bladder, pancreas
• Describe the various digestive secretions
• Describe the absorption of the major nutrients – carbohydrate, protein, fat
Digestive System: Overview

The alimentary canal or gastrointestinal (GI) tract digests and absorbs food.

Alimentary canal – mouth, pharynx, esophagus, stomach, small intestine, and large intestine.

Accessory digestive organs – teeth, tongue, gallbladder, salivary glands, liver, and pancreas.
Digestive Process

The GI tract is a “disassembly” line. Nutrients become more available to the body in each step.

Six Essential Elements
- Ingestion
- Propulsion
- Mechanical Digestion
- Chemical Digestion
- Absorption
- Defecation
Gastrointestinal Tract Activities

- Ingestion – taking food into the digestive tract
- Propulsion – swallowing and peristalsis
  - Peristalsis – waves of contraction and relaxation of muscles in the organ walls
- Mechanical digestion – chewing, mixing, and churning food
- Chemical digestion – catabolic breakdown of food
- Absorption – movement of nutrients from the GI tract to the blood or lymph
- Defecation – elimination of indigestible solid wastes
Mouth/Pharynx/Salivary Glands

Mastication
Initiation of swallowing reflex

Secretion: Function:
salt & water moisten food
mucus lubrication
amylase polysaccharide-digestion
Esophagus
Move food to stomach by peristaltic waves

Secretion: mucus
Function: lubrication
Stomach

Store, mix, dissolve, continue digestion of food
Regulate emptying of food into small intestine

Secretion: HCl, pepsin, mucus
Function: solubilize food particles, protein digestion, lubricate & protect epithelial surface
Secretion of enzymes & bicarbonate

Pancreas

Secretion:
- enzymes
- bicarbonate

Function:
- digest carbohydrates, fats, proteins, nucleic acids
- neutralize HCl entering small intestine from stomach
Secretion of bile

Liver

**Secretion:**
- bile salts
- bicarbonate

**Function:**
- solubilize fats
- neutralize HCl entering small intestine from stomach
Gallbladder

Store and concentrate bile between meals
Small intestine

Digestion and absorption of most substances
Mixing and propulsion of contents

Secretion:
- enzymes
- salt & water
- mucus

Function:
- food digestion
- maintain fluidity of luminal contents
- lubrication
Storage and concentration of undigested matter
Absorption of salt & water
Mixing and propulsion of contents
Defecation

Large intestine

Secretion: mucus
Function: lubrication
GI Tract

- External environment for the digestive process
- Regulation of digestion involves:
  - Mechanical and chemical stimuli – stretch receptors, osmolarity, pH and presence of substrate in the lumen
- Stimuli initiate reflexes that:
  - Activate or inhibit digestive glands
  - Mix lumen contents and move them along
  - Extrinsic control by CNS centers
  - Intrinsic control by local centers
Wall Structure of the GI Tract

From esophagus to the anal canal the walls of the GI tract have the same four tunics.

From the lumen outward they are: the mucosa, submucosa, muscularis externa, and serosa.

Each tunic has a predominant tissue type and a specific digestive function.
Wall Structure of the GI Tract

• **Mucosa**
  - Epithelial lining – villi, crypts, folds
  - Lamina propria – loose connective tissue with capillaries, mast cells
  - Muscularis mucosae – thin layer of smooth muscle

• **Submucosa**
  - Loose connective tissue & larger blood vessels

• **Muscularis externa**
  - Circular smooth muscle – constriction
  - Longitudinal smooth muscle - peristalsis

• **Serosa**
  - Enveloping layer of connective tissue
Mucosa

• Moist epithelial layer that lines the lumen of the alimentary canal
• Its three major functions are:
  – Secretion of mucus
  – Absorption of the end products of digestion
  – Protection against infectious disease
• Consists of three layers: a lining epithelium, lamina propria, and muscularis mucosae
Mucosa: Epithelial Lining

- Consists of simple columnar epithelium and mucus-secreting goblet cells
- The mucus secretions:
  - Protect digestive organs from digesting themselves
  - Ease food along the tract
- Stomach and small intestine mucosa contain:
  - Enzyme-secreting cells
  - Hormone-secreting cells (making them endocrine and digestive organs)
Mucosa: Lamina Propria and Muscularis Mucosae

