PATHOPHYSIOLOGY DIGESTIVE SYSTEM

Vital activity of the organism is possible with the constant intake of nutritional substances: proteins, fats, carbohydrates and, in addition, water, mineral salts and vitamins. At the same time, water, mineral salts and vitamins are absorbed in an unchanged form, in which they enter the food composition. Proteins, fats and carbohydrates undergo physical and chemical transformations in the gastrointestinal tract, after which the products of their metabolism are absorbed from the digestive tract and enter the blood and lymph. Digestion is the transformation of food products into compounds that lack species specificity, their absorption and participation in interchange.

The functions of the digestive tract include: secretory - the production of enzymes, hydrochloric acid, bile, etc., which provide digestion due to physical and chemical effects and enzymatic food processing; Motor and evacuation - mechanical processing of food due to grinding, mixing and movement along the gastrointestinal tract; Suction - active penetration of the final products of digestion, water, salts, vitamins, minerals through the mucosa of the gastrointestinal tract into the blood and lymph; Excretory (excretory) - first, it is one of the extrarenal ways of excretion of metabolites from the bloodstream to provide homeostasis (for example, uremia urea is excreted through the mucosa of the digestive tract, causing the formation of symptomatic ulcers); second, the excretory function ensures the participation of the digestive system in Interorganic metabolism of nutrients (nutrients are not food, but nutrients, in a day in the norm in the gastrointestinal tract is released up to 80 g of protein and 20 g of fat, which together with exogenous proteins and fat mi digested, absorbed and used by the body); Endocrine - the synthesis of their own hormones (cholecystokinin, secretin, enterogastron, etc.), as well as the function of the digestive tract, which is closely related to the blood system - the synthesis of the internal factor of Castle, with a lack of which develops B12-deficiency anemia.

Insufficiency of digestion - the state of the gastrointestinal tract, which does not provide sufficient assimilation of food coming into the body. As a result, the body develops a negative nitrogen balance, hypoproteinemia, hypovitaminosis, the phenomenon of incomplete starvation, exhaustion of the body, a violation of reactivity. Insufficiency of digestion can develop if the entire digestive tract or its departments are disrupted. Another IP Pavlov noted the remarkable coherence and regularity in the work of the digestive glands. This interdependence stands out particularly vividly in conditions of pathology, when the disruption of the activity of any part of the digestive tract causes, naturally, the breakdown of the functions of its other departments.

The main causes of digestive disorders

For digestive disorders can lead to:

1) congenital digestive tract;
2) inaccuracies in nutrition (poor quality, rough food, dry eating, unbalanced nutrition with protein deficiency, vitamins, trace elements, intake of excessively hot or cold food, etc.);
3) pathogens of certain infections (typhoid fever, dysentery, food poisoning, etc.);
4) entry into the gastrointestinal tract of poisons (salts of heavy metals, poisons of plant origin, etc.);
5) the effect of ionizing radiation;
6) tumors;
7) postoperative conditions;
8) psychotrauma, negative emotions, physical overstrain;
9) drug addiction, alcoholism, smoking.

Pathology of the gastrointestinal tract can develop as a result of direct or mediated damaging effects of etiological factors.

An example of a direct damaging effect of the etiologic factor may be the development of esophagitis and necrotic changes in the esophagus with the subsequent formation of stricture (cicatrical narrowing) of this organ after taking acetic essence.

The mediated damaging effect of the etiological factors is realized through disorders of the neurohumoral regulation of the digestive organs. At the same time, the damage can initially be

formed either in the nervous system or in some other organ of the gastrointestinal tract. If in the beginning there are disorders in the nervous system under the influence of poisons, chemicals, stressors, etc., it can lead to pathological impulses from the central nervous system to the periphery, which contributes to the disruption of the digestive system, for example, the development of a gastric ulcer after a head injury or contusion . If in the beginning there is damage to any organ of the gastrointestinal tract, this results in abundant afferent impulses from the diseased organ in the central nervous system. In the central nervous system a persistent pathological dominant is formed, pathological response impulse from the central nervous system to the periphery arises, which leads to a disruption of the function of other digestive organs. Disorder of the function of one part of the gastrointestinal tract causes disturbances in other departments by the mechanism of the viscero-visceral reflex. For example, if the stomach ulcer develops reactive pancreatitis or reactive hepatitis.

The main pathogenetic factors of insufficiency of digestion

The main pathogenetic factors in the development of digestive failure include:

1. Disorder of appetite.
2. Disruption of food processing in the oral cavity and its passage through the esophagus.
3. Disturbance of digestion in the stomach.
4. Disturbance of digestion in the intestine.

Disorders of appetite

The sensations of hunger and satiety are determined by the activity of the food center, which represents the functional union of several nerve formations at various levels of the central nervous system. It is a complex hypothalamic-limbic-reticulocortical complex. An important role in this complex is assigned to the hypothalamus. Normally, the process of food intake is regulated by two hypothalamic centers: ventrolateral - "hunger center" and ventromedial - "saturation center", which are in reciprocal relations. Leading department - the center of hunger, from him goes the excitation of the entire food center. There are several theories of the emergence of hunger.

It is considered, in particular, that the excitation of neurons of the center of hunger can arise as a result of a decrease in blood glucose (glucostatic theory) or in the action of metabolites of the Krebs cycle (metabolic). The feeling of hunger also comes with a decrease in the amino acid content (aminoacidostatic theory) and the level of fatty acids and triacylglycerols in the blood (lipostatic), and also with a decrease in body temperature (thermostatic) and as a result of impulse from the mechanoreceptors of the stomach with its "hungry" contractions (local theory) .

Peptide hormones play an important role in regulating food intake, creating hunger and feeling saturated. Strengthening of food motivation and activation of food behavior cause an excess of insulin, pentagastrin, oxytocin, activation of the parasympathetic nervous system. When the saturation center is activated, on the contrary there is a restraining impulse to the center of hunger, and the activity of the latter falls. Excitation of neurons of the saturation center is caused by glucose, leptin, cholecystokinin, pancreatic glucagon, somatostatin, sympathetic nervous system activation, and serotonin. The level of cerebral serotonin especially increases after eating foods rich in carbohydrates and proteins, when the passage through the blood-brain barrier of the precursor of serotonin - the amino acid tryptophan - increases. It is known that many depressions are caused by a decrease in the level of cerebral serotonin, and then hyperphagia develops in depression.

There are the following disorders of appetite: pathological exacerbation - hyperrexia (from Greek hyper - over, excessive, orexis - appetite), pathological decline right up to anorexia (from Greek an - denial) and aversion to food.

Pathological appetite enhancement is often combined with increased food intake - polyphagia (from the Greek poly - a lot, phagein - eat). With a sharp increase in appetite, they speak of bulimia (bus - bull, linos - hunger, a synonym - wolf hunger). In the experiment, hyperrexia is caused by destruction of the ventromedial nuclei of the hypothalamus or chemical damage by their aurotic glucose ($C_6HnAuSO_5$) administered parenterally. Pathological increase in appetite can be observed in a number of diseases of the central nervous system (dementia, neurosis, tumors of the posterior

cranial fossa) and endocrine glands (diabetes mellitus, pancreatic tumors producing insulin, insulomas, thyrotoxicosis).

A pathological decrease in appetite right up to anorexia can be reproduced in an experiment, destroying the ventrolateral nuclei of the hypothalamus. At the same time, the feeling of hunger disappears, and the animals refuse to eat until aphagia - a complete cessation of food intake.

There are the following types of anorexia:

1. Dynamic anorexia. It is one of the symptoms in diseases of the gastrointestinal tract and hepatobiliary system. It can be associated with a violation of the functions of the receptors of the digestive tract, and also have a conditioned reflex character, i.e. Cause pain, discomfort. This kind of dyspepsia can be a symptom of a number of stomach diseases (for example, stomach cancer) and intestines. In the latter case, it must be clearly differentiated from sitophobia, or Emcomfft's disease when the appetite is preserved, but eating can be reduced. Sitophobia develops, for example, in Crohn's disease (regional ileitis), especially in cases of partial intestinal obstruction, or in patients with gastric ulcer after partial or total gastrectomy. Anorexia may also precede jaundice syndrome with hepatitis.

2. Intoxication anorexia. It is noted for a number of intoxication poisonings and due to severe long-term diseases (tumors, infections). It is based on a decrease in the excitability of the food center. It is an important symptom in patients with chronic renal insufficiency, and is noted with intoxications with drugs, in particular cardiac glycosides, hypnotics, narcotic drugs.

3. Neurotic anorexia. The reason for its development are negative emotions, stressful situations, a strong brain stimulation.

4. Neuropsychiatric anorexia. It manifests itself in psychogenic disorders, in particular, with residual-organic lesions of the central nervous system. In these cases, anorexia often accompanies a depression syndrome and can be a manifestation of a consciously severe restriction of food intake with an obsessive idea of excessive completeness.

5. Neurodynamic anorexia. It develops as a result of reciprocal inhibition of the food center during vomiting, pain syndromes (hepatic, renal, intestinal colic, myocardial infarction, etc.).

In some cases, anorexia is difficult to attribute to one of the listed species, it often has a mixed character. For example, severe anorexia in severe chronic heart failure and pulmonary insufficiency is explained not only by the lack of oxygen in the organs of the gastrointestinal tract and the hepatobiliary system, but also by severe metabolic disorders, often the presence of drug intoxication.

Disorders of food processing in the oral cavity and its passage through the esophagus

Impaired chewing

The pathology of digestion can be caused by violations of its initial phase - chewing. Chewing is a mechanical process of grinding food in the mouth, performed by the temporomandibular joints, as well as teeth, the presence of which determines the area of the chewing surface. The most common cause of masturbation is tooth disease - caries and periodontitis. Caries is a bacterial-induced progressive deterioration of the mineral and organic components of the outer enamel and the underlying dentin and the main cause of tooth loss. The progressing course of caries is complicated by inflammation of the pulp and periodontal disease.

Parodontosis is a serious disease of the oral cavity, in which there are dystrophic changes in the periodontal, which leads to loosening and loss of teeth. The pathogenesis of caries and periodontitis is not completely clear. They play a role of disturbance in metabolism, especially protein, hypovitaminosis, nutrition imbalance, digestive disorders, absorption and other factors.

Disturbance of chewing is associated with a decrease in the number of teeth that experience functional overload. This leads to deformation of the dentition and bite, which further aggravates the chewing disorder. Chewing food is disturbed by anomalies of bite, injuries, gunshot wounds to the lower part of the face, when there are fractures of the jaw bones, dislocations and fractures of the teeth.

The violation of chewing arises in the pathology of the chewing musculature. Its function suffers with infections, innervation disorders, injuries, gunshot wounds. So, with tetanus, meningitis, there is a tonic spasm (trismus) of the chewing musculature. With trigeminal neuritis, there is a sharp pain when chewing (which can cause erroneous removal of healthy teeth), in a number of cases, peripheral paralysis of the masticatory muscles develops.

The chewing process is affected by disturbances in the temporomandibular joints, which occur, for example, in rheumatoid arthritis.

Inflammatory processes in the oral cavity - pulpitis, stomatitis, gingivitis disrupt the process of chewing, are the focus of infection, can cause sensitization of the body and allergic diseases of internal organs.

If there is a violation of the chewing food, there are changes in the activity of the stomach: his motor skills suffer, since poorly chewed food is slower to digest and lasts longer, causing changes in the mucosa. This is facilitated by a decrease in the reflex compartment of gastric and pancreatic juices. Rough, poorly crushed food injures the mucous membrane of the digestive tract, especially the esophagus and stomach, causing damage to the superficial epithelium.

Disturbance of salivation

Saliva secretion is important for the act of swallowing, as well as for wetting and forming a food lump. This is due to the content in the saliva of mucins (glycoproteins of saliva) enveloping the food lump. Saliva, except mucin, contains amylase (ptyalin), involved in the digestion of carbohydrates, lysozyme. Its secretion is also necessary for cleansing the oral cavity, which prevents the accumulation of bacteria. The bicarbonate buffer contained in it maintains a pH value of about 7 in the mouth. Saliva serves as a solvent for nutrients.

The increase in salivation (hypersalivation) occurs as a result of direct or reflex stimulation of the center of salivation in the medulla oblongata or the secretory nerves of the salivary glands. The strongest stimulants of salivation are taste sensations. When hypersalivation in an adult person for a day can stand up to 8-14 liters of saliva, which entails dehydration and loss of bicarbonates and potassium, which are in large quantities found in saliva. Hypersalivation is possible with the defeat of the central nervous system, inflammatory processes in the oral cavity, diseases of the esophagus (reflux-esophagitis), helminthiases, toxicosis of pregnant women, action of certain drugs (pilocarpine, physostigmine).

Salivation usually decreases at night. The volume of saliva released per day is 1000 ml or more, and about 90% of it is produced by the parotid (secretion of serous secretions with a small amount of organic components) and the submandibular glands (secretion of the mixed secretion - serous and mucous components). The composition of saliva is affected by the rate of its secretion and the action of hormones (estrogens, androgens, glucocorticoids, peptide hormones). When a large amount of saliva is ingested, the gastric juice is neutralized and digestion is disturbed in the stomach. A prolonged loss of saliva causes metabolic disorders, an acid-base balance, an exhaustion of the body. Usually, when hypersalivation of saliva is not completely swallowed. It flows outward, causing maceration and inflammation of the mucous lips and facial skin. It is possible that saliva enters the respiratory tract and infection with microbes in the oral cavity.

Reduction of salivation (hyposalivation) can occur in pathological processes in the tissues of salivary glands (siladenite, tumors). Inflammation of the salivary glands (siladenite) is usually associated with the presence in the duct of one of them salivary stone (sialolithiasis). Salivary stones have a mechanical impediment to the current of saliva and increase the pressure in the salivary ducts. At the same time, the current of saliva is disturbed, pain and swelling of the gland occur during meals; The parenchyma of the gland can atrophy. Hyposalivation noted when the central inhibition of the secretion of the salivary glands, which occurs with stress, pain. It is observed under the influence of a number of medications of anticholinergic action (atropine, metacin, scopolamine), some antidepressants. Salivation decreases with fever, a number of endocrine diseases (thyrotoxicosis, diabetes mellitus), lesions of the nervous system (damage to the base of the brain, spinal cord, etc.), exposure to ionizing radiation (due to radiotherapy of head and

neck tumors), dehydration. When the saliva secretion decreases or stops, xerostomia develops - dryness in the oral cavity. There is a violation of chewing food and swallowing it. Xerostomia is caused by dysfunction of the salivary glands and can be temporary or permanent. Factors that cause temporary xerostomia are emotional stress, certain medications, such as atropine, antihistamines, tricyclic antidepressants and phenothiazines. The development of persistent xerostomia occurs when the oral cavity is irradiated, which is associated with the atrophy of the salivary glands.

Hyposalivation and xerostomia are symptoms of Sjogren's disease - a systemic autoimmune disease, in which the secretion of the glands of the digestive tract, salivary glands is sharply reduced, the dryness of the synovial membranes (pleura, pericardium) is noted.

When hyposalization in the oral cavity - on the tongue and gums formed a plaque from the lowered epithelium, which serves as a nutrient medium for microflora. Normally, 1-10 ml of saliva contains 108-109 bacteria: streptococci, diplococci, spirochaetes, lactobacilli, actinomycetes, fungi of the genus Candida, often the Herpes simplex virus etc. In hyposalivation and xerostomia, the microflora of the oral cavity strengthens its growth and inflammatory processes of the oral cavity, Worsening digestion and serving as a hotbed of infection for possible septic complications.

Disturbance of swallowing

Swallowing is a complex reflex act having three phases: oral (arbitrary), pharyngeal (involuntary fast), and esophageal (involuntary slow).

Dysphagia (violation of swallowing) is defined as a feeling of "getting stuck" or obstructing the passage of food through the mouth, throat or esophagus.

Normal transport of the food lump through the swallowing canal, formed by the pharynx and esophagus, depends on the size of the food lump, the diameter of the lumen of the canal, its peristaltic contraction, the state of the swallowing center.

Dysphagia caused by too large a size of the food lump or narrowing the lumen of the swallowing canal is called mechanical dysphagia. Dysphagia associated with uncoordinated or weak peristaltic contractions of the canal walls, as well as with disturbance of the swallowing center, is called motor dysphagia.

