MINISTRY OF HEALTH OF UKRAINE

ODESA NATIONAL MEDICAL UNIVERSITY

Faculty *Medicine*

Department *of Surgery with Postgraduate Education*

APPROVED BY

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METHODOLOGICAL RECOMMENDATIONS FOR PRACTICAL CLASSES OF THE ACADEMIC DISCIPLINE

Faculty, course Medical 6th year

Academic discipline Surgery

(name of the discipline)

PRACTICAL CLASSES

Practical class № 7

Topic: "Dysphagia syndrome, vomiting and impaired act of defecation. Importance in diagnostics and differential diagnosis. Medical tactics"

Approved:

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PRACTICAL CLASSES

Practical class № 7

Topic of the practical class: "Dysphagia syndrome, vomiting and impaired act of defecation. Importance in diagnostics and differential diagnosis. Medical tactics"- 6 hours.

1.Relevance of the topic.

Dysphagia syndrome, which occurs in the practice of not only general, thoracic and oncological surgeons, but also general practitioners, gastroenterologists, neurologists, hematologists, is most often caused by a number of diseases of the esophagus, although there is also a list of extraesophageal causes of dysphagia. Among the diseases of the esophagus, the following most often lead to dysphagia: esophageal cancer, neuromuscular diseases of the esophagus (esophageal achalasia, cricopharyngeal achalasia, esophagospasm, nonspecific motor disorders), GERD and hiatal hernias, including those complicated by peptic stricture of the esophagus, post-burn cicatricial strictures of the esophagus, a number of forms of esophagitis, esophageal diverticula. The most acute problem is the problem of timely diagnosis of esophageal cancer and precancerous conditions. It is known that the resectability of esophageal cancer even in large clinics does not exceed 50%, while dysphagia - usually the first symptom - occurs with stenosis of the esophageal lumen in 70-80%, that is, most often in the case when the tumor is already unresectable due to germination into neighboring organs. The second most common disease of the esophagus is gastroesophageal reflux disease (GERD), in 90% of cases associated with a hiatal hernia. With a long course of these diseases, a number of complications may develop, including Barrett's esophagus, which is a precancerous condition. Late diagnosis is primarily due to the lack of the necessary endoscopic screening, which in turn requires not only the availability of special endoscopic equipment, but also a powerful morphological laboratory. But the most important thing is the high awareness of specialists about the clinical picture of esophageal diseases and the presence of a single diagnostic and treatment algorithm. For example, patients with hiatal hernia or achalasia of the esophagus are often diagnosed with angina pectoris, chronic pancreatitis, dyspepsia, intercostal neuralgia, neurosis and as a result, ineffective treatment is carried out for many years. Indeed, in the early stages, the symptoms of these diseases (heartburn, belching, retrosternal pain and dysphagia) can be moderately pronounced and patients adapt well to them. If the diagnosis is made at an early stage, then such patients are often given long-term conservative treatment, although endoscopic or surgical operations may be the method of choice. During this time, the disease progresses to the late stages, a number of complications develop, when even surgical treatment may have an unsatisfactory result.

Vomiting syndrome is one of the most frequent manifestations of surgical and non-surgical diseases. The list of causes of vomiting includes more than 70 different diseases and syndromes, the most significant of which are acute intestinal obstruction, stenosis of the gastric outlet, vomiting of "central" origin (for example, as a result of craniocerebral trauma or meningitis), infectious gastroenteritis, various intoxications (the most common example is alcohol intoxication), acute pancreatitis and other inflammatory diseases of the abdominal cavity. Such a variety of causes of vomiting obliges doctors of all specialties to be well-

versed in diagnostic search algorithms. For example, an infectious disease specialist who is admitted to a patient with repeated vomiting is obliged to suspect such surgical diseases as acute adhesive small intestinal obstruction or ulcerative pyloroduodenal stenosis, and if necessary, call a surgeon who will prescribe the appropriate studies. Or, conversely, a surgeon dealing with a patient complaining of repeated vomiting and fever is obliged to consult an infectious disease specialist, although at first glance the clinical picture may be consistent with acute pancreatitis, or to check for meningeal symptoms and call a neurologist. In this case, the doctor who bears the main responsibility for the patient (for example, the doctor of the reception department of a multidisciplinary hospital receiving the patient) often has to make the right decision himself, since the more consultants are involved, the more conflicting opinions can be expressed, and the more likely a diagnostic and tactical error is. Wrong tactics in such situations can cause not only a delay in the correct treatment of the patient, but even the death of the patient!

Disorders of the defecation act are a narrower problem, which is mainly dealt with by proctologists and, less often, neuropathologists. The most common forms of these disorders are fecal incontinence, anal sphincter stenosis and rectal prolapse. A clear knowledge of the semiotics of these disorders will allow a general practitioner to refer a patient for consultation to an appropriate specialist, who, in turn, will prescribe the correct treatment.

2. Lesson Objectives:

2.1. Learning Objectives:

Level 1 according to Bezpalko - to familiarize higher education students with the etiology, pathogenesis, main clinical manifestations and data of imaging methods of research in the main diseases that cause: 1) dysphagia: esophageal cancer, esophageal achalasia, hiatal hernia, post-burn esophageal stricture, peptic esophageal stricture, esophageal atony, esophageal polyps, esophagospasm; 2) vomiting: mechanical intestinal obstruction (tumor, adhesive, with strangulated hernias, volvulus, intussusception, cholelithiasis), dynamic intestinal obstruction (with peritonitis, pancreatitis, intoxications, disorders of water and electrolyte metabolism, metabolic and endocrine disorders), stenosis of the gastric outlet of ulcerative and tumor etiology, acute pancreatitis, acute cholecystitis, exacerbation of peptic ulcer disease, acute appendicitis, renal colic, acute infectious gastroenteritis (food poisoning, rotavirus infection, salmonellosis), consequences of craniocerebral trauma, intoxications (alcoholic, narcotic, medicinal), neuroinfections; 3) disorders of the act of defecation: spasm of the anal sphincter, anal sphincter insufficiency, rectal prolapse.

Level II according to Bezpalko - the student must master the features of the radiological picture, the data of endoscopic and functional methods of diagnosis of all forms of the above-mentioned diseases, and also, based on this knowledge, be able to conduct differential diagnostics and build treatment algorithms for these diseases depending on the variants of their course.

Level III according to Bezpalko - to give students the opportunity to learn the technique of performing the most typical surgical interventions for the above-mentioned diseases; perform the following diagnostic and therapeutic manipulations: placing a nasogastric tube, washing the stomach with an orogastric tube, performing

fibrogastroduodenoscopy, colonoscopy, rectoscopy together with an endoscopist, performing X-ray examination of the esophagus, stomach, small and large intestines, digital examination of the rectum; to provide an opportunity to assist in the simplest operations (for example, colostomy, gastroenterostomy, anal sphincterotomy, etc.).

2.2. Educational objectives are related to:

- the formation of a professionally significant substructure of the personality;
- relevant aspects of deontological, ecological, legal, psychological, patriotic, professional responsibility, etc.

3. Interdisciplinary integration

Table 1. Interdisciplinary integration

Disciplines	To Know	To Be able

Previous: 1) Anatomy 2) Histology 3) Normal Physiology	Anatomy of the esophagus, detailed anatomy of the gastroesophageal junction, anatomy of the stomach, duodenum, small and large intestines, detailed anatomy of the rectum and pelvic floor Histological structure of the esophagus, gastroesophageal junction, stomach, duodenum, small and large intestines, rectum, and pelvic floor Motor function of the esophagus and esophageal-gastric junction, including the physiology of the act of swallowing, motor and secretory functions of the stomach and duodenum, motor function of the small and large intestines, motor function of the rectum, including the physiology of the act of defecation. Physiology of the vomiting center of the CNS, vagus nerves.	
4) Pathological physiology	Pathophysiology of the vomiting process, pathogenesis of benign tumors, precancerous conditions and cancer of the esophagus and stomach, pathogenesis of gastric and duodenal ulcers, pathogenesis of inflammatory and cicatricial processes of the esophageal canal, various types of intestinal obstruction, acute diseases of the	Diagnose esophageal

5) Propaedeutics of internal diseases and internal diseases, neurology, endocrinology, surgical diseases (preliminary courses)	abdominal cavity (acute cholecystitis, pancreatitis, etc.) Semiotics, differential diagnosis of esophageal cancer, esophageal achalasia, hiatal hernia, diaphragm and gastroesophageal reflex disease, post-burn and peptic strictures of the esophagus, esophageal atony (against the background of central nervous system disorders, systemic scleroderma, diabetes mellitus, alcoholism), esophageal polyps, esophagitis, other diseases of internal organs that functionally (Plummer-Vinson syndrome) or mechanically lead to dysphagia (thyroid pathology, mediastinal tumors, enlargement of heart chambers). Semiotics, differential diagnostics of gastric and duodenal ulcer, various variants of intestinal obstruction, acute diseases of the abdominal cavity (acute cholecystitis, pancreatitis, etc.)	cancer, esophageal achalasia, hiatal hernias and gastroesophageal reflux disease, post-burn and peptic strictures of the esophagus, esophageal atony (on the background of central nervous system disorders, systemic scleroderma, diabetes mellitus, alcoholism), esophageal polyps, esophagitis, other diseases of internal organs that functionally (Plummer-Vinson syndrome) or mechanically lead to dysphagia (thyroid pathology, mediastinal tumors, enlargement of heart chambers). Diagnose gastric and duodenal ulcers, various types of intestinal obstruction, acute diseases of the abdominal cavity (acute cholecystitis, pancreatitis, etc.)

