**(Slide 1) Lecture 10**

**Digestive physiology**

**(Slide 2)** Lecture plan:

1. Digestive functions of the liver.
2. The role of bile in digestion.
3. Digestive functions of the pancreas.
4. Features of digestion in the small intestine.
5. Features of digestion in the large intestine.
6. The role of normal microflora of the large intestine in digestion processes.
7. Nervous and humoral regulation of digestion processes.

**(Slide 3)** The liver is the largest gland in the body, weighing about three pounds in an adult. It is also one of the most important organs. In addition to being an accessory digestive organ, it plays a number of roles in metabolism and regulation. The liver lies inferior to the diaphragm in the right upper quadrant of the abdominal cavity and receives protection from the surrounding ribs.

**(Slide 4)** The liver is divided into two primary lobes: a large right lobe and a much smaller left lobe. In the right lobe, some anatomists also identify an inferior quadrate lobe and a posterior caudate lobe, which are defined by internal features. The liver is connected to the abdominal wall and diaphragm by five peritoneal folds referred to as ligaments. These are the falciform ligament, the coronary ligament, two lateral ligaments, and the ligamentum teres hepatis. The falciform ligament and ligamentum teres hepatis are actually remnants of the umbilical vein, and separate the right and left lobes anteriorly. The lesser omentum tethers the liver to the lesser curvature of the stomach.

**(Slide 5)** The porta hepatis (“gate to the liver”) is where the hepatic artery and hepatic portal vein enter the liver. These two vessels, along with the common hepatic duct, run behind the lateral border of the lesser omentum on the way to their destinations.

As shown in **Slide 6**, the hepatic artery delivers oxygenated blood from the heart to the liver. The hepatic portal vein delivers partially deoxygenated blood containing nutrients absorbed from the small intestine and actually supplies more oxygen to the liver than do the much smaller hepatic arteries. In addition to nutrients, drugs and toxins are also absorbed. After processing the bloodborne nutrients and toxins, the liver releases nutrients needed by other cells back into the blood, which drains into the central vein and then through the hepatic vein to the inferior vena cava. With this hepatic portal circulation, all blood from the alimentary canal passes through the liver. This largely explains why the liver is the most common site for the metastasis of cancers that originate in the alimentary canal.

**(Slide 7)** Recall that lipids are hydrophobic, that is, they do not dissolve in water. Thus, before they can be digested in the watery environment of the small intestine, large lipid globules must be broken down into smaller lipid globules, a process called emulsification.

**(Slide 8)** Bile is a mixture secreted by the liver to accomplish the emulsification of lipids in the small intestine.

**(Slide 9) Video. Bile - What Is Bile?**

**(Slide 10)** Hepatocytes secrete about one liter of bile each day. A yellow-brown or yellow-green alkaline solution (pH 7.6 to 8.6), bile is a mixture of water, bile salts, bile pigments, phospholipids (such as lecithin), electrolytes, cholesterol, and triglycerides. The components most critical to emulsification are bile salts and phospholipids, which have a nonpolar (hydrophobic) region as well as a polar (hydrophilic) region. The hydrophobic region interacts with the large lipid molecules, whereas the hydrophilic region interacts with the watery chyme in the intestine. This results in the large lipid globules being pulled apart into many tiny lipid fragments of about 1 µm in diameter. This change dramatically increases the surface area available for lipid-digesting enzyme activity. This is the same way dish soap works on fats mixed with water. Bile salts act as emulsifying agents, so they are also important for the absorption of digested lipids. While most constituents of bile are eliminated in feces, bile salts are reclaimed by the enterohepatic circulation. Once bile salts reach the ileum, they are absorbed and returned to the liver in the hepatic portal blood. The hepatocytes then excrete the bile salts into newly formed bile. Thus, this precious resource is recycled.

**(Slide 11)** Bilirubin, the main bile pigment, is a waste product produced when the spleen removes old or damaged red blood cells from the circulation. These breakdown products, including proteins, iron, and toxic bilirubin, are transported to the liver via the splenic vein of the hepatic portal system. In the liver, proteins and iron are recycled, whereas bilirubin is excreted in the bile. It accounts for the green color of bile. Bilirubin is eventually transformed by intestinal bacteria into stercobilin, a brown pigment that gives your stool its characteristic color! In some disease states, bile does not enter the intestine, resulting in white (‘acholic’) stool with a high fat content, since virtually no fats are broken down or absorbed.