- Lamina Propria
  - Loose areolar and reticular connective tissue
  - Nourishes the epithelium and absorbs nutrients
  - Contains lymph nodes important in defense against bacteria

- Muscularis mucosae – smooth muscle cells that produce local movements of mucosa
Mucosa: Other Sublayers

- Submucosa – dense connective tissue containing elastic fibers, blood and lymphatic vessels, lymph nodes, and nerves
- Muscularis externa – responsible for segmentation and peristalsis
  - Inner circular smooth muscle – constriction
  - Outer longitudinal smooth muscle – peristalsis
- Serosa – the protective visceral peritoneum
  - Replaced by the fibrous adventitia in the esophagus
  - Retroperitoneal organs have both an adventitia and serosa
Structure of GI Wall
ANS Innervation of GI Tract

Enteric Nervous System is modulated by both sympathetic and parasympathetic branches of ANS.

Parasympathetic input is stimulatory
Sympathetic input is inhibitory
Enteric Nervous System

ENS consists of sensory neurons, interneurons, & motor neurons

Myenteric plexus situated between longitudinal and circular smooth muscle layers

Submucosal plexus situated between circular smooth muscle and muscularis mucosae layers
Enteric Nervous System

- Composed of two major intrinsic nerve plexuses
  - Submucosal nerve plexus – regulates glands and smooth muscle in the mucosa
  - Myenteric nerve plexus – Major nerve supply that controls GI tract mobility
Reflex Pathways in GI System

Segmentation and peristalsis are largely automatic involving local reflex arcs.

Linked to the CNS via long autonomic reflex arc.
Nervous Control of the GI Tract

Intrinsic controls
Nerve plexuses near the GI tract initiate short reflexes
Short reflexes are mediated by local enteric plexuses (gut brain)

Extrinsic controls
Long reflexes arising within or outside the GI tract
Involve CNS centers and extrinsic autonomic nerves
GI Function

Average amounts of solids and fluids ingested, secreted, absorbed, and excreted from the GI tract daily

99% of the fluid secreted into the GI tract is reabsorbed – only 100 ml is lost in feces

Almost all salts in secreted fluids are reabsorbed
Digestive enzymes are also digested and resulting amino acids are absorbed
Functions of the Mouth & Esophagus

- Food is ingested
- Mechanical digestion begins (chewing)
- Propulsion is initiated by swallowing
- Salivary amylase begins chemical breakdown of starch
- The pharynx and esophagus serve as conduits to pass food from the mouth to the stomach
Functions of the Stomach

- Storage of large quantities of food until food can be processed in the duodenum
- Mixing of food with gastric secretions to form chyme
- Regulate emptying of chyme into small intestine for proper digestion and absorption

Corpus (body) secretes mucus, pepsinogen, and HCl

Antrum secretes mucus, pepsinogen, and gastrin
Functions of Intestines

- Small intestine
  - Nutrient absorption
  - Peristaltic activity
  - Increased after a meal – gastroenteric reflex
  - Hormones – gastrin, cholecystokinin, insulin, serotonin

- Large intestine – colon
  - Absorption of water and electrolytes from chyme to form solid feces
  - Storage of fecal matter until it can be expelled
  - Movements are normally sluggish
Gastric Motility

• Motor activity of GI tract
  – Segmental contractions
    • Nonpropulsive movement of luminal contents
    • Increased mixing or churning
  – Peristaltic contractions
    • Propulsion of luminal contents in caudal direction
    • Elimination of nondigested, nonabsorbed material
  – Reservoirs for luminal contents
    • Stomach
    • Large intestine

Average time for food to pass through intestinal tract is 48 hrs (range is 24-96 hrs)
Digestive Secretions - Mouth

• Secretions
  – $\alpha$-amylase – breakdown of starches but little benefit because of short time food is in mouth
  – Mucin – lubrication & surface protection

• Mechanical breakdown of food

• Hydration of dry food

Action of salivary glands initiated by sight, smell, taste, thought of food
Motility of the Mouth