Mechanical dysphagia can be caused by internal or external compression of the lumen of the swallowing canal. The esophagus of a healthy person due to the elasticity of its wall has the ability to stretch up to 4 cm in diameter. If this ability is limited to 2.5 cm, dysphagia is possible, and with restriction to 1.3 cm, dysphagia will always develop. The causes of the development of mechanical dysphagia are numerous. They can be associated, first of all, with a change in the lumen of the canal (with too large a size of a food lump or foreign body entry). Possible internal narrowing of the channel due to the inflammatory process (stomatitis, pharyngitis, epiglottitis, esophagitis), benign strictures (peptic - under the influence of alkalis, acids or drugs, inflammatory - Crohn's disease, candidiasis, ischemic, postoperative, radiation, congenital), malignant Primary cancer, metastases) or benign (angioma, papilloma, polyp) tumors. External compression of the swallowing can be associated with cervical spondylitis, osteophytes of the spine, retropharyngeal abscess, mediastinal abscess, thyroid gland enlargement, aortic aneurysm, mediastinal tumors, pancreas, hematoma (after vagotomy), etc.

Motor dysphagia occurs due to a violation of the initiation of the swallowing reflex, damage to the skeletal muscles of the pharynx and esophagus or smooth muscles of the esophagus. So, many diseases of the central nervous system lead to dysphagia. The most severe disorders of swallowing are noted when the brainstem is damaged, where the nerve structures responsible for the innervation of the pharyngeal part of the passage of the food lump are located. At the same time, there are serious violations of the initial phase of swallowing, often difficult to reverse. In cerebrovascular diseases, aspiration pneumonia, dehydration of the body, loss of mass can become the results of dysphagia. The mechanisms of swallowing are disturbed in such diseases of the central nervous system as poliomyelitis and Parkinson's disease, which are characterized by dysarthria, dysphagia due to weakness of the pharynx and tongue muscles, amyotrophic lateral sclerosis with possible aspiration during or after swallowing. The cause of dysphagia can also be

dystrophy of the muscles of the tongue and pharynx, which is accompanied by nasopharyngeal regurgitation and nasal sound. Possible choke, aspiration of food because of the weakness of the muscles that raise the pharynx. Patients usually switch to slow reception of finely ground food, and with progression of swallowing disorders, feeding is done through the nasal probe. Similar conditions develop, for example, in dermatomyositis with disruption of the functions of the superior esophageal sphincter and the proximal striated musculature of the esophagus. With a number of diseases - tetanus, rabies, hysteria - there is a spasm of the swallowing musculature. With these diseases, phagophobia can develop - fear of swallowing and therefore rejection of it, which may be due to fear of aspiration. To the refusal of swallowing can lead and painful swallowing, for example, in the inflammatory process. Globus histericus is a sensation of a "coma" in the throat. Some patients feel the passage of food through the esophagus, which may be due to psychosomatic disorders.

Swallowing disorder is noted with botulism, which is caused by a violation of the transfer of impulses from the nerves to the muscles involved in the swallowing act. In swallowing disorders, the swallowing of water is more difficult, since it requires the maximum closure of the holes leading to the nose and the trachea, which is possible with an intensive reduction of the swallowing musculature. The ingestion of water is severely impaired in rabies, which has led to a judgment about "rabies" in this disease.

The reasons for the development of motor (neuromuscular) dysphagia is the damage to the smooth muscles of the esophagus. This is noted with achalasia of the esophagus, a number of collagen diseases, especially with scleroderma and metabolic neuromyopathy associated with alcohol intake, diabetes mellitus.

Disturbance of the motor function of the esophagus

The motor function of the esophagus may be decreased (hypokinesis, or atony) or elevated (hyperkinesis). Atony is reproduced in the experiment by high cutting of n.vagus (IP Pavlov), which causes a decrease in the peristalsis of the esophagus and, in connection with this, a delay in the progression of the food lump. The difficulty of moving food through the esophagus can occur due to its spasmodic contraction. Experimentally, spasm of the cardial part of the esophagus can be obtained by stimulation of the sympathetic nerve.

The main motor disorders affecting the body of the esophagus are observed in achalasia, gastroesophageal reflux disease, diffuse spasm of the esophagus and scleroderma.

With achalasia (lack of normal patency of the cardiac esophagus), coordination of peristalsis is impaired due to loss of inhibitory nervous regulation of the smooth muscles of the esophagus body and lower esophageal sphincter. The latter can not relax sufficiently when swallowed. In this regard, food is delayed in the esophagus, and it stretches (mesophagy). The main causes of achalasia of the esophagus may be a primary neurological disorder with damage to the brainstem, a vagus nerve, degenerative changes in the intramural nerve plexuses: Meissnerian and Auerbachian, as well as smooth muscles of the esophagus. The main mechanism of these disorders is the deficiency of the neurotransmitter necessary for muscle relaxation. Most likely they are a vasoactive intestinal polypeptide (VIP), which is confirmed by a decrease in its level in patients with cardiac achalasia in comparison with healthy patients. Under the influence of VIP, nitric oxide is released from the neurons of the esophagus, which is a vasodilator and simultaneously a relaxant of smooth muscles. It has been established that, with achalasia, the level of nitric oxide in the distal smooth muscles of the esophagus decreases.

Quite early signs of the disease are feelings of filling and squeezing in the chest. With the progression of the disease, the difficulty in swallowing food increases, associated with a feeling of esophagus overflow, it is possible to throw (reflux, regurgitate) food into the mouth, indicating that it is delayed in the enlarged esophagus. These disorders increase with emotional stress and fast food. Delayed food in the esophagus causes aversion to it and leads to a decrease in the patient's weight. Regurgitation of food increases with eating. Often in the mouth gets undigested food, eaten a few hours ago, it is possible to aspirate food in the respiratory tract (which can cause death or

cause aspiration pneumonia). Clinical manifestations of achalasia, in addition to dysphagia and regurgitation, may be pain in the chest, which is explained by the high contractile activity of the esophagus, as well as the inflammatory process in its mucosa due to stagnation of food. Secondary heartburn occurs, associated not with gastroesophageal reflux, but with enzymatic digestion of food in the esophagus itself and the formation of a large amount of lactic acid.

Another type of pathology - excessive relaxation of the lower esophageal sphincter (gastroesophageal sphincter), which contributes to gastroesophageal reflux. Decrease in pressure in the lower sphincter of the spine or an increase in the number of episodes of its spontaneous relaxation can be associated with a primary defect in the smooth muscles of the sphincter, including a conditioned violation of nervous regulation (with a decrease in the function of the vagus nerve) or a decrease in gastrin production regulating the function of this sphincter. In children of the first year of life, this sphincter is not developed enough, and therefore, after feeding, it is easy to regurgitate food, which increases with overfeeding. Insufficiency of the lower food sphincter, which contributes to gastroesophageal reflux, can be noted in systemic scleroderma, pregnancy, and also in smokers. Often underestimate the effect of a number of drugs that can reduce the tone of smooth muscles, including the lower food sphincter, and delay the emptying of the stomach, thus contributing to the appearance of gastroesophageal reflux. This can be the case with the use of nitrates, M-cholinolytics, tricyclic antidepressants, progesterone, prostaglandins, calcium antagonists, sedatives, euphyllin, β-adrenoblockers, narcotics. Reduces the tone of the sphincter and a number of foods, such as alcohol, chocolate, mint, fried and fatty foods, flour. Some drinks with a low pH increase the symptoms of reflux with esophagitis. Such beverages include Coca-Cola, Pepsi-Cola (pH 2.5), Red Wine (pH 3.25), Orange Juice (pH 3.5).

As a result of casting and prolonged exposure of gastric contents in the esophagus, inflammatory and degenerative changes occur, gastroesophageal reflux disease (reflux esophagitis) develops. In the development of this disease, an important role, in addition to reducing the tone of the lower sphincter of the spine, is the violation of esophageal peristalsis, the mechanisms of cleansing the esophagus from hydrochloric acid (esophageal clearance): chemical purification by reducing the neutralizing effect of saliva, bicarbonates of esophageal mucus; And volumetric purification - because of the inhibition of secondary peristalsis and a decrease in the tone of the wall of the thoracic esophagus. A certain value is attached to the damaging properties of the refluxant (hydrochloric acid, pepsin, bile acids), a decrease in the resistance of the mucosa of the esophagus to acido-peptic damage, an increase in the volume of gastric contents due to hypersecretion, food retention in the stomach, increased intra-abdominal pressure and a predisposition to sphincter failure.

The normal pH value in the esophagus (5.5-7.0) decreases in the case of reflux to below 4.0. Pathological reflux is indicated in the case when the number of episodes per day is more than 50 Thyles when the reflux duration exceeds 5 minutes, and the total duration of the period during which the intraepisitic pH drops below 4.0 is greater than 1 hour per day. When the degree of reflux-esophagitis is expressed, there are merging erosions, covered with exudate or rejecting necrotic masses, which are located in the distal esophagus. Erosive-ulcerative reflux esophagitis is complicated by strictures; Possibly replacing the flat nonkeratinized epithelium of the esophageal mucosa with a cylindrical epithelium (Barrett's esophagus). The presence of Barrett's esophagus leads to the development of the most formidable complication of reflux esophagitis-adenocarcinoma.

Diffuse spasm of the esophagus, as well as achalasia, is associated with the loss of inhibitory control over the smooth musculature of the esophagus during the normal functioning of the lower sphincter of the sphincter. There are indiscriminate intensive contractions of all parts of the esophagus, which can cause chest pain. These pains resemble angina and are also removed with nitrates, which reduce the tone of the esophagus. As a secondary complication, dysphagia is observed.

The cause of motor dysphagia can be systemic scleroderma with esophageal involvement. This disease refers to collagenosis. Along with the damage to the skin and muscles, internal organs

change. Most often among the digestive organs is affected by the esophagus. There is a progressive replacement of the smooth musculature of the esophagus and the lower food sphincter with a dense fibrous tissue, which leads to a loss of peristalsis of the esophagus and a decrease in the pressure of the lower food sphincter. Dysphagia and acidic gastroesophageal reflux (heartburn, sour belch, regurgitation) occur. Barrett's esophagus is possible.

The violation of the passage of food through the esophagus occurs when it forms diverticula - protrusion of the wall. In the diverticulum, food masses can stagnate and decay. Perhaps the thinning of the wall of the diverticulum followed by rupture, bleeding and infection of the mediastinum.

Hernias of the esophageal opening of the diaphragm - in 10% of cases it is a constant, circular diaphragmatic hernia, more often (in 90% of cases) there is a fickle, sliding hernia that appears in the case of increased peristalsis. The causes of the formation of hernias are: a sharp increase in intra-abdominal pressure with increased physical exertion and an inborn underdevelopment of connective tissue structures. With the development of hernia of the esophageal opening, reflux-esophagitis develops, it is possible to infringe the hernia with the occurrence of esophageal-gastric bleeding.

Hypertrophy of cardia is a hereditary disease characterized by an increase in the mass of the circular muscles of the lower part of the esophagus and a simultaneous increase in their tone. As a result, the speed of food movement slows down, the esophagus stretches, there is a feeling of discomfort and retrosternal pain.

Varicose veins inside the esophageal wall occur with portal hypertension. In the case of a sharp stretching of the esophagus during vomiting, these thin vessels can be ruptured, which in 40% of cases leads to a lethal outcome.

Digestive disorders in the stomach

Disorders of digestion in the stomach are associated with the disorder of its functions: secretory, reservoir, evacuation, motor, absorption, excretory, etc.

Violation of the secretory function of the stomach

Violation of the secretory function of the stomach includes changes in the amount of gastric juice, acidity, the formation of pepsin and mucus. Hydrochloric acid and pepsin are necessary for chemical processing of food. The main stimulator of the formation of hydrochloric acid is gastrin, produced G-cells of the gastrointestinal tract. Gastrin stimulates the release of HC1 and gastric enzymes, increases the blood circulation of the stomach (it is a trophic hormone), increases the motility of the antral stomach, but inhibits the emptying of the stomach, stimulates the release of insulin. The secretion of gastrin is increased: vagus irritation, protein intake, excess Ca ions, intake of caffeine, ethanol. The secretion of gastrin is reduced: hypersecretion of HC1, the action of somatostatin, secretin, glucagon.

The main stimulants of the secretion of hydrochloric acid in the stomach, in addition to gastrin, are histamine and acetylcholine. In response to n.vagus irritation, the concentration of gastrin produced by the G cells of the antrum of the stomach increases, which leads to an increase in the secretion of hydrochloric acid (a synthetic analogue of gastrin - pentagastrin is used as a stimulant for HC1 secretion). Gastrin and acetylcholine activate specific receptors associated with the calcium / protein kinase C system. After activation of the appropriate mechanisms, hydrogen-potassium (H + / K +) ATPase channels are stimulated, leading to production and release of hydrogen ions.

The stomach emits up to 2 liters of fluid per day. Quantitative changes in the secretion of gastric juice are expressed in an increase (hypersecretion) and a decrease (hyposecretion). This can be combined with changes in the production of hydrochloric acid by parietal cells and pepsinogen - the main cells located in the tubular glands mainly of the bottom and body of the stomach. The production of hydrochloric acid may increase (hyperchlorhydria) or decrease (hypochlorhydria).

Combinations of hypersecretion with hyperchlorhydria and hypoxecretion with hypo - and achlorhydria are possible.

Methods for the study of secretory function of the stomach include the method of fractional gastric sensing and PH-metry. The method of fractional gastric sounding is to obtain gastric juice through a probe injected into the stomach on an empty stomach 12 hours after ingestion. In this case, the following is obtained: a "rush" portion - the contents are sucked from the stomach 5 minutes after the introduction of the probe, "basal" secretion - 4 servings every 15 minutes for one hour, "stimulated secretion" - 4 servings every 15 minutes for one Hours after stimulation by the stimulus. Submaximal stimulation with histamine (0.01%, 0.1 ml / 10 kg of body weight) and maximum stimulation with pentagastrin (6 μg / kg of mass, synthetic preparation of gastrin) or histalkon (2 μg / kg) are used. In each serving of juice determine: volume, acidity (free, associated with proteins and total HC1), debit-HC1 - absolute acid production per hour, the content of pepsin (according to the ability of gastric juice to digest proteins), the stratification factor (by the ratio of liquid and dense Layers).

Normal indices of gastric juice

Indicator	On an empty stomach	Basal secretion	Stimulated secretion
Volume of gastric juice, ml	5-40		
Hourly voltage, ml		50-100	100-140
General HC1, titer. units	20-30	40-50	60-100
Free HO, title. units	0-15	20-40	65-85
The debit hour is HC1, meq / mmol / hr	Not determined	1,5-5,5 55-100	8-14 300-500
Debit hour of pepsin, mg / h	Not determined	10-40	50-90
Stratification coefficient, liquid / thick	Not determined	1:1-1:2	1:1-1:2

It should be noted that the term "free HC1" is conditional (hydrogen ions are bound by protein molecules and bicarbonate ions, so it is possible to separate hydrogen ions into bound and free ions). In this regard, the lack of free hydrochloric acid does not speak of achlorhydria, but only ascertains a decrease in the concentration of hydrogen ions to a pH of 3.5 or lower. To judge the increased or decreased acid-forming function of the stomach follows the definition of the absolute production (production rate) of hydrochloric acid (mmol / h), which takes into account the amount of secretion of gastric juice in milliliters (ml) and the concentration of total hydrochloric acid in millimoles (mmol) in each portion of the gastric Juice in the phases of basal and stimulated secretion.

The violation of acid formation is judged taking into account age, sex (in women acidity is lower by 20%) and the weight of the patient (with increasing mass, acidity increases). Considering the great variability in the parameters of the secretory function of the stomach, it is said to be disturbed only if there are gross deviations from the normal secretion parameters, taking into account the errors in the method for determining this function. The disadvantages of the method include: the need for continuous gastric juice sucking, in vitro research, the determination of mainly only the general HC1. In addition, when evaluating this method, the production rate of hydrochloric acid depends on what and what titrates (there may be impurities of mucus, bile, which has an alkaline pH).

A more accurate method for assessing the acid-forming function of the stomach is the pH-metry - the near-wall pH determination. It is carried out with the help of special instruments with pH probes, which are injected transnazalno (through the nose). It is possible to conduct a 3-hour pH-meter and 24-hour pH monitoring. The advantages of this method include: the ability to conduct pH-metry in each department of the stomach, functional stimulation and depressant tests and select

appropriate drug therapy, determine the true achlorhydria, which is of great importance in the diagnosis of precancerous state with atrophic gastritis.

Digestion in hypersecretion and hyperchlorhydria. When hypersecretion and hyperchlorhydria fasting is observed the presence of acidic gastric juice more than 50 ml with a concentration of hydrochloric acid up to 40 mmol.

Stimulation of the secretory function of the stomach is carried out with the participation of the vagus nerve through gastrin, histamine, glucocorticoids, insulin, thyroxine, etc. In addition, some drugs, acute and hot foods, specific food components such as peptides, amino acids, caffeine, alcohol, calcium, Which stimulate the production of gastrin, can stimulate gastric secretion.