**(Slide 12)** Hepatocytes work non-stop, but bile production increases when fatty chyme enters the duodenum and stimulates the secretion of the gut hormone secretin. Bile salts inside the lumen are absorbed into the blood from the distal small intestine. This bile salt recycling stimulates the liver to increase bile production. Between meals, bile is produced but conserved. The valve-like hepatopancreatic ampulla closes, allowing bile to divert to the gallbladder, where it is concentrated and stored until the next meal.

**(Slide 13)** The soft, oblong, glandular pancreas lies transversely in the retroperitoneum behind the stomach. Its head is nestled into the “c-shaped” curvature of the duodenum with the body extending to the left about 15.2 cm (6 in) and ending as a tapering tail in the hilum of the spleen. It is a curious mix of exocrine (secreting digestive enzymes) and endocrine (releasing hormones into the blood) functions.

**(Slide 14)** The exocrine part of the pancreas arises as little grape-like cell clusters, each called an acinus (plural = acini), located at the terminal ends of pancreatic ducts. These acinar cells secrete enzyme-rich pancreatic juice into tiny merging ducts that form two dominant ducts. The larger duct fuses with the common bile duct (carrying bile from the liver and gallbladder) just before entering the duodenum via a common opening (the hepatopancreatic ampulla). The smooth muscle sphincter of the hepatopancreatic ampulla controls the release of pancreatic juice and bile into the small intestine. The second and smaller pancreatic duct, the accessory duct (duct of Santorini), runs from the pancreas directly into the duodenum, approximately 1 inch above the hepatopancreatic ampulla. When present, it is a persistent remnant of pancreatic development.

**(Slide 15)** The pancreas produces over a liter of pancreatic juice each day. Unlike bile, it is clear and composed mostly of water along with some salts, sodium bicarbonate, and several digestive enzymes. Sodium bicarbonate is responsible for the slight alkalinity of pancreatic juice (pH 7.1 to 8.2), which serves to buffer the acidic gastric juice in chyme, inactivate pepsin from the stomach, and create an optimal environment for the activity of pH-sensitive digestive enzymes in the small intestine. Pancreatic enzymes are active in the digestion of sugars, proteins, and fats.

**(Slide 16)** The pancreas produces protein-digesting enzymes in their inactive forms. These enzymes are activated in the duodenum. If produced in an active form, they would digest the pancreas (which is exactly what occurs in the disease, pancreatitis). The intestinal brush border enzyme enteropeptidase stimulates the activation of trypsin from trypsinogen of the pancreas, which in turn changes the pancreatic enzymes procarboxypeptidase and chymotrypsinogen into their active forms, carboxypeptidase and chymotrypsin. The enzymes that digest starch (amylase), fat (lipase), and nucleic acids (nuclease) are secreted in their active forms, since they do not attack the pancreas as do the protein-digesting enzymes. Regulation of pancreatic secretion is the job of hormones and the parasympathetic nervous system. The entry of acidic chyme into the duodenum stimulates the release of secretin, which in turn causes the duct cells to release bicarbonate-rich pancreatic juice. The presence of proteins and fats in the duodenum stimulates the secretion of cholecystokinin, which then stimulates the acini to secrete enzyme-rich pancreatic juice and enhances the activity of secretin. Parasympathetic regulation occurs mainly during the cephalic and gastric phases of gastric secretion, when vagal stimulation prompts the secretion of pancreatic juice.

Usually, the pancreas secretes just enough bicarbonate to counterbalance the amount of hydrochloric acid produced in the stomach. Hydrogen ions enter the blood when bicarbonate is secreted by the pancreas. Thus, the acidic blood draining from the pancreas neutralizes the alkaline blood draining from the stomach, maintaining the pH of the venous blood that flows to the liver.

**(Slide 17) Video. All about the small intestine.**

**(Slide 18)** Chyme released from the stomach enters the small intestine, which is the primary digestive organ in the body. Not only is this where most digestion occurs, it is also where practically all absorption occurs. The longest part of the alimentary canal, the small intestine is about 3.05 meters (10 feet) long in a living person (but about twice as long in a cadaver due to the loss of muscle tone). Since this makes it about five times longer than the large intestine, you might wonder why it is called “small.” In fact, its name derives from its relatively smaller diameter of only about 2.54 cm (1 in), compared with 7.62 cm (3 in) for the large intestine. As we’ll see shortly, in addition to its length, the folds and projections of the lining of the small intestine work to give it an enormous surface area, which is approximately 200 m2, more than 100 times the surface area of your skin. This large surface area is necessary for complex processes of digestion and absorption that occur within it.