• Mastication (chewing)
  – Rhythmic movement of jaws, tongue, and lips when food is in the mouth
  – Force
    • Incisors create a pressure of 30-70 lbs/in$^2$
    • Molars create a pressure of 75-200 lbs/in$^2$
  – Control is voluntary
  – Rhythmicity is reflexly maintained through tactile stimuli in the mouth

• Deglutition (swallowing)
Deglutition (Swallowing)

Initiated voluntarily, then reflex process mediated by medulla oblongata

- Involves the coordinated activity of the tongue, soft palate, pharynx, esophagus and 22 separate muscle groups
- Buccal phase – bolus is forced into the oropharynx
- Pharyngeal-esophageal phase – controlled by the medulla and lower pons
  - All routes except into the digestive tract are sealed off
- Peristalsis moves food through the pharynx to the esophagus
Mechanics of Swallowing

1. Swallowing is initiated when tongue forces food into back of mouth

2. Soft palate elevates & lodges against back wall of pharynx blocking food from entering nasal cavity

3. Epiglottis closes preventing food entry into trachea; respiration is inhibited

4. Upper esophageal sphincter relaxes, food moves toward stomach by peristalsis (2-4 cm/sec), reaching LES in about 5-9 sec after swallowing
Motility of Esophagus

- **Esophageal Dysfunction**
  - Gastroesophageal reflux (heartburn)
    - Infrequent reflux of small volumes in all individuals
    - Reflux of air leads to belching
    - LES incompetence leads to inflammation of lower end of esophagus
  - Esophageal pain
    - Uncoordinated contraction (spasm) of esophagus
  - Dysphagia (difficulty in swallowing)
    - Decrease in propulsive force
    - Obstruction
    - Uncoordination of contraction and relaxation
Stomach

- Chemical breakdown of proteins begins and food is converted to chyme
- Cardiac region – surrounds the cardiac orifice
- Fundus – dome-shaped region beneath the diaphragm
- Body – midportion of the stomach
- Pyloric region – made up of the antrum and canal which terminates at the pylorus
- The pylorus is continuous with the duodenum through the pyloric sphincter
Microscopic Anatomy of the Stomach

- **Muscularis** – has an additional oblique layer that:
  - Allows the stomach to churn, mix, and pummel food physically
  - Breaks down food into smaller fragments
- **Epithelial lining** is composed of:
  - Goblet cells that produce a coat of alkaline mucus
    - The mucous surface layer traps a bicarbonate-rich fluid beneath it
- **Gastric pits** contain gastric glands that secrete gastric juice, mucus, and gastrin
Glands of the Stomach Fundus & Body

- Gastric glands of the fundus and body have a variety of secretory cells
  - Mucous neck cells – secrete acid mucus
  - Parietal cells – secrete HCl and intrinsic factor
  - Chief cells – produce pepsinogen
    - Pepsinogen is activated to pepsin by:
      - HCl in the stomach
      - Pepsin itself via a positive feedback mechanism
  - Enteroendocrine cells – secrete gastrin, histamine, endorphins, serotonin, cholecystokinin (CCK), and somatostatin into the lamina propria
Digestion in the Stomach

- The stomach:
  - Holds ingested food
  - Degrades this food both physically and chemically
  - Delivers chyme to the small intestine
  - Enzymatically digests proteins with pepsin
  - Secretes intrinsic factor required for absorption of vitamin $B_{12}$
Response of the Stomach to Filling

- Stomach pressure remains constant until about 1L of food is ingested
- Relative unchanging pressure results from reflex-mediated relaxation and plasticity
- Reflex-mediated events include:
  - Receptive relaxation – as food travels in the esophagus, stomach muscles relax
  - Adaptive relaxation – the stomach dilates in response to gastric filling
- Plasticity – intrinsic ability of smooth muscle to exhibit the stress-relaxation response
Response of the Stomach to Filling

Reception of Food

In conjunction with the relaxation of the LES, the fundus and body of the stomach relax (receptive relaxation). As stomach fills, it can relax further (stress-relaxation) and accommodate a full meal without any increase in internal pressure. This effect is mediated by vagus nerve (parasympathetic).
Stomach Mixing and Emptying