In the reflex phase, the secretion of gastric juice increases from the species, smell and taste of food, which occurs through the influence of vagus. In the gastric phase of secretion there is a mechanical stretching of the stomach by the food, perceived by the stretch receptors in the wall of the stomach, which is realized through the reflex arcs, including the vagus nerve.

Hyperchlorhydria in the stomach is noted in the Zollinger-Ellison syndrome (gastrinoma) caused by a gastrin-forming tumor located in the pancreas (65-75%) or in other organs (stomach, duodenum, liver, testicles, mesentery, lymph nodes, Fatty tissue of the abdominal cavity). Usually it is a multiple tumor. The isolation of gastrin by tumor cells causes persistent gastric hypersecretion, which is associated with the main manifestations of the disease: ulceration, digestive disorders and diarrhea. More than 90% of patients with gastrinomas develop ulcers of the upper gastrointestinal tract. Ulcers are resistant to standard therapy, have a continuously-recurrent course, are prone to severe complications: perforation, penetration and bleeding. Even a surgical operation for complications of ulcers does not stop the recurrence of the disease. In addition to peptic ulcers, the common manifestations of the syndrome Zollinger-Ellison are diarrhea and maldigestia syndrome (violation of the cavity digestion). The pathogenesis of diarrhea in Zollinger-Ellison syndrome is complex and is mainly associated with hypersecretion of gastric juice, reaching several liters per day. In addition, the cause of diarrhea can be inactivation of pancreatic enzymes with gastric acid juice of increased acidity, which leads to steatorrhea and to maldigestia syndrome. Due to a decrease in pH in the small intestine, the mucosa is damaged with the development of malabsorption syndrome (impaired absorption). Perhaps the development of "secretory" diarrhea, as hypergastrinemia increases the secretion of potassium and reduces the absorption of sodium and water in the small intestine.

In addition to the stimulating effect on the secretion of acid, gastrin has a pronounced trophic effect on the tissues of the gastrointestinal tract. It enhances the synthesis of DNA and proteins in the cells of the gastric mucosa and in other tissues. Hypergastrinemia in the syndrome of Zollinger-Ellison causes two synergistic effects: hyperstimulation of parietal cells of the stomach and, as a consequence, a significant increase in the secretion of acid and the number of secreting parietal cells.

The main differential-diagnostic sign of this syndrome is hypergastrinemia. The average level of gastrin in healthy and peptic ulcer patients is less than 150 ng / ml, while the level of gastrin in patients with Zollinger-Ellison syndrome is much higher -> 1000 ng / ml. At the same time, it is necessary to know that hypergastrinemia can be not only primary, contributing to a rise in the level of hydrochloric acid (as is the case with the Zollinger-Ellison syndrome), but also secondary due to hypo- and achlorhydria. The most common cause of hypergastrinemia is atrophy of the underlying gastric mucosa, as hydrochloric acid is the main inhibitor of gastrin release. The absence of hydrochloric acid leads to ineffable secretion of gastrin, hyperplasia of cells of the antrum of the stomach, which often occurs with pernicious anemia. In connection with this, the determination of gastric juice acid plays an important role in the differential diagnosis of the syndrome of hypergastrinemia.

Early detection and removal of the tumor is the basis for the treatment of Zollinger-Ellison syndrome.

When hypersecretion of hydrochloric acid there are conditions for a persistent spasm of the pylorus, since it takes a long time to neutralize the excessively acidic stomach contents in the

duodenum. When the gatekeeper spasms, food is in the stomach for a long time, the stomach is full, there may be heartburn, sour stomach, sometimes vomiting, pain syndrome occurs, and the evacuation function of the stomach decreases. In the intestine comes a more homogeneous food, decreases the peristalsis of the intestine, there is a tendency to constipation, autointoxication.

Digestion with hypoxecretion and hypochlorhydria. Decrease in secretion of gastric juice develops with an increase in the tone of the sympathetic nervous system, the action of glucagon, secretin, cholecystokinin, enterogastron. Secretin, cholecystokinin, enterogastron are referred to as the duodenal inhibitory mechanism of gastric secretion. Somatostatin normally inhibits the release of gastrin and the secretion of hydrochloric acid in the stomach; Reduction of gastric juice secretion also develops with significant structural changes related to the glandular layer of the stomach, a decrease in the number of cells producing gastric juice.

With a decrease in acid formation, pepsin is not active, and proteins are not digested. Patients can complain of belching "rotten", as the bactericidal action of hydrochloric acid decreases, the processes of putrefaction and fermentation intensify. Decreased production of hydrochloric acid leads to excessive colonization of the gastrointestinal tract by bacteria. Evacuation of food chyme from the stomach is usually accelerated, as its neutralization in the duodenum occurs rapidly. Conditions are created for the gaping of the gatekeeper. Quickly entering the duodenum large portions of gastric contents are poorly saturated with duodenal juice. Duodenal digestion suffers from a decrease in gastric secretion and secretion of pancreatic juice, the release of which is stimulated by hydrochloric acid. Food rough chyme, not prepared for absorption, enters the lower parts of the intestine. The peristalsis that causes diarrhea increases, maldigestia syndromes increase (mainly cavitary digestion) and malabsorption (impaired absorption). The early sign of the latter is steatorrhoea (after taking fatty foods). The body weight decreases, hypovitaminosis develops, electrolyte metabolism disorders, dehydration, significant metabolic disorders.

Achlorhydria can be combined with achilia (absence in the gastric juice of pepsin). Allocate a functional and organic Achilles. With functional achilias, the gastric activity and activity of the main cells are preserved, but their function is inhibited. This is a reversible condition, the occurrence of which is possible under stressful situations, avitaminosis (scurvy, pellagra). The unstable character of the Achilles is noted, changing depending on the stimulus of secretion and the conditions of the study. Organic Achilles develops with a pronounced atrophic gastritis. Functional inferiority, and then structural changes in the activity of the main cells develop later than in the lining cells. Organic Achilles is always associated with a severe syndrome of maldigestion and is often combined with pernicious (B12-deficient) anemia.

Violation of the reservoir and evacuation functions of the stomach

Evacuation of food masses from the stomach in the duodenum occurs when the food becomes liquid, and the previous portion of acidic chyme is neutralized by duodenal juice. The pathology of evacuation is expressed in the acceleration or deceleration of evacuation.

Acceleration of evacuation is observed when hyposecretion of gastric juice, achilles, achlorhydria, the intake of hypoosmolar food, as well as food rich in carbohydrates.

Deceleration of evacuation is noted with hypersecretion of gastric juice, ingestion of a large amount of food in the stomach, especially poorly chewed, for grinding it (up to a size of less than 1 mm) takes a long time. When a large volume of food enters the stomach, the large particles of the particles are "sieved" and grinded by reducing the antrum, which in turn worsens the absorption of nutrients. Stretching the stomach with a large amount of food strengthens the peristaltic contraction of the antrum and pushes food to the gatekeeper and duodenum, which can be accompanied by pain. Deceleration of evacuation is also noted when taking hypertensive solutions and hyperosmolar, protein and especially fatty foods, which contributes to the production of enterogastron in the intestinal mucosa, which is referred to as a duodenal braking mechanism (inhibits motility). Slows the emptying of the stomach with overexertion of duodenum and a decrease in the secretion of pancreatic juice and bile, which neutralize the acidic chyme. The reservoir and evacuation functions suffer from surgical interventions on the abdominal organs, stomach, partial resection, gastroenteric

anastomosis, cicatricial changes due to peptic ulcer or after chemical burns. All this prevents the normal passage of food chyme and disrupts the functions of mixing and evacuation.

Evacuation function is reduced after abdominal injuries, with circulatory disorders of the abdominal cavity. In addition, sometimes with acute, especially intestinal infections, reflex inhibition of tonus and peristalsis of the stomach is possible with violation of the evacuation function. This function is worsened in elderly people due to atrophy of the gastric mucosa and, possibly, under the influence of medications, with pyloric stenosis in adults as a result of tumors, scar scarring or congenital pyloric stenosis - hypertrophy of the muscles of the layer of the stomach.

When deceleration of evacuation, there is a delay in the stomach of food masses, liquids, gases. The wall of the stomach is stretched, thinned, its peristalsis and tone are weakened, the secretion of gastric juice decreases. With prolonged delay of food masses, the enlarged stomach exerts pressure on the diaphragm, PDC, nausea, vomiting, which leads to loss of fluid, chlorides. As a result, acid-base balance may be violated towards alkalosis, dehydration, collapse and coma.

Violation of the motor function of the stomach

Normally, the movements of the stomach are expressed in the form of peristalsis - a wave-like contraction of the stomach wall, which promotes food from the cardiac to the pyloric section, and the peristals - the tonic tension of the musculature, which facilitates the crushing of food.

In pathological conditions, the peristalsis of the stomach can be strengthened (hypertonus) or weakened (hypotonic, atony).

The occurrence of abnormalities on the part of the motor activity of the stomach is mainly due to the direct response of smooth muscles to the influence of a number of neurotransmitters and hormones with the participation of receptors. Possible pathology of smooth muscles and pacemaker stomach (for example, with surgical cutting of the vagus nerve). Stem vagotomy leads to an increase in the tone of the proximal parts of the stomach with a simultaneous decrease in the phase activity of its distal sections. From the stomach, the evacuation of the liquid is accelerated and the evacuation of solid chyme is slowed down. When the tone of vagus increases, the rhythm and strength of the stomach contractions increase, and the evacuation of its contents to the PDC is accelerated. On the contrary, the activation of sympathetic nerves reduces the rhythm, the force of the contractions of the stomach and the speed of the peristaltic wave.

The motor activity of the stomach is influenced by gastrointestinal hormones and hormones of general action. Secretin, cholecystokinin-pancreosimin, enterogastron, glucagon depress gastric motility and the rate of evacuation of food from it. Strengthening motility of the gastrointestinal tract occurs under the influence of gastrin, motilin, histamine, serotonin, insulin.

Oppress gastric motility hypoxecretion of gastric juice (hypo- and achlorhydria), bulbogastron, glucagon, fever, fasting.

Among other causes of gastric motility disorders, a number of drugs, particularly antihypertensive agents, especially calcium antagonists, more long-acting (prolonged), rauwolfia, a-methyldopa derivatives, psychotropic, anticholinergic drugs, nitrates, antispasmodics are often noted.

Violation of the motor activity of the stomach is noted for endocrine diseases (hypothyroidism, hyperparathyroidism, diabetes mellitus), nervous system (meningitis, encephalitis, brain tumor), a number of infectious diseases (Botkin's disease, intestinal infections), metabolic disorders, electrolyte disorders, often with mental illnesses (Neurogenic anorexia, indomitable vomiting). In each of these diseases, the presence of gastric motility disorders can be associated with a complex mechanism involving disorders of nervous, hormonal regulation, electrical rhythm, and smooth muscle functions of the stomach. So, for example, a violation of gastric emptying can develop with a prolonged course of diabetes mellitus, complicated by visceral neuropathy, which manifests itself in a disorder of the vegetative functions of not only the stomach, but also the gallbladder, bladder, and intestine. Gastroparesis in patients with diabetes is most often caused by visceral neuropathy, but the influence of sugar-lowering drugs, psychogenic factors, is not ruled out.

Primary change in the musculature of the stomach may occur with a number of collagenases, in particular with scleroderma and dermatomyositis. Significant violations of the motor activity of the stomach are noted during surgical operations.

Violations of the motor function of the stomach manifest such symptoms as heartburn, eructation, hiccups, nausea and vomiting.

Heartburn (pyrosis) - a sensation of heat or burning in the lower part of the esophagus (can be located behind the breastbone or in the upper part of the epigastric region), spreading from the bottom upwards - from the epigastric region to the neck. Heartburn is usually the result of throwing acidic (pH <4.0) contents of the stomach or bile into the esophagus with an anti-peristaltic wave with an open cardiac sphincter (ie associated with gastroesophageal reflux). At the level of contact with gastric contents there is a spasm of the esophagus, above - its antiperistaltic. The intensity of this manifestation depends on the concentration of acid in the gastric contents, the frequency and duration of its contact with the mucosa of the esophagus. Heartburn is worse after eating, especially abundant, with the torso in the supine position, with abdominal muscle abnormalities. It can be accompanied by a spontaneous appearance in the mouth of a liquid that may be acidic, brackish (stomach contents or "acidic eructation") or bitter, having a yellow or green color (bile).

Heartburn can occur after eating a number of foods: fats, sour fruit juices, tomatoes, garlic, onions, peppers, etc. Or medications that reduce the tone of the lower esophageal sphincter - theophylline, progesterone, antidepressants, nitrates, calcium antagonists, etc. Heartburn usually decreases when swallowing saliva, drinking water and most clearly when taking antacid preparations.

Erythra (eructatio) - a sudden ingestion in the mouth of a small portion of the contents of the stomach or esophagus. Usually, the stomach contains a small amount of gas (gas bubble), stimulating its motor and secretory functions. A small amount of air is swallowed while eating. From 20 to 60% of the gas in the intestine is due to the amount of swallowed air (the proof of this is the presence of nitrogen and oxygen present in the atmosphere and not produced in the digestive tract). The accumulation of air in the stomach can cause a feeling of overflow, overstretching it after eating, which is proved by radiographic examination of the abdominal cavity. Acute stretching of the stomach with swallowed air often occurs after a plentiful meal and is accompanied by a marked pain syndrome resembling angina. In the supine position, the stomach bladder syndrome can develop when the air in the stomach is trapped (below the junction of the esophagus with the stomach) by the pressure of the liquid above it, so that this air can not be regurgitated.

Aerophagia (ingestion of air outside food intake) is more often observed with neurogenic conditions.

Distinguish eructation by air and burping food. Eating food can be acidic or bitter (an impurity of bile), as well as putrefactive (with food stagnation in the stomach). Resistant burping of food is a characteristic symptom of a deficiency of the cardiac sphincter and a number of diseases of the abdominal cavity organs: peptic ulcer of stomach and duodenum, active gastroduodenitis, gastroesophageal reflux disease, stomach cancer, esophagus. With atrophic gastritis, when the gatekeeper gapes, increased gas formation in the stomach is often associated with gassing in the intestine, with the gas freely entering the stomach. An eructation, especially bitter, often occurs in the pathology of the hepatobiliary system. In addition, eructation can occur reflexively, for example, in diseases of the cardiovascular system.

A portion of the swallowed air passes further through the gatekeeper into the intestines, which causes its swelling. Air may be trapped in the splenic flexure of the large intestine (this is the syndrome of left bend of the colon) when there is a feeling of overflow in the left upper quadrant of the abdomen with possible irradiation into the left half of the thorax. Pain relief often occurs after defecation or secretion of gases from the intestine.

Hiccup (singultus) occurs as a result of a combination of rapid spasm of the diaphragm, convulsive contraction of the stomach and sudden strong inspiration with narrowing of the glottis. Hiccups can be observed in diseases of the gastrointestinal tract and other organs of the abdominal cavity, with more often there is a reflex when the center of the diaphragmatic nerve is excited.

Hiccups are also observed in diseases of the mediastinum, pleura, peritoneum, when the diaphragm or diaphragmatic nerve is directly irritated.

Nausea (nausea) is an unpleasant, painless subjective feeling of an impending desire to perform an emetic act. Nausea often precedes vomiting than accompanies it. However, nausea and vomiting can occur independently of each other. With nausea, various physiological reactions occur. In connection with the close location of the vomiting of the nuclei of the glossopharyngeal and facial nerves (which innervate the salivary glands), hypersalivation is often observed. With nausea, tachycardia often develops, probably as a result of a stressful reaction to possible vomiting. There is weakness, increased sweating, pale skin, cold extremities, a drop in blood pressure due to excitation of the parasympathetic, and then sympathetic parts of the autonomic nervous system. Possible development of hypotension with bradycardia (vasovagal syndrome). With nausea, the motor activity of the gastrointestinal tract is disrupted and the secretory function of the stomach decreases. The sensation of nausea is associated with anti-peristaltic movements of the stomach. Nausea is often accompanied by anorexia, i.e. A loss of desire to eat or a refusal to eat. Following the continuing for some time nausea and brief periods of urges for vomiting, a sequence of involuntary visceral and somatic motor acts, leading to the occurrence of vomiting, develops.

