GIT Physiology

Lec.1 **Dr.Latief fayadh**

The alimentary tract provides the body with a continual supply of water, electrolytes, and nutrients. To achieve this requires:

(1) Movement of food through the alimentary tract.

(2) Secretion of digestive juices and digestion of the food.

(3) Absorption of water, various electrolytes, and digestive products.

(4) Circulation of blood through the gastrointestinal organs to carry away the absorbed substances.

(5) Control of all these functions by local, nervous, and hormonal systems.

Figure below shows the entire alimentary tract. Each part is adapted to its specific functions: some to simple passage of food, such as the esophagus; others to temporary storage of food, such as the stomach; and others to digestion and absorption, such as the small intestine.

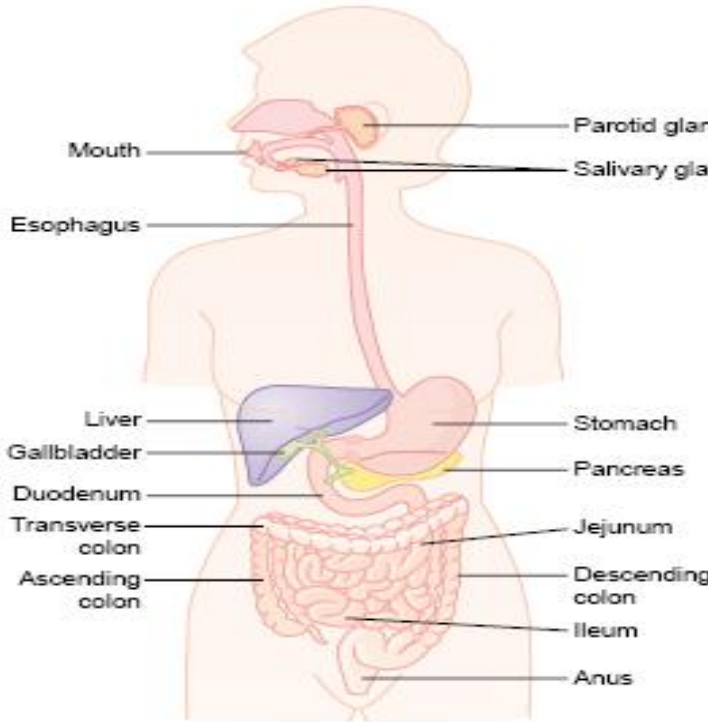


Figure 62–1

Alimentary tract.

General Principles of Gastrointestinal Motility

Physiologic Anatomy of the Gastrointestinal Wall

Figure below shows a typical cross section of the intestinal wall, including the following layers from outer surface inward:

(1) The serosa.

(2) A longitudinal muscle layer.

(3) A circular muscle layer.

(4) The submucosa.

(5) The mucosa.

In addition, sparse bundles of smooth muscle fibers, the mucosal muscle, lie in the deeper layers of the mucosa. The motor functions of the gut are performed by the different layers of smooth muscle.

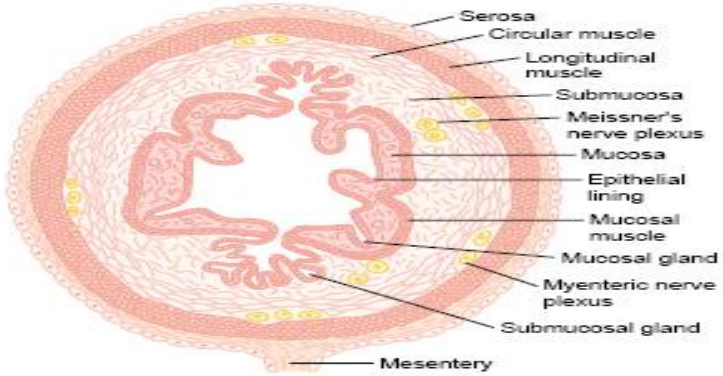


Figure 62–2

Typical cross section of the gut.

Neural Control f Gastrointestinal Function— Enteric Nervous System

The gastrointestinal tract has a nervous system all its own called the enteric nervous system. It lies entirely in the wall of the gut, beginning in the esophagus and extending all the way to the anus. The number of neurons in this enteric system is about 100 million, almost exactly equal to the number in the entire spinal cord. This highly developed enteric nervous system is especially important in controlling gastrointestinal movements and secretion. The enteric nervous system is composed mainly of two plexuses, shown in figure below:

(1) An outer plexus lying between the longitudinal and circular muscle layers, called the myenteric plexus or Auerbach's plexus.

(2) An inner plexus, called the submucosal plexus or Meissner's plexus, that lies in the submucosa.

The nervous connections within and between these two plexuses The myenteric plexus controls mainly the gastrointestinal movements, and the submucosal plexus controls mainly gastrointestinal secretion and local blood flow. Note the extrinsic sympathetic and parasympathetic fibers that connect to both the myenteric and submucosal plexuses. Although the enteric nervous system can function on its own, independently of these extrinsic nerves, stimulation by the parasympathetic and sympathetic systems can greatly enhance or inhibit gastrointestinal functions, as we discuss later. Also sensory nerve endings that originate in the gastrointestinal epithelium or gut wall and send afferent fibers to both plexuses of the enteric system, as well as:

(1) To the prevertebral ganglia of the sympathetic nervous system.

(2) To the spinal cord.

(3) In the vagus nerves all the way to the brain stem.

These sensory nerves can elicit local reflexes within the gut wall itself and still other reflexes that are relayed to the gut from either the prevertebral ganglia or the basal regions of the brain.

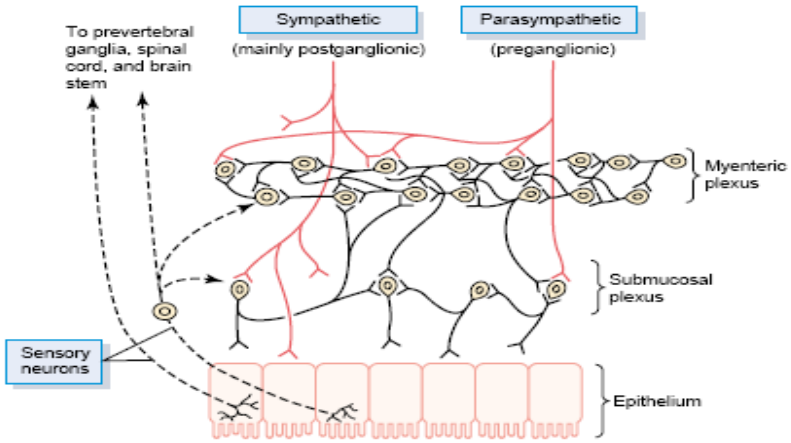


Figure 62–4

Neural control of the gut wall, showing (1) the myenteric and submucosal plexuses (*black fibers*); (2) extrinsic control of these plexuses by the sympathetic and parasympathetic nervous systems (*red fibers*); and (3) sensory fibers passing from the luminal epithelium and gut wall to the enteric plexuses, then to the prevertebral ganglia of the spinal cord and directly to the spinal cord and brain stem (*dashed fibers*).

Differences Between the Myenteric and Submucosal Plexuses

The myenteric plexus consists mostly of a linear chain of many interconnecting neurons that extends the entire length of the gastrointestinal tract. A section of this chain is shown in figure above. Because the myenteric plexus extends all the way along the intestinal wall and because it lies between the longitudinal and circular layers of intestinal smooth muscle, it is concerned mainly with controlling muscle activity along the length of the gut. When this plexus is stimulated, its principal effects are:

(1) Increased tonic contraction, or "tone," of the gut wall.

(2) Increased intensity of the rhythmical contractions.

(3) Slightly increased rate of the rhythm of contraction.

(4) Increased velocity of conduction of excitatory waves along the gut wall, causing more rapid movement of the gut peristaltic waves.

***The myenteric plexus** should not be considered entirely excitatory because some of its neurons are inhibitory; their fiber endings secrete an inhibitory transmitter, possibly vasoactive intestinal polypeptide or some other inhibitory peptidj e. The resulting inhibitory signals are especially useful for inhibiting some of the intestinal sphincter muscles that impede movement of food along successive segments of the gastrointestinal tract, such as the pyloric sphincter, which controls emptying of the stomach into the duodenum, and the sphincter of the ileocecal valve, which controls emptying from the small intestine into the cecum.*

***The submucosal plexus**, in contrast to the myenteric plexus, is mainly concerned with controlling function within the inner wall of each minute segment of the intestine. For instance, many sensory signals originate from the gastrointestinal epithelium and are then integrated in the submucosal plexus to help control local intestinal secretion, local absorption, and local contraction of the submucosal muscle that causes various degrees of infolding of the gastrointestinal mucosa.*

Types of Neurotransmitters Secreted by Enteric Neurons

In an attempt to understand better the multiple functions of the gastrointestinal enteric nervous system, research workers the world over have identified a dozen or more different neurotransmitter substances that are released by the nerve endings of different types of enteric neurons. Two of them with which we are already familiar are (1) acetylcholine and (2) norepinephrine. Others are (3) adenosine triphosphate, (4) serotonin, (5) dopamine, (6) cholecystokinin, (7) substance P, (8) vasoactive intestinal polypeptide, (9) somatostatin, (10) leu-enkephalin, (11) met-enkephalin, (12) bombesin.

The specific functions of many of these are not known well enough to justify discussion here, other than to point out the following.

Acetylcholine most often excites gastrointestinal activity. Norepinephrine almost always inhibits gastrointestinal activity.

Autonomic control of the gastrointestinal tract.

Parasympathetic stimulation increases activity of the enteric nervous system.

The parasympathetic supply to the gut is divided into cranial and sacral divisions, Except for a few parasympathetic fibers to the mouth and pharyngeal regions of the alimentary tract, the cranial parasympathetic nerve fibers are almost entirely in the vagus nerves. These fibers provide extensive innervations to the esophagus, stomach, and pancreas and somewhat less to the intestines down through the first half of the large intestine.

Sympathetic stimulation usually inhibits gastrointestinal tract activity.

The sympathetic fibers to the gastrointestinal tract originate in the spinal cord between segments T5 and L2. Most of the preganglionic fibers that innervate the gut, after leaving the cord, enter the sympathetic chains that lie lateral to the spinal column, and many of these fibers then pass on through the chains to outlying ganglia such as to the celiac ganglion and various mesenteric ganglia. Most of

the postganglionic sympathetic neuron bodies are in these ganglia, and postganglionic fibers then spread through postganglionic sympathetic nerves to all parts of the gut. The sympathetics innervate essentially all of the gastrointestinal tract, rather than being more extensive nearest the oral cavity and anus, as is true of the parasympathetics. The sympathetic nerve endings secrete mainly norepinephrine but also small amounts of epinephrine.

Afferent sensory nerve fibers from the gut.

Many afferent sensory nerve fibers innervate the gut. Some of them have their cell bodies in the enteric nervous system itself and some in the dorsal root ganglia of the spinal cord. These sensory nerves can be stimulated by:

(1) irritation of the gut mucosa.

(2) excessive distention of the gut.

(3) presence of specific chemical substances in the gut.

Signals transmitted through the fibers can then cause excitation or, under other conditions, inhibition of intestinal movements or intestinal secretion. In addition, other sensory signals from the gut go all the way to multiple areas of the spinal cord and even the brain stem. For example, 80 percent of the nerve fibers in the vagus nerves are afferent rather than efferent. These afferent fibers transmit sensory signals from the gastrointestinal tract into the brain medulla, which in turn initiates vagal reflex signals that return to the gastrointestinal tract to control many of its functions.

Gastrointestinal reflexes.

The anatomical arrangement of the enteric nervous system and its connections with the sympathetic and parasympathetic systems support three types of gastrointestinal reflexes that are essential to gastrointestinal control. They are the following:

***1.** Reflexes that are integrated entirely within the gut wall enteric nervous system. These include reflexes that control much gastrointestinal secretion, peristalsis, mixing contractions, local inhibitory effects, and so forth.*

***2.** Reflexes from the gut to the prevertebral sympathetic ganglia and then back to the gastrointestinal tract. These reflexes transmit signals long distances to other areas of the gastrointestinal tract, such as signals from the stomach to cause evacuation of the colon (the gastrocolic reflex), signals from the colon and small intestine to inhibit stomach motility and stomach secretion (the enterogastric reflexes), and reflexes from the colon to inhibit emptying of ileal contents into the colon (the colonoileal reflex).*

***3.** Reflexes from the gut to the spinal cord or brain stem and then back to the gastrointestinal tract. These include especially*

(1) Reflexes from the stomach and duodenum to the brain stem and back to the stomach by way of the vagus nerves 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 (the defecation reflexes).

Hormonal control of gastrointestinal motility

1. Gastrin

Is secreted by the "G" cells of the antrum of the stomach in response to stimuli associated with ingestion of a meal, such as 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:

(1) Stimulation of gastric acid secretion.

(2) Stimulation of growth of the gastric mucosa.

2.Cholecystokinin (CCK)

Is secreted by "I" cells in the mucosa of the duodenum and jejunum mainly in response to digestive products of fat, fatty acids, and monoglycerides in the intestinal contents. This hormone strongly contracts the gallbladder, expelling bile into the small intestine where the bile in turn plays important roles in emulsifying fatty substances, allowing them to be digested and absorbed. Cholecystokinin also inhibits stomach contraction moderately. Therefore, at the same time that this hormone causes emptying of the gallbladder, it also slows the emptying of food from the stomach to give adequate time for digestion of the fats in the upper intestinal tract.

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 products.

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.