6)Pharmacology	Mechanism of action, pharmacokinetics, pharmacodynamics, side effects of myotropic antispasmodics, M-cholinolytics, calcium channel blockers, nitrates, antimimetics, prokinetics, proton pump inhibitors, histamine receptor blockers, antacids, laxatives.	Prescribe and fill out a prescription form for the following drugs: myotropic antispasmodics, M-cholinolytics, calcium channel blockers, nitrates, antimimetics, prokinetics, proton pump inhibitors, histamine receptor blockers, antacids, laxatives.

7) Infectious diseases	Semiotics, differential diagnostics and treatment of acute infectious enterocolitis, neuroinfections	Diagnose and treat acute infectious enterocolitis, neuroinfectious diseases.
8)Urology	Semiotics, differential diagnostics and treatment of complications of urolithiasis	Diagnose and treat complications of urolithiasis
9)Psychiatry	Semiotics and treatment of alcoholism and drug addiction	Diagnose and treat alcoholism and drug addiction
10) Neurosurgery	Semiotics, differential diagnostics and treatment of traumatic brain injury	Diagnose and treat various types of traumatic brain injury
2. Next disciplines 1) Anesthesiology and intensive therapy	Pathogenesis, clinical, instrumental and laboratory semiotics, intensive therapy and prevention of water and electrolyte disorders	
2)Oncology	Etiology, pathogenesis, histopathological properties, clinical and instrumental semiotics, TNM classification, principles of complex and combined treatment of cancer and precancerous diseases of the esophagus and stomach, surgical techniques for esophageal and gastric cancer	
3. Intersubject integration 1) Acute intestinal obstruction syndrome: etiology, pathogenesis, diagnosis,	Etiology, pathogenesis, classification, semiotics, methods of conservative and surgical treatment of gastric and duodenal ulcer and its complications, tumours of the	Diagnose, conduct differential diagnose and treat (prescribe conservative

differential diagnosis, therapeutic tactics 2) Acute abdominal syndrome: diagnosis, differential diagnosis, treatment tactics	stomach, colon and rectum, adhesive disease of the abdominal cavity, acute cholecystitis, acute pancreatitis, strangulated hernias of the abdominal wall.	treatment and have a good knowledge of surgical treatment methods) gastric and duodenal ulcers and its complications, gastric, colon and rectal tumours, adhesive disease of the abdominal cavity, acute cholecystitis, acute pancreatitis, and strangulated hernias of the abdominal wall. Perform preoperative preparation and postoperative management of the above diseases.

4. Content of the lesson

The vomiting syndrome is a manifestation of a number of diseases of the abdominal cavity and other organs and systems, a list of which is given in Table 2, and also leads to water and electrolyte disorders, aspiration of vomit with the development of aspiration pneumonia, Mallory-Weiss syndrome and a number of other disorders. The pathogenesis of water-electrolyte disorders is complex; in general, dehydration, hypochloraemia, hypokalaemia and metabolic alkalosis, hyperuraemia occur, and in severe cases, hyponatraemia occurs. There are a number of features depending on the level of obstruction. Clinically, these disorders are manifested by hypotension, tachycardia, oliguria, decreased soft tissue turgor, convulsions, and may result in cardiac arrest. Correction of water-electrolyte disorders includes infusion of isotonic (in severe cases, hypertonic) saline solutions containing a large amount of potassium, chloride and other electrolytes into the central vein under the control of central venous pressure and diuresis. In severe cases, it is necessary to transfuse 5 or more litres of crystalloids. Colloidal solutions play a secondary role.

Table 2: List of diseases manifested by vomiting

Obstruction of the upper and middle parts of the gastrointestinal tract (diseases and conditions that manifest themselves as vomiting more often than constipation)

Mechanical obstruction
Intraluminal

Invagination
Gallstone ileus
Obstruction by foreign bodies
Bezoars
Helminths
Other foreign bodies
Intramural Congenital
Atresias and stenoses Duplications
Tumour
Benign (polyps, leiomyomas, lymphomas)
Malignant (cancer, sarcomas, carcinoids)
Inflammatory
Ulcerative pyloroduodenal stenosis
Small bowel stricture in Crohn's disease
Small bowel stricture in Crohn's disease
Extraluminal
Adhesive disease
Hernias (strangulated and unreducible)
Foreign bodies in the abdominal cavity
Cysts (including pancreatic pseudocysts) Neoplasms (including carcinomatosis)
Abscesses
Hematomas
Volvulus and nodules
Congenital changes
Annular pancreas
Abnormal vessels
Dynamic obstruction

Paralytic ileus
Pathology of the abdominal organs
Postoperative (transthoracic and abdominal operations)
Peritonitis
Pathology of the retroperitoneal space
Acute pancreatitis (edema stage)
Cysts
Tumours
Abscesses (including retroperitoneal phlegmon in destructive pancreatitis)

Hematoas
Ascites
Consequences of trauma (abdominal or other organs)
Spinal cord injury Spinal fracture
Traumatic brain injury
Anterior abdominal wall injury
Lumbar spine injury
Systemic diseases
Collagenosis
Multiple sclerosis
Systemic scleroderma
Amyloidosis
Electrolyte disorders
Hypercalcemia
Hypokalemia
Endocrine disorders
Hypothyroidism
Diabetic ketoacidosis
Hyperparathyroidism
Glucagonomia
Somatostatinoma
Pangipopituitarism
Pheochromocytoma
Multiple endocrine neoplasia syndromes (MEN) IIa, IIb
Addisonian crisis
Metabolic disorders
Porphyria
Uremia
Acute mesenteric ischemia/thrombosis Pseudoobstruction (Ogilvy syndrome)
Spastic obstruction (intoxications, e.g. lead poisoning)
Inflammatory diseases of the gastrointestinal tract
Hollow organs
Appendicitis
Cholecystitis
Gastric and duodenal ulcer disease
Gastroenteritis
Crohn's disease
Meckel's diverticulitis Parenchymal organs

Pancreatitis
Hepatitis
Liver abscess
Mesentery
Mesenteric lymphadenitis

Pelvic inflammatory disease
Tubo-ovarian abscess
Endometritis
Aborted ectopic pregnancy
Ovarian cysts

Impaired blood supply to the myomatous node
Neuroinfections
Traumatic brain injury
Renal colic / acute pyelonephritis
Intoxication / overdose
Narcotics
Psychotropic drugs
Antidepressants
Calcium channel blockers
Antispasmodics
Poisoning with heavy metal salts (mercury, lead, arsenic)

Psychogenic vomiting
Consequences of operations
Adductal loop syndrome
Anastomotic stricture / anastomosis
Gastrostasis (gastric resection, vagotomy)

Stenosis in peptic ulcer disease occurs when the ulcer is located in the duodenum or pyloric canal, and less commonly in the antrum or retrobulbar duodenum. Stenosis develops in chronic recurrent peptic ulcer disease. Clinical manifestations, in addition to the above-described signs of water and electrolyte disturbances, include weight loss, dull pain in the epigastrium after eating, vomit containing food eaten the day before, and a patient's history of ulcerative disease. Physical examination usually reveals moderate pain in the epigastrium and right hypochondrium, as well as a splashing noise. The diagnosis is made on the basis of endoscopy and gastric fluoroscopy. At the same time, endoscopy (with biopsy) plays a key

role in the differential diagnosis with tumour stenosis. The stenosis has three stages: compensated, subcompensated and decompensated, which determines the treatment tactics.

In compensated stenosis, vomiting is periodic, water-electrolyte disorders and weight loss are mild or absent; endoscopy reveals a peptic ulcer and moderate scar deformity, the device passes through the narrowed area; fluoroscopy reveals a scar-ulcer deformity, the stomach is not enlarged, its peristalsis is enhanced, and the passage of contrast through the narrowed segment is slightly slower. The surgery usually reveals a moderate deformation and infiltration around the ulcer, which allows for plastic surgery on the narrowed segment - pyloroplasty or duodenoplasty.

With subcompensated stenosis, vomiting is periodic or constant, water and electrolyte disorders and weight loss are moderate; endoscopy reveals peptic ulcer and gross scar deformity, the device passes through the narrowed area with difficulty; fluoroscopy reveals severe scar and ulcer deformity, the stomach is enlarged, peristalsis is preserved, the passage of contrast through the narrowed segment is slow, but complete emptying of the stomach from the contrast is observed no later than 12 hours later. The operation usually reveals a pronounced deformation and infiltration around the ulcer, which allows performing pyloroplasty or duodenoplasty with certain technical difficulties.

In case of decompensated stenosis, vomiting is constant, profuse, there are severe water and electrolyte disorders and weight loss; endoscopy reveals a gross scar-ulcer deformity, the device does not pass through the narrowed area; fluoroscopy reveals a gross scar-ulcer deformity, the stomach is sharply enlarged, peristalsis is absent, and the passage of contrast through the narrowed segment occurs later than 12 hours or does not occur at all. The surgery usually reveals a gross deformity and a massive infiltrate around the ulcer, which usually does not allow for pyloroplasty or duodenoplasty.