Vomiting (vomitus) is a complex reflex act, as a result of which the contents of the stomach erupt outward. In the process of vomiting, the stomach plays a relatively passive role. Pushing its contents is provided by the abdominal muscles. With the relaxation of the bottom of the stomach and gastroesophageal sphincter, there is an increase in intra-abdominal pressure due to an involuntary contraction of the diaphragm and the abdominal wall (external oblique muscles of the stomach). This reduction, together with the ongoing contraction of the gatekeeper, leads to the expulsion of the contents of the stomach into the esophagus. Increase in intra-abdominal pressure promotes further movement of the contents along the esophagus into the oral cavity. The reflex rise of the soft palate during vomiting prevents the contents of the stomach from getting into the nasal part of the pharynx, and the reflex closure of the glottis and respiratory depression inhibit the aspiration of the stomach contents into the respiratory tract.

When vomiting, there are violations of the motility of the gastrointestinal tract. The tone of the bottom of the stomach and the peristalsis of the stomach usually decrease, the tone of the DPC and the proximal jejunum increases, and the peristalsis may take the opposite direction (antiperistaltic). In the latter case duodenogastric reflux arises, and this explains the impurity of bile in the vomit from the duodenum. The role of anti-peristalsis in vomiting is well shown in experiments on animals (cats, dogs), which injected substances stimulating vomiting into the cavity of the ventricles of the brain. It has been established that before the act of emesis there is a change in the electrical activity of the intestine with an increase in the electro potential in the proximal direction. Clinically, the anti-peristalsis of the intestine is manifested by the frequent presence of intestinal contents in the vomit. With intestinal obstruction, vomiting with an admixture of feces is possible.

The emetic act is controlled by two functionally different centers located in the medulla oblongata: the vomiting center and the chemoreceptor trigger zone. These centers are located next to other centers of the brain stem that regulate autonomic functions. The afferent path of the emetic reflex follows the sensitive fibers of the vagus nerve into the center of vomiting, which is located in the lower part of the bottom of the IV ventricle, next to the respiratory and cough centers. Centrifugal impulses to the effectors spread along the motor fibers of the vagus nerve, along the diaphragmatic, dorsal and celiac nerves.

The vomiting center controls and unites the vomiting into a single whole. He receives afferent signals from the intestine, from other parts of the body, from the upper cortical centers, especially from the inner ear apparatus and the trigger chemoreceptor zone. Important efferent conductive pathways during vomiting are the diaphragm nerves (to the diaphragm), the spinal cord nerves (to the muscles of the abdominal wall), and the visceral efferent nerves (to the stomach and esophagus).

More often, vomiting occurs when gastric receptors are irritated by poor-quality food, toxic substances, in particular alcohol substitutes, and also with high excitability of these receptors in conditions of pathology. In such cases, vomiting is called gastric. Reflexogenic zones of the

vomiting act are also the posterior wall of the pharynx, the ileocecal region of the gut. It is possible to stimulate the center of vomiting from the peritoneal receptors, bile ducts, gallbladder, kidneys, urinary tract, coronary vessels, membranous inner labyrinth, etc. Vomiting caused by pulses from peripheral reflex zones is called peripheral vomiting.

Vomiting can be of central origin and occur in pathological processes in the region of the IV ventricle (tumor or inflammatory process). The center of vomiting can be irritated by poisons or toxins, with toxicoses of pregnant women, the use of toxic products, drugs, metabolic disorders in renal and hepatic insufficiency, in ketoacidosis, etc. Vomiting can occur by the mechanism of a conditioned reflex - with an unpleasant odor, a form of inedible food. In the laboratory of I.P. In 1914, Pavlov's conditional reflex vomiting was reproduced in a dog with a combination of an indifferent stimulus (sound of a pipe) with injections of apomorphine.

Vomiting can be acute, which is observed during poisoning as a protective reaction aimed at cleansing the gastrointestinal tract from toxins, substances of poor-quality food. Acute vomiting can be associated with an acute process in the abdominal cavity, such as intestinal obstruction, infringement of the hernia, which is also associated with pain syndrome. Acute pain with the phenomenon of vomiting occurs with perforation of the stomach and duodenal ulcers, acute appendicitis. Such a symptom is not always associated with the pathology of the gastrointestinal tract, but it can also be caused by a pathological process in the hepatobiliary system (acute cholecystitis, acute hepatitis, acute pancreatitis, cholelithiasis), cardiovascular pathology (acute myocardial infarction, exfoliating aortic aneurysm), kidneys (nephrolithiasis). Repeated vomiting, more often after a meal, facilitating the patient's condition, is typical for peptic ulcer of the stomach or duodenum during an exacerbation. With stenosis of the gatekeeper, vomiting occurs more often by evening and food, eaten the day before.

Vomiting, especially repeated, leads to a number of metabolic disorders. Most often it is metabolic alkalosis, hypokalemia and hyponatremia. Metabolic alkalosis is a consequence of an increase in the concentration of bicarbonates in the blood plasma, which is due to: 1) a decrease in the concentration of H + in the extracellular fluid; 2) loss of a liquid containing chlorides at higher concentrations than the concentration of bicarbonates in the extracellular fluid; 3) an increase in the concentration of bicarbonates when soda and other substances are converted into bicarbonate in the extracellular fluid.

Hypokalemia develops as a result of loss of potassium with vomiting and its small intake with food. Hyponatremia also develops as a result of excretion of sodium with vomit and, possibly, urine due to metabolic alkalosis.

Violation of the suction function of the stomach

Normally, this function is not large. With pathological conditions of the stomach, it can significantly increase. So, with food stagnation in the stomach, polypeptides can be absorbed through its wall, which causes intoxication syndrome and allergic organism. Strengthening of this function can be noted in inflammatory-dystrophic processes, in particular in chronic gastritis, when the gastric mucosa becomes permeable to toxins and food digesting products.

Violation of the excretory function of the stomach

The excretory (excretory) function of the stomach is one of the extrarenal ways of excreting metabolites from the bloodstream to provide homeostasis. Into the cavity of the stomach are released metabolic products, as well as substances that have entered the body, the stay of which is unnecessary or harmful. Excretory function of the stomach favors the kidneys, preventing them from excessive stress. The role of this function especially increases with various pathological conditions of the body or with extreme effects on it, which cause pronounced changes in the metabolism. Excretory function of the stomach is closely interrelated with its other functions, which is provided by general regulatory mechanisms.

The stomach wall can be released into its cavity circulating in the blood metabolites (urea, uric acid, creatine, creatinine). I.P. Pavlov stressed the role of gastric excretory function as an

important detoxification factor of the body, as a "physiological measure of protection." So, for example, in chronic renal failure, the content of urea, creatinine in the gastric juice and in saliva increases significantly. With the increase in gastric wall excretion of cellular decay products, the development of gastritic changes in the gastric mucosa is associated with severe infectious diseases and other diseases.

In dogs subjected to overheating, the appearance of lactic acid in the gastric juice. Excretion of a large number of nitrogen-containing substances by the stomach is observed in experimental animals with complete starvation. A similar situation with prolonged hunger occurs in humans. These substances are then absorbed into the intestines and used by the body to feed vital organs. Thus, in patients with prolonged chronic purulent processes, continuous secretion of gastric juice is noted, where the concentration of hydrochloric acid is reduced, and a high content of nitrogenous substances is present. The latter are absorbed into the intestine. However, the absorption process lags behind excretion, and this is one of the factors that contribute to the so-called wound depletion.

Gastric secretion is regulated by cholinergic structures of the autonomic nervous system. Stimulation of α- and β-adrenergic receptors mobilizes the excretory function of the stomach. In the starting phase of gastric digestion, the excretory capacity of the gastric glands is lower than in the completed one, in the mechanism of which the leading role is played by humoral factors. Stimulants of the excretory function of the stomach include corticosteroids, prostaglandins E, hypoxia; The inhibitory effect on it has mineralocorticoids.

The excretory function of the stomach is judged by the speed of appearance in the gastric juice of an intravenously injected solution of neutral red (neutral) powder, which normally appears there in 12-15 minutes. With secretory deficiency of the stomach, especially with atrophy of the mucosa, the release of the paint is significantly delayed (up to 30-45 min), with increased acidity - somewhat accelerated (up to 8-10 min).

Peptic Ulcer

The peptic ulcer is a chronic, cyclically recurring disease in which general and local mechanisms of nervous and humoral regulation of the secretory trophic activity of the gastrohepatopancreatic system are violated and ulcerative mucosal defects in the stomach or in the duodenum are formed, often against the background of active gastritis and duodenitis associated With Helicobacter pylori, prone to the progression and development of complications that threaten the life of the patient.

Ulcer disease occurs in 6-10% of the adult population, more often in men under the age of 50 years. In 60-70% of patients, peptic ulcer is formed in adolescence and young age. In adolescents and young men, duodenal ulcer is more likely to develop, in women and men of older age groups - peptic ulcer. In 1/3 patients with peptic ulcer disease, duodenal ulcer develops further.

Peptic ulcer is an independent disease. Acute (symptomatic) ulcers are always secondary, for example, when treated with steroid hormones, salicylates, butadione, with cirrhosis, acute renal failure, etc.

Etiology and pathogenesis of peptic ulcer

The peptic ulcer is polyethiologic. The main etiological factors of peptic ulcer of the stomach and duodenum are the bacterium Helicobacter pylori and neuropsychic stress.

At present, there is every reason to consider peptic ulcer as an infectious disease, since the link between the development of peptic ulcer and infection of Helicobacter pylori (HP) has been proved. Australian scientists R. Warren and B. Marshall in 2005 received the Nobel Prize for the "unexpected and startling" discovery that they made in 1982: they found that the cause of gastritis and peptic ulcer of the stomach and duodenum is the bacterium - HP. When B. Marshall singled out the pure culture of the bacterium, he conducted an experience of self-infection, and developed a sharp gastritis. As a means of treatment, he used antibiotic therapy. As a result of this discovery, a reasonable opportunity to treat ulcerous disease with antibiotics appeared, which increased the frequency of cure for peptic ulcer and reduced the number of relapses of the disease.

It is established that in patients with peptic ulcer of the duodenum HP is found in 90-95% of cases, in patients with peptic ulcer of the stomach - in 80% of cases. Evaluation of the presence of a bacterium is carried out with the help of a serological blood test, an enzyme immunoassay, a bacteriological study of the mucosal biopsy, a breath test,

Helicobacter pylori is a gram-negative anaerobic rod that has a flagella and is capable of producing urease. This pathogen is found in the mucous membrane of the antral part of the stomach, appearing sometimes in healthy, without any pathological changes, but much more often (up to 95%) in patients with gastritis or peptic ulcer. If you get into the lumen of the stomach with swallowed saliva or from the surface of the gastroscope, the gastric (duodenal) probe HP is in a difficult habitat (acid content of the stomach). However, due to their urease activity, bacteria can exist under these conditions. Urea, coming from the bloodstream, by sweating through the wall of capillaries, urease turns into ammonia and CO2, which neutralizes hydrochloric acid of gastric juice, creating local alkalinization around the bacterial cell. Ammonia acts irritatingly on the G cells of the APUD system, increasing the secretion of gastrin and, respectively, HC1.

Flagellum and spiral form of bacteria provide active advance, and HP in the environment of urease and ammonia penetrates from the lumen of the stomach into the layer of mucus, where the progress continues. In addition to local alkalization, around the bacteria there is a decrease in the viscosity of the gastric mucus - mucin is destroyed, and HP reaches through the protective mucous barrier of the gastrointestinal epithelium of the gastric mucosa. There is an adhesion of HP on the integumentary-pit epithelium of the antral part of the stomach. A part of the microbes penetrates into its own plate through interepithelial contacts. Dystrophic changes occur in the epithelial cells, which reduces their functional activity. Intensive reproduction and colonization of HP on the antrum mucosa of the stomach lead to damage to the epithelium due to the action of phospholipases. Isolate an ulcirogenous strain of HP, which synthesizes cytotoxins that activate the phospholipase. In this case, the probability of ulceration of the gastric mucosa is very high. There is a destruction of protective protein components, mucin, which opens the way of HP deep into the mucous membrane. Ammonia, affecting the endocrine cells of the antrum of the stomach, reduces the number of D-cells that produce somatostatin, and, accordingly, its concentration decreases. The release of gastrin leaves the control of D-cells, which leads to hypergastrinemia, an increase in the mass of parietal cells and hyperproduction of hydrochloric acid. Thus, infection with HP can be primary, and increased secretion of hydrochloric acid - a secondary link in the pathogenesis of gastric ulcer. In the submucosal layer, an inflammatory infiltrate is formed (consisting of neutrophils, lymphocytes, macrophages, plasma and mast cells), necrosis of the epithelium with the formation of a ulcerative defect.

Pathogenesis of duodenal ulcer is more difficult than stomach ulcers. HP selectively populates only the metaplastic epithelium and does not affect the normal mucosa of the duodenum. Gastric metaplasia (replacement of the cylindrical cells of the epithelium of the duodenum by cells of the gastric epithelium) is observed in 90% of patients with a duodenal ulcer. Metaplasia allows HP to penetrate the cells of the mucosa of the bulb of duodenum, making them less resistant to damage by hydrochloric acid, pepsin, bile. The prolonged casting of acidic gastric contents into the bulb of duodenum creates favorable conditions for the development of gastric metaplasia of its epithelium. The risk of developing duodenal ulcers with pronounced antral gastritis and proximal duodenitis associated with HP exceeds the control duodenal by 50 times, and in normal mucosa it is practically zero.

It is interesting to note that HP infection is high enough - infection in the north of Russia is 50%, in the south and east of Russia it reaches 80 and 90%, respectively. Only 1/8 of people infected with HP develop a peptic ulcer.

However, peptic ulcer is not a classic infection and one infection of HP is not enough for its occurrence.

The main etiological factors of peptic ulcer include neuropsychic stress. Under the influence of prolonged or often recurring psychoemotional overstrain (severe nervous shocks, professional failures and family dramas), the coordinating function of the cerebral cortex with respect to the

subcortical formations and especially the hypothalamus is disrupted. There is a persistent excitation of the centers of the autonomic nervous system. Abundant pathological parasympathetic impulse from the CNS leads to hypersecretion of HC1 and gastric hypermotorics. Abundant pathological sympathetic impulse from the central nervous system leads to the ejection of catecholamines in synapses and adrenal medulla that causes trophic and hemodynamic disturbances in the gastric mucosa. Activation of the hypothalamic-pituitary-adrenal system causes increased production of glucocorticoids, which entails hypersecretion of gastric juice, vasospasm, catabolic effect (increased disintegration and reduced protein synthesis). All of the above causes the formation of ulcerative defects, a decrease in mucus production and a decrease in regeneration.

Predisposing factors of peptic ulcer include genetic markers: high level of production of HC1 - maximum acid production of the stomach (as a result of genetically determined increase in the weight of the cells and their sensitivity to gastrin); High level of pepsinogen 1 in the blood serum - "ulcirogenic fraction of pepsinogen"; Excess gastrin release by G-cells in response to food intake; I blood group (these people have gastric adhesive mucosa with adhesive receptors for Helicobacter pylori); Genetically determined decrease in the production of a number of protective substances (protecting the mucosa from proteolysis), including α1-antitrypsin inhibitor of serine protease, and a2-macroglobulins (account for 97% of the total content of plasma macroglobulins - non-specific protease inhibitors and universal regulators of the immune system).

Factors contributing to the development of the disease are nutritional factors (acute, hot food, spices, seasonings), bad habits (smoking and abuse of strong alcoholic beverages, a role in the development of peptic ulcers take away coffee), ulciogenic drugs. Especially dangerous are long breaks in food intake, especially in individuals with increased secretion and acidity of gastric juice.

All etiological factors potentiate each other and lead to the formation of "aggression" factors. Ultimately, to be or not to be a peptic ulcer is determined by the ratio of the factors of "defense" and the factors of "aggression".

The factors of "aggression" include, first of all, HP infection and destruction of the mucous-bicarbonate barrier, as well as a high acid-peptic factor. The causes of hypersecretion of hydrochloric acid are hyperplasia of parietal cells, apparently, genetically conditioned vagotonia and hyperproduction of gastrin. It is known that the main stimulants of HCL secretion are histamine, gastrin, acetylcholine. In addition, it is known that inadequate production of glucagon and especially somatostatin also contributes to ulcer formation.

Pathogenetic factor in the realization of the disease, along with a high acid-peptic factor, is gastroduodenal dissotorics. If a healthy person has a rhythmic intake of gastric contents in the duodenum -3 contraction in 1 minute, then in patients with peptic ulcer in duodenum 15-minute periods of low pH are noted. High acidity can not maintain normal peristalsis, the "acidification" of the duodenum occurs. Prolonged contact of acidic contents with mucous leads to ulceration. "Oxidation" of duodenum is often associated with dyskinesia and a decrease in its alkalizing function due to a violation of the production of bicarbonates of pancreatic and biliary secret.