Functional types of movements in the gastrointestinal tract

Two types of movements occur in the gastrointestinal tract:

(1) Propulsive movements, which cause food to move forward along the tract at an appropriate rate to accommodate digestion and absorption,

(2) Mixing movements, which keep the intestinal contents thoroughly mixed at all times.

1. Propulsive movements—Peristalsis

The basic propulsive movement of the gastrointestinal tract is peristalsis, A contractile ring appears around the gut and then moves forward; this is analogous to putting one's fingers around a thin distended tube, then constricting the fingers and sliding them forward along the tube. Any material in front of the contractile ring is moved forward. Peristalsis is an inherent property of many syncytial smooth muscle tubes; stimulation at any point in the gut can cause a contractile ring to appear in the circular muscle, and this ring then spreads along the gut tube. (Peristalsis also occurs in the bile ducts, glandular ducts, ureters, and many other smooth muscle tubes of the body.)

The usual stimulus for intestinal peristalsis is distention of the gut. That is, if a large amount of food collects at any point in the gut, the stretching of the gut wall stimulates the enteric nervous system to contract the gut wall 2 to 3 centimeters behind this point, and a contractile ring appears that initiates a peristaltic movement. Other stimuli that can initiate peristalsis include chemical or physical irritation of the epithelial lining in the gut. Also, strong parasympathetic nervous signals to the gut will elicit strong peristalsis.

- Function of the myenteric plexus in peristalsis.

Peristalsis occurs only weakly or not at all in any portion of the gastrointestinal tract that has congenital absence of the myenteric plexus. Also, it is greatly depressed or completely blocked in the entire gut when a person is treated with atropine to paralyze the cholinergic nerve endings of the myenteric plexus. Therefore, effectual peristalsis requires an active myenteric plexus.

- Directional movement of peristaltic waves toward the anus.

Peristalsis, theoretically, can occur in either direction from a stimulated point, but it normally dies out rapidly in the orad direction while continuing for a considerable distance toward the anus. The exact cause of this directional transmission of peristalsis has never been ascertained, although it probably results mainly from the fact that the myenteric plexus itself is "polarized" in the anal direction, which can be explained as follows.

- Peristaltic reflex and the "Law of the Gut."

When a segment of the intestinal tract is excited by distention and thereby initiates peristalsis, the contractile ring causing the peristalsis normally begins on the orad side of the distended segment and moves toward the distended segment, pushing the intestinal contents in the anal direction for 5 to 10 centimeters before dying out. At the same time, the gut sometimes relaxes several centimeters downstream toward the anus, which is called "receptive relaxation," thus allowing the food to be propelled more easily anally than orad. This complex pattern does not occur in the absence of the myenteric plexus. Therefore, the complex is called the myenteric reflex or the peristaltic reflex. The peristaltic reflex plus the anal direction of movement of the peristalsis is called the "law of the gut."

2. Mixing movements

Mixing movements differ in different parts of the alimentary tract. In some areas, the peristaltic contractions themselves cause most of the mixing. This is especially true when forward progression of the intestinal contents is blocked by a sphincter, so that a peristaltic wave can then only churn the intestinal contents, rather than propelling them forward. At other times, local intermittent constrictive contractions occur every few centimeters in the gut wall. These constrictions usually last only 5 to 30 seconds; then new constrictions occur at other points in the gut, thus "chopping" and "shearing" the contents first here and then there. These peristaltic and constrictive movements are modified in different parts of the gastrointestinal tract for proper propulsion and mixing.

GIT Physiology

Lec. 2 **Dr.Latief fayadh**

Gastrointestinal blood flow— "splanchnic circulation"

The blood vessels of the gastrointestinal system are part of a more extensive system called the splanchnic circulation, shown in Figure below It includes the blood flow through the gut itself plus blood flows through the spleen, pancreas, and liver. The design of this system is such that all the blood that courses through the gut, spleen, and pancreas then flows immediately into the liver by way of the portal vein. In the liver, the blood passes through millions of minute liver sinusoids and finally leaves the liver by way of hepatic veins that empty into the vena cava of the general circulation. This flow of blood through the liver, before it empties into the vena cava, allows the reticuloendothelial cells that line the liver sinusoids to remove bacteria and other particulate matter that might enter the blood from the gastrointestinal tract, thus preventing direct transport of potentially harmful agents into the remainder of the body. The nonfat, water-soluble nutrients absorbed from the gut (such as carbohydrates and proteins) are transported in the portal venous blood to the same liver sinusoids. Here, both the reticuloendothelial cells and the principal parenchymal cells of the liver, the hepatic cells, absorb and store temporarily from one half to three quarters of the nutrients. Also, much chemical intermediary processing of these nutrients occurs in the liver cells. Almost all of the fats absorbed from the intestinal tract are not carried in the portal blood but instead are absorbed into the intestinal lymphatics and then conducted to the systemic circulating blood by way of the thoracic duct, bypassing the liver.

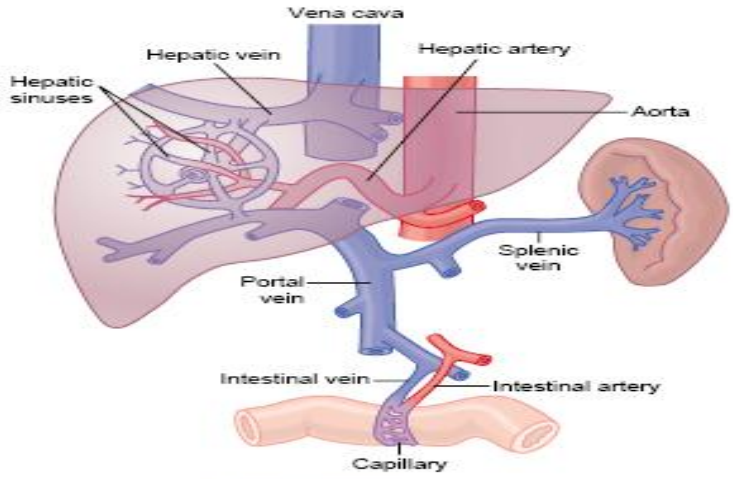


Figure 62–6

Splanchnic circulation.

Anatomy of the gastrointestinal blood supply.

Figure below shows the general plan of the arterial blood supply to the gut, including the superior mesenteric and inferior mesenteric arteries supplying the walls of the small and large intestines by way of an arching arterial system. Not shown in the figure is the celiac artery, which provides a similar blood supply to the stomach. On entering the wall of the gut, the arteries branch and send smaller arteries circling in both directions around the gut, with the tips of these arteries meeting on the side of the gut wall opposite the mesenteric attachment. From the circling arteries, still much smaller arteries penetrate into the intestinal wall and spread to:

(1) Along the muscle bundles

(2) Into the intestinal villi

(3) Into submucosal vessels beneath the epithelium to serve the secretory and absorptive functions of the gut. Figure below shows the special organization of the blood flow through an intestinal villus, including a small arteriole and venule that interconnect with a system of multiple looping capillaries. The walls of the arterioles are highly muscular and are highly active in controlling villus blood flow.

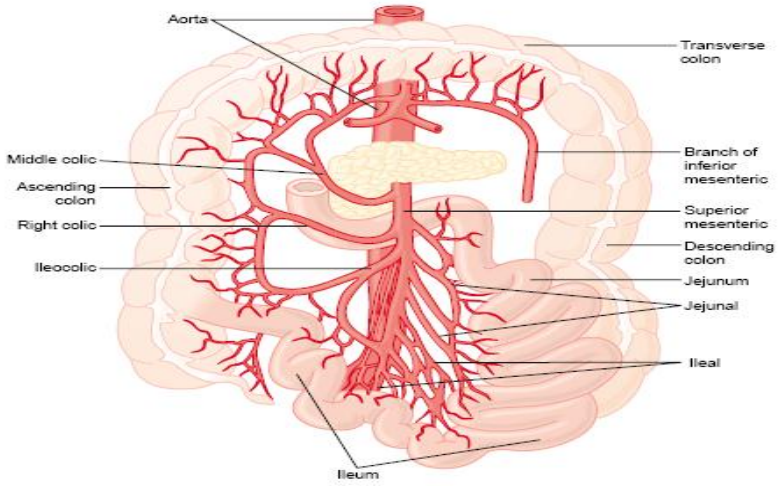


Figure 62–7

Arterial blood supply to the intestines through the mesenteric web.

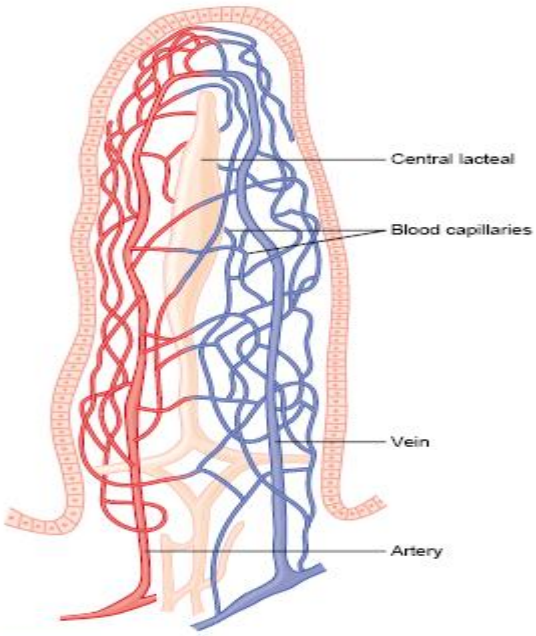


Figure 62–8

Microvasculature of the villus, showing a countercurrent arrangement of blood flow in the arterioles and venules.

Effect of gut activity and metabolic factors on gastrointestinal blood flow

Under normal conditions, the blood flow in each area of the gastrointestinal tract, as well as in each layer of the gut wall, is directly related to the level of local activity. For instance, during active absorption of nutrients, blood flow in the villi and adjacent regions of the submucosa is increased as much as eightfold. Likewise, blood flow in the muscle layers of the intestinal wall increases with increased motor activity in the gut. For instance, after a meal, the motor activity, secretory activity, and absorptive activity all increase; likewise, the blood flow increases greatly but then decreases back to the resting level over another 2 to 4 hours.

Possible causes of the increased blood flow during gastrointestinal activity.

Although the precise cause or causes of the increased blood flow during increased gastrointestinal activity are still unclear, some facts are known.

First, *several vasodilator substances are released from the mucosa of the intestinal tract during the digestive process. Most of these are peptide hormones, including cholecystokinin, vasoactive intestinal peptide, gastrin, and secretin. The same hormones control specific motor and secretory activities of the gut.*

Second, *some of the gastrointestinal glands also release into the gut wall two kinins, kallidin and bradykinin, at the same time that they secrete their secretions into the lumen. These kinins are powerful vasodilators that are believed to cause much of the increased mucosal vasodilation that occurs along with secretion.*

Third, *decreased oxygen concentration in the gut wall can increase intestinal blood flow at least 50 to 100 per cent; therefore, the increased mucosal and gut wall metabolic rate during gut activity probably lowers the oxygen concentration enough to cause much of the vasodilation. The decrease in oxygen can also lead to as much as a fourfold increase of adenosine, a wellknown vasodilator that could be responsible for much of the increased flow. Thus, the increased blood flow during increased gastrointestinal activity is probably a combination of many of the aforementioned factors plus still others yet undiscovered.*

"Countercurrent" blood flow in the villi.

Note in figure above that the arterial flow into the villus and the venous flow out of the villus are in directions opposite to each other, and that the vessels lie in close apposition to each other. Because of this vascular arrangement, much of the blood oxygen diffuses out of the arterioles directly into the adjacent venules without ever being carried in the blood to the tips of the villi. As much as 80 per cent of the oxygen may take this short-circuit route and therefore not be available for local metabolic functions of the villi. Under normal conditions, this shunting of oxygen from the arterioles to the venules is not harmful to the villi, but in disease conditions in which blood flow to the gut becomes greatly curtailed, such as in circulatory shock, the oxygen deficit in the tips of the villi can become so great that the villus tip or even the whole villus suffers ischemic death and can disintegrate. Therefore, for this reason and others, in many gastrointestinal diseases the villi become seriously blunted, leading to greatly diminished intestinal absorptive capacity.

Nervous control of gastrointestinal blood flow

Stimulation of the parasympathetic nerves going to the stomach and lower colon increases local blood flow at the same time that it increases glandular secretion. This increased flow probably results secondarily from the increased glandular activity and not as a direct effect of the nervous stimulation. Sympathetic stimulation, by contrast, has a direct effect on essentially all the gastrointestinal tract to cause intense vasoconstriction of the arterioles with greatly decreased blood flow. After a few minutes of this vasoconstriction, the flow often returns almost to normal by means of a mechanism called "autoregulatory escape." That is, the local metabolic vasodilator mechanisms that are elicited by ischemia become prepotent over the sympathetic vasoconstriction and, therefore, redilate the arterioles, thus causing return of necessary nutrient blood flow to the gastrointestinal glands and muscle.

Importance of nervous depression of gastrointestinal blood flow when other parts of the body need extra blood flow.

A major value of sympathetic vasoconstriction in the gut is that it allows shut-off of gastrointestinal and other splanchnic blood flow for short periods of time during heavy exercise, when increased flow is needed by the skeletal muscle and heart. Also, in circulatory shock, when all the body's vital tissues are in danger of cellular death for lack of blood flow—especially the brain and the heart sympathetic stimulation can decrease splanchnic blood flow to very little for many hours. Sympathetic stimulation also causes strong vasoconstriction of the large-volume intestinal and

mesenteric veins. This decreases the volume of these veins, thereby displacing large amounts of blood into other parts of the circulation. In hemorrhagic shock or other states of low blood volume, this mechanism can provide as much as 200 to 400 milliliters of extra blood to sustain the general circulation.