For compensated stenosis, the strategy is similar to that for uncomplicated, frequently recurrent peptic ulcer disease: continue conservative treatment in patients with a high surgical risk or perform elective selective proximal vagotomy (SPV) and pyloroduodenoplasty in patients with a low to moderate surgical risk. In sub- and decompensated stenosis, the initial tactics after diagnosis are gastric lavage with an orogastric tube to clear the stomach, followed by continuous nasogastric aspiration, and correction of water and electrolyte disturbances (see above). Then there are 2 options for the management of patients: the first, classical, is to perform surgery after 2-4 days of intensive preoperative preparation in patients with low and moderate surgical risk. In this case, in subcompensated stenosis, SPV or trunk vagotomy (SV) and pyloroduodenoplasty are performed, and in decompensated stenosis, SV and gastroenteroanastomosis (for example, extra-diabdominal anastomosis according to Gacker) are performed. In patients with a high surgical risk, a jejunostomy may be placed for nutrition under epidural or local anaesthesia.

The second, more modern, variant of the tactic is to establish enteral nutrition (percutaneous endoscopic jejunostomy for subcompensated stenosis or laparoscopic jejunostomy for decompensated stenosis) for the period necessary to compensate the patient and reduce inflammation in the pyloroduodenal area, which allows for a more functional

intervention with a lower risk of postoperative complications (the most common is gastrostasis and failure of pyloroduodenoplasty or bypass anastomosis in the setting of protein deficiency and dehydration) - SPV and pyloroduodenoplasty or resection of 2/3 of the stomach (if the latter is indicated). In patients with a high surgical risk and subcompensated stenosis, balloon dilatation and even stenting of the stenotic segment can be performed.

Stenosis of the antrum in gastric cancer, in addition to clinical symptoms characteristic of ulcerative stenosis, is manifested by a number of signs of cancer: anaemia, rapid weight loss, severe weakness, anorexia, subfibrillation, etc. In thin and emaciated patients, the tumour can be palpated. Diagnosis is best started with an endoscopic examination, which allows not only to detect a malignant tumour (polypoid, infiltrative or most often infiltrative ulcerative form) stenosing the gastric lumen, but also to perform a biopsy to confirm the diagnosis and clarify the histopathological form of cancer. It should be remembered that, in addition to cancer, stenosis of the antrum can be caused by MALT lymphoma, leiomyoma and large adenomatous polyps. Gastric cancer can also be diagnosed by gastric X-ray, which demonstrates narrowing of the antrum with indistinct corroded edges, sharp pathological changes in the relief and moderate stretching of the stomach. It is also necessary to perform a computed tomography of the abdominal cavity, which allows not only to visualise the tumour, but also to assess the possibility of its growth into the main vessels and neighbouring organs (i.e. tumour resectability) and to assess the possibility of distant metastases in the liver and retroperitoneal lymph nodes (to determine the patient's operability). By the way, to assess distant metastasis, it is also necessary to perform an X-ray examination of the chest.

The choice of treatment method for antral gastric cancer complicated by stenosis depends on the degree of stenosis, water and electrolyte disturbances and nutritional deficiencies, the extent of the cancer process, and the degree of surgical risk. If convincing signs of resectability are found and in the absence of a high surgical risk, it is necessary to plan a radical operation - subtotal gastrectomy (this operation is more often indicated for tumours of the antrum) or gastrectomy. The surgery is usually complemented by adjuvant polychemotherapy. In patients with complete stenosis, severe water-electrolyte disorders and nutritional deficiencies, enteral nutrition can be established (see above) to prepare for this intervention. Moreover, laparoscopic jejunostomy is the preferred option, as it is possible to assess the operability of the case more fully. If signs of unresectability are detected, you can immediately proceed to symptomatic surgery. If there are signs of unresectability at the preoperative examination or in patients with a high surgical risk, symptomatic surgery is immediately indicated - pre-jejunostomy with Brown's enteroenterostomy.

Dysphagia syndrome refers to a disorder of food passage through the oesophagus, which is associated with diseases of the oesophagus and a number of ‘extra-oesophageal’ causes (Table 3). This, in turn, leads to the development of water and electrolyte disturbances and nutritional deficiencies with the corresponding clinical and laboratory manifestations (see above). Stagnation of food in the esophagus and/or reflux of oesophageal contents into

the respiratory tract can contribute to a number of respiratory complications: bronchial asthma, pneumonia, chronic laryngitis, even lung abscesses. If the oesophagus is irritated by stagnant contents or reflux, congestive esophagitis occurs, against which bleeding and foci of metaplasia, dysplasia and neoplasia often occur. The so-called oesophageal-cardiac syndrome, manifested by coronary spasm and arrhythmias, is also common. All these complications are called extra-oesophageal complications.

Table 3: List of diseases manifested by dysphagia

Tumours of the oesophagus
Benign
Polyps Papilloma
Lipomas Malignant
Cardioesophageal cancer (adenocarcinoma) Squamous cell carcinoma
Primary dyskinesia of the oesophagus
Achalasia of the upper oesophageal sphincter
Achalasia of the oesophagus (achalasia of the cardia), ‘vigorous’ achalasia Diffuse esophagospasm
Segmental esophagospasm (nutcracker oesophagus) Cardiospasm
Nonspecific movement disorders
Secondary esophageal dyskinesia (esophageal atony)
Collagenoses
Scleroderma Multiple sclerosis Dermatomyositis Rheumatoid arthritis
Systemic lupus erythematosus
Consequences of central nervous system trauma
Consequences of neuroinfections
Consequences of acute cerebrovascular accident
Tumours of the central nervous system
Encephalitis

Poliomyelitis

Consequences of other infections Botulism

Diphtheria by alcohol

Diabetes mellitus

Gastroesophageal reflux disease, hernia of the esophageal orifice of the diaphragm, reflux esophagitis

Other esophagitis

Chronic specific

Syphilitic Tuberculosis

Acute infectious esophagitis Diphtheria

Scarlet fever Herpetic

In immunodeficiency states and dysbiosis Candida esophagitis

Scarring strictures of the oesophagus

Peptic strictures as a result of reflux esophagitis post-burn

Strictures of anastomoses

Crohn's disease with predominant oesophageal involvement

Congenital anomalies

Shatsky ring

Membranes

Extra-oesophageal causes

Abnormal vessels

Aneurysm of the thoracic aorta

Enlarged heart chambers with defects

Enlarged mediastinal lymph nodes

Lymphogranulomatosis

Lymphosarcoma
Metastatic lesions in lung cancer
Tuberculous lymphadenitis
Enlargement of the thyroid gland
Tumours and cysts of the mediastinum
Cyst of the pericardium
Branchogenic cyst
Neurogenic tumours
Teratomas
Deforming spondylosis of the cervical spine
Iron deficiency anaemia (sideropenic dysphagia or Plummer-Vinson syndrome)

Esophageal achalasia. The etiology of the disease is not known and has more than 10 different theories, of which the most noteworthy is the theory of damage to the parasympathetic nerve pathways: the intermuscular (Auerbach's) plexus in the area of the esophagus and lower esophageal sphincter, as well as the trunks of the vagus nerves. As a result, on the one hand, the ability of the esophagus to peristaltic contractions is lost, and on the other hand, the ability of the lower esophageal sphincter to relax during swallowing is simultaneously lost. As the disease progresses, the oesophageal-gastric junction narrows and the oesophagus dilates, which forms a characteristic radiological picture.

The diagnosis is made mainly by X-ray and anamnesis (dysphagia, slowly progressing over several years, and regurgitation, moderate pain, symptoms of respiratory complications, onset of the disease after a stressful situation), although endoscopy and oesophageal manometry are important tests. Radiological and endoscopic findings distinguish 4 stages, on the basis of which the treatment tactics are built. Stage I (functional) is characterised by periodic impaired patency of the oesophagogastric junction. There is no narrowing of the oesophagogastric junction, the amplitude of peristaltic contractions of the oesophageal body is slightly reduced or unchanged, the diameter of the oesophagus is normal or does not exceed 2.5 cm, the shape of the oesophagus is unchanged. Phenomena of congestive esophagitis are not expressed at this stage. Stage II (also called the stage of persistent spasm) is characterised by persistent impaired patency of the oesophagogastric junction. There is a moderate narrowing of the esophagogastric junction, the amplitude of peristaltic contractions of the esophageal body is moderately reduced, the diameter of the esophagus is from 2.5 to 4 cm, the shape of the esophagus is spindle-shaped. Mild congestive esophagitis is usually noted.

Stage III (scarring changes) is characterised by the initial phenomena of fibrosis in the lower oesophageal sphincter. There is a marked narrowing of the esophagogastric junction, the amplitude of peristaltic contractions of the esophageal body is significantly reduced, the diameter of the esophagus is from 4 to 8 cm, the shape of the esophagus is flask-shaped. Esophagitis is moderate. Stage IV (megaesophagus) is characterised by scarring narrowing of the esophagogastric junction up to complete obstruction. The amplitude of peristaltic contractions of the esophagus is sharply reduced up to atony, the diameter of the esophagus exceeds 8 cm, the shape of the esophagus is S-shaped. Esophagitis is moderate or severe up to the appearance of mucosal leukoplakia, which predisposes to the development of squamous cell carcinoma.

