Introduction to Digestion

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qums
Digestive System is composed of:

1. Digestive tract
2. Accessory Organs
Digestive System

The primary function of the gastrointestinal (alimentary) tract is:

to process ingested food and provide the body with nutrients, water, and electrolytes.
Function of the Digestive System

(A) motility

(B) Secretion and Digestion
Dissolving and breaking-down of nutrients into smaller units

(c) Absorption
Absorption of these units (+ water) through the intestinal tract into the blood or lymph system
Muscularis
Mucosae
Gland in submucosa
Serosa
Submucosa
Lymph node
Lamina propria
Muscularis Mucosae
Epithelium
Villus
Gland in submucosa
Longitudinal
Circular
Smooth Muscle
Form hollow tubes.
Form a syncitium - electrically coupled, joined by gap junctions.
Contractions synchronous.
Actin:myosin ratio 15:1 (skeletal muscle 2:1).
Contractile elements not arranged in sarcomeres.
Not striated.
Stimulated by neurotransmitter released from varicosities.
Have slow wave activity.
Gap junctions

Syncitium - electrically coupled, joined by gap junctions.

Helps synchronous contractions.

Diagram of gap junctions in animal cells, showing the structure and function of gap junctions.
Electrical Activity
Slow Waves in GI smooth muscles

They don’t cause contraction

They are present in GI smooth muscle except in esophagus and proximal part of Stomach

They oscillate between -50 to -65 mV

Oscillation of ?
Slow waves are produced by interstitial cells of Cajal

They are located in a thin layer between the longitudinal and circular layers of muscularis externa.

They have properties of both fibroblast and smooth muscle.

They have junctions with both circular and longitudinal smooth muscle.

Structures
- Interstitial cells of Cajal
- Smooth muscle cells
- Autonomic axon

Functions
- Production of slow waves
- Conduction of slow waves to smooth muscle
- Depolarization and opening of Ca²⁺ channels, production of action potentials
- Neural input to ICC and smooth muscle
Slow Wavenumber in each part

- In Stomach it is 3 cpm
- In Duodenum it is 12 cpm
- Proximal jejunum is 10.8 cpm
- Distal jejunum is 9.6 cpm
- Distal ileum is 8 cpm

- In terminal ileum it is
Acetylcholine Stretch

Spikes with contractions

Slow waves at rest

Hyperpolarization

Noradrenaline

Adrenaline

Seconds
GI Innervation

- Intrinsic (Enteric Nervous System)
- Extrinsic (Autonomic)
Entric Nervous System
Myenteric plexus (Auerbach’s)

Submucosal plexus (Meissner’s)

Longitudinal

circula
Functions of extrinsic nervous system

- **Parasympathetic stimulation:**
  A general increase in activity of the entire enteric nervous system

- **Sympathetic stimulation:**
  Generally inhibits GI system activity
  (There are some exceptions: in muscularis mucosa, sphincter, and GI vessels it stimulates smooth muscle contraction)
Afferent Nerves from Gut

- There are sensory nerves in epithelium or gut wall.
- They can be stimulated by:
  1. distention
  2. specific chemical substances
  3. irritation of gut mucosa
Types of Movements

• Propulsive (Propels the materials)

• Mixing (Mixes the materials)
Mechanism of Peristaltic Contraction
When sensory fibers are stimulated, sensory fibers (stretch) stimulate excitatory motor neurons. The effect on the tissue depends on the type of sensory fibers involved:

- **Excitatory Intereurons** toward Aboral release Ach or Substance P, leading to Contraction.
- **Inhibitory Intereurons** toward Anal release NO or VIP or ATP, leading to Relaxation.

The interneurons further modulate the response through the release of CGRP, Ach, Substance P, NO, VIP, and ATP.
Hormonal Control of Gastrointestinal Motility

1) **Gastrin** is secreted by the "G" cells of the antrum of the stomach in response to distention of the stomach, the products of proteins, and gastrin releasing peptide, which is released by the nerves of the gastric mucosa during vagal stimulation.

The primary actions of gastrin are:
(A) stimulation of gastric acid secretion
(B) stimulation of growth of the gastric mucosa.