**(Slide 19)** The wall of the small intestine is composed of the same four layers typically present in the alimentary system. However, three features of the mucosa and submucosa are unique. These features, which increase the absorptive surface area of the small intestine more than 600-fold, include circular folds, villi, and microvilli. These adaptations are most abundant in the proximal two-thirds of the small intestine, where the majority of absorption occurs. Also called a plica circulare, a circular fold is a deep ridge in the mucosa and submucosa. Beginning near the proximal part of the duodenum and ending near the middle of the ileum, these folds facilitate absorption. Their shape causes the chyme to spiral, rather than move in a straight line, through the small intestine. Spiraling slows the movement of chyme and provides the time needed for nutrients to be fully absorbed.

**(Slide 20)** Within the circular folds are small (0.5–1 mm long) hairlike vascularized projections called **villi** (singular = villus) that give the mucosa a furry texture. There are about 20 to 40 villi per square millimeter, increasing the surface area of the epithelium tremendously. The mucosal epithelium, primarily composed of absorptive cells, covers the villi. In addition to muscle and connective tissue to support its structure, each villus contains a capillary bed composed of one arteriole and one venule, as well as a lymphatic capillary called a lacteal. The breakdown products of carbohydrates and proteins (sugars and amino acids) can enter the bloodstream directly, but lipid breakdown products are absorbed by the lacteals and transported to the bloodstream via the lymphatic system.

**(Slide 21)** As their name suggests, **microvilli (singular = microvillus)** are much smaller (1 µm) than villi. They are cylindrical apical surface extensions of the plasma membrane of the mucosa’s epithelial cells, and are supported by microfilaments within those cells. Although their small size makes it difficult to see each microvillus, their combined microscopic appearance suggests a mass of bristles, which is termed the brush border. Fixed to the surface of the microvilli membranes are enzymes that finish digesting carbohydrates and proteins. There are an estimated 200 million microvilli per square millimeter of small intestine, greatly expanding the surface area of the plasma membrane and thus greatly enhancing absorption.

**(Slide 22)** In addition to the three specialized absorptive features just discussed, the mucosa between the villi is dotted with deep crevices that each lead into **a tubular intestinal gland (crypt of Lieberkühn)**, which is formed by cells that line the crevices. These produce intestinal juice, a slightly alkaline (pH 7.4 to 7.8) mixture of water and mucus. Each day, about 0.95 to 1.9 liters (1 to 2 quarts) are secreted in response to the distention of the small intestine or the irritating effects of chyme on the intestinal mucosa. The submucosa of the duodenum is the only site of the complex mucus-secreting duodenal glands (Brunner’s glands), which produce a bicarbonate-rich alkaline mucus that buffers the acidic chyme as it enters from the stomach.

**(Slide 23)** The movement of intestinal smooth muscles includes both segmentation and a form of peristalsis called migrating motility complexes. The kind of peristaltic mixing waves seen in the stomach are not observed here. If you could see into the small intestine when it was going through segmentation, it would look as if the contents were being shoved incrementally back and forth, as the rings of smooth muscle repeatedly contract and then relax. Segmentation in the small intestine does not force chyme through the tract. Instead, it combines the chyme with digestive juices and pushes food particles against the mucosa to be absorbed. The duodenum is where the most rapid segmentation occurs, at a rate of about 12 times per minute. In the ileum, segmentations are only about eight times per minute. When most of the chyme has been absorbed, the small intestinal wall becomes less distended. At this point, the localized segmentation process is replaced by transport movements. The duodenal mucosa secretes the hormone motilin, which initiates peristalsis in the form of a migrating motility complex. These complexes, which begin in the duodenum, force chyme through a short section of the small intestine and then stop. The next contraction begins a little bit farther down than the first, forces chyme a bit farther through the small intestine, then stops. These complexes move slowly down the small intestine, forcing chyme on the way, taking around 90 to 120 minutes to finally reach the end of the ileum. At this point, the process is repeated, starting in the duodenum.