Propulsion and mixing of food in the alimentary tract

For food to be processed optimally in the alimentary tract, the time that it remains in each part of the tract is critical. Also, appropriate mixing must be provided. Yet because the requirements for mixing and propulsion are quite different at each stage of processing, multiple automatic nervous and hormonal feedback mechanisms control the timing of each of these so that they will occur optimally, not too rapidly, not too slowly. The purpose of this lecture is to discuss these movements, especially the automatic mechanisms of this control.

Ingestion of Food

The amount of food that a person ingests is determined principally by intrinsic desire for food called hunger. The type of food that a person preferentially seeks is determined by appetite. These mechanisms in themselves are extremely important automatic regulatory systems for maintaining an adequate nutritional supply for the body. The current discussion of food ingestion is confined to the mechanics of ingestion, especially mastication and swallowing.

Mastication (Chewing)

The teeth are admirably designed for chewing, the anterior teeth (incisors) providing a strong cutting action and the posterior teeth (molars), a grinding action. All the jaw muscles working together can close the teeth with a force as great as 55 pounds on the incisors and 200 pounds on the molars. Most of the muscles of chewing are innervated by the motor branch of the fifth cranial nerve, and the chewing process is controlled by nuclei in the brain stem. Stimulation of specific reticular areas in the brain stem taste centers will cause rhythmical chewing movements. Also, stimulation of areas in the hypothalamus, amygdala, and even the cerebral cortex near the sensory areas for taste and smell can often cause chewing. Much of the chewing process is caused by a chewing reflex, which may be explained as follows: The presence of a bolus of food in the mouth at first initiates reflex inhibition of the muscles of mastication, which allows the lower jaw to drop. The drop in turn initiates a stretch reflex of the jaw muscles that leads to rebound contraction. This automatically raises the jaw to cause closure of the teeth, but it also compresses the bolus again against the linings of the mouth, which inhibits the jaw muscles once again, allowing the jaw to drop and rebound another time; this is repeated again and again. Chewing is important for digestion of all foods, but especially important for most fruits and raw vegetables because these have indigestible cellulose membranes around their nutrient portions that must be broken before the food can be digested. Also, chewing aids the digestion of food for still another simple reason: Digestive enzymes act only on the surfaces of food particles; therefore, the rate of digestion is absolutely dependent on the total surface area exposed to the digestive secretions. In addition, grinding the food to a very fine particulate consistency prevents excoriation of the gastrointestinal tract and increases the ease with which food is emptied from the stomach into the small intestine, then into all succeeding segments of the gut.

Swallowing (Deglutition)

Swallowing is a complicated mechanism, principally because the pharynx subserves respiration as well as swallowing. The pharynx is converted for only a few seconds at a time into a tract for propulsion of food. It is especially important that respiration not be compromised because of swallowing. In general, swallowing can be divided into:

(1)Voluntary stage, which initiates the swallowing process.

(2)Pharyngeal stage, which is involuntary and constitutes passage of food through the pharynx into the esophagus.

(3)Esophageal stage, another involuntary phase that transports food from the pharynx to the stomach.

- Voluntary Stage of Swallowing.

When the food is ready for swallowing, it is "voluntarily" squeezed or rolled posteriorly into the pharynx by pressure of the tongue upward and backward against the palate, as shown in Figure below. From here on, swallowing becomes entirely—or almost entirely—automatic and ordinarily cannot be stopped.

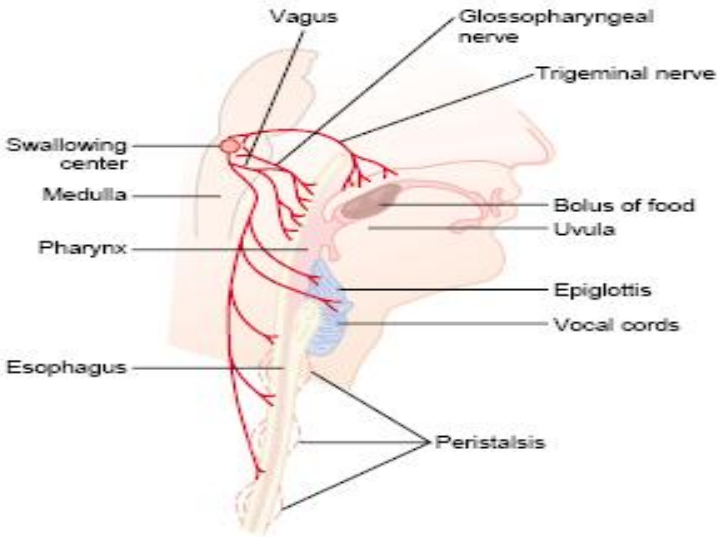


Figure 63–1

Swallowing mechanism.

- Pharyngeal Stage of Swallowing.

As the bolus of food enters the posterior mouth and pharynx, it stimulates epithelial swallowing receptor areas all around the opening of the pharynx, especially on the tonsillar pillars, and impulses from these pass to the brain stem to initiate a series of automatic pharyngeal muscle contractions as follows:

***1.** The soft palate is pulled upward to close the posterior nares, to prevent reflux of food into the nasal cavities.*

***2.** The palatopharyngeal folds on each side of the pharynx are pulled medially to approximate each other. In this way, these folds form a sagital slit through which the food must pass into the posterior pharynx. This slit performs a selective action, allowing food that has been masticated sufficiently to pass with ease. Because this stage of swallowing lasts less than 1 second, any large object is usually impeded too much to pass into the esophagus.*

***3.** The vocal cords of the larynx are strongly approximated, and the larynx is pulled upward and anteriorly by the neck muscles. These actions, combined with the presence of ligaments that prevent upward movement of the epiglottis, cause the epiglottis to swing backward over the opening of the larynx. All these effects acting together prevent passage of food into the nose and trachea.*

***4.** The upward movement of the larynx also pulls up and enlarges the opening to the esophagus. At the same time, the upper 3 to 4 centimeters of the esophageal muscular wall, called the upper esophageal sphincter (also called the pharyngoesophageal sphincter) relaxes, thus allowing food to move easily and freely from the posterior pharynx into the upper esophagus. Between swallows, this*

sphincter remains strongly contracted, thereby preventing air from going into the esophagus during respiration.

5. *Once the larynx is raised and the pharyngoesophageal sphincter becomes relaxed, the entire muscular wall of the pharynx contracts, beginning in the superior part of the pharynx, then spreading downward over the middle and inferior pharyngeal areas, which propels the food by peristalsis into the esophagus.*

To summarize *the mechanics of the pharyngeal stage of swallowing: The trachea is closed, the esophagus is opened, and a fast peristaltic wave initiated by the nervous system of the pharynx forces the bolus of food into the upper esophagus, the entire process occurring in less than 2 seconds.*

Esophageal stage of swallowing.

The esophagus functions primarily to conduct food rapidly from the pharynx to the stomach, and its movements are organized specifically for this function. The esophagus normally exhibits two types of peristaltic movements:

1. primary peristalsis

Is simply continuation of the peristaltic wave that begins in the pharynx and spreads into the esophagus during the pharyngeal stage of swallowing. This wave passes all the way from the pharynx to the stomach in about 8 to 10 seconds. Food swallowed by a person who is in the upright position is usually transmitted to the lower end of the esophagus even more rapidly than the peristaltic wave itself, in about 5 to 8 seconds, because of the additional effect of gravity pulling the food downward.

2.The secondary peristaltic waves

If the primary peristaltic wave fails to move into the stomach all the food that has entered the esophagus, secondary peristaltic waves result from distention of the esophagus itself by the retained food; these waves continue until all the food has emptied into the stomach. These waves are initiated partly by intrinsic neural circuits in the myenteric nervous system and partly by reflexes that begin in the pharynx and are then transmitted upward through vagal afferent fibers to the medulla and back again to the esophagus through glossopharyngeal and vagal efferent nerve fibers. The musculature of the pharyngeal wall and upper third of the esophagus is striated muscle. Therefore, the peristaltic waves in these regions are controlled by skeletal nerve impulses from the glossopharyngeal and vagus nerves. In the lower two thirds of the esophagus, the musculature is smooth muscle, but this portion of the esophagus is also strongly controlled by the vagus nerves acting through connections with the esophageal myenteric nervous system.

Motor Functions of the Stomach

The motor functions of the stomach are threefold:

(1) Storage of large quantities of food until the food can be processed in the stomach, duodenum, and lower intestinal tract.

(2) Mixing of this food with gastric secretions until it forms a semifluid mixture called chyme

(3) Slow emptying of the chyme from the stomach into the small intestine at a rate suitable for proper digestion and absorption by the small intestine. Figure below shows the basic anatomy of the stomach. Anatomically, the stomach is usually divided into two major parts:

(1) The body

(2) The antrum.

Physiologically, it is more appropriately divided into:

(1) The "orad" portion, comprising about the first two thirds of the body.

(2) The "caudad" portion, comprising the remainder of the body plus the antrum.

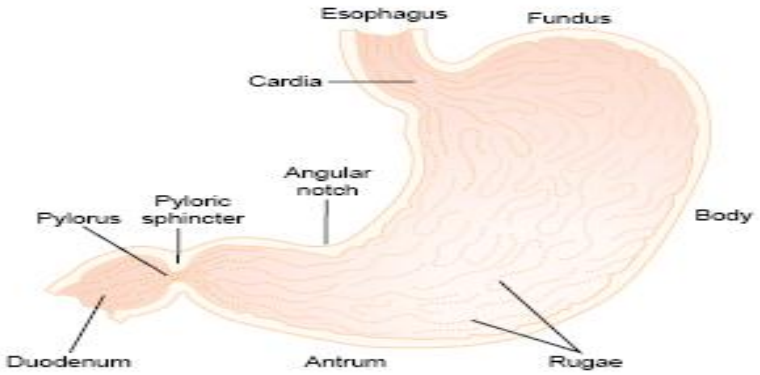


Figure 63–2

Physiologic anatomy of the stomach.

Storage function of the stomach

As food enters the stomach, it forms concentric circles of the food in the orad portion of the stomach, the newest food lying closest to the esophageal opening and the oldest food lying nearest the outer wall of the stomach. Normally, when food stretches the stomach, a "vagovagal reflex" from the stomach to the brain stem and then back to the stomach reduces the tone in the muscular wall of the body of the stomach so that the wall bulges progressively outward, accommodating greater and greater quantities of food up to a limit in the completely relaxed stomach of 0.8 to 1.5 liters. The pressure in the stomach remains low until this limit is approached.

Mixing and propulsion of food in the stomach—the basic electrical Rhythm of the stomach wall

The digestive juices of the stomach are secreted by gastric glands, which are present in almost the entire wall of the body of the stomach except along a narrow strip on the lesser curvature of the stomach. These secretions come immediately into contact with that portion of the stored food lying against the mucosal surface of the stomach. As long as food is in the stomach, weak peristaltic constrictor waves, called mixing waves, begin in the mid- to upper portions of the stomach wall and move toward the antrum about once every 15 to 20 seconds. These waves are initiated by the gut wall basic electrical rhythm, consisting of electrical "slow waves" that occur spontaneously in the stomach wall. As the constrictor waves progress from the body of the stomach into the antrum, they become more intense, some becoming extremely intense and providing powerful peristaltic action potential–driven constrictor rings that force the antral contents under higher and higher pressure toward the pylorus. These constrictor rings also play an important role in mixing the stomach contents in the following way:

Each time a peristaltic wave passes down the antral wall toward the pylorus, it digs deeply into the food contents in the antrum. Yet the opening of the pylorus is still small enough that only a few milliliters or less of antral contents are expelled into the duodenum with each peristaltic wave. Also, as each peristaltic wave approaches the pylorus, the pyloric muscle itself often contracts, which further impedes emptying through the pylorus. Therefore, most of the antral contents are squeezed upstream through the peristaltic ring toward the body of the stomach, not through the pylorus. Thus, the moving peristaltic constrictive ring, combined with this upstream squeezing action, called "retropulsion," is an exceedingly important mixing mechanism in the stomach.

Chyme.

After food in the stomach has become thoroughly mixed with the stomach secretions, the resulting mixture that passes down the gut is called chyme. The degree of fluidity of the chyme leaving the

stomach depends on the relative amounts of food, water, and stomach secretions and on the degree of digestion that has occurred.The appearance of chime is that of a murky semifluid or paste.

Hunger Contractions.

Besides the peristaltic contractions that occur when food is present in the stomach, another type of intense contractions, called hunger contractions, often occurs when the stomach has been empty for several hours or more. They are rhythmical peristaltic contractions in the body of the stomach. When the successive contractions become extremely strong, they often fuse to cause a continuing titanic contraction that sometimes lasts for 2 to 3 minutes. Hunger contractions are most intense in young, healthy people who have high degrees of gastrointestinal tonus; they are also greatly increased by the person's having lower than normal levels of blood sugar. When hunger contractions occur in the stomach, the person sometimes experiences mild pain in the pit of the stomach, called hunger pangs. Hunger pangs usually do not begin until 12 to 24 hours after the last ingestion of food; in starvation, they reach their greatest intensity in 3 to 4 days and gradually weaken in succeeding days.

Stomach emptying

Stomach emptying is promoted by intense peristaltic contractions in the stomach antrum. At the same time,
emptying is opposed by varying degrees of resistance to passage of chyme at the pylorus.