2) **Cholecystokinin** is secreted by "I" cells in the mucosa of the duodenum and jejunum mainly in response to digestive products of fat, fatty acids, and mono glycerides in the intestinal contents. This hormone strongly contracts the gallbladder, and also inhibits stomach contraction moderately.
Hormonal Control of Gastrointestinal Motility

3) **Secretin** was the first gastrointestinal hormone discovered and is secreted by the "S" cells in the *mucosa of the duodenum* in response to acidic gastric juice emptying into the duodenum from the pylorus of the stomach. Secretin has a mild effect on motility of the gastrointestinal tract and acts to promote pancreatic secretion of bicarbonate which in turn helps to neutralize the acid in the small intestine.

4) **Gastric inhibitory peptide** is secreted by the *mucosa of the upper small intestine*, mainly in response to fatty acids and amino acids but to a lesser extent in response to carbohydrate. It has a mild effect in decreasing motor activity of the stomach and therefore slows emptying of gastric contents into the duodenum when the upper small intestine is already overloaded with food.
• **5) Motilin** is secreted by the *upper duodenum* during fasting, and the only known function of this hormone is to *increase gastrointestinal motility*. Motilin is released cyclically and stimulates waves of gastrointestinal motility called *interdigestive myoelectric complexes* that move through the stomach and small intestine.

• every 90 minutes in a fasted person. Motilin secretion is inhibited after ingestion by mechanisms that are not fully understood.
Gastrointestinal Reflexes

1) Reflexes that are integrated entirely within the gut wall enteric nervous system. Propulsive Movements - Peristalsis,

2) Reflexes from the gut to the prevertebral sympathetic ganglia and then back to the gastrointestinal tract.

3) Reflexes from the gut to the spinal cord or brain stem and then back to the gastrointestinal tract.
Gastrointestinal Reflexes

3) Reflexes from the gut to the spinal cord or brain stem and then back to the gastrointestinal tract.

1) Vago-vagal reflex to control gastric motor and secretory activity;

2) Pain reflexes that cause general inhibition of the entire gastrointestinal tract

3) Defecation reflexes that travel from the colon and rectum to the spinal cord and back again to produce the powerful colonic, rectal, and abdominal contractions required for defecation.
1) Effect of Gut Activity and Metabolic Factors on Gastrointestinal Blood Flow (oxygen concentration, adenosine)

2) Nervous Control of Gastrointestinal
Stimulation of the parasympathetic nerves increases local blood flow. Sympathetic stimulation, decreased blood flow

3) Possible Causes of the Increased Blood Flow During Gastrointestinal Activity:
1) several vasodilator released from the mucosa of the intestinal tract: CCK-PZ, VIP, gastrin, and secretin.
2) kallidin and bradykinin, from Gland.
3) Hypoxia in the gut wall
Basic Digestive Processes

(1) Motility

Muscles in GI tract contract

- Mix and move contents

Propulsive movements

- Mix with digestive juices - greater digestion
- Greater exposure to absorptive surface ↑ absorption
Chewing Reflex

- Food presence ➔ Reflex inhibition of chewing muscles
- Lower Jaw drops
- This drops initiates a stretch reflex ➔ Jaw muscles contract
- Continues
SWALLOWING

• Oral Phase (voluntarily)
• Pharyngeal Phase (Involuntarily)
• Esophageal Phase (Involuntarily)
Once food has been chewed, it is swallowed. When you swallow, a series of reflexes allow food to enter the esophagus and proceed to the stomach (rather than enter the trachea).
Esophageal Phase of Swallowing (Pressure Changes)
Achalasia

- Problem in opening of LES (defects in release of NO & VIP)

- Treatment
  - Surgical
  - Balloon
  - Botulinum toxin
Stomach

Functions

(A) Storage

(B) Mixing

(C) Initiates secretion & digestion

(D) Carefully controls emptying of contents to the Small Intestine
Major Areas of the Stomach

- Esophagus
- Fundus
- Lower esophageal sphincter
- Oxyntic glandular mucosa
- Pacemaker zone
- Body
- Pylorus
- Antrum
- Pyloric glandular mucosa
- Duodenum
Motor behavior of the stomach is determined by dominant pacemaker in the corpus

**Pacemaker Potentials Determine Contractile Parameters:**
- Maximal Frequency
- Propagation Velocity
- Propagation Direction

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**Diagram:**
- **Fundus**
- **Corpus**
- **Antrum**
- **Pylorus**
- **Pacemaker Region**
Gastric Motility