There are 4 main methods of treating oesophageal achalasia: medical treatment, endoscopic injection of botulinum toxin into the lower oesophageal sphincter, cardiodilation and surgery. Drug treatment (M-cholinolytics, myotropic antispasmodics, prolonged nitrates and prolonged calcium channel blockers) is not used as an independent treatment and is indicated as an adjunctive method in preparation for and after other treatments.

The method of endoscopic injection of botulinum toxin into the lower oesophageal sphincter is based on the ability of botulinum toxin type A to block cholinergic synapses, thereby causing sphincter relaxation. Due to its limited efficacy, the method is justified in patients with stage I and II esophageal achalasia in case of failure or contraindications to dilation and surgical treatment as more effective methods.

Cardiodilatation is based on the partial rupture of the circular fibres of the lower oesophageal sphincter and the scar tissue present in the later stages. The immediate results are characterised by relief of dysphagia in the vast majority of patients. However, many patients experience relapses within 3 to 24 months, depending on the stage of the disease. Therefore, repeated courses are necessary. Thus, the absolute indication for balloon dilation is stage I esophageal achalasia, as well as any stage in case of recurrence after cardiomyotomy, failure or contraindications to surgery; the relative indication is stage II esophageal achalasia.

Before the advent of video endoscopic technologies, the main methods of surgical treatment of esophageal achalasia were Heller cardiomyotomy using transthoracic or laparotomy access with the use of various methods of fundoplication (according to Belsi). Cardiomyotomy is the most effective method of treatment, but transthoracic and laparotomy cardiomyotomy are characterised by a high incidence of postoperative complications, which cannot be said about laparoscopic cardiomyotomy, which is the operation of choice at present. Thus, the indications for laparoscopic cardiomyotomy according to Heller with Dohr or Toupé fundoplication are stages II, III and IV, as well as stage I, when there are contraindications to balloon dilatation and endoscopic injection of botulinum toxin.

Gastroesophageal reflux disease (GERD) is a chronic disease caused by acid or alkaline gastroesophageal reflux, in most cases (up to 90%) associated with hiatal hernia, which leads to the development of severe complications: reflux esophagitis, peptic ulcers of the oesophagus, peptic stricture of the oesophagus, Barrett's esophagus, anaemia and extra-

esophageal complications. *Hiatal hernia* is a condition in which the abdominal part of the esophagus (I degree of axial hernia of the hiatal hiatus), the cardia (II degree of axial hernia), the stomach floor or part of the stomach body (III degree of axial hernia), or any abdominal organ, including the stomach, penetrates the posterior mediastinum through the dilated hiatal hiatus. including the stomach, while maintaining the normal location of the esophagogastric junction and the cardia (paraesophageal hernia), which leads to a violation of the closing function of the esophagogastric junction.

Reflux esophagitis, confirmed by biopsy, develops in all patients with GERD. However, endoscopic examination does not reveal esophagitis in all cases of GERD, which is associated with minimal changes in the oesophageal epithelium, especially in the early stages of the disease. This is the so-called endoscopy-negative variant of GERD, the frequency of which ranges from 10 to 30%. A chronic inflammatory process leads to gastric or small intestinal metaplasia of the epithelium of the lower third of the oesophagus. Gastric metaplasia of the oesophageal epithelium contributes to a more severe course of esophagitis and peptic ulcers of the oesophagus. More dangerous is small intestinal metaplasia or Barrett's esophagus, which occurs in 8-30% of cases of GERD, against which adenocarcinoma of the lower third of the esophagus develops in 1-2% of cases.

Not all patients have all the typical symptoms: heartburn, belching, pain behind the sternum and in the epigastrium, regurgitation when the body is tilted, and occasional dysphagia. Up to 15 % of patients present only atypical complaints (heart pain, cough and shortness of breath, hoarseness of voice), which are typical of extra-oesophageal complications.

The main methods of diagnosis are fluoroscopy of the oesophagus and stomach with examination in the Trendelenburg position and endoscopy. If a hiatal hernia is detected during fluoroscopy and reflux oesophagitis during endoscopy, the diagnosis is usually unquestionable. If there are indications for surgical treatment, daily intra-esophageal pH monitoring can be performed before surgery (to confirm the diagnosis) and after surgery (to assess the results of surgical treatment and select the necessary drug correction). If the patient does not have a hiatal hernia or an axial hiatal hernia of the diaphragm, and endoscopic examination shows no signs of reflux esophagitis and excludes other diseases with a similar clinic (chronic gastritis, peptic ulcer disease, coronary artery disease, intercostal neuralgia, chronic pancreatitis, gastrointestinal diseases), then there may be an endoscopically negative form of GERD. According to the Rome criteria, in this case, a proton pump inhibitor is prescribed in a standard dosage for 2 weeks. If the complaints significantly decrease or disappear, the diagnosis of GERD is confirmed. At the patient's request, it can be confirmed by pH monitoring. If the complaints do not disappear after a course of proton pump inhibitors, pH monitoring is mandatory. If the DeMeester index calculated in this study is higher than normal, the diagnosis of GERD is confirmed. If the DeMeester index is within the normal range, it is necessary to continue the diagnostic search.

In the absence of a hiatal hernia or in the presence of a grade I axial hernia, conservative treatment is first performed for 2 to 4 months. It includes lifestyle changes (avoiding coffee,

smoking, overeating, eating spicy and fatty foods, losing weight) and prescribing proton pump inhibitors in combination with prokinetics and antacids. If conservative treatment is ineffective, laparoscopic antireflux surgery is indicated. If there is a high risk of surgery or if the patient refuses surgery, conservative therapy is continued. In the presence of an axial hiatal hernia of the esophageal orifice of the diaphragm of stage II - III or paraesophageal hernia, surgical treatment is immediately indicated - laparoscopic crurorrhaphy or plastic surgery of the esophageal orifice of the diaphragm with a mesh graft (depending on the size of the hernial defect) and Nissen fundoplication. In the presence of Barrett's esophagus, laser ablation or endoscopic resection of the metaplastic epithelium is performed, followed by laparoscopic anti-reflux surgery.

Esophagospasm is a rare disease characterised by spastic contraction of the oesophagus after eating, and often before meals, at night. In diffuse esophagospasm, the entire esophagus is spasmodically contracted, and in segmental esophagospasm (Barsoni-Teschendorf syndrome), multiple limited areas of the esophagus 1-2 centimetres long are affected. In esophagospasm, patients complain of sharp intense pain behind the sternum, radiating to the lower jaw, back, which can be relieved only by large doses of narcotic analgesics and antispasmodics. Dysphagia and regurgitation occur during eating. Outside of a painful attack, patients may complain of transient moderate dysphagia. Radiological examination outside the attack reveals a slight dilation of the esophagus, the passage of primary and secondary peristaltic waves is not disturbed, and the lower oesophageal sphincter opens in a timely manner, to the full extent. Short-term spastic contractions are detected in different parts of the oesophagus. During an attack with diffuse esophagospasm, the oesophagus is spasmodic throughout, and the contrast medium is pushed out in a retrograde direction. In segmental esophagospasm, such contractions are numerous, covering the entire oesophagus, completely bringing its walls together, making the oesophagus look clear. Hence the synonyms of diffuse esophagospasm: corkscrew esophagus, corkscrew esophagus, pseudodiverticulosis of the esophagus. Endoscopy performed outside of an attack does not provide specific information. Esophageal manometry during a painful attack demonstrates multiple spastic contractions of moderate amplitude or high-amplitude contractions of the entire esophagus, and outside the attack - a normal primary swallowing complex, against which segmental contractions can be registered in different parts of the esophagus. The basal and residual pressure of the lower oesophageal sphincter is not affected. Treatment is conservative (analgesics, antispasmodics). In severe cases, transthoracic esophagomyotomy can be performed.

Oesophageal atony is relatively common and develops as a result of impaired innervation of the oesophagus in other diseases. Most often, esophageal atony occurs in systemic scleroderma and other connective tissue diseases, as well as due to traumatic brain injury and cerebrovascular disorders. Less commonly, esophageal atony develops as a result of diabetes mellitus, poliomyelitis, brain tumours, encephalitis, diphtheria, botulism, and alcoholism (Table 3). Esophageal atony decreases the tone of the esophagus and lower oesophageal sphincter. On the one hand, there is a violation of food passage, on the other

hand, gastroesophageal reflux, which is complicated by severe reflux esophagitis and peptic stricture of the esophagus, which leads to further impaired food passage.