**(Slide 24)** The digestion of proteins and carbohydrates, which partially occurs in the stomach, is completed in the small intestine with the aid of intestinal and pancreatic juices. Lipids arrive in the intestine largely undigested, so much of the focus here is on lipid digestion, which is facilitated by bile and the enzyme pancreatic lipase. Moreover, intestinal juice combines with pancreatic juice to provide a liquid medium that facilitates absorption. The intestine is also where most water is absorbed, via osmosis. The small intestine’s absorptive cells also synthesize digestive enzymes and then place them in the plasma membranes of the microvilli. This distinguishes the small intestine from the stomach; that is, enzymatic digestion occurs not only in the lumen, but also on the luminal surfaces of the mucosal cells.

**(Slide 25)** For optimal chemical digestion, chyme must be delivered from the stomach slowly and in small amounts. This is because chyme from the stomach is typically hypertonic, and if large quantities were forced all at once into the small intestine, the resulting osmotic water loss from the blood into the intestinal lumen would result in potentially life-threatening low blood volume. In addition, continued digestion requires an upward adjustment of the low pH of stomach chyme, along with rigorous mixing of the chyme with bile and pancreatic juices. Both processes take time, so the pumping action of the pylorus must be carefully controlled to prevent the duodenum from being overwhelmed with chyme.

**(Slide 26) Video. All about the large intestine.**

**(Slide 27)** **The large intestine** is the terminal part of the alimentary canal. The primary function of this organ is to finish absorption of nutrients and water, synthesize certain vitamins, form feces, and eliminate feces from the body. The large intestine is subdivided into four main regions: the cecum, the colon, the rectum, and the anus. The ileocecal valve, located at the opening between the ileum and the large intestine, controls the flow of chyme from the small intestine to the large intestine. Upon entering the colon, the food residue first travels up the ascending colon on the right side of the abdomen. At the inferior surface of the liver, the colon bends to form the right colic flexure (hepatic flexure) and becomes the transverse colon. The region defined as hindgut begins with the last third of the transverse colon and continues on. Food residue passing through the transverse colon travels across to the left side of the abdomen, where the colon angles sharply immediately inferior to the spleen, at the left colic flexure (splenic flexure). From there, food residue passes through the descending colon, which runs down the left side of the posterior abdominal wall. After entering the pelvis inferiorly, it becomes the s-shaped sigmoid colon, which extends medially to the midline. The ascending and descending colon, and the rectum (discussed next) are located in the retroperitoneum. The transverse and sigmoid colon are tethered to the posterior abdominal wall by the mesocolon.

**(Slide 28)** Most bacteria that enter the alimentary canal are killed by lysozyme, defensins, HCl, or protein-digesting enzymes. However, trillions of bacteria live within the large intestine and are referred to as the bacterial flora. Most of the more than 700 species of these bacteria are nonpathogenic commensal organisms that cause no harm as long as they stay in the gut lumen. In fact, many facilitate chemical digestion and absorption, and some synthesize certain vitamins, mainly biotin, pantothenic acid, and vitamin K. Some are linked to increased immune response. A refined system prevents these bacteria from crossing the mucosal barrier. First, peptidoglycan, a component of bacterial cell walls, activates the release of chemicals by the mucosa’s epithelial cells, which draft immune cells, especially dendritic cells, into the mucosa. Dendritic cells open the tight junctions between epithelial cells and extend probes into the lumen to evaluate the microbial antigens. The dendritic cells with antigens then travel to neighboring lymphoid follicles in the mucosa where T cells inspect for antigens. This process triggers an IgA-mediated response, if warranted, in the lumen that blocks the commensal organisms from infiltrating the mucosa and setting off a far greater, widespread systematic reaction.

**(Slide 29)** The residue of chyme that enters the large intestine contains few nutrients except water, which is reabsorbed as the residue lingers in the large intestine, typically for 12 to 24 hours. Thus, it may not surprise you that the large intestine can be completely removed without significantly affecting digestive functioning. For example, in severe cases of inflammatory bowel disease, the large intestine can be removed by a procedure known as a colectomy. Often, a new fecal pouch can be crafted from the small intestine and sutured to the anus, but if not, an ileostomy can be created by bringing the distal ileum through the abdominal wall, allowing the watery chyme to be collected in a bag-like adhesive appliance.

**(Slide 30)** Neural and endocrine regulatory mechanisms work to maintain the optimal conditions in the lumen needed for digestion and absorption. These regulatory mechanisms, which stimulate digestive activity through mechanical and chemical activity, are controlled both extrinsically and intrinsically.