A. When filled with food, vigorous peristaltic contractions cause mixing and homogenizing of food
B. As the peristaltic wave moves from the antrum towards the pyloris, the pressure in that area increases
C. The closed pyloric sphincter prevents expulsion of the chyme into the duodenum and retropulsion occurs towards the fundus

The continual forward and backward movement of the chyme through narrowed contracted portions of the stomach leads to homogenization of the contents.
Gastric Contractile Activity

- Peristaltic waves move toward the pylorus at the rate of 3 per minute
- This basic electrical rhythm (BER) is initiated by pacemaker cells (cells of Cajal)
- Most vigorous peristalsis and mixing occurs near the pylorus
- Chyme is either:
  - Delivered in small amounts to the duodenum or
  - Forced backward into the stomach for further mixing (grinding)
Regulation of Gastric Emptying

- Gastric emptying is regulated by:
  - The neural enterogastric reflex
  - Hormonal (enterogastrone) mechanisms
- These mechanisms inhibit gastric secretion and duodenal filling
- Carbohydrate-rich chyme quickly moves through the duodenum
- Fat-laden chyme is digested more slowly causing food to remain in the stomach longer

Key:
- Initial stimulus
- Physiological response
- Result
- Stimulate
- Inhibit

Presence of fatty, hypertonic, acidic chyme in duodenum

Duodenal entero-endocrine cells

Chemoreceptors and stretch receptors

Enterogastrones (secretin, CCK, GIP)

Target

Enteric neurons

CNS centers: SNS activity; parasympathetic activity

Duodenal stimuli decline

Contractile force and rate of stomach emptying decline
Gastric Emptying

Water/saline empty rapidly; solids must be reduced to < 2 mm before emptying

In general, gastric contents stimulate and duodenal contents inhibit motility

Acid in duodenum inhibits via sympathetic and vagus nerves (enterogastric reflex)
Fat elicits secretin, cholecystokinin, & gastric inhibitory peptide which inhibit gastric motility
Regulation of Gastric Emptying

Gastric emptying is inhibited by
- distension of the duodenum
- presence of fat
- high acidity
- hypertonic solutions

About 3% of stomach contents are evacuated per minute
Gastric Dysfunction

• Vomiting
  • Reflex stimulated from stomach irritation, other abdominal viscera, noxious sights or smells, pyloric obstruction
  • Vomiting center in dorsolateral reticular formation in medulla
  • Center can also be stimulated from changes in brain
  • Sequence of events: few deep respirations → glottis closes, soft palate elevates → contraction of diaphragm and abdominal muscle; relaxation of body & fundus of stomach and esophagus; pyloric antrum contracts → stomach contents are emptied

  – Gastric pain due to excessive contractions – peptic ulcer
Digestive Secretions - Stomach

- Gastric secretions
  - Stimulated by acetylcholine, gastrin, histamine
  - HCl secreted by parietal cells
    - 0.2-0.5% HCl, pH1.0 – denatures proteins
  - Pepsin secreted by peptic cells
    - Chief cells → pepsinogen → pepsin when pH 3-5
    - Initiates protein digestion – breaks proteins into polypeptides
    - Secretion stimulated by cAMP (secretin, $\beta_2$ receptors, PGE$_2$) and Ca$^{2+}$ (Ach, gastrin/CCK)
  - End product of gastric digestion is chyme
Regulation of Gastric Secretion

• Neural and hormonal mechanisms regulate the release of gastric juice
• Stimulatory and inhibitory events occur in three phases
  – Cephalic (reflex) phase: prior to food entry
  – Gastric phase: once food enters the stomach
  – Intestinal phase: as partially digested food enters the duodenum

Signals for acid secretion
Acetylcholine (+)
Gastrin (+)
Histamine (+)
Somatostatin (-)
Gastric Secretion

Non-parietal cell secretion – Na⁺ rich basal secretion

Parietal cell secretion – HCl

The proximal portion of the stomach secretes acid, pepsinogens, bicarbonate & mucus

The distal portion releases gastrin & somatostatin
Parietal Cell Secretion of HCl

H⁺ derived from CO₂ - H₂O dissociation by the carbonic anhydrase reaction is actively transported into lumen via H/K ATPase