Regulation of stomach emptying

The rate at which the stomach empties is regulated by signals from both the stomach and the duodenum. However, the duodenum provides by far the more potent of the signals, controlling the emptying of chyme into the duodenum at a rate no greater than the rate at which the chyme can be digested and absorbed in the small intestine.

Summary of the control of stomach emptying

Emptying of the stomach is controlled only to a moderate degree by stomach factors such as the degree of filling in the stomach and the excitatory effect of gastrin on stomach peristalsis. Probably the more important control of stomach emptying resides in inhibitory feedback signals from the duodenum, including both enterogastric inhibitory nervous feedback reflexes and hormonal feedback by CCK. These feedback inhibitory mechanisms work together to slow the rate of emptying when:
(1) Too much chyme is already in the small intestine.
(2)The chyme is excessively acidic, contains too much unprocessed protein or fat, is hypotonic or hypertonic, or is irritating In this way, the rate of stomach emptying is limited to that amount of chyme that the small intestine can process.

GIT Physiology

Dr.Latief fayadh **Lec. 3**

Movements of the Small Intestine.

The movements of the small intestine, like those elsewhere in the gastrointestinal tract, can be divided into mixing contractions and propulsive contractions. To a great extent, this separation is artificial because essentially all movements of the small intestine cause at least some degree of both mixing and propulsion. The usual classification of these processes is the following.

- Mixing Contractions (Segmentation Contractions).

When a portion of the small intestine becomes distended with chyme, stretching of the intestinal wall elicits localized concentric contractions spaced at intervals along the intestine and lasting a fraction of a minute. The contractions cause "segmentation" of the small intestine, as shown in figure below. That is, they divide the intestine into spaced segments that have the appearance of a chain of sausages. As one set of segmentation contractions relaxes, a new set often begins, but the contractions this time occur mainly at new points between the previous contractions. Therefore, the segmentation contractions "chop" the chyme two to three times per minute, in this way promoting progressive mixing of the food with secretions of the small intestine. The maximum frequency of the segmentation contractions in the small intestine is determined by the frequency of electrical slow waves in the intestinal wall, which is the basic electrical rhythm. Because this frequency normally is not over 12 per minute in the duodenum and proximal jejunum, the maximum frequency of the segmentation contractions in these areas is also about 12 per minute, but this occurs only under extreme conditions of stimulation. In the terminal ileum, the maximum frequency is usually 8 to 9 contractions per minute. The segmentation contractions become exceedingly weak when the excitatory activity of the enteric nervous system is blocked by the drug atropine. Therefore, even though it is the slow waves in the smooth muscle itself that cause the segmentation contractions, these contractions are not effective without background excitation mainly from the myenteric nerve plexus.

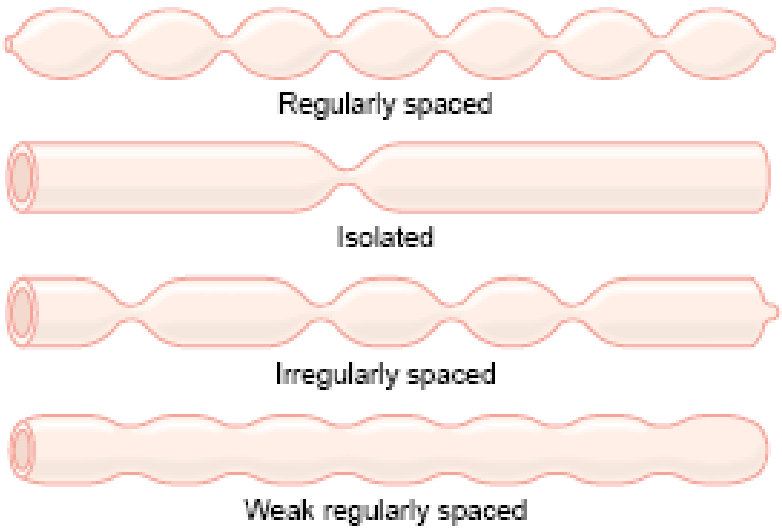


Figure 63–3

Segmentation movements of the small intestine.

-Propulsive Movements

Peristalsis in the Small Intestine.

Chyme is propelled through the small intestine by peristaltic waves. These can occur in any part of the small intestine, and they move toward the anus at a velocity of 0.5 to 2.0 cm/sec, faster in the proximal intestine and slower in the terminal intestine. They normally are very weak and usually die out after traveling only 3 to 5 centimeters, very rarely farther than 10 centimeters, so that forward movement of the chyme is very slow, so slow in fact that net movement along the small intestine normally averages only 1 cm/min. This means that 3 to 5 hours are required for passage of chyme from the pylorus to the ileocecal valve.

Movements of the Colon

The principal functions of the colon are:

(1) Absorption of water and electrolytes from the chyme to form solid feces

(2) Storage of fecal matter until it can be expelled.

The proximal half of the colon, is concerned principally with absorption, and the distal half with storage. Because intense colon wall movements are not required for these functions, the movements of the colon are normally very sluggish. Yet in a sluggish manner, the movements still have characteristics similar to those of the small intestine and can be divided once again into mixing movements and propulsive movements.

Mixing Movements—"Haustrations."

In the same manner that segmentation movements occur in the small intestine, large circular constrictions occur in the large intestine. At each of these constrictions, about 2.5 centimeters of the circular muscle contracts, sometimes constricting the lumen of the colon almost to occlusion. At the same time, the longitudinal muscle of the colon, which is aggregated into three longitudinal strips called the teniae coli, contracts. These combined contractions of the circular and longitudinal strips of muscle cause the unstimulated portion of the large intestine to bulge outward into baglike sacs called haustrations. Each haustration usually reaches peak intensity in about 30 seconds and then disappears during the next 60 seconds. They also at times move slowly toward the anus during contraction, especially in the cecum and ascending colon, and thereby provide a minor amount of forward propulsion of the colonic contents. After another few minutes, new haustral contractions occur in other areas nearby. Therefore, the fecal material in the large intestine is slowly dug into and rolled over in much the same manner that one spades the earth. In this way, all the fecal material is gradually exposed to the mucosal surface of the large intestine, and fluid and dissolved substances are progressively absorbed until only 80 to 200 milliliters of feces are expelled each day.

Propulsive Movements—"Mass Movements."

Much of the propulsion in the cecum and ascending colon results from the slow but persistent haustral contractions, requiring as many as 8 to 15 hours to move the chime from the ileocecal valve through the colon, while the chyme itself becomes fecal in quality, a semisolid slush instead of semifluid. From the cecum to the sigmoid, mass movements can, for many minutes at a time, take over the propulsive role. These movements usually occur only one to three times each day, in many people especially for about 15 minutes during the first hour after eating breakfast. A mass movement is a modified type of peristalsis characterized by the following sequence of events:

First, a constrictive ring occurs in response to a distended or irritated point in the colon, usually in the transverse colon. Then, rapidly, the 20 or more centimeters of colon distal to the constrictive ring lose their haustrations and instead contract as a unit, propelling the fecal material in this segment en masse further down the colon. The contraction develops progressively more force for about 30 seconds, and relaxation occurs during the next 2 to 3 minutes. Then, another mass movement occurs, this time perhaps

farther along the colon. A series of mass movements usually persists for 10 to 30 minutes. Then they cease but return perhaps a half day later. When they have forced a mass of feces into the rectum, the desire for defecation is felt.

Defecation

Most of the time, the rectum is empty of feces. This results partly from the fact that a weak functional sphincter exists about 20 centimeters from the anus at the juncture between the sigmoid colon and the rectum. There is also a sharp angulation here that contributes additional resistance to filling of the rectum. When a mass movement forces feces into the rectum, the desire for defecation occurs immediately, including reflex contraction of the rectum and relaxation of the anal sphincters. Continual dribble of fecal matter through the anus is prevented by tonic constriction of :

(1) An internal anal sphincter, a several-centimeters-long thickening of the circular smooth muscle that lies immediately inside the anus.

(2) An external anal sphincter, composed of striated voluntary muscle that both surrounds the internal sphincter and extends distal to it. The external sphincter is controlled by nerve fibers in the pudendal nerve, which is part of the somatic nervous system and therefore is under voluntary, conscious or at least subconscious control; subconsciously, the external sphincter is usually kept continuously constricted unless conscious signals inhibit the constriction.

Defecation Reflexes.

Ordinarily, defecation is initiated by defecation reflexes. One of these reflexes is an intrinsic reflex mediated by the local enteric nervous system in the rectal wall. This can be described as follows:

When feces enter the rectum, distention of the rectal wall initiates afferent signals that spread through the myenteric plexus to initiate peristaltic waves in the descending colon, sigmoid, and rectum, forcing feces toward the anus. As the peristaltic wave approaches the anus, the internal anal sphincter is relaxed by inhibitory signals from the myenteric plexus; if the external anal sphincter is also consciously, voluntarily relaxed at the same time, defecation occurs. The intrinsic myenteric defecation reflex functioning by itself normally is relatively weak. To be effective in causing defecation, it usually must be fortified by another type of defecation reflex, a parasympathetic defecation reflex that involves the sacral segments of the spinal cord, When the nerve endings in the rectum are stimulated, signals are transmitted first into the spinal cord and then reflexly back to the descending colon, sigmoid, rectum, and anus by way of parasympathetic nerve fibers in the pelvic nerves. These parasympathetic signals greatly intensify the peristaltic waves as well as relax the internal anal sphincter, thus converting the intrinsic myenteric defecation reflex from a weak effort into a powerful process of defecation that is sometimes effective in emptying the large bowel all the way from the splenic flexure of the colon to the anus. Defecation signals entering the spinal cord initiate other effects, such as taking a deep breath, closure of the glottis, and contraction of the abdominal wall muscles to force the fecal contents of the colon downward and at the same time cause the pelvic floor to relax downward and pull outward on the anal ring to evaginate the feces. When it becomes convenient for the person to defecate, the defecation reflexes can purposely be activated by taking a deep breath to move the diaphragm downward and then contracting the abdominal muscles to increase the pressure in the abdomen, thus forcing fecal contents into the rectum to cause new reflexes. Reflexes initiated in this way are almost never as effective as those that arise naturally, for which reason people who too often inhibit their natural reflexes are likely to become severely constipated. In newborn babies and in some people with transected spinal cords, the defecation reflexes cause automatic emptying of the lower bowel at inconvenient times during the day because of lack of conscious control exercised through voluntary contraction or relaxation of the external anal sphincter.

Secretory functions of the alimentary tract

Throughout the gastrointestinal tract, secretory glands subserve two primary functions:

***First,** digestive enzymes are secreted in most areas of the alimentary tract, from the mouth to the distal end of the ileum.*

***Second**, mucous glands, from the mouth to the anus, provide mucus for lubrication and protection of all parts of the alimentary tract. Most digestive secretions are formed only in response to the presence of food in the alimentary tract, and the quantity secreted in each segment of the tract is almost exactly the amount needed for proper digestion. Furthermore, in some portions of the gastrointestinal tract, even the types of enzymes and other constituents of the secretions are varied in accordance with the types of food present. The purpose of this chapter, therefore, is to describe the different alimentary secretions, their functions, and regulation of their production.*

General Principles of Alimentary Tract Secretion

Anatomical Types of Glands

Several types of glands provide the different types of alimentary tract secretions

***First,** on the surface of the epithelium in most parts of the gastrointestinal tract are billions of single-cell mucous glands called simply mucous cells or sometimes goblet cells because they look like goblets. They function mainly in response to local irritation of the epithelium: they extrude mucus directly onto the epithelial surface to act as a lubricant that also protects the surfaces from excoriation and digestion.*

***Second,** many surface areas of the gastrointestinal tract are lined by pits that represent invaginations of the epithelium into the submucosa. In the small intestine, these pits, called crypts of Lieberkühn, are deep and contain specialized secretory cells.*

***Third,** in the stomach and upper duodenum are large numbers of deep tubular glands. A typical tubular gland can shows an acid- and pepsinogen-secreting gland of the stomach (oxyntic gland).*

***Fourth**, also associated with the alimentary tract are several complex glands—the salivary glands, pancreas, and liver—that provide secretions for digestion or emulsification of food. The liver has a highly specialized structure The salivary glands and the pancreas are compound acinous glands, These glands lie outside the walls of the alimentary tract and, in this, differ from all other alimentary glands. They contain millions of acini lined with secreting glandular cells; these acini feed into a system of ducts that finally empty into the alimentary tract itself.*

Table 64–1

Daily Secretion of Intestinal Juices

	Daily Volume (ml)	pH
Saliva	1000	6.0–7.0
Gastric secretion	1500	1.0–3.5
Pancreatic secretion	1000	8.0–8.3
Bile	1000	7.8
Small intestine secretion	1800	7.5–8.0
Brunner's gland secretion	200	8.0–8.9
Large intestinal secretion	200	7.5–8.0
Total	6700	

Basic Mechanisms of Stimulation of the Alimentary Tract Glands

Effect of Contact of Food with the Epithelium—Function of Enteric Nervous Stimuli.

The mechanical presence of food in a particular segment of the gastrointestinal tract usually causes the glands of that region and often of adjacent regions to secrete moderate to large quantities of juices. Part of this local effect, especially the secretion of mucus by mucous cells, results from direct contact stimulation of the surface glandular cells by the food. In addition, local epithelial stimulation also activates the enteric nervous system of the gut wall. The types of stimuli that do this are

(1) Tactile stimulation.

(2) Chemical irritation.

(3) Distention of the gut wall.

The resulting nervous reflexes stimulate both the mucous cells on the gut epithelial surface and the deep glands in the gut wall to increase their secretion.