**(Slide 31)** Functions of the digestive system, coordination of motility, secretion and absorption are regulated by a complex system of neural and humoral mechanisms.

**(Slide 32)** There are three basic mechanisms of regulation of the digestive apparatus: reflex, humoral and local. These mechanisms have different significance in different parts of the gastrointestinal tract. Central reflex mechanisms (both conditioned and unconditioned) are most evident in the upper parts of the gastrointestinal tract. Further down the digestive tract their participation in regulation of digestion progressively decreases, while the role of humoral mechanisms increases and reaches maximum in the stomach, duodenum, pancreas, in regulation of production and excretion of bile. In the small intestine and especially in the large intestine, most significant are the local regulatory mechanisms (mechanical and chemical stimulations).

**(Slide 33)** Food activates secretion and motility of the gastrointestinal tract directly at the site of its action and downstream and inhibits the activity upstream. Afferent impulses from mechano-, chemo-, osmo- and thermoreceptors located in the wall of the digestive tract travel to the neurons of intra- and extramural ganglia, of the spinal cord and the brain. From there impulses go via the efferent autonomic fibers to the effector cells of digestive organs: glandulocytes, myocytes, enterocytes.

**(Slide 34)** Digestive processes are controlled by sympathetic, parasympathetic and metasympathetic (enteric) nervous systems. Enteric innervation is represented by nervous plexuses, of which the most important for regulation of digestive functions are myenteric plexuses (Auerbach’s plexuses) and submucosal plexuses (Meissner’s plexuses). They participate in realization of local reflexes terminating on the intramural ganglia. Sympathetic preganglionic neurons release acetylcholine, enkephalin, neurotensin; sympathetic postsynaptic neurons release norepinephrine, acetylcholine, vasoactive intestinal polypeptides (VIPs); parasympathetic preganglionic neurons release acetylcholine and enkephalin; postganglionic parasympathetic neurons release acetylcholine, enkephalin, VIPs. Other neurotransmitters in the stomach and intestine are substance P, gastrin, somatostatin, cholecystokinin.

**(Slide 35)** Motility and secretion of the gastrointestinal tract are stimulated by cholinergic neurons and inhibited by adrenergic neurons.

**(Slide 36)** Important for humoral regulation of digestive functions are gastrointestinal hormones. They are peptides and amines produced by endocrine cells of the mucous membrane of the stomach, duodenum, pancreas. By common to all these cells ability to absorb and carboxylate amine precursor, they are combined into APUD system1.

**(Slide 37)** Gastrointestinal hormones regulate the activity of target cells by different ways: by endocrine way (being delivered to the target organs with systemic and regional blood flow), and by paracrine way (being diffused through interstitial tissue to the adjacent and closely located cells). Some of these hormones are produced by nerve cells and serve as neurotransmitters. Gastrointestinal hormones participate in regulation of secretion, motility, absorption, trophism, in release of other regulatory peptides, and produce general effects: changes in metabolism, in the activity of cardiovascular and endocrine systems and in feeding behavior.

**(Slide 38)** In the absence of digestion, gastric glands secrete only mucus and pyloric juice. Secretion of gastric juice is stimulated by sight, odor of food and by entry of food into the mouth. Secretion of gastric juice passes three phases: reflex (cephalic), gastric and intestinal phases.

**(Slide 39)** Reflex (cephalic) phase is based on conditioned and unconditioned reflexes. Conditioned reflex-based secretion of gastric juice is elicited by stimulation of olfactory, visual, auditory receptors (odor, sight of food, sounds associated with cooking, talks about food). Synthesis of afferent visual, auditory and olfactory stimuli in the thalamus, hypothalamus, limbic system and cerebral cortex increases excitability of neurons of the bulbar digestive center. This activates gastric glands, which secrete juice which was called “priming” (initiating) juice by I. Pavlov.

**(Slide 40)** Unconditioned reflex-based gastric secretion starts at the moment of entry of food into the mouth, and is induced by stimulation of receptors of the mouth, pharynx and esophagus. Impulses from these receptors travel through the afferent fibers of glossal (V cranial nerve), glossopharyngeal (IX cranial nerve) and superior laryngeal nerve (X nerve) to the center of gastric secretion in the medulla oblongata. From this center impulses go through efferent fibers of the vagus to gastric glands with the result of increase in secretion (Fig.1). The juice secreted in the first phase, possesses high proteolytic activity and is very important for digestion because it prepares the stomach for receiving food. Secretion of gastric juice is inhibited by stimulation of efferent sympathetic fibers going from the centers of the spinal cord.