1. Fasting State
   - Pyloric Sphincter
   - Proximal Stomach
   - Distal Stomach
   - Antrum

2. Meal Enters Stomach
   - Receptive Relaxation
   - Food Bolus

3. Peristalsis Begins
   - Peristaltic Wave
   - Food Bolus

4. Antral Systole Grinds Food and Mixes Food
   - Pyloric Sphincter partially closes
   - Reflux and mixing of gastric contents
Movements in Stomach
Types of Motility

• Mixing movement (Segmental contractions)

• Propulsive movements
In stomach it is 3 contraction per minute

In Duodenum it is 12 contraction per minute

In Jejunum it is 12 contraction per minute
Peristaltic Rush

• Powerful peristalsis

• Irritation of intestinal mucosa by Infectious diarrhea

• Partly by extrinsic nervous reflexes to the brain stem and back again to the intestine and partly by potentiation of intrinsic myenteric reflexes
Migrating Motor Complex

- From Stomach to the end of illeum
- Every 75-90 min
- After processing of the last food
Colon

Function: Absorption of water & electrolyte Reservoir
Types of Colonic Motility

• Mixing movement (Segmental contractions)

• Propulsive movements (Mass contraction)
Mixing movement

(Haustra contractions)

They help absorption
Mass Movements

In most cases
15 min after breakfast

1- a contracted ring in transverse colon

2- 20 Cm after the contracted ring losses its haustra and contract simultaneously
Propulsive movements (Mass contraction)

- Gastrocolic and Duodenocolic reflexes facilitate mass movement appearance after meals
- If extrinsic autonomic nerves are dissected these movement disappear
- Irritation of colon can also brings about mass movement
Defecation

• Is initiated by defecation reflexes

• 1- intrinsic defecation reflexes

• 2- Parasympathetic defecation reflexes
Defecation

Intrinsic defecation reflexes

Parasympathetic defecation reflexes

Voluntary motor nerve to external anal sphincter

From cerebral cortex (conscious) control

Sensory nerve fibers

Involuntary motor nerve (parasympathetic division)

Rectum

Sigmoid colon

Internal anal sphincter (smooth muscle)

External anal sphincter (skeletal muscle)

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Many digestive juices are secreted into the digestive tract by glands along the route.

Contain water, electrolytes and enzymes, bile salts, mucous etc.

Act on nutrients or start/stop other substances acting on the nutrients

Released in response to hormones or neurons
Salivary Secretion

- FUNCTIONS:
  Lubrication & Protection (Mucus)
  Digestion (alpha amylase)
  Speech
  Tasting
  Defense (IJA & Lysozym)

- GLANDS: Parotid, Submandibular, Sublingual

- FUNCTIONAL UNIT: Salivon
Amylase-containing PRIMARY SECRETION (nearly isotonic; levels of $\text{Na}^+$, $\text{K}^+$, $\text{Cl}^-$, and [probably] $\text{HCO}_3^-$ similar to plasma)

Modification of ionic content
Gastric Secretion

• Acid (Kills bacteria & Changes inactive Pepsinogen to active form and digest protein)
• Mucus (protection)
• Bicarbonate (protection)
• Pepsinogen (enzyme)
• Intrinsic Factor (Vit B12 absorption)
Microscopic Anatomy

- The otherwise smooth lining is dotted with millions of gastric pits which lead to gastric glands that produce gastric juice.
- The glands of the stomach body are substantially larger and produce the majority of the stomach secretions.
Gastric Gland ➞ Gastric Pit

- Mucous surface cell (secretes mucus)
- Mucous neck cell (secretes mucus)
- Parietal cell (secretes hydrochloric acid and intrinsic factor) 
  (oxyntic cell)
- Chief cell (secretes pepsinogen)
- Hormone-producing G cell (secretes gastrin)

Gastric pit

Gastric gland

Muscularis mucosae
Submucosa

Simple columnar epithelium
Areolar connective tissue

SEM about 1000x
Stomach mucosa

Mucous surface cells
• Vagus via parasympathetic (Ach)
Gastric Phase

- Presence of food in stomach
- 1- Distention $\rightarrow$ central (vagovagal) & local reflexes $\rightarrow$ Ach & Gastrin
- Amino acid & peptide $\rightarrow$ Gastrin
FIGURE 34-10. Control of gastric secretion of HCl. Histamine stimulates gastrin release from G cells, which in turn stimulates parietal cells to secrete HCl. Parasympathetic nerve stimulation results in Ach release, which stimulates ECL cells to release histamine. Peptides and amino acids also stimulate gastrin release. D cells release somatostatin, which inhibits gastrin release. Luminal hydrogen ions stimulate parietal cells to secrete HCl.
Intestinal Phase