A definite value in the development of peptic ulcer is given to duodenogastral reflux (DGR) - casting of bile (bile acids) into the stomach. Bile, affecting the gastric mucosa, leads to a disruption of the mucous barrier and to an increase in the acid-peptic properties of gastric juice due to stimulation of the endocrine apparatus of the stomach (first of all, the production of gastrin is enhanced). Dyskinesia duodenum, especially hypomotor type, lowering the tone of the antral part of the stomach promote DGR, make it long and intense. It is proved that DGR occurs much more often when a peptic ulcer is combined with diseases of the hepatobiliary system, especially with cholelithiasis.

The factors of "aggression" include the violation of the duodenal inhibitory mechanism (insufficient production of secretin, cholecystokinin, enterogastron) in duodenum, a disturbance in the exchange of biogenic amines - histamine and serotonin, which are released mainly from enterochromafin cells of the gastric mucosa. Histamine stimulates the secretion of HC1 through H2 receptors associated with cAMP. During the peptic ulcer exacerbation, histamine synthesis processes usually increase, which leads to the appearance of free histamine in the blood. According

to one hypothesis, histamine acts as a mediator of the parasympathetic nervous system. According to another widely held view, histamine is an intermediate link in the realization of gastrin action on secretory cells. The capillary circulation changes, the permeability of the vascular wall increases, the production of pepsin increases (histamine is a potent stimulant of the main cells). Histamine and serotonin, acting as activators of the kinin system (activate bradykinin), cause significant microcirculation disorders, blood circulation and trophism of the gastric mucosa suffer. Normally biogenic amines are rendered harmless by the amine oxidase of the intestinal wall.

The protective barrier of the gastric mucosa consists of three parts: 1) epineothelial (mucus, bicarbonates); 2) epithelial (epithelial cells and their repair, prostaglandins, growth hormones); 3) subepithelial (blood supply, microcirculation).

Mucous stomach is constantly exposed to hydrochloric acid, pepsin, and with duodenogastric reflux - and the effects of bile acids, pancreatic enzymes. In the protective barrier of the stomach, the first line of defense against damaging factors is the cells of the mucous membrane. These are surface cells and secretory additional, secreting mucus and bicarbonates. Due to these substances, a physicochemical barrier is created, which is a gel that maintains the pH of the neutral medium at the surface of the epithelium. All superficial epithelial cells lining the stomach and duodenum synthesize and secrete bicarbonates. The mucosa of the proximal part of the duodenum produces bicarbonates 2 times more than the entire gastric mucosa. An important role in maintaining the basal level of secretion of bicarbonates and mucus is also assigned to endogenous prostaglandins. Slime, its insoluble fraction, bicarbonates protect the gastric mucosa from the effects of hydrochloric acid and pepsin. The mucous barrier prevents the reverse diffusion of H + from the lumen of the stomach into the blood. Prolonged contact of the mucosa with acidic medium and changes in the composition of mucus (during the peptic ulcer exacerbation in the mucus the content of sialic acids and glycoproteins neutralizing hydrochloric acid decreases) lead to the breakthrough of the mucous barrier and the appearance of reverse diffusion of hydrogen ions. In response, histamine is released from the mast cells (tissue basophils of the stomach) and the cholinergic system is reflexively excited, venous stasis, capillary overflow, hydrochloric acid and pepsin production are enhanced - all this contributes to peptic ulcer formation.

In maintaining the stability of the mucous membrane of the stomach and the duodenum, the ability of cells to rapidly renew (repair), a good state of circulation and the secretion of chemical mediators of protection (prostaglandins, growth hormone) play an important role in the factors of aggression. It is known that the mucous membrane of the stomach and duodenum after the damage is usually quickly restored (within 15-30 minutes). This process is not due to cell division, but as a result of their movement from the gastrointestinal epithelium of the stomach along the basal membrane and the closure of the defect in the area of the damaged epithelium. Prostaglandins, especially prostaglandin E2, contribute to improving the protective properties of the gastric mucosa, as they inhibit the activity of parietal cells, stimulate the secretion of mucus and bicarbonates and improve the blood supply to the mucous membrane, reducing the reverse diffusion of hydrogen ions and accelerating regeneration. Their secretion is carried out by the main, additional and parietal cells of the gastric mucosa.

The subepithelial part of the protective barrier of the gastric mucosa includes the optimal blood supply and microcirculation.

In addition, the factors of "protection" include the alkaline reaction of saliva, pancreatic juice, bile; Optimal motor and evacuation of the stomach; As well as the mechanism of duodenal inhibition of acid and pepsin formation (the production of cholecystokinin, secretin, enterogastrone duodenum).

When the factors of "aggression" outweigh the scales on the scales, an ulcer is formed, it becomes the focus of afferent impulsation in the central nervous system, where a pathological dominant arises. Other organs and systems of the body (liver, pancreas, etc.) are involved in the process, the disease becomes chronic.

The clinic of peptic ulcer includes pain syndrome, which is characterized by periodicity (depending on food intake, "hungry" pain), seasonality (exacerbation in spring and autumn),

rhythmicity (night, daytime - from daily rhythms of gastrointestinal juices). Pain syndrome is the leading subjective manifestation of the disease in the phase of exacerbation. The syndrome of dyspeptic disorders is characterized by heartburn, eructation, often regurgitation with salivation. Appetite remains good, with duodenal ulcers even increases (a painful feeling of hunger). Constipation occurs in 50% of patients, they worry even more than pain.

Complications of peptic ulcer include bleeding (small - up to 500 ml, average - up to 1000 ml, large - up to 1500 ml, massive - more than 1500 ml), posthemorrhagic anemia (mild, moderate, severe), penetration (into the small omentum, Pancreas, liver, gall bladder, etc.), perforation (into the free abdominal cavity, small gland cavity), stenosis (compensated, subcompensated, decompensated), malignancy (typical For peptic ulcer, I Duodenal ulcer is not malignant), reactive hepatitis, reactive pancreatitis, perivistseritis (perigastritis, periduodenitis).

Outcomes of peptic ulcer: scarring and healing; Stenosis of the pylorus and deformation of the stomach as a result of scarring; Lifelong existence of peptic ulcer; Malignization; A lethal outcome is usually a result of bleeding or perforation.

Experimental stomach ulcers. To reproduce the stomach ulcer in the experiment, the following methods are most often used:

1. Damage to the gastric mucosa by physical and chemical irritants (hot water, lapis, acids, croton oil, etc.). In the wall of the stomach develops acute inflammation and the formation of ulcerative defects, which usually quickly heal.

Disturbance of blood circulation in the wall of the stomach or duodenum (dressing, embolism, sclerosing of blood vessels). Blood flow is usually restored by anastomoses, and the resulting ulcers quickly heal.

Long-term administration of drugs that enhance gastric secretion (atofan, histamine, pentagastrin, pilocarpine, etc.), followed by the formation of a ulcerative defect.

Chronic irritation of vagus with increased gastric secretion and impaired microcirculation in the wall of the stomach.

Experimental neuroses with additional administration of gastric juice. In dogs, stomach ulcers arose when a burst of higher nervous activity was combined with a daily two-hour irrigation of the gastric mucosa with gastric juice.

Imposition of ligature on the gatekeeper with preservation of its patency (Shey's method). At the same time, erosions and sometimes ulcers appeared in the stomach of rats after 1-2 days as a result of vasodilation and the irritant effect of the ligature on n. Vagus, which caused a significant violation of blood circulation.

Introduction of gastro-cytotoxic serum obtained by immunizing animal donors with homogenate of gastric tissue.

For example, a rabbit is immunized with a dog's stomach tissue and the resulting serum containing anti-gastric antibodies is administered intravenously to an intact recipient dog. Antibodies interact with the stomach tissue of the recipient animal and cause damage to this tissue as a result of the antigen-antibody reaction.

The described methods of experimental modeling of ulcers cause mainly acute ulcerative defects. By the mechanism of origin and flow characteristics (usually quickly heal), they are fundamentally different from peptic ulcer, more reproducing the picture of symptomatic human ulcers. However, it is partially possible to model individual manifestations of this disease, which orientates in the developed antiulcer therapy.

Digestive disorders in the intestine

Digestion in the small intestine provides depolymerization of nutrients to the stage in which they are absorbed into the blood and lymph. Digestion in the small intestine first occurs in its cavity (cavity digestion), and then in the zone of the intestinal epithelium with the help of enzymes fixed on its microvilli and in the glycocalysis (parietal digestion). Cavity and parietal digestion is carried out by intestinal enzymes and enzymes of the pancreas.

An important role in the violation of digestion in the intestine is the violation of bile secretion, external secretion of the pancreas, as well as impaired secretory, absorption, motor, excretory functions of the intestine.

Violation of bile secretion

Bile is produced by hepatocytes and secreted into the intestine (in duodenum) in a volume of 500 ml per day. It contains bile acids, bile pigments, cholesterol and other lipids, as well as alkaline phosphatase.

Bile acids and their salts (sodium and potassium) are necessary for fat absorption. When bile acids enter through the duct and sphincter of Oddi in the duodenum, they are mixed with digestible lipids and fat-soluble vitamins, forming micelles (water-soluble complexes). Micelles are involved in the emulsification of fats, increase the surface area for hydrolysis and prepare fats for absorption. Hepatocytes produce cholic and chenodeoxycholic acids - primary bile acids. Under the influence of bacteria of the small intestine, they are modified into secondary bile acids (Deoxycholic, Methocholic and Ursocholic). The bile acids are reabsorbed in the small intestine and enter the portal vein system for recirculation. When entering the liver by the mechanism of negative feedback, they inhibit the synthesis of new bile acids, i.e. There is a process of intestinal-hepatic circulation of bile acids. Without this circulation, there is a violation of fat absorption, since the liver is not able to provide the synthesis of new bile acids necessary for the lipids entering the intestines. Changes in the composition of bile occur under the action of bile duct cells, which secrete bicarbonate and water (regulated by secretin) in bile. The final bile secreted in the duodenum has an alkaline reaction and is isosmolar to the blood plasma. This process is also regulated by cholecystokinin. These hormones also have a synergistic effect on the secretion of pancreatic juice.

Insufficient intake of bile in the intestine is called hypochole, and complete cessation of its admission is called achiolia. This is possible with plugging of the common bile duct with a stone, less often with worms, due to inflammation or compression of the tumor, enlarged lymph nodes, scar tissue of the liver gates. With hypocholism, especially acholia, digestion and absorption of fats are disrupted. Lipase pancreatic juice in the absence of bile is inactive, fats are not emulsified, and their contact with lipolytic enzymes is difficult. The process of absorption of fatty acids suffers, since for this, the formation of water-soluble complexes with bile acids is necessary. The absorption of cholesterol and fat-soluble vitamins is also disturbed, as they are absorbed, like food fats. Violation of the digestion of fats is manifested by steatoria (stear, atos - fat, fat, rhoe - flow) - excess fat in feces. With feces at the same time up to 70-80% of eaten fats. In the intestine, unsplit fats envelop the food chyme and hamper the action of amylolytic and proteolytic enzymes of duodenal juice, the activity of which decreases with insufficient intake of bile into the intestine. Sorption properties of the intestinal epithelium also suffer from a lack of bile acids, disturbed parietal digestion. This entails a violation of digestion and absorption of proteins and carbohydrates. Non-fat absorption promotes loss through the intestine of fat-soluble vitamins. Developing hypovitaminosis. Due to hypovitaminosis A, dermatitis occurs, growth slows down, vision is reduced until blindness (xerophthalmia). Lack of vitamin K leads to blood clotting and increased bleeding, vitamin D, which regulates the absorption of Ca2 + in the small intestine, to rickets and osteomalacia, and vitamin E deficiency to disorders of the nervous system (in the form of cerebellar disorders).

With hypocholia, peristaltic activity of the intestine weakens, which leads to increased flatulence, putrefaction, fermentation, as the bactericidal action of bile decreases.

Violation of the external secretion of the pancreas

The volume of the secretion of the pancreas is 1500 ml per day. It is secreted into the small intestine and contains enzymes, hydrolyzing proteins, fats and carbohydrates. Regulation of secretion is carried out by hormones - cholecystokinin (stimulates the secretion of enzymes) and

secretin (stimulates the secretion of bicarbonates). Regulation of pancreatic secretion is carried out through the vagus nerve.

The main causes of violations of external secretion of the pancreas are: 1) insufficient production of secretin in achlorhydria; 2) neurogenic inhibition of the pancreas function (with vagotomy, atropine poisoning); 3) development of allergic reactions; 4) exposure to various chemicals (poisoning with phosphorus, lead, mercury, cobalt); 5) injuries of the abdominal cavity; 6) toxicinfections (typhoid fever, paratyphus); 7) chronic infections (tuberculosis, malaria); 8) alimentary factors (excessive intake of food, animal fat, etc.); 9) destruction of the pancreas by the tumor process; 10) obstruction and compression of the duct with a tumor; 11) duodenitis - inflammatory processes in the DPC of any etiology (infectious, parasitic, etc.), leading to a decrease in secretin formation, followed by hypoxecretion of the pancreas; 12) exposure to alcohol, increasing the release of hydrochloric acid, which leads to stimulation of secretin secretion with excessive secretion of pancreatic secretions; 13) acute and chronic pancreatitis.

Etiology and pathogenesis of acute pancreatitis. The main etiological factors (in 70% of cases) of acute pancreatitis are cholelithiasis and alcohol intake. The emergence of acute alcoholic pancreatitis is due not only to the toxic effects of alcohol. Alcohol stimulates the release of hydrochloric acid, which, affecting the mucosa of the duodenum, increases the secretion of secretin. The latter is a potent stimulator of pancreatic secretion, the excess release of which leads to an increase in pressure in the ducts of the gland and the development of acute pancreatitis. In addition, strong alcoholic drinks contribute to the edema of the mucosa of the duodenum, which causes spasm of the faterov nipple with the subsequent increase in pressure in the pancreatic ducts. It is also known the direct effect of alcohol on the vessels of the pancreas, which causes their spasm. This leads to ischemia of the organ with death of the acinous cells and activation of enzymes in the gland tissue. Taking alcohol at a dose exceeding 100 g / day for several years can lead to the precipitation of pancreatic enzymes in small channels and the formation of protein caps. The more rare causes of acute pancreatitis are abdominal trauma, hyperlipidemia (especially type I and IV), the use of certain medications (nitropium, sulfasalazine, furosemide, corticosteroids, estrogens), infections (mumps, Botkin's disease, salmonellosis), surgical interventions, diagnostic Retrograde cholangiopancreatography, anatomical abnormalities of the pancreatic duct (strictures, tumors), hypercalcemia, uremia, vascular lesions, hereditary predisposition.

Three mechanisms of development of acute pancreatitis are considered. The most accepted theory of self-digestion of the gland tissue, according to which proteolytic enzymes - trypsinogen, chymotrypsinogen, proelastase and phospholipase A are activated inside the duct of the pancreas. It is believed that some factors (endo- and exotoxins, in particular alcohol, viral infections, ischemia and trauma) activate proenzymes, i.e. In conditions of pathology, trypsinogen can be activated in the gland under the influence of coenzyme cytokinase released from damaged parenchyma cells. An important role in the development of pancreatitis is played by the trypsin inhibitor, which is normally contained in a sufficient amount in the pancreas and prevents the conversion of trypsinogen to trypsin. With a high activity of trypsin, inhibitors of the anti- enzyme system are depleted, and their deficiency arises. This is used as a test in the diagnosis of acute pancreatitis: the higher the serum trypsinogen content, the less the trypsin inhibitor. When this factor is deficient, there is an active transition of trypsinogen to trypsin. The increased activity of proteolytic enzymes, especially trypsin, leads to the digestion of pancreatic tissue and the activation of other enzymes - elastase and phospholipase. The active enzymes of cell membranes are digested, proteolysis, edema, interstitial inflammation, vascular damage, coagulation, fatty necrosis (steatoneecrosis) and necrosis of the parenchyma of the gland develop. Damage and destruction of cells lead to the release of activated enzymes. The digestive action of enzymes also affects the periphery. This is connected with the phenomenon of "enzyme evasion into the blood", which causes the development of necrotic processes in other organs. When lipase enters the blood, necrosis of distant organs with severe subsequent intoxication is possible. The process can be complicated by peritonitis and abscesses of the abdominal cavity. Trypsin activates pancreatic kallikrein, which causes the formation of callidin and bradykinin, which increase the damage to the gland tissue. There is a

further activation of the kinin system. Activation and release of bradykinin and histamine cause various hemodynamic disorders. Vessels are dilated, the permeability of their walls rises and the swelling of the gland develops. The release of fluid and protein into the tissue leads to a reduction in oncotic pressure and the development of pancreatic collapse, sometimes fatal. This collapse can be replicated in an experiment with intravenous administration of pancreatic juice to an animal. If the juice is pre-boiled, the collapse will not develop.