Autonomic Stimulation of Secretion

Parasympathetic Stimulation.

Stimulation of the parasympathetic nerves to the alimentary tract almost invariably increases the rates of alimentary glandular secretion. This is especially true of the glands in the upper portion of the tract (innervated by the glossopharyngeal and vagus parasympathetic nerves) such as the salivary glands, esophageal glands, gastric glands, pancreas, and Brunner's glands in the duodenum. It is also true of some glands in the distal portion of the large intestine, innervated by pelvic parasympathetic nerves. Secretion in the remainder of the small intestine and in the first two thirds of the large intestine occurs mainly in response to local neural and hormonal stimuli in each segment of the gut.

Sympathetic Stimulation.

Stimulation of the sympathetic nerves going to the gastrointestinal tract causes a slight to moderate increase in secretion by some of the local glands. But sympathetic stimulation also results in constriction of the blood vessels that supply the glands. Therefore, sympathetic stimulation can have a dual effect: ***First,*** *sympathetic stimulation alone usually slightly increases secretion*

second*, if parasympathetic or hormonal stimulation is already causing copious secretion by the glands, superimposed sympathetic stimulation usually reduces the secretion, sometimes significantly so, mainly because of vasoconstrictive reduction of the blood supply.*

Regulation of Glandular Secretion by Hormones.

In the stomach and intestine, several different gastrointestinal hormones help regulate the volume and character of the secretions. These hormones are liberated from the gastrointestinal mucosa in response to the presence of food in the lumen of the gut. The hormones then are absorbed into the blood and carried to the glands, where they stimulate secretion. This type of stimulation is particularly valuable to increase the output of gastric juice and pancreatic juice when food enters the stomach or duodenum. Chemically, the gastrointestinal hormones are polypeptides or polypeptide derivatives.

Gastric Secretion

Characteristics of the Gastric Secretions

In addition to mucus-secreting cells that line the entire surface of the stomach, the stomach mucosa has two important types of tubular glands: oxyntic glands (also called gastric glands) and pyloric glands. The oxyntic (acid-forming) glands secrete hydrochloric acid, pepsinogen, intrinsic factor, and mucus. The pyloric glands secrete mainly mucus for protection of the pyloric mucosa from the stomach acid. They also

secrete the hormone gastrin. The oxyntic glands are located on the inside surfaces of the body and fundus of the stomach, constituting the proximal 80 per cent of the stomach. The pyloric glands are located in the antral portion of the stomach, the distal 20 per cent of the stomach.

Secretions from the Oxyntic (Gastric) Glands

A typical stomach oxyntic gland is shown in Figure below. It is composed of three types of cells:

(1) Mucous neck cells, which secrete mainly mucus

(2)Peptic (or chief) cells, which secrete large quantities of pepsinogen

(3) Parietal (or oxyntic) cells, which secrete hydrochloric acid and intrinsic factor.

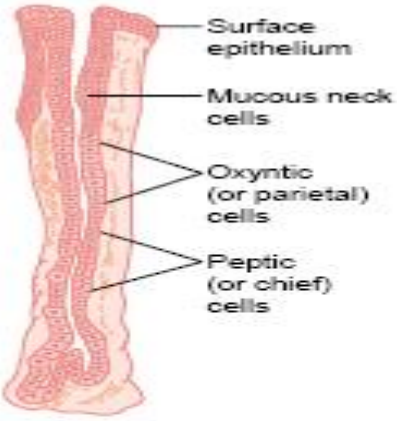


Figure 64–4

Oxyntic gland from the body of the stomach.

Basic Mechanism of Hydrochloric Acid Secretion.

When stimulated, the parietal cells secrete an acid solution that contains about 160 millimoles of hydrochloric acid per liter, which is almost exactly isotonic with the body fluids. The pH of this acid is about 0.8, demonstrating its extreme acidity. At this pH, the hydrogen ion concentration is about 3 million times that of the arterial blood. To concentrate the hydrogen ions this tremendous amount requires more than 1500 calories of energy per liter of gastric juice. Figure below shows schematically the functional structure of a parietal cell (also called oxyntic cell), demonstrating that it contains large branching intracellular canaliculi. The hydrochloric acid is formed at the villus-like projections inside these canaliculi and is then conducted through the canaliculi to the secretory end of the cell.

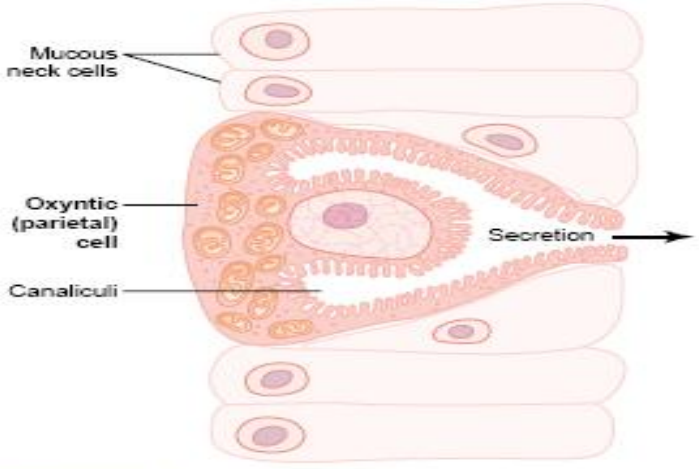


Figure 64–5

Schematic anatomy of the canaliculi in a parietal (oxyntic) cell.

Secretion and Activation of Pepsinogen.

Several slightly different types of pepsinogen are secreted by the peptic and mucous cells of the gastric glands. Even so, all the pepsinogens perform the same functions. When pepsinogen is first secreted, it has no digestive activity. However, as soon as it comes in contact with hydrochloric acid, it is activated to form active pepsin. In this process, the pepsinogen molecule, having a molecular weight of about 42,500, is split to form a pepsin molecule, having a molecular weight of about 35,000. Pepsin functions as an active proteolytic enzyme in a highly acid medium (optimum pH 1.8 to 3.5), but above a pH of about 5 it has almost no proteolytic activity and becomes completely inactivated in a short time. Hydrochloric acid is as necessary as pepsin for protein digestion in the stomach.

Pyloric Glands—Secretion of Mucus and Gastrin

The pyloric glands are structurally similar to the oxyntic glands but contain few peptic cells and almost no parietal cells. Instead, they contain mostly mucous cells that are identical with the mucous neck cells of the oxyntic glands. These cells secrete a small amount of pepsinogen, and an especially large amount of thin mucus that helps to lubricate food movement, as well as to protect the stomach wall from digestion by the gastric enzymes. The pyloric glands also secrete the hormone gastrin, which plays a key role in controlling gastric secretion.

Surface Mucous Cells

The entire surface of the stomach mucosa between glands has a continuous layer of a special type of mucous cells called simply "surface mucous cells." They secrete large quantities of a very viscid mucus that coats the stomach mucosa with a gel layer of mucus often more than 1 millimeter thick, thus providing a major shell of protection for the stomach wall as well as contributing to lubrication of food transport. Another characteristic of this mucus is that it is alkaline. Therefore, the normal underlying stomach wall is not directly exposed to the highly acidic, proteolytic stomach secretion. Even the slightest contact with food or any irritation of the mucosa directly stimulates the surface mucous cells to secrete additional quantities of this thick, alkaline, viscid mucus.

Stimulation of Gastric Acid Secretion

Parietal Cells of the Oxyntic Glands Are the Only Cells That Secrete HCL, The parietal cells, located deep in the oxyntic glands of the main body of the stomach, are the only cells that secrete hydrochloric acid. the acidity of the fluid secreted by these cells can be very great, with pH as low as 0.8. However, secretion of this acid is under continuous control by both endocrine and nervous signals. Furthermore, the parietal cells operate in close association with another type of cell called enterochromaffin- like cells (ECL cells), the primary function of which is to secrete histamine. The ECL cells lie in the deep recesses of the oxyntic glands and therefore release histamine in direct contact with the parietal cells of the glands. The rate of formation and secretion of hydrochloric acid by the parietal cells is directly related to the amount of histamine secreted by the ECL cells. In turn, the ECL cells can be stimulated to secrete histamine in several different ways:

(1) Probably the most potent mechanism for stimulating histamine secretion is by the hormonal substance gastrin, which is formed almost entirely in the antral portion of the stomach mucosa in response to proteins in the foods being digested.

(2) In addition, the ECL cells can be stimulated by

(a) Acetylcholine released from stomach vagal nerve endings

(b) Probably also by hormonal substances secreted by the enteric nervous system of the stomach wall. Let us discuss first the gastrin mechanism for control of the ECL cells and their subsequent control of parietal cell secretion of hydrochloric acid.

Stimulation of Acid Secretion by Gastrin.

Gastrin is itself a hormone secreted by gastrin cells, also called G cells. These cells are located in the pyloric glands in the distal end of the stomach. Gastrin is a large polypeptide secreted in two forms: a large form called G-34, which contains 34 amino acids, and a smaller form, G-17, which contains 17 amino acids. Although both of these are important, the smaller is more abundant. When meats or other protein-containing foods reach the antral end of the stomach, some of the proteins from these foods have a special stimulatory effect on the gastrin cells in the pyloric glands to cause release of gastrin into the digestive juices of the stomach. The vigorous mixing of the gastric juices transports the gastrin rapidly to the ECL cells in the body of the stomach, causing release of histamine directly into the deep oxyntic glands. The histamine then acts quickly to stimulate gastric hydrochloric acid secretion.

Regulation of Pepsinogen Secretion

Regulation of pepsinogen secretion by the peptic cells in the oxyntic glands is much less complex than regulation of acid secretion; it occurs in response to two types of signals:

(1) Stimulation of the peptic cells by acetylcholine released from the vagus nerves or from the gastric enteric nervous plexus.

(2) Stimulation of peptic cell secretion in response to acid in the stomach. The acid probably does not stimulate the peptic cells directly but instead elicits additional enteric nervous reflexes that support the original nervous signals to the peptic cells. Therefore, the rate of secretion of pepsinogen, the precursor of the enzyme pepsin that causes protein digestion, is strongly influenced by the amount of acid in the stomach. In people who have lost the ability to secrete normal amounts of acid, secretion of pepsinogen is also decreased, even though the peptic cells may otherwise appear to be normal.

GIT Physiology

Dr.Latief fayadh **Lec. 4**

Phases of gastric secretion

Gastric secretion is said to occur in three "phases" as shown in Figure below a cephalic phase, a gastric phase, and an intestinal phase.

Cephalic phase.

The cephalic phase of gastric secretion occurs even before food enters the stomach, especially while it is being eaten. It results from the sight, smell, thought, or taste of food, and the greater the appetite, the more intense is the stimulation. Neurogenic signals that cause the cephalic phase of gastric secretion originate in the cerebral cortex and in the appetite centers of the amygdala and hypothalamus. They are transmitted through the dorsal motor nuclei of the vagi and thence through the vagus nerves to the stomach. This phase of secretion normally accounts for about 20 per cent of the gastric secretion associated with eating a meal.

Gastric phase.

Once food enters the stomach, it excites

(1) Long vagovagal reflexes from the stomach to the brain and back to the stomach.

(2) Local enteric reflexes.

(3) The gastrin mechanism.

all of which in turn cause secretion of gastric juice during several hours while food remains in the stomach. The gastric phase of secretion accounts for about 70 per cent of the total gastric secretion associated with eating a meal and therefore accounts for most of the total daily gastric secretion of about 1500 milliliters.

Intestinal phase.

The presence of food in the upper portion of the small intestine, particularly in the duodenum, will continue to cause stomach secretion of small amounts of gastric juice, probably partly because of small amounts of gastrin released by the duodenal mucosa.

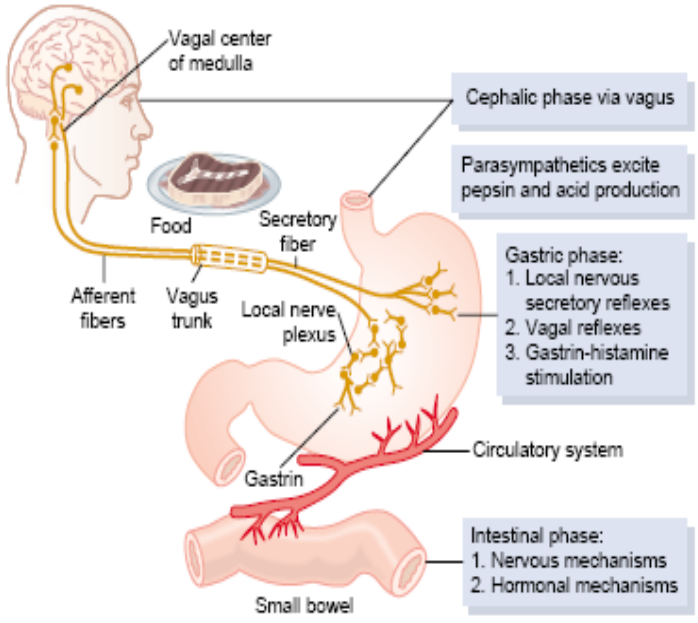


Figure 64–7

Phases of gastric secretion and their regulation.

Gastric secretion during the interdigestive period.

The stomach secretes a few milliliters of gastric juice each hour during the "interdigestive period," when little or no digestion is occurring anywhere in the gut. The secretion that does occur usually is almost entirely of the nonoxyntic type, composed mainly of mucus but little pepsin and almost no acid. Unfortunately, emotional stimuli frequently increase interdigestive gastric secretion (highly peptic and acidic) to 50 milliliters or more per hour, in very much the same way that the cephalic phase of gastric secretion excites secretion at the onset of a meal. This increase of secretion in response to emotional stimuli is believed to be one of the causative factors in development of peptic ulcers.