Resulting bicarbonate is secreted into blood in exchange for chloride

K⁺ and Cl⁻ move into lumen via membrane channels
Stimulation of HCl Secretion

- HCl secretion is stimulated by ACh, histamine, and gastrin through second-messenger systems
- Release of hydrochloric acid:
  - Is low if only one ligand binds to parietal cells
  - Is high if all three ligands bind to parietal cells
- Antihistamines block H$_2$ receptors and decrease HCl release
HCl Secretion by Parietal Cell

- Metabolism → CO₂
  - HCO₃⁻ + H⁺ → H₂CO₃
- Cl⁻ → ATP → H⁺ → Lumen of gland
- Cl⁻ → K⁺
- Cl⁻ → Na⁺ → ATP → K⁺
Gastric Acid Secretagogues

ACh, gastrin, & histamine bind directly to parietal cell receptors
ACh released from vagus nerve endings, gastrin from D cells
Histamine (synthesized from histidine) & released from enterochromaffin-like cells by action of ACh and gastrin (indirect pathway)
Signal Transduction in Parietal Cell

ACh (muscarinic receptor) & Gastrin (CCK\textsubscript{B} receptor) bind to GTP binding protein → phospholipase C → phosphatidylinositol 4,5 bisphosphate → inositol 1,4,5 triphosphate → Ca\textsuperscript{2+} and diacylglycerol → protein kinase C

Histamine binds to GTP binding protein → adenylyl cyclase → cAMP → protein kinase A

Somatostatin binds to GTP binding protein → inhibits adenylyl cyclase
Pepsinogen Secretion

ACh and gastrin also stimulate chief cells to release pepsinogen via IP$_3$ – DAG pathway
Other stimuli include VIP, secretin, nor-epinephrine, and CCK

Release of acid and pepsinogen are highly correlated
Gastric Acid Secretion Phases
Basal Phase

Interdigestive phase
Circadian rhythm
(lowest in morning, highest at night)

Intragastic pH is a function of:
- gastric acid secretion
- buffering power of food
- rate of gastric emptying
Gastric Acid Secretion Phases
Cephalic Phase

• Initiated by
  – Sight
  – Smell
  – Taste
  – Chewing
  – Swallowing
  – Thought
  – Other stimuli (ie sound)

• Mediated by vagus nerve
  – ACh stimulated acid release by parietal cells
  – ACh triggered release of histamine from ECL cells
  – Gastrin stimulated acid release by parietal cells and histamine release from ECL cells
  – Inhibited D cell release of somatostatin, reducing somatostatin’s inhibitory effect on gastrin release

30% of total acid secretion occurs prior to food entering stomach
Distension reflex activates (1) local ENS pathway which secretes ACh and (2) vagal afferent ↔ vagal efferent loop → acid secretion via 4 pathways.

Chemical reflex – peptides and amino acids → G cells → gastrin release → acid secretion.

50-60% of total gastric acid secretion.
Gastric Acid Secretion Phases
Intestinal Phase

Peptones & amino acids:
1. Stimulate duodenal G cells to secrete gastrin → acid
2. Stimulate intestinal endocrine cells to secrete entero-oxyntin → acid
3. Amino acids stimulate acid secretion by?

5-10% of total gastric acid secretion
Gastric Inhibitory Mechanisms

- Acidified chyme exerts a negative effect on gastrin release in the stomach
- Enterogastrones are hormones responsible for this effect
  - Secretin released from duodenal mucosa when chyme pH < 4.5 inhibits parietal cell secretion of acid, inhibits gastrin release, stimulates chief cell secretion of pepsingens
  - Peptides, amino acids, & free fatty acids stimulate duodenal release of cholecystokinen-pancreozymin, a competitive inhibitor of gastrin stimulated acid secretion
  - Gastric inhibitory peptide released from duodenal mucosa in response to fat and carbohydrate inhibits gastrin release
Protection of Gastric Surface

• The stomach is exposed to the harshest conditions in the digestive tract
• Gastric gradients
  – intraluminal pH in stomach = 1; pH of gastric epithelial cells = 7.2
  – Intragastric [Na⁺] = 5 mM; plasma [Na⁺] = 140 mM
• How are these gradients maintained?
• Why aren’t epithelial cells destroyed by luminal acidity?
• Why don’t luminal pepsins digest the epithelial cells?