**(Slide 41)** Gastric phase of secretion begins at the moment of entry of food into the stomach. This phase is mediated by the vagus nerve, enteric nervous system and humoral factors. Gastric secretion in this phase is induced by stimulation of receptors of the gastric mucosa by food. Impulses proceed via afferent fibers of the vagus to the medulla oblongata and then to secretory cells via efferent branches of the vagus.

**(Slide 42)** The vagus nerve influences gastric secretion in several ways: by direct contact with the principal, parietal and accessory cells of gastric glands (excitation of M-cholinoreceptors by acetylcholine), through the enteric nervous system and through humoral factors since the vagus fibers innervate G-cells of pyloric part of the stomach which produce gastrin. Gastrin increases activity of principal cells, but primarily of parietal cells. At the same time production of gastrin is stimulated by extractive substances of meat, vegetables, products of digestion of proteins, and by bombesin. Decrease in pH in the antral region of the stomach leads to decrease in release of gastrin.

**(Slide 43)** Vagal stimulation increases secretion of histamine by EC2-cells of the stomach. Histamine interacts with H2-histamine receptors of parietal cells to increase secretion of gastric juice of high acidity with low content of pepsin. Secretory activity of gastric mucosa is directly controlled by chemical substances such as meat and vegetable extractions, alcohol and products of cleavage of proteins (albumoses and peptones).

**(Slide 44)** Intestinal phase of secretion starts with emptying of chyme from the stomach into the intestine. The chyme acts on chemo-, osmo-, mechanoreceptors of the intestine and reflexly changes intensity of gastric secretion.

Depending on the extent of hydrolysis of food substances, the stomach receives signals that increase or, on the contrary, inhibit gastric secretion. Secretion is stimulated by local and central reflexes realized through the vagus nerve, enteric nervous system and humoral factors (release of gastrin by G-cells of the duodenum).

**(Slide 45)** This phase is characterized by a prolonged latent period and by long duration. Acidity of gastric juice in this period is low. Gastric secretion is inhibited by release of secretin and CCK-PS, which suppress secretion of HCl, but enhance secretion of pepsinogen. Secretion of HCl is also inhibited by glucagon, gastric inhibitory peptide (GIP), vasoactive intestinal polypeptide (VIP), neurotensin, somatostatin, serotonin, bulbogastron, products of hydrolysis of fats. Duration of secretion, the quantity of gastric juice, its digestive power and acidity strictly depend on the kind of food and are regulated by neural and humoral influences. This dependence was proved by classic experiments conducted in Pavlov’s laboratory on dogs with an isolated small stomach. An animal received bread as carbohydrate food, lean meat as protein food, and milk as mixed food containing proteins, fats and carbohydrates. The highest amount of gastric juice was obtained in digestion of meat, digestion of bread produced moderate secretion of juice, and the lowest amount was secreted in digestion of milk (due to fats contained in it). Duration of secretion was also different: it required 10 hours to digest bread, 8 hours to digest meat, and 6 hours to digest milk. The digestive power of the juice reduced in the following order: meat, bread, milk; acidity: meat, milk, bread. It was also found that high-acidity gastric juice more effectively cleaves animal proteins, and low-acidity gastric juice more effectively cleaves plant proteins. These data are taken into consideration in prescribing diets for patients with hypo- and hypersecretion of gastric glands. Thus, patients with hypersecretion are recommended to use milk products, and those with hyposecretion are recommended to use vegetable and meat products containing many extractive substances.

**(Slide 46)** Lesson assignment:

Lauralee Sherwood. Fundamentals of Human Physiology: On the website of the department

Pages: 437 – 475.

Questions that we will analyze for a lesson on this topic:

1. Digestive functions of the liver.

2. The role of bile in digestion.

3. Digestive functions of the pancreas.

4. Features of digestion in the small intestine.

5. Features of digestion in the large intestine.

6. The role of normal microflora of the large intestine in digestion processes.

7. Nervous and humoral regulation of digestion processes.

Finish for today

The full lecture is at the indicated website.

**Thank you for attention**