In the first phase of the disease, when the lower oesophageal sphincter is insufficient, patients complain of periodic moderate dysphagia, constant heartburn, belching and regurgitation. Radiographically, a moderate dilatation of the esophagus, a decrease in the amplitude of peristaltic contractions, which leads to a slight slowdown in food passage, and a gaping esophagogastric junction are detected. Gastroesophageal reflux is always detected in the Trendelenburg position. On X-ray examination, there are no tertiary contractions characteristic of esophageal achalasia. Endoscopy reveals esophageal dilatation, severe reflux oesophagitis, gaping oesophageal junction and gastroesophageal reflux. Oesophageal manometry reveals a decrease in the amplitude of peristaltic contractions, a decrease in the basal pressure of the lower esophageal sphincter and the absence of tertiary contractions. Daily intra-esophageal pH monitoring allows to detect signs of pathological gastroesophageal reflux. In the second stage of the disease, when peptic stricture of the lower third of the esophagus develops, patients complain of severe dysphagia, regurgitation and moderate sternal pain. Heartburn and belching disappear. Radiological examination reveals esophageal dilation, decreased peristalsis and narrowing of the esophagogastric junction, reminiscent of esophageal achalasia. Endoscopically, moderate esophageal dilation and scarring stricture are detected against the background of severe reflux oesophagitis. At the same time, the endoscope has difficulty passing into the stomach or does not pass at all. Manometry reveals a decrease in the amplitude of peristaltic contractions, increased basal pressure of the lower oesophageal sphincter and the absence of its relaxation during swallowing. Daily pH monitoring is not informative in this phase of the disease. Thus, in the later stages, esophageal atony is very similar to esophageal achalasia.

The difference lies in the fact that in esophageal atony there is a certain causative factor (from among the above), there is no cardiac opening and tertiary contractions, and the narrowing of the lower third of the esophagus is of scarred origin as a result of reflux oesophagitis.

The treatment in the first phase is initially conservative: it should be aimed at eliminating the cause of the disease and eliminating reflux (proton pump inhibitors, prokinetics, anatacids, diet). If ineffective, laparoscopic antireflux surgery is indicated.

Oesophageal cancer is one of the most common diseases of the oesophagus that manifest as dysphagia. The clinical picture of oesophageal cancer, especially cancer of the lower third of the oesophagus (squamous cell carcinoma or adenocarcinoma) and cardioesophageal cancer (adenocarcinoma), is very similar to the manifestations of achalasia, but there are a number of differences. Patients suffering from oesophageal carcinoma usually do not live more than a year without treatment. On the contrary, patients with oesophageal achalasia seek medical attention on average 2 years after the onset of the disease. Dysphagia in oesophageal cancer progresses rapidly; at first it occurs when taking solid food, then when taking liquids, and then complete oesophageal obstruction occurs. Then dysphagia may decrease and, in some cases, even disappear completely, which is associated with the

tumour's decay. Oesophageal cancer pain, especially in the later stages, is constant, gnawing, localised deep behind the sternum, radiating to the back, and is also progressive. Patients lose weight rapidly, complain of weakness and loss of appetite. They develop anaemia, the degree of which is not typical for esophageal achalasia. On examination, cachexia and pallor of the skin are notable.

Instrumental examination of such patients is best started with endoscopy. The following types of oesophageal cancer are distinguished: superficial (polypoid or non-polypoid), polypoid, ulcerative, infiltrative-ulcerative, diffuse-infiltrative (skirotic) and unclassified. Superficial polypoid or non-polypoid tumours are early cancers and are mostly incidental findings during screening. A polypoidal tumour appears as a dense, sedentary nodule with an uneven surface, often with an ulcer accompanied by contact bleeding. The ulcerative type is characterised by the presence of an ulcer with clearly demarcated and raised edges, a dirty grey bottom, and often small thrombosed vessels. The infiltrative ulcerative form is characterised by the presence of an ulcer without clear edges. In large tumours, the endoscope usually does not pass below the tumour or passes with considerable difficulty. Skeletal cancer can be difficult to diagnose endoscopically. In this case, the oesophageal wall is stiff, the mucous membrane loses its folds, may change its colour, and may be accompanied by contact bleeding. Biopsy confirms the diagnosis of malignancy. Endoscopic ultrasound examination is of great importance in the diagnosis of oesophageal cancer. X-ray examination in cancer, similar to achalasia, shows narrowing and suprastenotic dilation of the oesophagus. However, the esophageal dilation (unlike achalasia) is moderate, no more than 3 to 4 cm; there is no pronounced hypotension or atony, as well as spastic contractions. There is no esophageal peristalsis in the area of the tumour itself. Depending on the type, the tumour looks like a niche with uneven edges and bottom, a filling defect with an uneven surface or ulcer, or a circular narrowing with an uneven surface. If the tumour is located in the oesophageal-gastric junction, there is no opening of the lower oesophageal sphincter in response to swallowing.

The choice of treatment for oesophageal cancer primarily depends on the location and stage of the disease. *Cancer of the cervical esophagus.* In stages 0 - I (see TNM classification of cancer), endoscopic mucosectomy is indicated (if it is not possible, resection of the upper third of the oesophagus with reconstruction of a segment of the small or large intestine) with adjuvant radiotherapy. In stages II - III, with a resectable tumour and low surgical risk, resection of the upper third of the oesophagus with reconstruction of a segment of the small or large intestine with adjuvant radiotherapy is indicated. This operation can be divided into 2 stages: gastrostomy with resection, and then a reconstructive stage. A good effect is also obtained from preoperative radiotherapy, especially in case of borderline resectability of the tumour. In case of an unresectable tumour or high surgical risk, as well as in stage IV, gastrostomy with subsequent curative radiotherapy is indicated. *Cancer of the upper and middle thirds of the thoracic oesophagus*. In stages 0 - I, endoscopic mucosectomy is indicated (if it is not possible, resection of the oesophagus with plastic surgery with a segment of the small or large intestine is indicated). In stages II - III, with a resectable tumour and low

surgical risk, esophageal extirpation in the form of Torek's operation (with the imposition of a cervical esophagostomy and gastrostomy followed by reconstructive surgery) is indicated in weakened patients or with primary cervical esophagocolon or esophagogastrointestinal anastomosis in strong patients. In case of unresectable tumour or high surgical risk, as well as in stage IV, gastrostomy or endoscopic stenting of the esophagus with a metal stent and curative chemoradiotherapy are indicated. *Cancer of the lower third of the thoracic and abdominal oesophagus.* In stages 0 - I, endoscopic mucosectomy is indicated (if this is not possible, oesophageal resection with cervical or intrathoracic esophagogastroanastomosis (Lewis, Garlock, Gavrilio operations) is indicated. In stages II - III, with a resectable tumour and low peracute risk, oesophageal resection with esophagogastroesophageal anastomosis is indicated. In case of an unresectable tumour or high surgical risk, as well as in stage IV, gastrostomy or endoscopic stenting of the esophagus with a metal stent and curative chemoradiotherapy are indicated.

Among the **benign tumours of the oesophagus**, leiomyomas are the most common, and polyps (adenomatous, hyperplastic, fibrovascular), papillomas, and lipomas are much less common. With a significant increase, they are manifested by dysphagia and regurgitation, and less often by pain. Polyps are often manifested by bleeding. Due to the slow growth of neoplasms, dysphagia increases very slowly, but is characterised by constancy. In case of small tumours, it can be periodic. During endoscopic examination, leiomyoma looks like a round or oval, sedentary mass, the mucous membrane over which is often unchanged. Polyps look like rounded formations on a narrow or wide base with a smooth or villous surface displaced by the apparatus. The mucous membrane of the polyp may be hyperemorrhagic and eroded. Esophageal dilatation is observed only in case of complete esophageal obstruction, which is quite rare. The device usually passes through the compression zone. An X-ray examination reveals a filling defect corresponding to the contours of the leiomyoma or polyp; esophageal dilatation is not pronounced, narrowing is rarely complete: in benign tumours, esophageal peristalsis is not affected.

Treatment of polyps consists of endoscopic polypectomy. One or another variant of oesophageal resection is necessary in the case of large polyps on a wide base, which is rare. Leiomyomas are treated with transthoracic enucleation or oesophageal resection.

Scarring strictures of the oesophagus (post-burn and peptic) are always manifested by dysphagia and regurgitation. Dysphagia progresses slowly and is characterised by persistence. In the case of post-burn strictures, which occur more often in the thoracic region at the level of the aortic constriction and in the area of the oesophageal-gastric junction, the diagnosis is made on the basis of anamnesis. However, it should be remembered that squamous cell carcinoma of the oesophagus can occur in the setting of post-burn strictures. Peptic strictures occur as a result of reflux oesophagitis and affect the lower third of the oesophagus and the oesophagogastric junction. Such patients have a history of prolonged complaints of belching, heartburn, and indications of a previously detected hiatal hernia. Again, it is necessary to remember the possibility of adenocarcinoma of the lower third of the oesophagus of reflux esophagitis, which leads to cylindrical metaplasia of the

oesophageal epithelium. X-ray examination reveals narrowing of various lengths in the lower third of the oesophagus and in the area of the oesophagogastric junction up to complete obstruction.

The contours of the narrowing are uneven, contrast passes through it in a thin stream, and there is no opening of the cardia. Esophageal dilatation is moderate, motility is somewhat weakened. Endoscopy reveals a pronounced narrowing, not passable for the endoscope, at different levels and lengths. The wall of the oesophagus in the area of the stricture is rigid, whitish in colour. The mouth of the stricture is often located eccentrically, and there are always signs of esophagitis, often ulcerative. In case of scarring strictures, given the similarity of radiological data and endoscopic picture with esophageal cancer, biopsy should be performed without fail. Partial and limited strictures are treated with a bougie (in case of peptic strictures, an antireflux surgery is additionally performed). In case of recurrence after the banding, as well as in case of long and complete strictures, one or another variant of resection or esophageal extirpation is indicated. In patients with a recurrence after gastric bypass and a high surgical risk, gastrostomy or stenting with a metal stent can be performed.