The second theory is the theory of the "common channel". Thanks to anatomical features, most people (80%) have a common biliary and pancreatic duct that facilitates reflux of bile into the pancreatic duct. However, normal pressure in the pancreatic duct is 2 times higher than in the common bile duct (200 mm of water). This prevents from casting bile and intestinal contents into the ducts of the pancreas. The casting of bile may be noted with hypertension of the sphincter of Oddi or hypermotor dyskinesia of the biliary tract. The frequent development of pancreatitis in cholelithiasis is due to the increased pressure in the biliary system. This ensures the transfer of infected bile under high pressure into the pancreatic duct, which causes chemical damage to the gland tissue, increases its enzymatic activity. The bile phospholipase activates trypsinogen. In cholelithiasis, attacks of acute pancreatitis may be associated with the transient obturation of the falcon nipple with gallstones. The casting of intestinal contents is possible with the festering nipple gaping or with hypertonic dyskinesia of duodenum arising from inflammation, the effects of nutritional and other factors. At the same time enteropeptidase, which enters the gland, activates trypsinogen. The resulting trypsin has an autocatalytic effect - it activates trypsinogen and other proteolytic enzymes. So, if in the experiment to introduce a small amount of trypsin into the pancreatic duct, then a pronounced necrosis of its tissue occurs, since active proteolytic enzymes are formed.

The third theory explains the development of pancreatitis by pancreatic duct obstruction and hypersecretion. Obstruction (spasm of the sphincter of Oddi, edema of the duodenum, etc.) causes a delay in the secretion of pancreatic secretions followed by activation of enzymes within the gland.

In pancreatitis, 3 stages develop: acute attack (edema, possibly pancreatic necrosis), incomplete cure with persistent chronic inflammation or destruction of the pancreatic duct, and then the stage of chronic inflammation with exocrine pancreatic insufficiency. With the development of fibrotic changes in the gland tissue associated with acute pancreatitis, an exocrine (exocrine) insufficiency of the pancreas occurs, characteristic of chronic pancreatitis. In iron, decreases, and then completely stops (with sclerosis, wrinkling of the body) the formation of digestive enzymes (pancreatic achilles). Violated cavity digestion (in the cavity of the small intestine) and absorption. First of all, the digestion and absorption of fat sharply suffer. Fats up to 60-80% are not digested and in high amounts are excreted with feces (steatorrhea - excretion with feces more than 5 g per day or more than 5-6% of the introduced isotope - trioleate-glycerin). There is polyphecal, with coprologic examination in the feces of a lot of neutral fat (as broken fat splitting to fatty acids). Steatorrhea causes loss of calcium by the body, which is excreted together with fats in the form of insoluble soaps (in feces, in addition to neutral fat, there will be soaps). Along with calcium ions, magnesium and zinc ions are also lost, which also form soaps with unsweetened fats. Developed syndromes of hypocalcemia, hypomagnesemia. To a lesser extent and later digestion of protein is disrupted (not digested up to 30-40%). This is evidenced by the appearance of a large number of muscle fibers in the feces (creators), especially after eating meat. Digestion of carbohydrates is also impaired. There is a decrease in the volume of pancreatic secretion, bicarbonate in pancreatic juice (after stimulation with secretin of 1 mg / kg of weight) and enzymes - amylase, trypsin, lipase (after pancreosimine stimulation 1.5 mg / kg body weight). Digestive disorders are aggravated by a dyspeptic symptom complex. There is a syndrome of diarrhea, maldigestia syndrome develops, there is a progressive loss of body weight (in the absence of substitution therapy).

Violation of the secretory function of the small intestine

Disorders of the secretory function of the intestine may depend on a decrease in the amount of juice that is separated, reducing the content and activity of its enzymes and disturbances in the wall

digestion. They are often caused by intestinal enzymes-inadequate production of enzymes in the small intestine. Enzymopathy can be congenital and acquired.

More often there is a disaccharide deficiency (congenital deficiency of enzymes - disaccharidases) and especially a deficiency of lactase, sucrose and isomaltase. Significantly less frequent is the insufficiency of trehalase, an enzyme that breaks the disaccharide of trehalose found in fungi, algae and insects (in some Eastern peoples its share in food is considerable). With a deficiency of trehalase, mushrooms, especially young ones, are poorly tolerated. Rare forms of peptidase deficiency include congenital enterokinase deficiency (enteropeptidase). Enterokinase is the key enzyme of proteolytic processes in the intestine. It activates pancreatic trypsinogen, converting it into an active proteolytic enzyme called trypsin. In this case, children have severe disorders of protein metabolism, hypoproteinemia, edema, diarrhea, malabsorption syndrome. Patients are treated with pancreas extracts.

Congenital enteropathy includes gluten disease. When this disease develops, the splitting of gluten is broken (a gluten protein component gluing the constituents of some cereals: wheat, rye, barley, oats). There are two main theories of the pathogenesis of gluten disease. According to the first, intestinal epithelial cells involved in the process of digesting gluten are devoid of the corresponding peptidase or protease. In this regard, there is no splitting and subsequent absorption of gluten. The disease is considered as a metabolic defect, because of which undigested gluten and products of its incomplete cleavage have a toxic effect on the mucosa of the small intestine.

According to the second theory, the primary role is played by immunological reactions to gluten. Undivided gluten, interacting with mucosal immunocytes, leads to their sensitization, in particular to the sensitization of lymphocytes. As a result, various products of immunogenesis are formed - antibodies to gluten, immunized lymphocytes, lymphokines, causing damage to the intestinal epithelium with a violation of its digestive and suction functions. There is evidence of the involvement of genetic factors in the pathogenesis of gluten disease. Its main diagnostic criteria are: malabsorption, subtotal or total atrophy of the small intestine mucosa, the clinical effect of the gluten-free diet.

Acquired enzymes can be associated with inadequate production of both (monoenzymopathy) and several (polyenzymopathy) enzymes of intestinal juice. They are accompanied by bloating (flatulence), diarrhea and lead to other manifestations of the syndrome of maldigestia.

Manifestations of intestinal enzimopathy - flatulence, diarrhea and the development of Maldigestia syndrome. Violation of predominantly cavitary digestion (Maldigestia syndrome) occurs due to many reasons: uncompensated reduction of the secretory function of the stomach, small intestine, pancreas, bile secretion. An important role in its occurrence is played by violations of the motor function of the gastrointestinal tract: congestion due to spasm, stenosis or compression of the intestine, acceleration of the passage of food chyme due to increased peristalsis.

In the clinical picture of maldigestia, signs of digestion disorders in various parts of the gastrointestinal tract may predominate. There are gastric, intestinal and pancreatic forms. The appearance of the gastric form is usually associated with atrophic gastritis, leading to secretory insufficiency. Perhaps the development of the gastric form and with decompensated stenosis of the pylorus, stomach cancer. Clinically, it is characterized by loss of appetite, a sense of heaviness, bursting and pressure in the epigastric area after eating, flatulence, diarrhea, belching, air, food with a rotten smell. In the study of gastric secretion reveal Achilia, achlorhydria.

When intestinal form associated with a chronic inflammatory process in the small intestine, with the development of intestinal enzymes, rumbling, intestinal transfusion, bloating, flatulence, unstable stool with prevalence of diarrhea are revealed. When intestinal cavity digestion is disturbed, intestinal steatorea is most often found, when fatty acids, soaps, amylorrhea, creators, and high content of ammonia predominate. The degree of impairment of the cavity digestion is judged by the level of enzymes (enterokinase and alkaline phosphatase) in intestinal contents and feces, as well as by the nature of the glycemic curve under starch loading and by the study of fat absorption by successive loading with trioleate-glycerin and oleic acid labeled.

The emergence of the pancreatic form of maldigestia is associated with exocrine insufficiency of the pancreas. The clinic is dominated by anorexia, meteorism, colicky abdominal pains, abundant "pancreatogenic" diarrhea. When coprological analysis revealed steatorrhea pancreatic type (due to neutral fat), amylorea, creatorrhea. In diseases of the intestine, there is often a combination of all three forms of impaired cavitary digestion.

Disturbance of parietal (membrane) digestion in the intestine

In addition to the violation of the cavity digestion in the intestine (Maldigestia syndrome), there may be a disturbance of the parietal (membrane) digestion, which occurs in the zone of the intestinal epithelium with the help of enzymes fixed on its microvilli and in the glycocalysis. In the glycocalysis enzymes break down the products of cavity hydrolysis - oligomers, formed from large-molecule substances and adsorbed in the zone of striated border of enterocytes, to dimers. On the cytoplasmic membrane of the microvilli, the cleavage proceeds to the final product - monomers that enter the enterocytes and then into the blood and lymph, i.e. Absorbed.

Microvilli of the apical membrane of enterocytes are the smallest cytoplasmic outgrowths, the length of which is 1 μm, the width is 0.1 μm. Thanks to this structure, the active digestive surface increases by 30 times. The distance between the villi varies from 10 to 20 nm, and therefore only small molecules penetrate the brush rim. Microbes, the size of which is several micrometers, are not able to penetrate it - it's a kind of bacterial filter. The processes of wall digestion are performed on a huge surface. The mucosa of the small intestine has folds, villi and microvilli, increasing its inner surface by 300-500 times.

Enzymes that consistently perform parietal digestion have a twofold origin. Some of them are adsorbed from the cavity of the small intestine (where they enter into the pancreatic and intestinal juices), and they bind to the glycocalysis of the microvilli. The other part is transferred from enterocytes (intestinal epithelium), fixing on cytoplasmic membranes of microvilli. The main intestinal enzymes involved in parietal hydrolysis of carbohydrates are D-glucosidases (maltase, trehalase, etc.), β-galactase (lactase), glucoamylase (γ-isoamylase), invertase, etc. Hydrolysis of oligo- and dipeptides is carried out by several pesticides of phosphorus Esters (for example, alkaline phosphatase), and lipids - lipases.

The causes of violation of parietal digestion can be:

1) violations of the structure of villi and microvilli, a decrease in their number per unit surface (AM Ugolev). This is a characteristic sign of chronic diseases of the small intestine, where the morphological substrate is inflammatory, dystrophic and sclerotic changes in the mucosa. The development of atrophic changes in the mucosa of the small intestine, predominantly villi, is noted in dysentery, cholera;

2) alteration of the enzymatic layer of the intestinal surface as a result of genetic or acquired deficiency of enzymes involved in parietal digestion. Primary insufficiency of parietal digestion, as a rule, develops in children at an early age with the expansion of the diet with the inclusion of new products containing the intolerant disaccharide. Acquired insufficiency is more often a consequence of diseases of the small intestine - chronic enteritis, as well as viral hepatitis and other infections;

3) disorders of intestinal peristalsis, which leads to disruption of the transfer of nutrients from the intestinal cavity to the surface of enterocytes, for example, chronic enteritis, Whipple's disease, Crohn's disease and other diseases of the small intestine;

4) Insufficiency of the cavitary digestion, when the little-split large molecules do not pass into the brush border of the villous epithelium.

The clinical picture of insufficiency of parietal digestion is similar to dyspepsia in the syndrome of insufficiency of absorption. There are persistent diarrhea, feces liquid, abundant, foamy. In order to clarify the diagnosis, the activity of enzymes (amylase, lipase) is determined upon their sequential disorption in homogenates of the biopsy of the small intestine mucosa obtained by inoscopy. Part of the biopsy specimen is examined morphologically, which allows detecting inflammatory, atrophic changes in the mucosa. Comparison of the activity of the enzymes in the desorbed fractions allows us to derive the activity curves of the enzymes, which characterize

the relationship between cavity and membrane digestion. The violation of parietal digestion in chronic enteritis is also determined by other methodical techniques.

Disturbance of absorption in the intestine

Disturbances of absorption are manifested in its slowing down or pathological enhancement. Slowing down the suction is the basis of the malabsorption syndrome (from the French mal - disease), caused by a violation of absorption in the small intestine of one or several nutrients. The range of clinical manifestations of malabsorption syndrome varies from the absence of its visible signs to the expressed loss of body weight. It combines the symptoms of diarrhea, steatorrhea, protein deficiency, hypovitaminosis. Malabsorption syndrome may be primary (congenital or hereditary) or secondary (acquired). Congenital malabsorption is rare in clinical practice. Most often this is the pathology of childhood, due, for example, to congenital disruption of transport (insufficiency of transporter vectors) of amino acids in the small intestine. So, this syndrome is associated with a violation of absorption of neutral amino acids (Hartnap's disease - pellagra skin changes, cerebellar ataxia); Syndrome of absorption disorders of cysteine and basic amino acids, syndrome of decreased absorption of many amino acids (Low syndrome - congenital cataract, glaucoma, hypertension, osteoporosis, mental retardation), decreased absorption of lysine (congenital lysinuria - protein intolerance, diarrhea, vomiting, retardation) and Other congenital malabsorption of glucose and galactose is possible. In the small intestine mucosa of such patients, the enzyme glucose-6-phosphatase is absent. With a congenital impairment of absorption of fructose in the mucosa, there is a deficiency of fructose-1-phosphataldolase, responsible for its transport. There is an isolated violation of absorption of these substances, there is diarrhea and abdominal pain. Primary malabsorption of vitamin B12 or folic acid leads to the development of megaloblastic anemia.

Secondary impairment of absorption is more common. It is associated with such diseases of the intestine, liver, pancreas and other organs as:

1) insufficient digestion of food in the stomach (due to achlorhydria, subtotal resection of the stomach, trunk vagotomy) or duodenum;

2) exocrine pancreatic insufficiency (chronic pancreatitis, cancer, cystic fibrosis, pancreas resection);

3) liver disease (chronic hepatitis, cirrhosis) and biliary tract obstruction (gall bladder stones or pancreatic head cancer), which is associated with insufficiency of bile acids entering the duodenum;

4) ischemic enteropathy with possible intestinal infarction (eg, lead poisoning, mesenteric atherosclerosis);

5) inflammation of the small intestine of various etiologies (acute and especially chronic enteritis with the development of changes in the small intestine mucosa down to atrophy, which reduces its suction surface), Crohn's disease (with duodenum or ileum injury);

6) dysbacteriosis, when absorption of fat and vitamin B12 is particularly affected, as microbes cause deconjugation of bile acids in the intestine and absorb vitamin B12;

7) radiation (radiation) enteropathy associated with irradiation of the intestine, for example, in the treatment of cancer, which causes swelling of the mucosa, later - atrophy of the villi and thinning of the mucous membrane. The defeat of the ileum leads to a deficiency of vitamin B12 and impaired intestinal hepatic exchange of bile acids;

8) resection of the small intestine (short bowel syndrome) associated with trauma, small intestinal obstruction, vascular thromboembolism, severe Crohn's disease, etc.;

9) intestinal obstruction in the upper parts of the intestine, when the food masses do not enter the distal parts of the gut;

10) movement disorders of the intestine, in particular with accelerated peristalsis, when the contact time of the chyme with the absorption surface of the small intestine decreases;

11) lymphatic obstruction (lymphangiectasia of the intestine, Whipple's disease, lymphoma);

12) cardiovascular diseases (pericarditis, congestive heart failure, vasculitis);

13) immunodeficiency, endocrine disorders (diabetes mellitus, hypo- and hyperparathyroidism, Zollinger-Ellison syndrome).

As a result of malabsorption, malabsorption syndrome develops, which, in addition to changes in the gastrointestinal tract, is characterized by pathological changes from other organs and systems.

Often there is bloating, usually after eating, associated with milk intake, increased gas production. Diarrhea is noted in connection with the accumulation of osmotically active substances in the intestinal cavity, acceleration of transit through the intestine and hyperexudation. There is polyphecal with the remnants of undigested food. There is a steatorrhea - a sign of a violation of fat absorption (fat loss with feces is more than 5 g / day, reaching 10 g / day or more).

An important clinical symptom of malabsorption is weight loss (at I degree of malabsorption - up to 5-10 kg, at grade II - over 10 kg, at grade III - over 20 kg). There are signs of hypoavitaminosis, trophic disorders. The skin becomes dry, with a decreased turgor, hair - dry, dull, hair loss is noted. There are changes in nail plates, their fragility, as well as gum disease, hyperemia of the tongue, smoothness of its papillae, which is explained by the deficiency of vitamins B2, B6, B12, nicotinic acid. There is bleeding gums associated with vitamin C deficiency. Polyneuritis, visual impairment caused by vitamin A deficiency is often developed. In severe malabsorption syndrome, the absorption of trace elements deteriorates. As a result of calcium deficiency, osteoporosis occurs, right up to osteomalacia. The violation of iron absorption leads to the development of iron deficiency anemia. Due to impaired absorption of proteins, hypoproteinemia develops with subsequent edematous syndrome. There may be abnormalities in the endocrine glands activity as a type of plurigandular insufficiency - the development of endocrinopathy with damage to the pituitary gland, adrenals, and gonads.