- At first  \( \text{PH} > 3 \)
- Then  \( \text{PH} < 3 \)
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<th>Phase</th>
<th>Stimuli</th>
<th>Mechanisms of Stimulation of HCl Secretion</th>
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<tr>
<td>Cephalic</td>
<td>Chewing, swallowing, taste, smell of food</td>
<td>Vagal impulses excite enteric secretomotor neurons to parietal, G, and ECL cells</td>
</tr>
<tr>
<td>Gastric</td>
<td>Gastric distention</td>
<td>Local and vagovagal reflexes stimulate parietal cells and release of histamine and gastrin</td>
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<td>Peptides and amino acids in lumen</td>
<td>Peptides and amino acids release gastrin from G cells in stomach</td>
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<td>Protein digestion products in duodenum</td>
<td>Release of gastrin from G cells in intestine and enterooxyntin</td>
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<td>Distention of duodenum</td>
<td>Enteric and vagovagal reflexes to ECL, G, and parietal cells</td>
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<td>Amino acids and peptides in blood</td>
<td>Release of gastrin from G cells in stomach</td>
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Gastric Pits

Acidic Environment

- pepsinogen

- pepsin

- protein

- peptides

- gastrin

- HCl

- stomach lumen

- parietal cell

- chief cell

- G cell

- mucous cells

- surface of gastric mucosa

- gastric gland

- mucosa

- stomach lining
Gastric secretions

- **Mucus**
  - pH < 2
  - pH 7
  - HCO_3^-

  Physical/chemical barrier to attack by gastric juice

  Stimulated by:
  - Ach
  - Mechanical stim
  - Chemicals (ethanol)

  If breached e.g. hypersecretion of acid → ulceration
Causes:
1. High acid and peptic content
2. Irritation
3. Poor blood supply
4. Poor secretion of mucus
5. Infection, *H. pylori*

Figure 66-1

Peptic ulcer. *H. pylori, Helicobacter pylori.*
When the stomach contents are emptied into small intestine they are mixed with the secretions from the pancreas and liver that are emptied at the beginning of the small intestine (duodenum)
Pancreas

Pancreatic secretions

(1) Enzymes

Trypsin
Chymotrypsin
Carboxypeptidase

Lipase

ONLY enzyme that digests FAT - Free fatty acids + Monoglycerides
Figure 29-2 Locations of important transport processes involved in the elaboration of pancreatic juice. Acinar fluid is isotonic and resembles plasma in its concentrations of Na⁺, K⁺, Cl⁻, and HCO₃⁻. Secretion of acinar fluid and the proteins that it contains is stimulated primarily by cholecystokinin. The hormone secretin stimulates secretion of water and electrolytes from the cells that line the extralobular ducts. The secretin-stimulated secretion is richer in HCO₃⁻ than the acinar secretion because of Cl⁻/HCO₃⁻ exchange. (Adapted from Swanson CH, Solomon AK: J Gen Physiol 62:407, 1973.)
(2) Alkaline secretion (Sodium Bicarbonate)

Neutralise acids from stomach

• enabling enzymes to function
Activation of pancreatic proteases

Enterokinase

Trypsinogen → Trypsin

Trypsinogen
Chymotrypsinogen
Proelastase
Procarboxypeptidase

Trypsin
Chymotrypsin
Elastase
Carboxypeptidase

Active proteases inactivated by trypsin
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<td>Prophospholipase A₂</td>
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<td>Deoxyribonuclease</td>
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<td>Ribonuclease</td>
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<td>Trypsin inhibitors</td>
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<td>Monitor peptide</td>
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Bicarbonate secretion

Lumen

Blood

H₂O ⇌ CO₂

H₂CO₃⁻

HCO₃⁻

Cl⁻

Na⁺

H⁺

ATP

H₂O
Regulation of secretion

- Neural (Parasympa & sympa)
- Hormonal Secretin (aqueous) & CCK (enzyme)
Pahses

• Cephalic 25%
• Gastric 10%
• Intestinal 65%
Intestinal phase of secretion

CCK

Secretin

HCO₃⁻

Peptides
Amino acids
Fat, H⁺

Enzymes

VAGUS

ACh
Liver (Bile)