Pancreatic secretion

The pancreas, which lies parallel to and beneath the stomach , is a large compound gland with most of its internal structure similar to that of the salivary glands .The pancreatic digestive enzymes are secreted by pancreatic acini, and large volumes of sodium bicarbonate solution are secreted by the small ductules and larger ducts leading from the acini. The combined product of enzymes and sodium bicarbonate then flows through a long pancreatic duct that normally joins the hepatic duct immediately before it empties into the duodenum through the papilla of Vater, surrounded by the sphincter of Oddi. Pancreatic juice is secreted most abundantly in response to the presence of chyme in the upper portions of the small intestine, and the characteristics of the pancreatic juice are determined to some extent by the types of food in the chyme. (The pancreas also secretes insulin, but this is not secreted by the same pancreatic tissue that secretes intestinal pancreatic juice. Instead, insulin is secreted directly into the blood—not into the intestine—by the islets of Langerhans that occur in islet patches throughout the pancreas.

Pancreatic digestive enzymes

Pancreatic secretion contains multiple enzymes for digesting all of the three major types of food: proteins, carbohydrates, and fats. It also contains large quantities of bicarbonate ions, which play an important role in neutralizing the acidity of the chyme emptied from the stomach into the duodenum. The most important of the pancreatic enzymes for digesting proteins are trypsin, chymotrypsin, and carboxypolypeptidase. By far the most abundant of these is trypsin. Trypsin and chymotrypsin split whole and partially digested proteins into peptides of various sizes but do not cause release of individual amino acids. However, carboxypolypeptidase does split some peptides into individual amino acids, thus completing digestion of some proteins all the way to the amino acid state. The pancreatic enzyme for digesting carbohydrates is pancreatic amylase, which hydrolyzes starches, glycogen, and most other carbohydrates (except cellulose) to form mostly disaccharides and a few trisaccharides. The main enzymes for fat digestion are

(1) Pancreatic lipase, which is capable of hydrolyzing neutral fat into fatty acids and monoglycerides;

(2) Cholesterol esterase, which causes hydrolysis of cholesterol esters.

(3) Phospholipase, which splits fatty acids from phospholipids.

When first synthesized in the pancreatic cells, the proteolytic digestive enzymes are in the inactive forms trypsinogen, chymotrypsinogen, and procarboxypolypeptidase, which are all inactive enzymatically. They become activated only after they are secreted into the intestinal tract. Trypsinogen is activated by an enzyme called enterokinase, which is secreted by the intestinal mucosa when chyme comes in contact with the mucosa. Also, trypsinogen can be autocatalytically activated by trypsin that has already been formed from previously secreted trypsinogen. Chymotrypsinogen is activated by trypsin to form chymotrypsin, and procarboxypolypeptidase is activated in a similar manner.

Secretion of trypsin inhibitor prevents digestion of the pancreas itself.

It is important that the proteolytic enzymes of the pancreatic juice not become activated until after they have been secreted into the intestine because the trypsin and the other enzymes would digest the pancreas itself. Fortunately, the same cells that secrete proteolytic enzymes into the acini of the pancreas secrete

simultaneously another substance called trypsin inhibitor. This substance is formed in the cytoplasm of the glandular cells, and it prevents activation of trypsin both inside the secretory cells and in the acini and ducts of the pancreas. And, because it is trypsin that activates the other pancreatic proteolytic enzymes, trypsin inhibitor prevents activation of the others as well. When the pancreas becomes severely damaged or when a duct becomes blocked, large quantities of pancreatic secretion sometimes become pooled in the damaged areas of the pancreas. Under these conditions, the effect of trypsin inhibitor is often overwhelmed, in which case the pancreatic secretions rapidly become activated and can literally digest the entire pancreas within a few hours, giving rise to the condition called acute pancreatitis. This sometimes is lethal because of accompanying circulatory shock; even if not lethal, it usually leads to a subsequent lifetime of pancreatic insufficiency.

Secretion of bile by the liver:

Functions of the biliary tree

One of the many functions of the liver is to secrete bile, normally between 600 and 1000 ml/day. Bile serves two important functions:

***First,** bile plays an important role in fat digestion and absorption, not because of any enzymes in the bile that cause fat digestion, but because bile acids in the bile do two things:*

(1) they help to emulsify the large fat particles of the food into many minute particles, the surface of which can then be attacked by lipase enzymes secreted in pancreatic juice.

(2) they aid in absorption of the digested fat end products through the intestinal mucosal membrane.

***Second**, bile serves as a means for excretion of several important waste products from the blood. These include especially bilirubin, an end product of hemoglobin destruction, and excesses of cholesterol.*

Physiologic anatomy of biliary secretion

Bile is secreted in two stages by the liver:

(1) The initial portion is secreted by the principal functional cells of the liver, the hepatocytes; this initial secretion contains large amounts of bile acids, cholesterol, and other organic constituents. It is secreted into minute bile canaliculi that originate between the hepatic cells.

(2) Next, the bile flows in the canaliculi toward the interlobular septa, where the canaliculi empty into terminal bile ducts and then into progressively larger ducts, finally reaching the hepatic duct and common bile duct. From these the bile either empties directly into the duodenum or is diverted for minutes up to several hours through the cystic duct into the gallbladder. In its course through the bile ducts, a second portion of liver secretion is added to the initial bile. This additional secretion is a watery solution of sodium and bicarbonate ions secreted by secretory epithelial cells that line the ductules and ducts. This second secretion sometimes increases the total quantity of bile by as much as an additional 100 per cent. The second secretion is stimulated especially by secretin, which causes release of additional quantities of bicarbonate ions to supplement the bicarbonate ions in pancreatic secretion (for neutralizing acid that empties into the duodenum from the stomach).

Storing and concentrating bile in the gallbladder.

Bile is secreted continually by the liver cells, but most of it is normally stored in the gallbladder until needed in the duodenum. The maximum volume that the gallbladder can hold is only 30 to 60 milliliters. Nevertheless, as much as 12 hours of bile secretion (usually about 450 milliliters) can be stored in the gallbladder because water, sodium, chloride, and most other small electrolytes are continually absorbed through the gallbladder mucosa, concentrating the remaining bile constituents that contain the bile salts, cholesterol, lecithin, and bilirubin. Most of this gallbladder absorption is caused by active transport of sodium through the gallbladder epithelium, and this is followed by secondary absorption of chloride ions, water, and most other diffusible Water and constituents. Bile is normally concentrated in this way about 5-fold, but it can be concentrated up to a maximum of 20-fold.

Function of bile salts in fat digestion and absorption

The liver cells synthesize about 6 grams of bile salts daily. The precursor of the bile salts is cholesterol, which is either present in the diet or synthesized in the liver cells during the course of fat metabolism. The cholesterol is first converted to cholic acid or chenodeoxycholic acid in about equal quantities. These acids in turn combine principally with glycine and to a lesser extent with taurine to form glyco- and tauroconjugated bile acids. The salts of these acids, mainly sodium salts, are then secreted in the bile. The bile salts have two important actions in the intestinal tract:

First, *they have a detergent action on the fat particles in the food. This decreases the surface tension of the particles and allows agitation in the intestinal tract to break the fat globules into minute sizes. This is called the emulsifying or detergent function of bile salts.*

Second, *and even more important than the emulsifying function, bile salts help in the absorption of*

(1) Fatty acids.

(2) Monoglycerides.

(3) Cholesterol.

(4) Other lipids from the intestinal tract.

They do this by forming very small physical complexes with these lipids; the complexes are called micelles, and they are semi-soluble in the chyme because of the electrical charges of the bile salts. The intestinal lipids are "ferried" in this form to the intestinal mucosa, where they are then absorbed into the blood, Without the presence of bile salts in the intestinal tract, up to 40 per cent of the ingested fats are lost into the feces, and the person often develops a metabolic deficit because of this nutrient loss.

Liver secretion of cholesterol and gallstone formation

Bile salts are formed in the hepatic cells from cholesterol in the blood plasma. In the process of secreting the bile salts, about 1 to 2 grams of cholesterol are removed from the blood plasma and secreted into the bile each day. Cholesterol is almost completely insoluble in pure water, but the bile salts and lecithin in bile combine physically with the cholesterol to form ultramicroscopic micelles in the form of a colloidal solution, When the bile becomes concentrated in the gallbladder, the bile salts and lecithin become concentrated along with the cholesterol, which keeps the cholesterol in solution. Under abnormal conditions, the cholesterol may precipitate in the gallbladder, resulting in the formation of cholesterol gallstones, The amount of cholesterol in the bile is determined partly by the quantity of fat that the person eats, because liver cells synthesize cholesterol as one of the products of fat metabolism in the body. For this reason, people on a high-fat diet over a period of years are prone to the development of gallstones. Inflammation of the gallbladder epithelium, often resulting from low-grade chronic infection, may also change the absorptive characteristics of the gallbladder mucosa, sometimes allowing excessive absorption of water and bile salts but leaving behind the cholesterol in the bladder in progressively greater concentrations. Then the cholesterol begins to precipitate, first forming many small crystals of cholesterol on the surface of the inflamed mucosa, but then progressing to large gallstones.

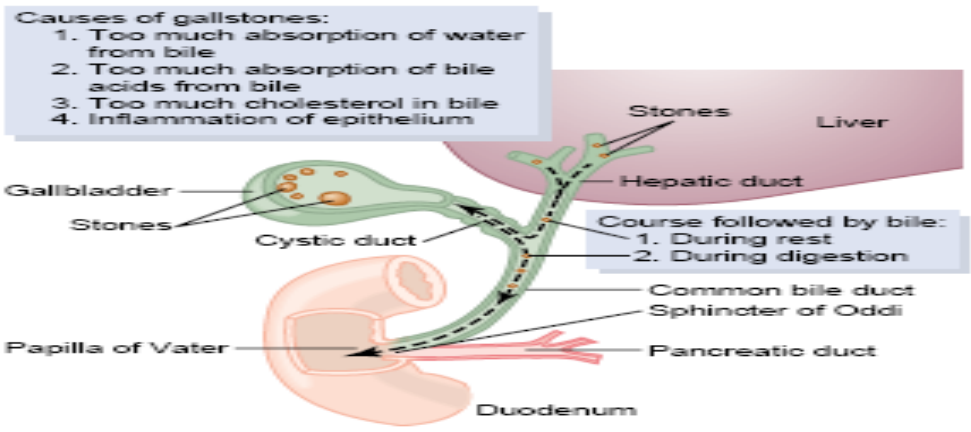


Figure 64–12

Formation of gallstones.

Secretions of the Small Intestine

Secretion of mucus by brunner's glands in the duodenum

An extensive array of compound mucous glands, called Brunner's glands, is located in the wall of the first few centimeters of the duodenum, mainly between the pylorus of the stomach and the papilla of Vater where pancreatic secretion and bile empty into the duodenum. These glands secrete large amounts of alkaline mucus in response to

(1)Tactile or irritating stimuli on the duodenal mucosa.

(2) Vagal stimulation, which causes increased Brunner's glands secretion concurrently with increase in stomach secretion.

(3) Gastrointestinal hormones, especially secretin

The function of the mucus secreted by Brunner's glands is to protect the duodenal wall from digestion by the highly acid gastric juice emptying from the stomach. In addition, the mucus contains a large excess of bicarbonate ions, which add to the bicarbonate ions from pancreatic secretion and liver bile in neutralizing the hydrochloric acid entering the duodenum from the stomach. Brunner's glands are inhibited by sympathetic stimulation; therefore, such stimulation in very excitable persons is likely to leave the duodenal bulb unprotected and is perhaps one of the factors that cause this area of the gastrointestinal tract to be the site of peptic ulcers in about 50 per cent of ulcer patients.

Secretion of intestinal digestive juices by the crypts of lieberkühn

Located over the entire surface of the small intestine are small pits called crypts of Lieberkühn, one of which is illustrated in Figure below. These crypts lie between the intestinal villi. The surfaces of both the crypts and the villi are covered by an epithelium composed of two types of cells:

(1)A moderate number of goblet cells, which secrete mucus that lubricates and protects the intestinal surfaces.

(2) A large number of enterocytes, which, in the crypts, secrete large quantities of water and electrolytes and, over the surfaces of adjacent villi, reabsorb the water and electrolytes along with end products of digestion. The intestinal secretions are formed by the enterocytes of the crypts at a rate of about 1800 ml/day. These secretions are almost pure extracellular fluid and have a slightly alkaline pH in the range of 7.5 to 8.0. The secretions also are rapidly reabsorbed by the villi. This flow of fluid from the crypts into the villi supplies a watery vehicle for absorption of substances from chyme when it comes in contact with the villi. Thus, the primary function of the small intestine is to absorb nutrients and their digestive products into the blood.

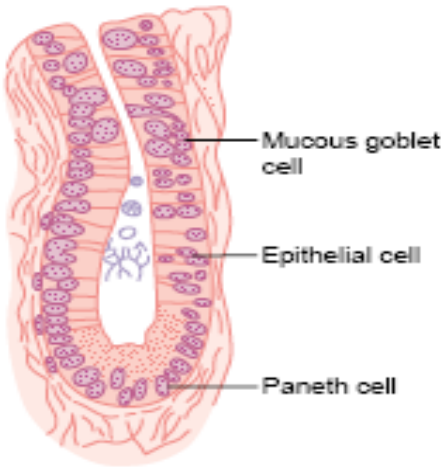


Figure 64–13

A *crypt of Lieberkühn*, found in all parts of the small intestine between the villi, which secretes almost pure extracellular fluid.