• Answer is gastric diffusion barrier
• To keep from digesting itself, the stomach has a mucosal barrier with:
  – A thick coat of bicarbonate-rich mucus on the stomach wall
  – Epithelial cells that are joined by tight junctions
  – Gastric glands that have cells impermeable to HCl
• Damaged epithelial cells are quickly replaced
Protection of Gastric Surface

1. Apical membrane is relatively impermeable to acid
2. Mucous-gel layer overlying surface epithelial cells
3. Bicarbonate microclimate adjacent to surface epithelial cells maintaining a high pH

Mucous acts as diffusion barrier for $\text{H}^+$ & pepsins, and traps alkaline solution of $\text{HCO}_3^-$
Release of Gastric Juice

**Stimulatory Events**

1. **Cephalic phase**
   - Sight and thought of food → Cerebral cortex → Hypothalamus → Vagus nerve and medulla oblongata
   - Stimulation of taste and smell receptors → Hypothalamus → Vagus nerve

2. **Gastric phase**
   - Stomach distension activates stretch receptors → Vagovagal reflexes → Medulla → Vagus nerve → G cells → Gastrin release to blood
   - Food chemicals (especially peptides and caffeine) and rising pH activate chemoreceptors → Vagovagal reflexes → Medulla → Vagus nerve → G cells → Gastrin release to blood

3. **Intestinal phase**
   - Presence of low pH and partially digested foods in duodenum when stomach begins to empty → Intestinal (enteric) gastrin release to blood

**Inhibitory Events**

1. **Cerebral cortex**
   - Lack of stimulatory impulses to parasympathetic center → Gastrin secretion declines → G cells

2. **Sympathetic nervous system (SNS) activation**
   - Excessive acidity (pH 2) in stomach → G cells
   - Emotional upset

**Key:**

- Stimulate
- Inhibit

*Distension of duodenum; presence of fatty, acidic, hypertonic chyme, and/or irritants in the duodenum*
Small Intestine

- Runs from pyloric sphincter to the ileocecal valve
- Has three subdivisions: duodenum, jejunum, and ileum
- The bile duct and main pancreatic duct:
  - Join the duodenum at the hepatopancreatic ampulla
  - Are controlled by the sphincter of Oddi
- The jejunum extends from the duodenum to the ileum
- The ileum joins the large intestine at the ileocecal valve
Small Intestine: Microscopic Anatomy

• Structural modifications of the small intestine wall increase surface area
  – Plicae circulares: deep circular folds of the mucosa and submucosa
  – Villi – fingerlike extensions of the mucosa
  – Microvilli – tiny projections of absorptive mucosal cells’ plasma membranes

• The epithelium of the mucosa is made up of:
  – Absorptive cells and goblet cells
  – Enteroendocrine cells
  – Interspersed T cells called intraepithelial lymphocytes (IELs)
    • IELs immediately release cytokines upon encountering antigens

• Cells of intestinal crypts secrete intestinal juice
  – Secreted by intestinal glands in response to distension or irritation of the mucosa
  – Slightly alkaline and isotonic with blood plasma
  – Largely water, enzyme-poor, but contains mucus
Small Intestine: Microscopic Anatomy

(a) Vein carrying blood to hepatic portal vessel
Muscle layers
Large circular folds
Villi
Lumen

(b) Absorptive cells
Lacteal
Goblet cell
Blood capillaries
Intestinal crypt
Muscularis mucosae
Duodenal glands
Submucosa

(c) Microvilli

(d) Crypt
Goblet cells

68
Structure of Small Intestine Villi

Epithelium lining intestine is not flat – surface area is increased by villi – finger like projections about 0.5 mm long
Apical surface is covered with a brush border of about 1,500 microvilli
Brush border contains enzymes and carrier proteins for absorption of monosaccharides and amino acids

Fat is absorbed via lacteals – blind-ended lymphatic vessels
Other nutrients are absorbed via capillary network
Digestion in the Small Intestine

- As chyme enters the duodenum:
  - Carbohydrates and proteins are only partially digested
  - No fat digestion has taken place