Shatsky's ring has also been described - a limited ring-shaped narrowing (the so-called ring B) located in the distal third of the oesophagus and causing moderate intermittent dysphagia. The ring narrows the lumen of the esophagus by 1/4 - 1/3; during endoscopy, the mucous membrane above it is not changed; during X-ray examination, the motility of the esophagus is not impaired. It is believed that Shatsky's ring is congenital, but it can also develop in the setting of gastroesophageal reflux. Treatment consists of esophagoplasty with local tissue. There are also **congenital strictures and membranes** located in the middle and lower third that can cause dysphagia. The diagnosis is easily established with the help of X-ray and endoscopic examination and relevant history. Esophagoplasty is performed using local tissue with membrane excision.

The syndrome of defecation disorder is expressed in several variants, which are the main symptoms: difficulty defecation, tenesmus, painful defecation, faecal incontinence (Table 4). A separate disorder is rectal prolapse. The diagnosis of each type of disorder is established by taking anamnesis, finger examination of the rectum, rectoscopy, defecography and anal manometry. Strictures of the anal canal are treated by plication of the stricture. An anal fissure that is not amenable to conservative treatment requires excision and lateral internal sphincterotomy. Impaired relaxation of the puborectal muscle is treated with physiotherapy. Faecal incontinence associated with damage to the anal sphincters is corrected by plastic surgery of the external anal sphincter or relocation of other muscles. Faecal incontinence associated with neurogenic causes and collagenosis is initially treated conservatively, and if the effect is not effective, plastic surgery is performed. Rectal prolapse requires complex reconstructive surgery to fix the rectum to the pelvic structures and strengthen the pelvic floor muscles.

Table 4. List of diseases manifested by defecation disorders

Difficult defecation
Anal sphincter strictures
Consequences of operations
Hemorrhoidectomy
Removal of polyps, early cancer
Consequences of sphincter trauma
Radiation stricture
Inflammatory stricture
Lymphogranuloma venereum
Tuberculosis
Cancer of the rectum and anal canal
Retrorectal tumours
Sphincter spasm with chronic anal fissure
Disorders of the puborectal muscle relaxation
Internal invagination (internal rectal prolapse)
Tenesmus
Crohn's disease
Ulcerative colitis
Cancer of the rectum and anal canal
Retrorectal tumours
Fecal incontinence
Consequences of the operation
Episiotomy Fistulotomy, excision of a rectal fistula
Consequences of sphincter injury
Neurogenic causes
Neuritis of the scrotal nerve due to prolonged labour
Injury of the spermatic nerve
Multiple sclerosis
Collagenosis
Cancer of the rectum and anal canal
Retrorectal tumours Prolapse of the rectum

5. **Plan and organisational structure of the lesson**

№	The main stages of the lesson, their functions and content	Learning objectives in terms of learning levels	Training and control tools	Materials for methodological support of class visibility, control of students' knowledge.	Time (in minutes or %) of the total training time.
	2	3	4	5	6
	Preparatory	1)Setting learning objectives for level I according to Bespalko (see point 2.1)	Collection of questions on baseline knowledge and skills	A set of questions on baseline knowledge and skills (see 6.1)	15 % 1
		2) Control of the initial level of knowledge and skills of higher education students, their readiness to perceive the material of the current lesson			
	Main	1Setting learning objectives of levels II, III according to Bespalko (see point 2.1)	Work with patients in the department of surgical gastroenterology, admission department and department of of proctology	1) Diagrams of algorithms for diagnosis, differential diagnosis and treatment	70 % 7

		2) Supervision of patients and discussion of algorithms for diagnosis, differential diagnosis and treatment of patients with different variants of diseases manifested by dysphagia		2) Illustrations of operation techniques in figures and tables		
	Final stage	1) Control of theoretical knowledge and practical skills	1) A collection of Krok-2 type control test tasks (see paragraph 6.3)	Collection of control test tasks of the Krok-2 type	15 %	1
		2) Summary of the class 3) Assignment	2) Reading list for the next session			

6. Materials for methodological support of the class

6.1. Control materials for the preparatory stage of the class - a collection of questions

1. What are the diseases (surgical and 'non-surgical') that are accompanied by vomiting?
2. What are the diseases (surgical and non-surgical) associated with dysphagia?
3. What are the forms of defecation disorders and what are their causes?
4. Briefly describe the anatomy, histology and physiology of the oesophagus and the oesophagogastric junction.
5. Briefly describe the anatomy, histology and physiology of the stomach and duodenum.
6. Briefly describe the anatomy, histology and physiology of the small and large intestine.
7. Briefly describe the anatomy, histology and physiology of the small rectum and pelvic floor.
8. Describe the physiology of the vomiting centre, vagus nerves, pathophysiology of vomiting and its consequences.
9. Describe the pathogenesis of benign tumours, precancerous conditions and cancer of the oesophagus and stomach.

10. Briefly describe the pathogenesis of gastric ulcer and duodenal ulcer.
11. Briefly describe the pathogenesis of various variants of intestinal obstruction, acute abdominal diseases (acute cholecystitis, pancreatitis, appendicitis)
12. Describe the semiotics of oesophageal cancer, oesophageal achalasia, hiatal hernia and gastroesophageal reflex disease, post-burn and peptic strictures of the oesophagus, and oesophageal polyps.
13. Describe the semiotics of stenosis of the outlet due to gastric ulcer and duodenal ulcer and gastric cancer.

6.2. Materials for the methodological support of the main stage of the lesson

6.2.1. See the text of point 4.

6.2.2 Tables of differential diagnosis of dysphagia syndrome

Table 1: Differential diagnosis of oesophageal achalasia with GERD and hiatal hernia (without peptic stricture)

Sign	Oesophageal achalasia not associated with GERD	GERD and GORD not complicated by peptic stricture
Symptoms, medical history and clinical data:		
Dysphagia	always	rarely
Transthoracic pain	often	often
Heartburn	rarely	very often
Eructation	rarely	very often
Regurgitation	very often	often
Reducing body weight	often	very rarely
Pain in the epigastrium	very rarely	often
Respiratory complication	often	often
Cardiac syndrome	very rarely	often
Otorhinolaryngological complication	often	often
pains at palpation in the epigastrium and/ or to the left hypochondrium	never	rarely
Concomitant peptic ulcer disease, chronic gastritis, cholelithiasis, chronic pancreatitis, factors predisposing to high intra-abdominal pressure	rarely	very often
Concomitant mental and vegetative disorders	very often	often
X-ray research:		
Esophageal dilatation	always except for the I st	rarely

A decrease in the amplitude of the primary esophagus peristalsis	always	rarely
Narrowing of the oesophageal-gastric junction, incomplete and delayed relaxation of the lower oesophageal sphincter	always	never
gastroesophageal reflux	never	always
Radiological signs of hiatal hernia	never	very often
Endoscopic research:		
Dilatation of the oesophageal lumen	always except for the I st	rarely
Stagnant esophagitis	often	never
Reflux esophagitis	never	often
Narrowing of the esophagogastric junction	very often	never
gastroesophageal reflux	never	very often
Endoscopic signs of oesophageal hernia of the diaphragmatic opening	never	very often
Barrett's esophagus	never	often
Esophagus manometry:		
Increased basal pressure of the lower oesophageal sphincter (LES)	very often	never
Reducing the basal pressure of the lower oesophageal sphincter	rarely	always
Increased residual pressure of the lower oesophageal sphincter	always	never
Reducing the amplitude of peristaltic contractions of the oesophageal body	always	rarely
Presence of two zones of increased pressure in the area of the oesophageal-gastric junction	never	very often
Daily pH monitoring	norm	higher th an norm

Table 2. Differential diagnosis of esophageal achalasia and esophagospasm

Sign	achalasia esophagus	Esophagospas m
Symptoms, anamnesis:		
Dysphagia	always	often

Sternum pain during or after eating	often	very often
Spontaneous and nocturnal sternum pain	very rarely	always
Regurgitation	very often	often
Weight loss	often	rarely
Extra-oesophageal complications	often	rarely
Concomitant mental and vegetative disorders	very often	very often
X-ray research:		
Dilatation esophagus	always except for the I st	very rarely
Reduction in the amplitude of primary esophageal peristalsis	always	rarely
Multiple segmental contractions of the esophageal body	never	always
Narrowing of the oesophageal-gastric junction, incomplete and delayed relaxation of the lower oesophageal sphincter	always	never
Endoscopic research:		
Dilatation of the esophageal lumen	always except for the I st	very rarely
Stagnant esophagitis	often	very rarely
Reflux esophagitis	never	never
Narrowing of the esophageal-gastric transition	very often	never
Esophagus manometry:		
Increased basal pressure of the lower oesophageal sphincter	very often	never
Increased residual pressure of the lower oesophageal sphincter	always	never
Reduction of the amplitude of peristaltic contractions of the esophageal body	always	rarely
Multiple segmental contractions of the oesophagus	rarely	always
Daily pH monitoring	norm	norm