Mechanism of secretion of the watery fluid.

The exact mechanism that controls the marked secretion of watery fluid by the crypts of Lieberkühn is not known. It is believed to involve at least two active secretory processes:

(1) Active secretion of chloride ions into the crypts.

(2) Active secretion of bicarbonate ions.

The secretion of both of these ions causes electrical drag as well of positively charged sodium ions through the membrane and into the secreted fluid. Finally, all these ions together cause osmotic movement of water.

Digestive enzymes in the small intestinal secretion.

When secretions of the small intestine are collected without cellular debris, they have almost no enzymes. The enterocytes of the mucosa, especially those that cover the villi, do contain digestive enzymes that digest specific food substances while they are being absorbed through the epithelium. These enzymes are the following:

(1) Several peptidases for splitting small peptides into amino acids,

(2)Four enzymes sucrase, maltase, isomaltase, and lactase for splitting disaccharides into monosaccharides

(3) Small amounts of intestinal lipase for splitting neutral fats into glycerol and fatty acids.

The epithelial cells deep in the crypts of Lieberkühn continually undergo mitosis, and new cells migrate along the basement membrane upward out of the crypts toward the tips of the villi, thus continually replacing the villus epithelium and also forming new digestive enzymes. As the villus cells age, they are finally shed into the intestinal secretions. The life cycle of an intestinal epithelial cell is about 5 days. This rapid growth of new cells also allows rapid repair of excoriations that occur in the mucosa.

Regulation of small intestine secretion—local stimuli

By far the most important means for regulating small intestine secretion are local enteric nervous reflexes, especially reflexes initiated by tactile or irritative stimuli from the chyme in the intestines.

Secretions of the Large Intestine

Mucus secretion.

The mucosa of the large intestine, like that of the small intestine, has many crypts of Lieberkühn; however, unlike the small intestine, there are no villi. The epithelial cells contain almost no enzymes. Instead, they consist mainly of mucous cells that secrete only mucus. The great preponderance of secretion

in the large intestine is mucus. This mucus contains moderate amounts of bicarbonate ions secreted by a few non–mucus-secreting epithelial cells. The rate of secretion of mucus is regulated principally by direct, tactile stimulation of the epithelial cells lining the large intestine and by local nervous reflexes to the mucous cells in the crypts of Lieberkühn. Stimulation of the pelvic nerves from the spinal cord, which carry parasympathetic innervation to the distal one half to two thirds of the large intestine, also can cause marked increase in mucus secretion. This occurs along with increase in peristaltic motility of the colon, During extreme parasympathetic stimulation, often caused by emotional disturbances, so much mucus can occasionally be secreted into the large intestine that the person has a bowel movement of ropy mucus as often as every 30 minutes; this mucus often contains little or no fecal material. Mucus in the large intestine protects the intestinal wall against excoriation, but in addition, it provides an adherent medium for holding fecal matter together. Furthermore, it protects the intestinal wall from the great amount of bacterial activity that takes place inside the feces, and, finally, the mucus plus the alkalinity of the secretion (pH of 8.0 caused by large amounts of sodium bicarbonate) provides a barrier to keep acids formed in the feces from attacking the intestinal wall.

Diarrhea caused by excess secretion of water and electrolytes in response to irritation.

Whenever a segment of the large intestine becomes intensely irritated, as occurs when bacterial infection becomes rampant during enteritis, the mucosa secretes extra large quantities of water and electrolytes in addition to the normal viscid alkaline mucus. This acts to dilute the irritating factors and to cause rapid movement of the feces toward the anus. The result is diarrhea, with loss of large quantities of water and electrolytes. But the diarrhea also washes away irritant factors, which promotes earlier recovery from the disease than might otherwise occur

Oral physiology

Dr.Latief fayadh **Lec.5**

Saliva

Saliva is a watery fluid consists mainly of water in addition to proteins and ions.

Proteins of saliva:

Mucin and ptyalin → act on starch and convert it to other saccharides.

The saliva is secreted by 3 pairs of salivary glands:

Parotid, sublingual & submaxillary glands.

Θ When there is cancer in the salivary glands cause the fibrosis and stop the secretion of the gland.

Θ When the parotid gland inflamed causes mumps.

Quantity of saliva secretion:

The quantity of saliva secreted per day is about (0.8-1.5 L) and this quantity is depending on the conditions of the individual.

Θ The secretion of saliva is controlled by nervous system.

Note:

The saliva is very important for oral hygiene because it has important functions in swallowing (lubrication for bolus so bolus will slip toward the esophagus. In addition to lubrication the saliva moist the tongue and mouth and makes speech possible.

Glandular cell:

It is a cell secretes a hormone or enzyme, and it is surrounded by a large amount of capillaries

***Q**// how protein is formed inside the cell?*

A// In capillaries (which are surround and adjacent the glandular cell) there are nutrient materials, these materials transported from capillaries toward the glandular cell.

The glandular cell receives the nutrient materials on rough endoplasmic reticulum (R.E.R.)

Which convert the nutrient materials to proteins which transported to Golgi apparatus which is reservoir for proteins which are formed in Ribosomes. The proteins which are in Golgi apparatus either diluted or concentrated see this figure:-

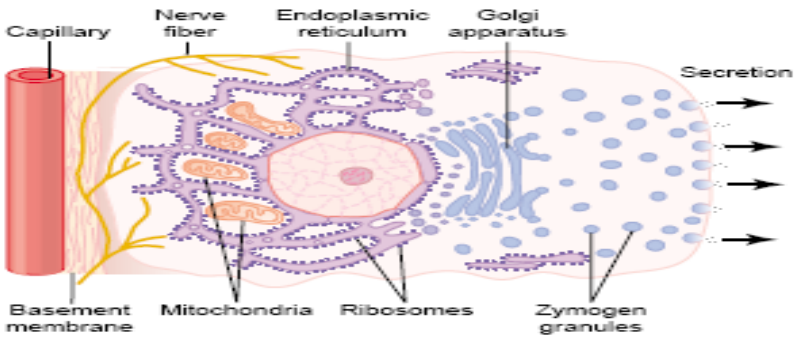


Figure 64–1

Typical function of a glandular cell for formation and secretion of enzymes and other secretory substances.

Θ When the protein is needed it goes out from GA. And form vesicles which are transported to the cell membrane and unite with it to become one piece then the vesicles will go further and expelled externally and ruptured releasing the proteins found inside vesicle, this process called "Exocytosis".

Note:

1. Sympathetic → decrease secretion of saliva.

2. Parasympathetic →increase secretion of saliva.

After releasing of protein from vesicles, it is transported to a lumen and it is very viscous so they must be converted by good amount of water to facilitate their flow in the ducts.(see this figure):

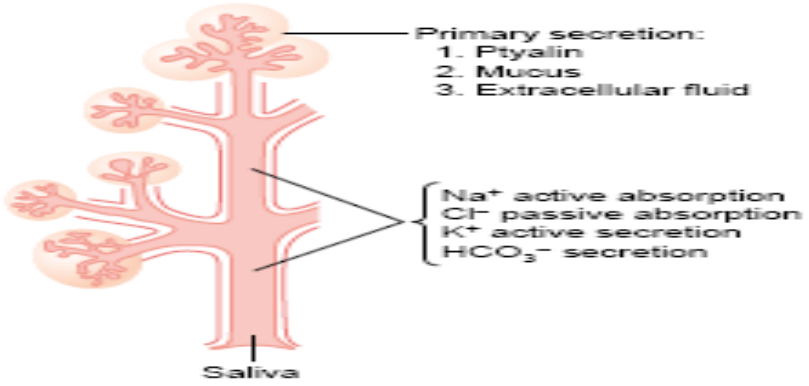


Figure 64–2

Formation and secretion of saliva by a submandibular salivary gland.

Water formation & electrolytes concentration

There must be water formation to flush out the protein into duct then secrets the saliva to oral cavity.

The Na+ and Cl- that enter the cell lead to high concentration of Nacl and less H2O concentration, so H2O molecules transport from capillaries or (ECF) to the cell by osmotic pressure and the cell swells. To prevent the rupture of the cell there will be micro punctures in the wall of the cell, so the water will exit from the cell by forceful way.

The H2O entered has Na+, Cl-, other ions, and when it enters and exits toward the lumen is similar to (ECF) because it carries similar compounds.

Saliva differs from (ECF) in:

*1. **Na+** in saliva is 7-10 times less than in ECF.*

*2. **Cl^-** in saliva is 10 times less than in ECF.*

*3. **K+** in saliva is more 4-7 times than in ECF.*

*4. **HCO3-** in saliva is 2times more than in ECF.*

Q) Why the concentration of ions saliva is differ from (ECF)?

A) Because there is reabsorption and re-execretion processes take place by the cells of duct.

Active reabsorption of Na+ by the cells of the duct leads to decrease in the Na+.

Active re-execretion of K+ from the cells of duct causes increase in the concentration of K+ in saliva.

Note:

Active (reabsorption or re-execretion) mean this process needs energy.

Passive reabsorption takes place for Cl- along the duct wall (because Cl- is negative in charge and every Na+ absorption take with it Cl- reabsorption.

HCO3- :-

Either passive excretion with K+ or active excretion (the active excretion of HCO3-is more than the passive excretion)The passive (reabsorption and excretion) mean this process doesn't need any energy.

Factors that stimulate salivary secretion

1. Tactile sensation of oral tissue.

2. Pain sensation.

3. Psychological effects.

4. Thinking by food or looking to it.

Sometimes the body needs saliva as 20 times more than the normal range , and this cause lack of ***K+,*** *and when this occurs, the condition is called (hypokalemia)and this condition causes dangerous symptoms like:*

1. Abnormal heart beats.

2. Paralysis in the muscles.

In some cases when there is cancer needs radiotherapy this therapy will affect on the salivary gland and causes fibrosis of the gland.

The fibrosis of salivary glands is called (Xerostomia) and it causes dryness of mouth due to lack in the secretion of gland.

The innervations of tongue & stimulation of salivary glands

The anterior two third of the tongue is innervated by chrodatympanic nerve.

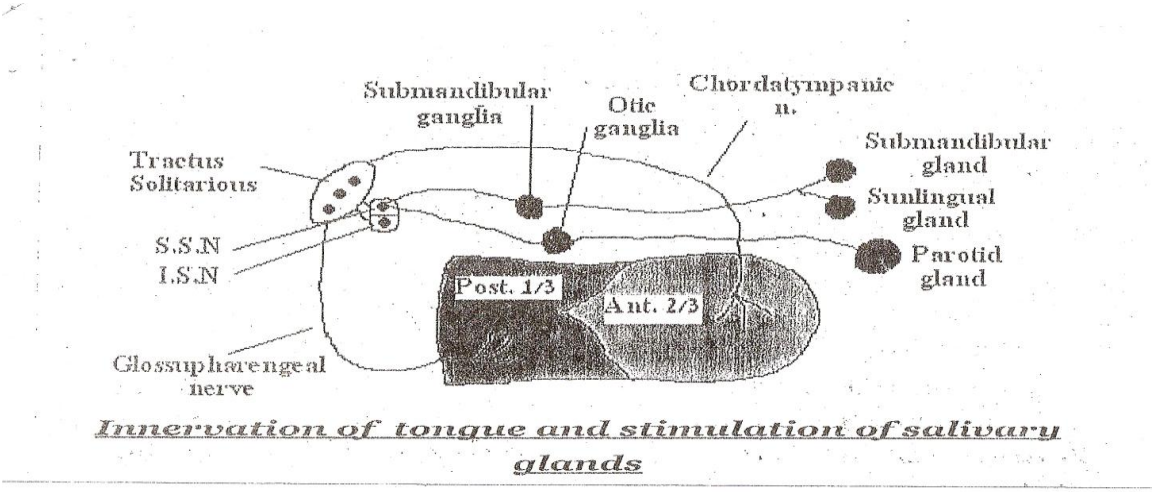
The posterior one third of the tongue is innervated by glossopharyngeal nerve.

1. The stimulation from the anterior 2/3 of the tongue is transmitted by the chrodatympanic nerve to the Tractus Solitarious, then orders from S.S.N. is go to the Submandibular Ganglia, then from it to the SubmandibularGland and Sublingual Gland.

Note:

1.There is a connection between Tractus Solitarious and (S.S.N. & I.S.N.)

2. The stimulation of posterior 1/3 of the tongue is transmitted by the glossopharyngeal nerve to the Tractus Solitarious and from the S.S.N. orders go to the otic ganglia then to the parotid gland.



Innervation of tongue and stimulation of salivary glands

Functions of saliva

1. Facilitates speech. (the person with dry mouth has difficulties in speech).

2. As a lubricant to facilitate the swallowing of bolus.

3. Cleaning function.(when person has Xyrostomia –dry mouth condition, caries will be in all his teeth.

Θ *in some teeth the caries are little like in labial, buccal, lingual sides of teeth, while more caries in the interproximal area because the saliva couldn't reach there.*

4. Has an antibacterial effect.

5. PH of saliva is about 7 is the best PH for digestion of ptyalin, and also has a buffering effect not only in the oral cavity but also in the stomach and small intestine.

Θ *Buffering effect of saliva means when the acidity in the stomach is high it will lower the acidity.*

Saliva collection

Q) Why we do such experiment to the saliva?

A) There are two reasons:

1. Either to collect the saliva especially from the parotid gland.

2. Or to collect the saliva in general (whole saliva) or (mixed saliva) from parotid gland, submandibular gland, sublingual gland and from minor glands.