Liver produces bile salts & gallbladder stores & concentrates it

Bile

(A) Converts large fat globule into an emulsion of fat droplets - greater area

(B) Transport of fat droplets - fat is difficult to transport
Microscopic Anatomy of the Liver

Figure 24.24c, d
Bile Acid Synthesis

Major bile acids

Cholesterol (Liver) → 7α-Hydroxylase (12α-hydroxylase) → C27 dehydroxylase

Primary

Cholic acid

Intestinal bacteria

Deoxycholic acid

Secondary

Chenodeoxycholic acid

Lithocholic acid

Ursodeoxycholic acid

Conjugation of bile acids

Glycine, $pK_a = 3.7$

Taurine, $pK_a = 1.5$
Cartoon of a Bile Acid

Hydrophobic face

- OH groups
- Peptide bond
- Carboxyl or sulfonic acid

Hydrophilic face
Form micelle
Helps lipid absorption
Storage and concentration of bile between meals

Electrolytes secreted

Active absorption of bile acids

Bile acids return to liver in portal circulation

Emulsification and digestion of fats

Micelle formation and fat absorption
FIGURE 35–8. Absorption of bile salts by epithelial cells of the terminal ileum. Bile salts are absorbed both by simple diffusion and by Na⁺–bile salt symporter. Conjugated bile salts are absorbed mainly by this symporter; unconjugated bile salts are absorbed chiefly by diffusion.
Causes of gallstones:
1. Too much absorption of water from bile
2. Too much absorption of bile acids from bile
3. Too much cholesterol in bile
4. Inflammation of epithelium

Course followed by bile:
1. During rest
2. During digestion

Figure 64-12
Formation of gallstones.
Small Intestine

Site at which most digestion and absorption takes place, no further digestion or absorption of nutrients occurs from here.
Intestinal secretion
Duodenum

- Bruner’s gland $\rightarrow$ HCO$_3$ & Mucus
- Stimulated by parasympathetic & mechanical & secretin
- Sympathetic stimulation $\rightarrow$ decreases
Crypt of lieberkuhn

- Secretes Na & Cl & water
- Stimulated by:
  Local reflexes & mechanical stimuli

Goblet cells:
  - Mucus
a) COMPLETE DIGESTION
b) ABSORPTION

Absorption and Secretion occur simultaneously.
Histology of Large Intestine

- Teniae coli
- Haustra
- Epiploic appendages
- Opening of crypts
- Epithelium
- Submucosa
  - Circular muscle
  - Longitudinal muscle
- Serosa
- Crypts
- Lamina propria
- Goblet cells
- Lymphatic nodule
- Goblet cells in crypt
Colonic secretion

- Low volume
- High mucus by goblet cells
- Aqueous → Rich of K & Bicarbonate
- Stimulated by mechanical & cholinergic
- Sympathetic stimulation → Decrease secretion
Large Intestine

Stores and concentrates faecal material before defecation

Secretion of mucous for protection of mucosa

No nutrient absorption, only salts and relatively small amounts of water

Faeces: Water, unabsorbed food e.g. cellulose, bacteria
Small Intestine

- Mixing and segmentation of contents - no secretion
  - Brings contents in contact with epithelial surface
- Digestion in the lumen is accomplished by secretions from the pancreas
- Digestion occurs within the epithelial cells of the intestine
- Minerals are also absorbed
The structure of the small intestine

Vein carrying blood to hepatic portal vessel

Muscle layers
Large circular folds
Villi

Epithelial cells
Lumen
Blood capillaries
Lacteal

Microvilli (brush border)

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Absorption

Lipids

Amino Acids

Carbohydrates

Liver
Small Intestinal Lumen

Carbohydrates

H₂O

Intestinal cells

Capillary

Proteins

Lipids

Lacteal
Enzymes

- Biological catalysts which greatly increase the rate of a chemical reaction but are not themselves changed during the process.
- Enzymes are central to the digestion process.

Digestion

Protein → Pepsin → Peptide fragments

Glands in stomach
Proteins

Structure

Amino Acids - glycine
Amino acids combine to form peptides which combine to form proteins.
Protein digestion & absorption

Protein digestion involves the enzymatic degradation of proteins to di-, or tri- peptides & finally amino acids.