Pathogenesis of clinical manifestations of insufficiency of absorption

Pathogenesis	Manifestations of insufficiency of absorption
Infringement of absorption of fats, carbohydrates, proteins, decrease in receipt in an organism of calories	Weight loss
Impaired absorption of amino acids, hypoproteinemia	Peripheral edema
Vitamin D deficiency, osteoporosis and osteomalacia as a result of malabsorption of proteins and calcium.	Ossalgia (pain in the bones), myopathy
Deficiency of B vitamins	Peripheral neuritis
Impaired absorption of calcium and magnesium	Paresthesia, tetany
Reduction of absorption of proteins, vitamin B12, folic acid, iron	Anemia
Impaired absorption of vitamin K, vitamin A deficiency	Hemorrhages. Night blindness (Hemerallopia, xerophthalmia)
Deficiency of riboflavin (B2)	Heilit
Deficiency of vitamins B2, B6, B12, nicotinic acid	Glossitis
Deficiency of nicotinic acid	Dermatitis

Pathological increase in absorption can be associated with increased permeability of the intestinal wall (for example, with its arterial hyperemia or irritation of the intestinal epithelium). Intensification of absorption is easily developed in young children, when the permeability of the intestinal wall is quite high. At the same time, products of incomplete cleavage of nutrients are rapidly absorbed and intoxicated. Unchanged form can be absorbed protein cow's milk or chicken eggs, which causes sensitization of the body with the development of allergic reactions.

Violation of the motor function of the intestine

Motor activity of the small intestine provides mixing of food contents with digestive secretions, promotion of chyme and increased intestinal pressure, which facilitates the filtration of certain components into the blood and lymph.

Disorders of the motor function of the intestine are manifested in the acceleration or deceleration of peristalsis and the alternation of these processes, as well as in the disturbance of rhythmic segmentation, which occurs due to a predominantly circular layer of muscles and pendulum contractions that ensure the interaction of the longitudinal and circular layers of muscles. On the length of the intestine, several peristaltic waves are moving at the same time. In disorders of motor activity of the intestine, anti-peristaltic contractions are noted, when the wave of movement goes in the opposite (oral) direction. Tonic contractions can have a very small speed and sometimes do not spread, which causes the narrowing of the lumen of the gut on a large extent.

Motor activity of the intestine is excited through parasympathetic nerve fibers. An important role of the cerebral cortex in the regulation of motor activity is proved by the fact that motor activity is strengthened even at the thought of tasty food, and, in the negative attitude towards food, on the contrary, is inhibited. With fear, there is sometimes a turbulent peristalsis of the intestine ("nervous diarrhea"). The motor activity of the small intestine depends on the physical and chemical properties of the chyme. So, its activity is increased by rough food (black bread, vegetables) and fats. Intestinal motility is affected by a number of humoral substances, acting directly on the muscle fibers and through the receptors on neurons of intramural nervous ganglia. Thus, increased motility of the small intestine is observed with an increase in the level of vasopressin, bradykinin, serotonin, histamine, cholinomimetics, cholecystokinin - pancreosimin and peptides (motilin, gastrin). Usually the main empirical rhythm is constant - about 8 cuts per minute. However, in a number of cases it is more frequent, for example, with thyrotoxicosis.

The inhibition of motor activity of the intestine occurs under the influence of sympathetic fibers. Motor activity decreases with fasting. In a person after 24-36 h fasting, it is 34% of the initial.

The motor (motor) function of the small intestine plays an important role in the effectiveness of nutrient absorption from its lumen. Due to the contractile function of the intestine, the content is mixed and promoted in the intestinal cavity, which does not allow the formation of a high concentration of hydrolysis products in one wall layer, creating a diffusion barrier. Under experimental conditions, it has been proved that at a high rate of transit of chyme in the gut, its ability to absorb decreases. For example, this happens when including coarse-fiber products in the diet. The content of glucose in the blood becomes 2 times less than with a diet without coarse fibers.

Acceleration of peristalsis. As a result of the acceleration of peristalsis, the food gruel advances through the intestine more rapidly and develops diarrhea (diarrhoea).

Diarrhea can be acute (not exceeding 2-3 weeks) and chronic (lasts 4-6 weeks or more), infectious and non-infectious, inflammatory and non-inflammatory. By the mechanism of development, the following types of diarrhea are distinguished: hypersecretory (hyperexudative) and hyperosmolar, hypo - and hyperkinetic.

Hypersecretory type of diarrhea is characterized by increased secretion of water and electrolytes into the lumen of the intestine. This is due to the effect on the intestinal mucosa of bacterial endotoxins (with cholera, intestinal infections), bile and fatty acids, glucagon, prostaglandins and a whole series of laxatives (bisacodyl, castor oil, phenolphthalein). The pathogenesis of bacterial diarrhea is due to two mechanisms: invasion of bacteria in the mucosa and hypersecretion caused by enterotoxins. Hyper-secretory diarrhea is also observed with an increase in hydrostatic pressure due to damage to the lymphatic system of the intestine (with lymphoectasis, intestinal amyloidosis, lymphoma, Whipple's disease) and right ventricular heart failure. The stool is usually abundant in this type of diarrhea, watery.

Very severe diarrhea (the so-called aqueous diarrhea) can be caused by excessive production of the vasoactive intestinal polypeptide, which is normally contained in the gastrointestinal tract,

mainly in the wall of the small intestine. It suppresses the secretion of hydrochloric acid, stimulates intestinal and pancreatic secretions, increases the concentration of cAMP in the small intestine mucosa. The vasoactive intestinal polypeptide (VIP) produces some tumors - ganglioneuroblastoma (more often in children) and adenoma of islet tissue (not α- and not β-cell) of the pancreas - WIPoma (Werner-Morrison syndrome - "pancreatic cholera"). By affecting specific receptors of the intestinal epithelium, the VIP activates adenylate cyclase and increases the level of cAMP. This causes an increase in the secretion of water and electrolytes, resulting in the development of profuse watery diarrhea (the osmotic density of the stool is close to the osmotic density of the plasma). There comes dehydration (more than 3 liters per day, sometimes up to 20 liters), hypokalemia (increased loss of potassium and stool), metabolic acidosis, cachexia (in the absence of steatorrhea) progresses. Due to the influence of VIP on the vascular tone, some of the patients experience hot flashes (a feeling of heat for 2-3 min) with a purple dyeing of the face and upper half of the trunk, while the other part develops diabetes mellitus. An elevated level of circulating VIP is defined, but this index has a high percentage of false positive and false negative results. Chronic diarrhea can also be a manifestation of other endocrine tumors that produce secretion stimulants. For example, in thyroid carcinoma diarrhea is caused by increased secretion of calcitonin and other peptides.

With excessive formation of cAMP, diarrhea is associated with cholera. The cholera vibrio toxin (cholerogen) in combination with the specific receptor with C1m1-ganglioside activates adenylcyclase catalyzing the formation of cAMP. Heavy watery diarrhea develops. It should be noted that with cholera, the intestinal mucosa remains normal and its absorption capacity is preserved. This creates a basis for oral rehydration with solutions containing simple sugars and sodium chloride "(the former stimulate the absorption of the latter).

With a hyperosmolar type of diarrhea, there is a decrease in the absorption of water and electrolytes. This type of diarrhea is noted in cases of absorption disorders, which is observed in cases of gluten, ischemic disease of the small intestine, congenital suction defects, chronic pancreatitis, pancreatic cancer, bile acid deficiency (eg, mechanical jaundice), insufficient contact time of the chyme with the intestinal wall With resection of the small intestine, enteroanastomoses), etc. There are polyphecal and steatorrhea. Thus, with resection of the ileum and some diseases of the small intestine (for example, with Crohn's disease) diarrhea can occur due to a violation of absorption of bile acids and free fatty acids that stimulate the secretion of fluid in the large intestine. In mild cases, the absorption of bile acids is inhibited. In particularly severe cases (resection of more than 100 cm of the terminal ileum), absorption of both bile acids and salts deteriorates, which in turn causes a violation of the digestion and absorption of fatty acids. The latter, getting into the large intestine, cause diarrhea. In other forms of steatorrhoea, for example in pancreatogenic insufficiency, unabsorbed triacylglycerols reach the colon, where they are hydrolyzed by microorganisms to fatty acids, which also causes diarrhea.

Hypo - and hyperkinetic types of diarrhea are caused by stimulation: neurogenic, for example, in irritable bowel syndrome, diabetic enteropathy; Hormonal (serotonin, secretin, pancreosimine); Pharmacological (laxatives isofenin, phenolphthalein). It is possible to slow the transit of intestinal contents in scleroderma, the syndrome of the cecum. The stool is usually liquid or mushy, ungrowth.

Slowing of peristalsis. When the peristalsis slows down, the food chyme moves along the intestine, and constipation (obstipatio) develops. With constipation, the intervals between acts of defecation increase in comparison with the individual physiological norm or the intestine is systematically insufficiently emptied. The frequency of the stool is very variable and can vary depending on the habit of emptying the intestine after a certain time, the nature of nutrition, climatic and other factors. Most people have a chair once a day, a part - 2 times and a significantly smaller percentage (7%) - 3 times a day or more. As a rule, a chronic bowel evacuation delay of more than 48 hours is considered as constipation.

The terminal section of the food tract, carrying out the absorption of water and mineral salts, takes part in the regulation of water-salt metabolism. The main functions of the colon (formation, promotion, retention and release of stool) are realized by the interaction of the following

components of motility - the tone of the intestinal wall, different in strength and length of peristaltic waves, their coordination and discoordination. The motor activity of the large intestine is influenced by nervous, endocrine, physical and nutritional factors. In addition, it is characteristic to participate in the regulation of the motility of the microflora and the emotional-psychological sphere of man.

The etiology and pathogenetic factors are most fully taken into account by the classification of A.V. Frolkis, which produces constipation: 1) alimentary; 2) neurogenic (dyskinetic, reflex), due to suppression of urge to defecate, with organic diseases of the central nervous system; 3) hypodynamic; 4) due to inflammatory bowel diseases; 5) proctogenic; 6) mechanical; 7) due to anomalies in the development of the large intestine; 8) toxic; 9) medicamentous; 10) endocrine; 11) due to violations of water-electrolyte exchange.

Constipation may be a manifestation of mechanical obstruction in the intestine: sigmoid colon swelling, diverticulitis, invagination, hernia, swelling, scars, stools, etc. Isolate proctogenic constipation, i.e. Caused by pathological processes in the anal area of the rectum (hemorrhoids, anal fissures and perianal abscesses). They are associated with the suppression of urge to defecate because of severe pain and spasm of the anal sphincter, which mechanically prevents the release of stool. Possible so-called senile constipation associated with intestinal atony. Disorders of the motor activity of the intestine can also be associated with endocrine pathology. Constipations of endocrine origin include dyskinesia of the intestine in women during pregnancy, after childbirth, in the climacteric period. Thus, during pregnancy and after childbirth there is a relative hypotonia of the musculature of the intestine, caused by hormonal changes in the body of a woman. Chronic constipation develops in hypothyroidism, which is associated with a characteristic slowing of transit through the intestines. A similar situation occurs with hypercalcemia.

By the nature of motor disorders, hyper- and hypokinetic constipation is distinguished. Hyperkinetic constipation occurs with spasm of the intestinal wall, which hinders the progress of food chyme along the intestine. Spasm often develops in areas of the intestine, where there are strengthened contractions (sphincter of Bali, passage of the caecum into the ascending and colon, etc.). Hyperkinetic type of constipation is possible in case of poisoning with mercury, lead, sulemoy, when taking medications (iron, calcium, tranquilizers, ganglion blockers, etc.). Perhaps the development of this type of constipation under the influence of emotions and psychotic states (psychogenic constipation). They arise as a reaction to unfavorable conditions for evacuation of the intestine, i.e. Negative emotions, for example, when it is necessary to perform an act of defecation in unhygienic conditions, can lead to its involuntary suppression. With multiple "braking" defecation, desires disappear and habitual constipation develops. This type of constipation can occur under the influence of other psychogenic factors (mental overstrain, depression, schizophrenia, drug addiction), and also can be associated with the influence of viscer-visceral reflexes from the stomach, pancreas, biliary tract, etc.

The change in the volume of intestinal contents, the composition of the intestinal microflora, and the breakdown of gastrointestinal reflux can lead to a weakening of propulsive motility, i.e. To the development of hypokinetic constipation. Quite often, constipation leads to a meager diet, the intake of easily digestible, fiber-poor food (mechanically and chemically sparing diets). The use of chemically purified, completely water-soluble products used in space flights, causes a reduction in stool frequency up to 1 time in 5-7 days, there are alimentary constipation. In the summer, when a lot of fruits and vegetables are consumed, the frequency of constipation decreases. The role of dietary fiber in the stimulation of bowel evacuation is proved. Bran increases the daily amount of feces, accelerates intestinal transit. Constipation aggravated dryness (drying of stool), a lack of calcium and potassium in the diet, excessive digestion of food masses in the stomach, for example, with hyperchlorhydria. In addition, inadequate, as well as untimely consumption of food leads to a violation of gastrointestinal reflux, which stimulates large peristaltic waves. Therefore, people neglecting breakfast, irregularly eating food, often suffer from constipation. Hypokinetic constipation occurs in the absence of physical exercises (with hypodynamia).

Primary motor disorders of the anorectal region and pelvic floor include congenital disorders of intestinal motility in Hirschsprung's disease, the mobile blind and sigmoid colon and congenital

splanchnoptosis. With Hirschsprung's disease, an anomaly of colon development is noted, characterized by chronic congestion of intestinal contents, expansion of the colon with hypertrophy of its wall. The essence of the disease is the complete absence or deficiency of intramural nerve ganglia. Thus, there are no ganglion cells of Auerbach's plexus completely absent in the internal anal sphincter, rectus and sigmoid colon. The affected area of the intestine is narrowed, not peristaltic, the intestinal contents over the site of the lesion stagnate, the overlying parts of the large intestine (megacolon) expand. The wall of the intestine is hypertrophic, since the peristalsis is enhanced by the need to overcome the narrowed non-irritating site. It is established that in this area the concentrations of VIP and substance P are sharply reduced, which normally stimulate intestinal motility. With a long zone of damage, the picture of intestinal obstruction grows. Stools usually do not happen 3-7 days, in rare cases it is independent, mostly - only after enema.

Intestinal obstruction (ileus) - impaired intestinal passability due to a violation of its functions or mechanical obstruction. Intestinal obstruction can be congenital, which is caused by abnormal development of the intestinal tube during the intrauterine period, and acquired. Acquired intestinal obstruction by pathogenesis is divided into mechanical, dynamic and thromboembolic.

Mechanical obstruction is associated with mechanical closure of the lumen of the intestine with a tumor, calic stones (coprostasis), helminths, foreign bodies, or due to compression of the gut from the outside by a tumor, scar. Mechanical obstruction develops when the intestine turns, intussusception, infringement of the intestinal loop in the hernial opening, with adhesive process in the abdominal cavity. Allocate its following reasons: 1) intestinal compression of the gut, for example, with adhesions of the abdominal cavity, hernia (external and internal); 2) internal compression of the intestine (diverticulosis, cancer, regional enteritis, or Crohn's disease); 3) Obturation, for example, gallstones or with intussusception.

Most often, the causes of the obstruction of the small intestine are adhesions of the abdominal cavity and external hernias, and the colon - a cancerous tumor, diverticulitis (sigmoid colon) and a curvature. Mechanical obstruction can be obturation and strangulation. With obstructive obstruction, the lumen of the intestine is closed, but the circulation in its wall is not initially disturbed, with strangulation, along with obstruction of the intestinal lumen, compression of the vessels and nerves of the mesentery occurs, which causes an extremely severe clinical picture. Rapid disruption of intestinal wall feeding leads to its necrosis. With mixed obstruction, along with the overlap of the lumen of the gut, there is a gradual compression of her mesentery with a violation of the blood supply to the intestinal wall.

Dynamic obstruction occurs with spasm (spasmodic), which can occur when heavy metals are poisoned by diseases, biliary tract and other abdominal organs, or paralysis of the intestinal musculature (paralytic), when the intestinal peristalsis sharply weakens until complete cessation. This occurs with severe long-term operations on the abdomen, trauma.

Thromboembolic (haemostatic) obstruction of the intestine develops as a result of circulatory disturbances in the intestinal wall with thrombosis (embolism) or paralysis of its vessels. Thrombosis or embolism of the intestinal arteries can be a manifestation of severe atherosclerosis, heart failure, may complicate atrial fibrillation, implantation of artificial heart valves or severe heart defects. Involvement in the process of large arterial vessels of the intestine is possible with systemic vasculitis.