Table 3. Differential diagnosis of esophageal achalasia and esophageal atony in the first phase (in the absence of peptic stricture of the lower third of the esophagus)

Sign	achalasia esophagus	Atony esophagus

Symptoms, history and clinical data:		
Dysphagia	always	often
Transthoracic pain	often	rarely
Heartburn	rarely	very often
Eructation	rarely	very often
Regurgitation	very often	very often
Reducing body weight	often	rarely
Pain in the epigastrium	very rarely	rarely
Respiratory complication	often	often
Cardiac syndrome	very rarely	often
Otorhinolaryngological complication	rarely	often
Concomitant mental and vegetative disorders	very often	rarely
Association with collagenosis (systemic scleroderma), consequences of TBI, OMT and neuroinfections, diabetes mellitus, brain tumours, alcoholism	missing	present
X-ray research:		
Dilatation esophagus	always except for the I st	always
Reduction in the amplitude of primary esophageal peristalsis	always	always
Narrowing of the esophageal-gastric junction, incomplete and delayed relaxation of the lower esophageal sphincter	always	never
Gaping SSHP, gastroesophageal reflux	never	always
Endoscopic research:		
Dilatation enlightenment esophagus	always except for the I st	always
Stagnant esophagitis	often	very rarely
gastroesophageal reflux	never	always
Narrowing esophageal-gastric transition	very often	never

Table 4. Differential diagnosis of oesophageal achalasia and cancer of the lower third of the oesophagus (cardioesophageal cancer)

Sign	achalasia esophagus	Cancer esophagus
Symptoms, history and clinical data:		
Dysphagia	always	always
Paradoxical dysphagia	often	rarely
Regurgitation	very often	often
Transthoracic pain	often	often
Pain in the back	rarely	often
Pain in the epigastrium	never	often
Cachexia	very rarely	often
Cough, stridor, hoarseness	very rarely	often
Anemia	rarely	often
Dynamics symptoms	slow	ambulance
Disease duration	few years	few months
X-ray research:		
Dilatation esophagus	always, except for the I st .	often, moderate
Reduction in the amplitude of primary esophageal peristalsis	always	rarely
Character of lower oesophageal sphincter motility	incomplete and slow relaxation	functions absence

The nature of narrowing of the lower third of the oesophagus and the oesophagogastric junction-	circular, with smooth edges	circular with uneven, corroded edges
Endoscopic research:		
Dilatation of the esophageal lumen:	always, except for the I st	often, moderate
Congestive esophagitis	often	rarely
Nature of narrowing of the oesophageal-gastric junction	functional, passage for the device	as a result, tumours, no passing for device
Esophagus manometry:		
Increased basal pressure of the lower oesophageal sphincter	very often	always
Character of lower oesophageal sphincter motility	incomplete and slow relaxation	absence of function
Histological research	degenerative - inflammatory changes	tumorous growth

Table 5. Differential diagnostics of esophageal achalasia and scarring strictures of the lower third of the esophagus (peptic, post-burn, stage II esophageal atony)

Sign	achalasia esophagus	Stricture
Symptoms, history and clinical data:		
Dysphagia	always	always
Regurgitation	very often	often
Transthoracic pain	often	very rarely
GERD, esophageal atony, a history of oesophageal burns	few years	few months
X-ray research:		
Dilatation esophagus	always, except I Art	often moderate
Reduction in the amplitude of primary esophageal peristalsis	always	rarely, except atony

		esophagus
Character of lower oesophageal sphincter motility	incomplete slow relaxation	absence of function
Narrowing of the lower third of the oesophagus and esophagus	circular, with smooth edges	circular, with smooth edges
Endoscopic research:		
Dilatation of the esophageal lumen:	always, except I stage	often moderate
The nature of gastric narrowing	functional, passable for apparatus	scarred, not passable for the apparatus
Esophagus manometry:		
Increased basal LGS pressure	very often	always
Character of motility of the lower esophageal sphincter	incomplete and slowed down relaxation	absence functions
Histological research	degenerative - inflammatory changes	inflammatory and cicatricial changes

Table 6. Differential diagnosis of GERD and hiatal hernia and esophageal atony in the first phase (in the absence of peptic stricture of the lower third of the esophagus)

Sign	GERD and GORD	Atony esophagus
Symptoms, history and clinical data:		
Dysphagia	rarely	often
Transthoracic pain	often	rarely
Heartburn	very often	very often
Eructation	very often	very often
Regurgitation	often	very often
Pain in the epigastrium	often	rarely
Extraesophageal complication	often	often
Accompanying mental and vegetative disorders	often	rarely
Connection with collagenoses (systemic	missing	present

scleroderma), consequences of TBI,neuroinfections , diabetes , tumors main brain, alcoholism		
Concomitant peptic ulcer disease, chronic gastritis, cholelithiasis, chronic pancreatitis, factors predisposing to increased intra-abdominal pressure	very often	rarely
X-ray research:		
Dilatation esophagus	rarely	always
A decrease in the amplitude of the primary peristalsis esophagus	rarely	always
Radiological signs of hiatal hernia	very often	never
gastroesophageal reflux	always	always
Endoscopic research:		
Dilatation of the oesophageal lumen	rarely	always
Reflux esophagitis	often	always
Endoscopic signs of hiatal hernia	very often	never
gastroesophageal reflux	very often	always
Esophagus manometry:		
Reducing the basal pressure of the lower oesophageal sphincter	always	always
Reducing the amplitude of peristaltic contractions of the esophageal body	rarely	always
Daily pH monitoring	higher than norm	higher than norm

Table 7. Differential diagnosis of GERD and hiatal hernias and oesophageal cancer

Sign	GERD and hiatal hernia	Oesophageal cancer
Symptoms, history and physical data:		
Dysphagia	rarely	always
Heartburn	very often	very rarely
Regurgitation	often	often

Transthoracic pain	often	often
Back pain	rarely	often
Pain nature	pressing burning	gnawing, scratching
Reduction of body weight	very rarely	often
Cough, stridor, hoarseness	often	often
Anemia	rarely	often
Dynamics of symptoms	slow	ambulance
Duration of the disease	few years	few months
X-ray research:		
Dilatation esophagus	rarely	often, moderate
gastroesophageal reflux	always	never
Radiological signs of a herniated disc	very often	never
Tumour stenosis of the oesophagus	missing	present
Endoscopic research:		
Dilatation enlightenment esophagus	rarely	often, moderate
Tumour of the oesophagus	never	always
gastroesophageal reflux	very often	never
Endoscopic signs of a hiatal hernia	very often	never
Esophagus manometry:		
Decrease of basal pressure	always	never
Daily pH monitoring	above by norm	norm
Histological research	inflammatory changes	tumorous growth

Table 8. Differential diagnosis of GERD and hiatal hernias and post-burn esophageal strictures

Sign	GERH and GSOD	Stricture
Symptoms, history and clinical data:		
Dysphagia	rarely	always
Heartburn	very often	very rarely
Regurgitation	often	often
Transthoracic pain	often	very rarely

Decrease masses bodies	very rarely	very often
Burn history of esophagus	missing	present
X-ray research:		
Dilatation esophagus	rarely	often, moderate
Gastroesophageal reflux	always	never
Radiological signs of a hiatal hernia	very often	never
Cicatricial stenosis of esophagus	missing	present
Endoscopic research:		
Dilatation of the esophageal lumen	rarely	often, moderate
Congestive esophagitis	never	often
Esophageal stricture	never	always
gastroesophageal reflux	very often	never
Endoscopic signs of a hiatal hernia	very often	never
Esophagus manometry:		
Decrease basal NSS pressure	always	never
Daily pH monitoring	above by norm	norm
Histological research	inflammatory changes	scar-inflammatory changes

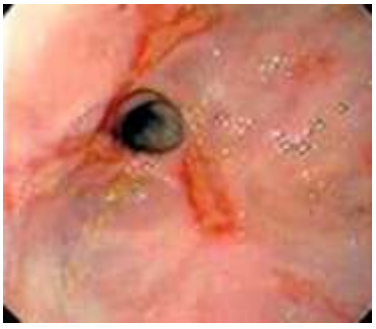
6.2.3. Schemes of surgical interventions (tables, atlases, electronic images).

6.3. Control materials for the final stage of the lesson

6.3.1. Situational tasks

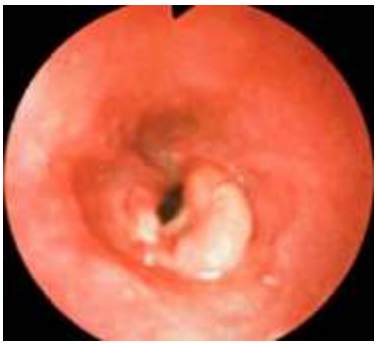
Task 1.

Patient M., 45 years old, consulted a doctor with complaints of belching, severe salivation, vomiting after eating. In the past, he was treated for ulcerative esophagitis. An endoscopic examination of the oesophagus was performed (see photo). What is the preliminary diagnosis? What treatment should be prescribed to the patient?