- Digestion begins in the stomach with the interaction with pepsin.

- Further proteolytic cleavage occurs in the intestinal lumen by pancreatic trypsin, chymotrypsin, and carboxy peptidases.

- Final degradation occurs on the membrane of the intestinal microvilli by the action of aminopeptidases.

- Protein absorption occurs through active transport.
Protein → peptides → Di/tri peptides → transporters
Protein → peptides → amino acids → transporters
Cytoplasmic peptidase
Amino acids
peptidases, aminopolypeptidase
transporters
The result of pancreatic and brush border enzyme

- AA & small peptides (mostly di & tri)
- They are entered into the cells
- In the cell peptides are digested
- Finally entered into blood as AA and a few as dipeptide

3 sites for digestion of protein
How AA are entered into the cells

- Some by Na (co-transport) (5 types)
- Some facilitated (2 types)
- Some diffusion
How Di & Tri peptides are entered into the cells

• Co-transport by Na

Co transport by H
Carbohydrates

Monosaccharides

Disaccharides

Polysaccharides

Glycogen

(A)

(B)

Glucose subunit

Glycogen
Carbohydrates

Structure

Monosaccharides
- Glucose
- Fructose

Disaccharides
- Sucrose
- Maltose
- Lactose

Polysaccharides
- Starch
- Glycogen
- Fibre
FIGURE 35–1. Structure of a branched starch molecule and the action of α-amylase. The circles represent glucose monomers. The colored circles show glucose units linked by α-1,6 linkages at the branch points. The α-1,6 links and terminal α-1,4 bonds cannot be cleaved by α-amylase.
Carbohydrate digestion & absorption

- Carbohydrate digestion involves the enzymatic degradation of di-, tri, and polysaccharides to monosaccharides (glucose, fructose, galactose).

- Digestion begins in the mouth with salivary amylase.
- Polysaccharides are then further broken down by pancreatic amylase in the small intestine.
- Final degradation occurs on the surface (brushborder) of the absorptive cells in the jejunum. lactase, sucrase, and maltase.
- Monosaccharides absorbed by facilitated (membrane carrier) or active transport (membrane carrier, Na+, and ATP).
- Fructose = facilitated diffusion
- Glucose and galactose = active transport
Fats

Fatty Acid

Fat (Triglyceride)
Lipid digestion and absorption

- Fats triacylglycerols (triglycerides) to monoacylglycerols and 2 fatty acids.
- Small amount of lipase in saliva begins digestion which continues in the stomach with (slow acting) gastric lipases.
- Pancreatic lipases and bile are mixed with hydrolyzed product in the duodenum. The bile emulsifies the fat into fat droplets (micelles) making it easier for colipase and pancreatic lipase to breakdown triglycerides.
- Monoacylglycerol and fatty acids are packaged into mixed micelles (cholesterol, bile salts, and fat soluble vitamins) and diffuse across the cellular membrane (of the jejunum).
- Triglycerides are reformed (re-esterified) then combine with cholesterol and phospholipids to form chylomicrons for transportation.
Lipid Absorption
Intracellular Metabolism of Absorbed Lipids
FIGURE 35–9. Overall fluid balance in the human gastrointestinal tract. Approximately 2 L of water is ingested each day, and 7 L of various secretions enter the gastrointestinal tract. Of this total of 9 L, about 8.5 L is absorbed in the small intestine. Approximately 500 mL is passed on to the colon, which normally absorbs 80% to 90% of the water presented to it.
**Figure 25-8.** Absorption of iron. Fe$^{3+}$ is converted to Fe$^{2+}$ by ferric reductase, and Fe$^{2+}$ is transported into the enterocyte by the apical membrane iron transporter DMT1. Heme is transported into the enterocyte by a separate heme transporter (HT), and heme oxidase (HO) releases Fe$^{2+}$ from the heme. Some of the intracellular Fe$^{2+}$ is converted to Fe$^{3+}$ and bound to ferritin. The rest binds to the basolateral Fe$^{2+}$ transporter ferroportin (FP) and is transported to the interstitial fluid. The transport is aided by hephaestin (Hp). In plasma, Fe$^{2+}$ is converted to Fe$^{3+}$ and bound to the iron transport protein transferrin (TF).