- Digestion continues in the small intestine
  - Chyme is released slowly into the duodenum
  - Because it is hypertonic and has low pH, mixing is required for proper digestion
  - Required substances needed are supplied by the liver
  - Virtually all nutrient absorption takes place in the small intestine
Motility in the Small Intestine

• Segmental contraction – mixing of chyme
  • Graded frequency of contraction: duodenum 12/min, ileum 8/min
  • Thicker muscle proximally = more forceful contractions
  • Chyme propelled distally
  • Fasted = synchronized, rhythmic contractions
  • Fed = segmental + peristaltic contractions

• Dysfunction
  • Inflammation and obstruction may cause excessive contraction and pain (cramping)
  • Inflammation may cause diarrhea
  • Paralysis may occur with handling during surgery
Motility in the Small Intestine

• The most common motion of the small intestine is segmentation
  – It is initiated by intrinsic pacemaker cells (Cajal cells)
  – Moves contents steadily toward the ileocecal valve
• After nutrients have been absorbed:
  – Peristalsis begins with each wave starting distal to the previous
  – Meal remnants, bacteria, mucosal cells, and debris are moved into the large intestine
• Local enteric neurons of the GI tract coordinate intestinal motility
• Cholinergic neurons cause:
  – Contraction and shortening of the circular muscle layer
  – Shortening of longitudinal muscle
  – Distension of the intestine
• Other impulses relax the circular muscle
• The gastroileal reflex and gastrin:
  – Relax the ileocecal sphincter
  – Allow chyme to pass into the large intestine
Large Intestine

- Is subdivided into the cecum, appendix, colon, rectum, and anal canal
- Colon has distinct regions: ascending colon, transverse colon, descending colon, and sigmoid colon
- The sigmoid colon joins the rectum
- The anal canal, the last segment of the large intestine, opens to the exterior at the anus
Large Intestine

• Has three unique features:
  – Teniae coli – three bands of longitudinal smooth muscle in its muscularis
  – Haustra – pocketlike sacs caused by the tone of the teniae coli
  – Epiploic appendages – fat-filled pouches of visceral peritoneum

Primary functions
  - Absorb fluid and electrolytes and solidify ileocecal material
  - Absorb short-chain fatty acids formed by fermentation of dietary carbohydrates not absorbed in small intestine
  - Storage of colonic content – reservoir function
  - Elimination of colonic contents in a regulated and controlled fashion, largely under voluntary control

Though essential for comfort, the colon is not essential for life
Large Intestine

• Two distinct organs
  • Proximal (ascending and transverse colon)
    – Fluid and electrolyte absorption
    – Fermentation
    – Non-propulsive segmentation (mixing)
    – Mass peristalsis (20 cm distally, 2-3 times/day)
  • Distal (descending colon, sigmoid colon, rectum)
    – Final desiccation
    – Reservoir function
    – Non-propulsive segmentation
    – Mass peristalsis
Large Intestine: Microscopic Anatomy

- Colon mucosa is simple columnar epithelium except in the anal canal
- Colon has numerous deep crypts lined with goblet cells
- Anal canal mucosa is stratified squamous epithelium
- Anal sinuses exude mucus and compress feces
- Superficial venous plexuses are associated with the anal canal
- Inflammation of these veins results in itchy varicosities called hemorrhoids
Motility of the Large Intestine

• Haustral contractions
  – Slow segmenting movements that move the contents of the colon
  – Haustra sequentially contract as they are stimulated by distension

• Presence of food in the stomach:
  – Activates the gastrocolic reflex
  – Initiates peristalsis that forces contents toward the rectum

• Dysfunction
  – Inflammation may cause diarrhea
  – ‘Irritable bowel’ or ‘spastic colon’ may cause pain and diarrhea
  – Constipation may be due to obstruction but commonly due to dietary factors
  – Diseases – cholera – decreased fluid reabsorption $\rightarrow$ dehydration
    $\rightarrow$ death?
Defecation

- Distension of rectal walls caused by feces:
  - Stimulates contraction of the rectal walls
  - Relaxes the internal anal sphincter
- Voluntary signals stimulate relaxation of the external anal sphincter and defecation occurs