The pathogenesis of intestinal obstruction is complicated. There is a stretching of the intestine with the accumulation of gases and liquid contents in it proximal to the septic segment. The accumulated fluid in the intestine consists of saliva, gastric juice, bile and pancreatic enzymes. In the first 12-24 hours of obstruction the motor activity of the intestine decreases, the transport of sodium and, consequently, the water from the lumen of the enlarged colon into the blood slows. After 24 hours, sodium and water accumulate in the gut lumen, which is accompanied by its stretching and loss of fluid. Intestinal pressure increases, vomiting occurs. There comes a strangulation (blood circulation is sharply disturbed) in connection with the expressed stretching of a gut proximal to a site of occlusion. Intramural blood flow is reduced to such an extent that bowel necrosis occurs. When the blood supply is disturbed, the pathogenic bacterial flora multiplies with

subsequent development of peritonitis. The high standing of the diaphragm due to swelling of the intestine causes a violation of pulmonary ventilation with the development of atelectasis in the lungs. The outflow of blood in the system of the inferior vena cava is disturbed. Loss of body tissues fluid and electrolytes can be pronounced. As a result, dehydration and thickening of blood quickly occur. In the blood, the chloride content decreases, which, together with water, passes into the abdominal cavity, the content of ammonia, urea and other rotting products that are formed in the intestine and is absorbed into the blood increases. Heavy intestinal toxicity develops. Increasing hypovolemia leads to the development of acute renal failure, shock and death of the patient. With complete obturation, gases and feces do not depart. Blood in the feces is rarely detected, only occasionally with an invagination form of occlusion. It is possible to vomit, more often with the obstruction of the small intestine than thick.

Isolate pseudo-obstruction of the intestine, which is based on pronounced motor disorders, which contributes to stretching of the intestine, the occurrence of abdominal pain, nausea and even vomiting. Pseudo-obstruction can be primary and secondary. In primary, or idiopathic, pseudo-obstruction, impaired motor activity of the intestine is caused by an anomaly of sympathetic innervation or its muscular layer, and the patient does not have any systemic disease. In secondary pseudo-prolapse, the expansion of the thick and / or small intestine is associated with the involvement of the muscle layer in the process, for example, in autoimmune diseases (dermatomyositis, scleroderma, amyloidosis) or autonomic visceral nervous system in endocrine diseases - diabetes, myxedema. Secondary pseudo-obstruction may develop in chronic diseases of the nervous system (Parkinson's disease, cerebrovascular disease), and may also be associated with side effects of a number of drugs (calcium antagonists, cholinolytics, (5-adrenoblockers, psychotropic, etc.).

Violations of defecation. May occur in the following situations:

1) with strong mental shocks (fear, fright), the influence of the cerebral cortex on the spinal center of defecation falls, and at the same time defecation occurs involuntarily (reflexively);

2) with n damage. Pelvici, n. Hypogastrici defecation is disrupted, as the function of the muscles participating in this act is upset;

3) inflammatory processes in the rectum (with proctitis of any etiology) increase the sensitivity of its receptors, and there are false desires for defecation (tenesmus);

4) with injuries of the lumbosacral spinal cord due to the deenergizing of the center of defecation, incontinence occurs, or there is no urge to defecate. In addition, in connection with the abnormality of the muscles of the abdominal press, emptying the rectum may be incomplete, and constipation may occur;

5) with a decrease in muscle tone and physical activity in elderly, recumbent patients, the act of defecation is broken, there is a feeling of overflow of the rectum, the urge to defecate, constipation develops or paradoxical diarrhea (when liquid feces passes through the distal fecal stones).

Flatulence (accumulation of gases in the intestine, its swelling). A large number of gases pass through the digestive tract of healthy people every day. Gases enter the intestine along with the inhaled air and partially diffuse out of the blood. A certain amount of gases is formed in the intestine as a result of enzymatic processes and the vital activity of the intestinal microflora. On average, about 500 cm3 of gas is generated per day. However, this can be enhanced by aerophagia or increased formation of gases by intestinal bacteria. Gas formation increases with malabsorption syndrome, especially when eating foods such as beans, peas, cauliflower and cabbage, characterized by a high content of indigestible polysaccharides. When the gases in the intestine are held in a state of fermentation and decay, the amount of carbon dioxide, methane, and hydrogen sulphide increases. At the same time, blood circulation in the intestinal wall is disrupted, its mechano - and chemoreceptors are re-stimulated. There can be a happy reflex shifts: inhibition of diuresis, instability of arterial pressure. Due to the high standing of the diaphragm, breathing is disturbed. The secretion of digestive glands is inhibited, hypotension of the intestine is intensified, which further aggravates flatulence. A "vicious circle" is being created.

Violation of the excretory function of the intestine

The main mechanisms of excretion (sorption of substances by membranes, various types of membrane transport) underlie the interorganic exchange of nutrients, the leading role in which the digestive system plays. The term "nutrients" (nutrients) in this case is more accurate, since it comes from the word "nutrition" - nutrition. It is known that in the activity of the gastrointestinal tract, in addition to proper digestion, there is another side associated with the release into its cavity of a significant amount of endogenous substances which, together with the exogenous digest, are absorbed and promote assimilation of nutrients throughout the body. The body has developed a coordinated interaction between different bodies to obtain the necessary nutrients and supply them with each other, which contributes to the normal course and good coordination of metabolism throughout the body. Some tissues, thanks to their specialized metabolism, are able to synthesize intensively certain substances and not only satisfy their own need, but also export them with blood for use by other tissues. For example, the muscles in fasting release into the blood amino acids with a branched carbohydrate chain, heavily used by the brain. The kidneys are intensively secreted into the blood of the series, which is then used by almost all the tissues of the body.

Participation of the digestive system in the interorgan exchange of nutrients is expressed: a) in the protein substance circulation between the blood and the digestive system; B) in the circulation of certain mineral substances, in particular zinc; C) in the export of phospholipids and other compounds by the liver for use by other, rapidly proliferating tissues; D) in the isolation of endogenous nutrients during the periodic activity of the gastrointestinal tract in conditions of famine.

The nutritional cycle is of clinical interest. Wherever there is a violation of their circulation (in places where endogenous substance is released, its transport or in the intestinal absorption zone), in all cases a secondary insufficiency of the substance in the body may occur.

The role of the intestine in the metabolism is determined not only by its absorbing activity, but also by the ability to secrete protein and other substances from the blood. The role of the digestive tract in the loss of endogenous protein is proved. For an objective assessment of the role of the gastrointestinal tract in the digestion and absorption of protein, it is necessary to know the rate of synthesis and decomposition of the protein in the body. The results of the study with labeled albumin indicate that in norm 10-20% of albumins can be excreted through the intestine. Isolation of protein from the bloodstream into the gastrointestinal tract is accomplished by simple diffusion. In this case, transudation occurs not through the cells of the mucosa, but through the intercellular space of its epithelium. The amount of protein passing into the intestine is proportional to the hydrostatic pressure and plasma protein concentration in the extravascular space. It is estimated that for a day in physiological conditions, 80 g of protein are excreted in the human cavity of the digestive tract. However, in a healthy person, its loss with feces is negligible. Most of the protein released into the gastrointestinal tract undergoes enzymatic digestion to amino acids that are absorbed along with the products of hydrolysis of exogenous nutrients. This process helps to ensure the consistency of amino acid homeostasis. Part of the protein enters the gastrointestinal tract with the secretions of the digestive glands, part is lost with the cells of the desquamated epithelium. In addition, in the lumen of the intestine, about 20 g of fat are released per day. Experimental studies have shown that cholesterol, triacylglycerols, a number of phospholipids are excreted only in the composition of the sloughing epithelial cells, and only free fatty acids can be released by transudation. In healthy people, lipids released into the intestine are almost completely reabsorbed, and their loss with feces is negligible. With depleted cells enter the lumen of the gastrointestinal tract and other substances - iron, folic acid, etc.

With an increase in the excretory function, the syndrome of "exudative enteropathy" can develop - protein-destroying enteropathy - protein loss due to increased release into the gastrointestinal tract. The main mechanism of increased protein loss with feces is an increase in its transudation into the lumen of the gastrointestinal tract, which occurs when the pressure in the lymphatic vessels of the intestine increases. In this regard, protein intussusception in the intestine is

enhanced in patients with obstruction or stasis of the intestinal lymphatic pathways (granulomatosis, neoplastic processes, lymphangiectasia in the intestine, constrictive pericarditis). It is possible to exudate the protein through the inflamed or ulcerated mucosa. Proteins are lost when the mucous membrane is destroyed (villous and superficial epithelium), which disturbs the regulation of their diffusion through the intercellular spaces.

As an independent disease, this syndrome is rare (primary exudative enteropathy - idiopathic intestinal lymphangiectasia). Secondary exudative enteropathies are quite common - about 90 diseases are accompanied by this syndrome. Symptomatic exudative enteropathies are observed in giant hypertrophic gastritis (Menetries disease), in conditions after gastrectomy, in a number of bowel diseases (tropical, nontropical spruce, acute gastroenteritis, Crohn's disease, ulcerative colitis), with cirrhosis, constrictive pericarditis, heart failure, with Generalized lymphosarcomas, nephrotic syndrome, radiation sickness, etc.

Due to the loss of protein with feces (proteinuria) in exudative enteropathy, pronounced hypoproteinemia with a significant decrease in albumin content in the blood and dysproteinemia takes place. The pronounced hypoproteinemia causes a decrease in the blood oncotic pressure followed by the transudation of liquid from the capillaries into the tissues, with the development of edema, secondary aldosteronism with a delay in the release of water and sodium ions from the body. With feces, a number of important biologically active substances of protein nature are lost, primarily immunoglobulins. This causes a decrease in the concentration of immunoglobulins of various classes, transferrin, ceruloplasmin, etc. Due to hypogammaglobulinemia, immunoglobulin deficiency, there are immunodeficiency states with a tendency to secondary infections. The content of lipoproteins and cholesterol in the blood also decreases. Along with proteinuria, a marked enzyme is noted. Patients lose digestive enzymes (enterokinase, etc.), a number of enzyme inhibitors (alpha-antitrypsin, etc.), calcium and other substances with feces.

The rate of synthesis and destruction of albumin is determined by the administration of radioactive albumin intravenously to study the level of radioactivity in serum. Exudative enteropathy is confirmed by a reduced amount of intravascular and total pool of albumin, its constant or accelerated synthesis, a markedly shortened period of "life" and an increase in protein losses with feces. A non-invasive method for diagnosing exudative enteropathy, which does not require the use of radioactive isotopes, was also developed. The method is based on the determination of the intestinal clearance of alpha1-antitrypsin. This antiferment, when it is determined in feces and blood, becomes an endogenous marker of protein loss with feces. Its content in healthy individuals is 0.4 mg / g dry weight of stool, with intensive lymphangiectasia - 16.2 mg / g, in patients with graft-versus-host disease with exudative enteropathy - 18.8 - 38.8 mg / g.

Intestinal Intoxication

Intestinal autointoxication develops with a decrease in intestinal secretion, intestinal obstruction, mechanical and toxic damage to the intestinal mucosa, etc. Gastrointestinal tract in humans and animals is a natural habitat for microorganisms. Especially rich in the microflora is the large intestine. In vertebrates, the number of microbes in it is 1010-1011 / g of intestinal contents. In the small intestine, their amount is much less due to the bactericidal properties of gastric juice and, probably, endogenous antimicrobial factors of the small intestine. For a day with feces, trillions of bacteria are excreted. The microflora of the intestine causes in it the processes of fermentation and putrefaction, but in norm they are not expressed clearly. The resulting toxic substances are eliminated from the body or rendered harmless, and intoxication does not occur. The processes of fermentation and putrefaction intensify with a decrease in intestinal secretion and an increase in flatulence, which usually accompanies constipation. The most pronounced intoxication is with intestinal obstruction. Essential is the mechanical and toxic damage to the intestinal mucosa. In the pathological process, the nervous apparatus of the intestine is involved, which leads to disruption of its motor and secretory functions and aggravates trophic disorders in the intestinal wall. Develops dysbacteriosis, characterized by a decrease in the number of microorganisms that are constantly

present in the intestine (bifidumbacterium, E. coli, lactobacilli). The ratio of bacteria in different parts of the intestine with increased reproduction is conditional-pathogenic and the appearance of pathogenic flora. Secondary fermentopathy occurs. All this leads to an intensification of the processes of fermentation and decay. Amino acids turn into toxic substances: hydrogen sulphide, skatole, cresol, indole, phenol, etc. When decarboxylating amino acids, biogenic amines are formed: histamine, cadaverine, putrescine. Partially they are rendered harmless in the intestinal wall under the influence of aminoxidase. However, with an excess of these substances, they are absorbed into the blood and through the portal vein system enter the liver. In the liver, indole and scatol are rendered harmless by binding sulfuric and glucuronic acids (indoxylsulfur, skatoxylsulfur, indoxylglucuronic acid and skatoxyl glucuronic acid are formed). Other toxic substances in the liver are deaminated, oxidized, and also transformed into harmless compounds. Partly they are excreted by the kidneys. If a lot of toxic substances are formed and the processes of putrefaction in the intestine continue for a long time, then there is an overload of the detoxifying function of the liver. With the development of hepatic insufficiency, the main importance for the removal of toxins circulating in the blood is acquired by the kidneys. But if the functional state of the kidneys suffers, then the phenomena of intestinal intoxication are increasing. Being in the intestine, toxic substances reflexively influence various organs and systems. In addition to feelings of spreading in the abdomen, swelling, rumbling in the intestines, nausea, there is an unpleasant taste in the mouth, there are weakness, weakness, fatigue, headaches, decreased appetite, insomnia, depression. With chronic intestinal intoxication, dystrophic changes in organs, including the myocardium, can occur.

The toxic substances circulating in the blood act on the receptors of the vessels and the centers of the brain. This can lead to violations of the cardiovascular system in the form of lowering blood pressure, weakening heartbeats. Possible respiratory depression. Reducing glycogen stores in the liver and hypoglycemia can lead to a coma. Chronic intestinal intoxication leads to anorexia and severe digestive disturbances due to suppression of the glands of the digestive tract.

CONSEQUENCES OF DELETING VARIOUS DEPARTMENTS OF THE GASTROINTESTINAL TRACT

The first experiments to study this issue were carried out in the experimental laboratory of E.S. London, Removing the cardiac part of the stomach in dogs led to bulimia and polyphagia. Eating often ended with vomiting, which resembled an attack of suffocation and was accompanied by a contraction of the neck muscles. The peculiarities of vomiting were explained by the fact that it arose in the presence of food in the esophagus (esophageal vomiting).

Turning off the gatekeeper (imposing gastroenteroanastomosis) caused a violation of the evacuation of food chyme from the stomach, the more pronounced anastomosis was applied to the lower part of the small intestine. Removal of the bottom of the stomach led to a violation of its reservoir and secretory functions. The disorders were more pronounced with complete removal of the stomach, but its functions were gradually taken over by the underlying parts of the digestive tract.

After surgical operations such as pyloroplasty, gastrectomy, anthrectomy leading to disruption of the functions of the antral part of the stomach or pylorus, a specific symptom complex known as "dumping syndrome" is observed due to rapid evacuation of stomach contents into the intestine. Early dumping syndrome develops within the first hour after eating, when there is a feeling of rapid saturation, loose stools, bloating and abdominal pain. There are also vegetative reactions in the form of facial flushing, sweating, and tachycardia. These symptoms are associated with the intake of a large amount of hypertonic chyme from the stomach or its stump into the proximal parts of the small intestine. In this case, there is a reflex stimulation of the motility, which causes diarrhea and a sudden increase in the fluid content in the small intestine, which leads to its extension. As a consequence, hypovolemia is possible. Symptomatics is enhanced by the release of intestinal hormones and vasoactive mediators (bradykinin, serotonin, etc.), which cause vegetative disorders. Late dumping syndrome includes dizziness, headaches, trembling, palpitations, sweating, hunger, confusion and sometimes fainting after 1.5-3 hours after eating. Symptoms can appear earlier, if

food products contain a large number of simple carbohydrates, especially sucrose. This syndrome is caused by hypoglycemia. Initially, there is a rapid increase in the amount of glucose in the blood (hyperglycemia) because of the sudden intake of sugar-containing food components from the stomach into the proximal parts of the small intestine, which is followed by hypoglycemia. Rapid absorption of carbohydrates stimulates the release of insulin, which circulates in the blood longer than glucose, and eventually develops hypoglycemia.