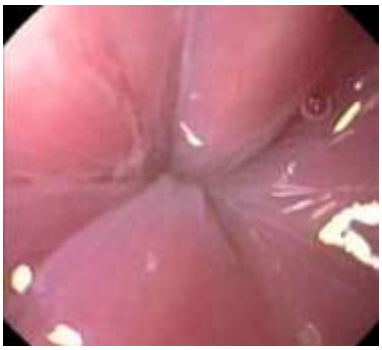
Task 2

The patient complains of difficulty in swallowing solid food, weight loss, nausea, vomiting, which occurs periodically. The patient has been ill for 2 months. Fibroesophagoscopy was performed (see photo). What preliminary diagnosis can be made? What additional research methods are needed to clarify the diagnosis?



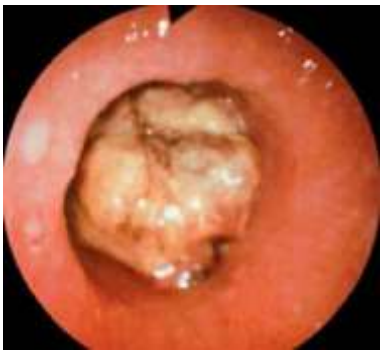
Task 3.

The patient complains of difficulty swallowing, vomiting after eating. Solid food enters the stomach better than liquid food. During the process of eating, spastic pain occurs behind the sternum. An X-ray examination of the oesophagus revealed an ‘inverted candle’ symptom. Fibrogastroscopy was performed (see photo). Your diagnosis and treatment?



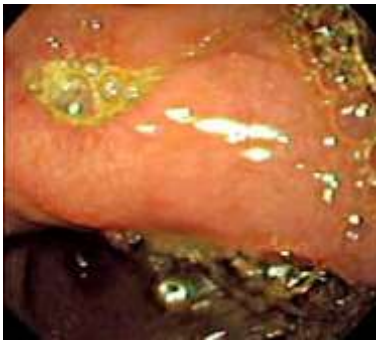
Task 4

Patient K, 63 years old, came to the hospital with complaints of pain behind the sternum that occurs when eating, regurgitation of undigested food with mucus and a small amount of blood, bad breath. Over the past 6 months, she has lost 15 kg. Fibroesophagoscopy was performed (see photo). What was your previous diagnosis and treatment?



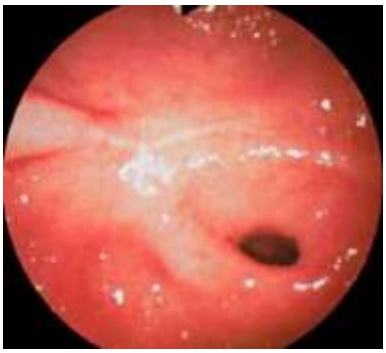
Task. 5

Patient P., 47 years old, came to the clinic with complaints of pain in the epigastric region, aggravated by eating, heartburn, nausea, vomiting, which occurs periodically. He has been ill for 4 years. Outpatient treatment provided temporary relief. Fibrogastroscopy (see photo) revealed an ulcer in the antrum of the stomach. Your tactics?



Task 6.

Patient K., 44 years old, complains of frequent vomiting with old food, a feeling of heaviness in the abdomen, weight loss of 20 kg over the past 3 months. Suffers from duodenal ulcer for 7 years. He was treated irregularly. During fibrogastroscopy (see photo), the remains of old food were found in the stomach. The gatekeeper is scarred, it is impossible to pass an endoscope into the duodenum. What is your diagnosis and treatment tactics?



6.3.1. Control test tasks of the Krok-2 type

1. Patient P., 40 years old, complains of dysphagia, which periodically occurs when taking certain foods, periodic sternum pain, regurgitation during the day. He has been ill for 3 years; the onset of the disease is associated with a stressful situation. An endoscopy is performed, with food residues found in the stomach, congestive esophagitis is noted, the mucosa of the esophagogastric junction is slightly hyperaemic, the endoscope passes into the stomach with little effort. Radiology shows a significant dilation of the esophagus, its peristalsis is significantly reduced, the esophagogastric junction is narrowed, and the contrast passes slowly to the stomach. What is your diagnosis?

A. Esophageal achalasia
B. Hernia of the esophageal hiatus of the diaphragm
C. Oesophageal cancer
D. Zenker's diverticulum
E. Post-burn esophageal stricture

2. Patient D., 64 years old, complained of heartburn, belching, periodic pain in the area of the xiphoid process, constant regurgitation when bending the body after eating. She became ill 2 years ago. Taking antacids and omeprazole slightly reduces the symptoms. During endoscopy, hyperaemia of the lower third of the oesophagus and insufficiency of the oesophagogastric junction are noted. Fluoroscopy in the Trendelenburg position was performed, and prolapse of the stomach floor and part of the stomach body into the mediastinum was detected. What is your diagnosis?

A. Hernia of the esophageal hiatus of the diaphragm
B. Esophageal achalasia
C. Esophageal cancer
D. Zenker's diverticulum
E. Post-burn esophageal stricture

3. Patient M., 50 years old, complained of constant rapidly progressive dysphagia, moderate constant sternal pain, regurgitation after eating. Abuses spicy food. He has been ill for 3 months. Initially, dysphagia was noted during solid food intake, now it is noted during even liquid food intake. He has lost 15 kg. Endoscopy revealed an irregularly shaped tumour-like mass in the middle third of the oesophagus, which completely narrowed the lumen of the oesophagus. X-ray of the oesophagus in the middle third of the oesophagus revealed a filling defect, a narrowing with fuzzy contours, through which contrast flows in a thin stream. What is your diagnosis?

A. Esophageal cancer
B. Esophageal achalasia
C. Hernia of the oesophageal hiatus of the diaphragm
D. Zenker's diverticulum
E. Post-burn esophageal stricture

4.Patient S., 45 years old, complained of constant progressive dysphagia, constant regurgitation immediately after eating. More than 6 months ago, he mistakenly drank an unknown liquid, after which the man was disturbed by intense pain behind the sternum. Over the past 2 months, the pain disappeared and progressive dysphagia appeared. During endoscopy, a concentric narrowing was found in the middle third of the oesophagus, which could not be passed with an endoscope. An esophagoscopy was performed, which revealed a narrowing with indistinct contours in the middle third of the esophagus. What is your diagnosis?

A. Post-burn esophageal stricture
B. Esophageal achalasia
C. Esophageal cancer
D. Esophageal hernia
E. Zenker's diverticulum

5.Patient A., 45 years old, complained of recurrent dull pain in the left side of the neck after eating, the presence of a tumour-like mass that appears after eating and disappears when pressed, accompanied by ‘throwing’ food into the throat, grunting. She became ill for the first time 4 years ago after a stressful situation. The symptoms of the disease are gradually

progressing. During the endoscopy, an additional mouth leading to a cavity with food debris was found on the back wall at the entrance to the oesophagus. A lateral projection X-ray of the oesophagus is performed, which reveals that a sac-like cavity located behind the oesophagus is filled with contrast in the cervical region; the cavity compresses the oesophagus and pushes it forward. What is your diagnosis?

A. Zenker's diverticulum
B. Esophageal achalasia
C. Esophageal cancer
D. Esophageal hernia
E. Post-burn esophageal stricture

6. The patient, 55 years old, complains of constant heartburn, which worsens after eating, especially fatty and spicy foods. Endoscopic examination revealed the presence of ulcers in the lower third of the oesophagus. X-ray examination confirmed the presence of reflux of gastric contents into the oesophagus. What is your diagnosis?

A. Gastroesophageal reflux disease
B. Hernia of the esophageal hiatus of the diaphragm
C. Esophageal achalasia
D. Post-burn esophageal stricture
E. Esophageal cancer

7. A 60-year-old patient complains of belching after eating and burning sensation behind the sternum. Endoscopy reveals a small protrusion of a part of the stomach into the oesophagus through the oesophageal opening of the diaphragm. What is your preliminary diagnosis?

A. Hernia of the esophageal hiatus of the diaphragm
B. Esophageal achalasia
C. Esophageal cancer
D. Esophageal diverticulum
E. Gastritis

8. A 47-year-old patient complained of pain in the cervical region after eating and grumbling. Endoscopy revealed a sac-like cavity located behind the oesophagus. What is your diagnosis?

A. Zenker's diverticulum
B. Hernia of the esophageal hiatus of the diaphragm
C. Esophageal achalasia
D. Esophageal cancer
E. Gastroesophageal reflux disease

9. The patient, 50 years old, complains of constant dysphagia, especially when eating solid food. Endoscopy revealed a concentric narrowing of the oesophagus. What is your preliminary diagnosis?

A. Post-burn esophageal stricture
B. Esophageal achalasia
C. Esophageal cancer
D. Zenker's diverticulum
E. Hernia of the esophageal hiatus of the diaphragm

10. A 42-year-old patient complains of frequent belching, pain behind the sternum, especially after eating. Fluoroscopy revealed the presence of a part of the stomach protruding into the oesophagus. What is your diagnosis?

A. Hernia of the esophageal opening of the diaphragm
B. Esophageal achalasia
C. Esophageal cancer
D. Post-burn esophageal stricture
E. Diverticulum

Literature:

1. **https://www.saudedireta.com.br/catinc/tools/e_books/Oxford%20Handbook%20of%20Clinical%20Surgery,%204th%20Edition.pdf**
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