Θ If we are going to measure quantity of saliva, then there is no need to distinguish it.

Θ if we are going to distinguish the saliva secreted by single gland, then we take it and make chemical analysis on it.

Methods of Collection of Saliva

1. Spitting method:

Tell the patient to spit in a beaker (regard this quantity-zero time for collection), then clean the mouth with a piece of cotton and wait 1 minute, then tell the patient to spit again and collect the saliva to study it.

2. Cotton Roll Method:

In this method we clean the patient mouth with cotton and place the cotton roll in the patient's mouth (regard this as zero time) until this roll filled with saliva and take another roll for some time, and then we weight the rolls.

3. Drooling method:

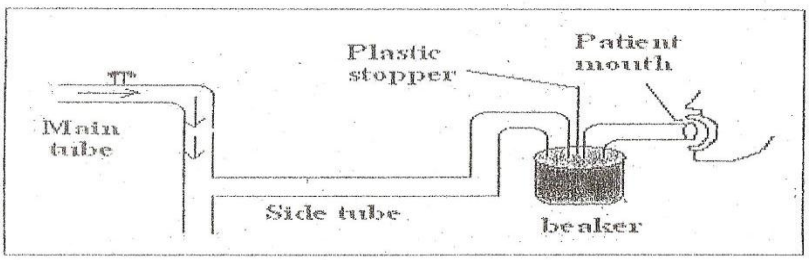
We asked the patient to spit and clean his mouth with cotton, open the patient's mouth and place beaker under his mouth for sometime(1,2,3,....) minutes ,so the patient will drool and the saliva will pass to the beaker and finally we tell him to spit for last time then measure the quantity of saliva.

4. Suction method:

This method is done by saliva ejector, placed in the patient's mouth. Water flow in main tube makes negative pressure in the side tube and the side tube placed in beaker, so negative pressure transmits to the beaker, and beaker takes saliva from patient's mouth and calculates the amount in certain time.

Θ all these methods are applied to collect the saliva from major glands.

Θ for minor glands that found in mucous membrane of the lip when we dried the lip from saliva by fingers then we will observe droplets of saliva on the inner surface of the lip and this is the secretion from the minor glands, we collect the saliva by micro pipette to study it.



Taste function

Taste sensation:

Is a function of taste buds in the oral cavity? There is about (10,000) taste buds, and this amount decreases with age due to degeneration of taste buds with age, like most human tissues which degenerate with increase of age, or decrease of taste buds may be due to atrophy the number of taste buds in child are more than in adult so taste sensation in children is higher than in adult. Taste process is a very complex process that achieved not only by taste buds but has a close association to the smell sensation, so when the person has influenza, his taste sensation will weaken because his smell sensation is weakened also due to the over secretion of mucous that cover the villi and make them difficult to move, so the smell sensation will be weak, therefore the taste sensation will be weak also.

The taste function depends on:

1. ***Texture**: if it is hard, semifluid or fluid.*
2. ***Pain effect**: like chili taste.*

Primary taste sensations

We have primary taste sensations:

1. Sour sensation.
2. Salty sensation.
3. Sweet sensation
4. Bitter sensation.

But there is no separated sensation of one of these four types of sensation, because there is no mater in the world has pure one sensation that means all food has many sensations.

Sour taste

*It depends on **PH strength**, (**H** +ions concentrations) that present in the acidic matters. When **H**+ ions concentration is high, PH is high also, so the taste is acidic (sour).*

Salty taste

*All salts in nature consist of positive ions and negative ions, for example **NaCl**, **Na**+ Cation, and **Cl**- Anion. The **Cations** are responsible for salty taste mostly, but **Anions** responsible for either salty with Cation, or for other taste.*

Sweet taste

*There is along list of materials that cause sweet taste, like: **sucrose, mannose, maltose, arabinose, glucose, lactose, amino acids, alcohol, glycogen** and other materials, so the scientists made an index and gave the cane sugar **(sucrose)** the number 1 in the index, and others measured relative to the cane sugar, for example*

Sucrose	*1*
Fructose	*1.7*
Glucose	*0.8*
Saccharin	*600*
P400	*5000*

Saccharin:

*Has sweetness more than cane sugar, so it has index of 600 but this matter has o problem called **After bitter taste**, that fell bitter after period of time, and it may be slightly Carcinogenic, but this problem is not sure because the experiments achieved were on rats, pigs and dogs by giving them a high amounts of saccharine for about 10 years then examine their stomach and intestine*

P4000:

Has sweetness of about 5000 of gm of sucrose for each gm of this substance, so it has be given an index 5000, but it is highly toxic and not used for human taken.

Bitter taste

It caused by:

*1. Long chain of Organic substances containing **Nitrogen**.*
*2. **Alkaloids** that cause bitter taste like Caffeine, Strychnine, Nicotine and Quinine, and other drugs. Most of those substances that cause bitter sensation taste are toxic to be avoided by human and animals to not taken for eating.*

Metallic and alkaline sensations

In addition to these primary sensations, we have another two sensations that we may consider them as primary sensations, they are:

1. Metallic sensation.

When we put a piece of metal in our mouth, we will sense there is special taste different from that of any other primary sensations, like the ***frame matter*** *in prosthetic patient.*

2. Alkaline sensation.

When we put a positive part of a battery in our mouth, and make it to touch our tongue, we will taste alkaline taste, and the ***alkaloids*** *will give us alkaline taste.*

Taste Blindness

Taste Blindness (PTC taste):

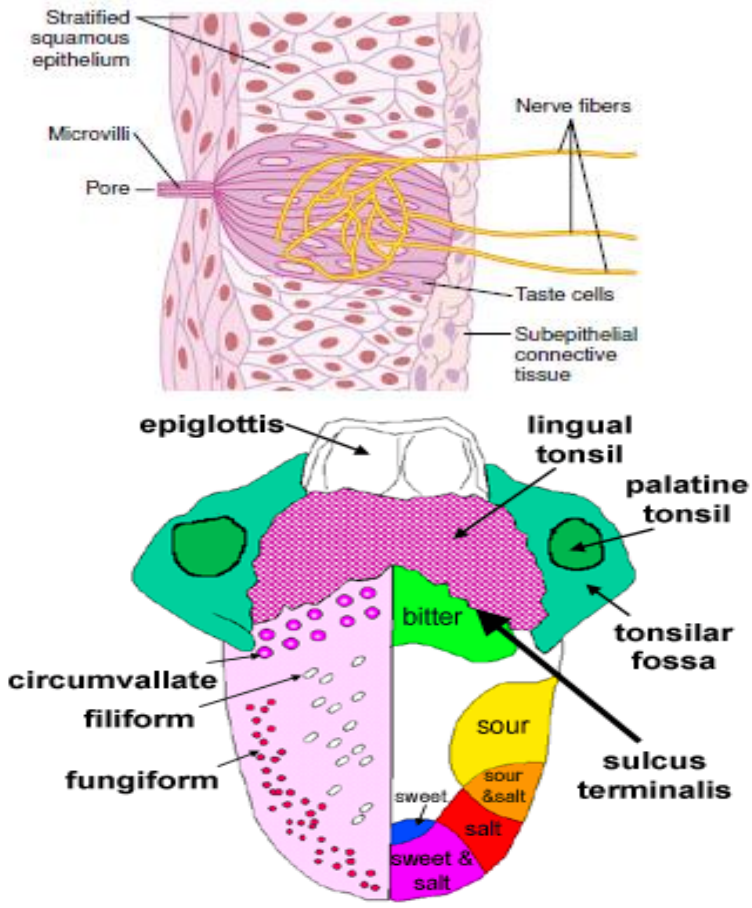
15-30% of people have taste blindness, which means inability to taste certain substances at the taste threshold, especially for different types of ***Thio urea compound*** *to diagnose it, doctor use* ***PTCmaterial*** *(**P**henyl **T**hio**C**arbomids), (which is very bitter material by putting it on the patient tongue.*

Causes of taste sensation blindness:

1. Problem or injury in the specific taste nerve.
2. Damage in the taste center in the brain.
3. Atrophy or degenerated of the taste bud.
4. Tumor in the nerve responsible for taste, which prevent taste reach to CNS.

Taste Bud

Taste bud is the organ responsible for taste. Each taste bud consists of 40-50 modified epithelial cells, some of them are supporting cells, called ***Sustentacular cells****, and others are receptor cells called* ***Taste cells*** *embedded in connective tissue of taste bud.*



The newly formed taste cells are lied outside while the degenerated taste cells are lied inside.

Life spam of each taste cell is about 10 days. The length of taste cell is 1/16 mm, and its diameter is 1/3 mm. they are arranged in pores, and on the tip of each taste cell, there is moveable microvill of 2- microns

in length, 0.1-0.2 in width, located on the top of each taste cell. These microvilli move according to the movement of saliva and tissue.

*Among the taste cells, there is branching network of several taste nerve fibers; stimulated by taste cell. Each cell of taste buds is connected by nerve fibers to form nerve trunk that transmits stimuli to the **CNS**. The taste bud degenerates when taste nerve fibers are destroyed.*

Location of Taste Buds

Taste buds are located on Papilla in Four types:

1. Circumvallate Papillae:

Their number is about 8-11, found in the posterior one third of the tongue as V-shaped line. Large number of taste buds on the walls surrounding each circumvallate papilla, there is about 100 taste buds. They are mainly taste Bitter.

2. Fungiform papillae:

Lie in the front of the tongue, have a moderate number of taste buds, about 10 taste buds. They are mainly taste sweet &salty.

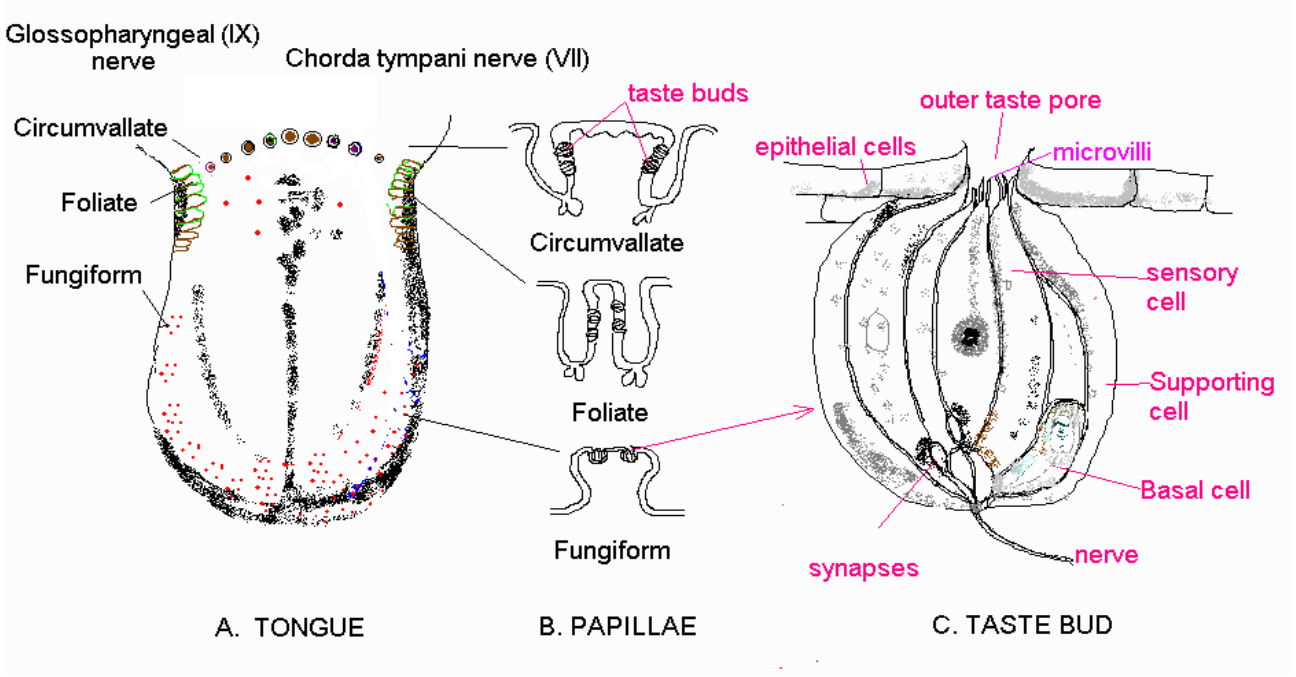
3. Foliate papillae:

Found on the lateral dorsum of the tongue, on each of them there have a moderate number of about 10 taste buds. They are mainly sour.

4. Filiform papillae:

Located on the central of the tongue. They have no taste buds.

*Θ Also there are taste buds in the **Soft palate**, **Nasopharynx** and **Tonsils**.*



Mechanism of taste

*When the substance pass through the villi, it will move them by specific way that give information about the taste for certain like sweet, salty or other, so there will be **action potential** that will pass through taste cells, then to the nerves, then to the CNS.*

Innervations of tongue

Anterior 2/3:

*Is supplied by **Lingual nerve** then it will become **Chorda tympani nerve**, then **Facial nerve**, then goes to **Tractus Solitarius**, then it will become **Medial Leminiscus**, then goes to the **Thalamus**, then finally goes to the **Taste Center** in the **Brain**.*

***Posterior 1/3*:**

Is supplied by ***Glossopharyngeal Nerve*** *, then this nerve will go also to the* ***Tractus Solitarius***, ***Thalamus*** *until reach the* ***Taste Center***.

The vagus nerve: *Supply the* ***floor of the mouth***, ***nasopharynx***, ***tonsils*** *and also the mucous membrane of the oral cavity, then this nerve go to the same regions too.*